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Human Evoked Cortical Activity to Signal-to-Noise Ratio and Absolute Signal Level

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Abstract

The purpose of this study was to determine the effect of signal level and signal-to-noise ratio (SNR) on the latency and amplitude of evoked cortical activity to further our understanding of how the human central auditory system encodes signals in noise. Cortical auditory evoked potentials (CAEPs) were recorded from 15 young normal-hearing adults in response to a 1000 Hz tone presented at two tone levels in quiet and while continuous background noise levels were varied in five equivalent SNR steps. These 12 conditions were used to determine the effects of signal level and SNR level on CAEP components P1, N1, P2, and N2. Based on prior signal-in-noise experiments conducted in animals, we hypothesized that SNR, would be a key contributor to human CAEP characteristics. As hypothesized, amplitude increased and latency decreased with increasing SNR; in addition, there was no main effect of tone level across the two signal levels tested (60 and 75 dB SPL). Morphology of the P1-N1-P2 complex was driven primarily by SNR, highlighting the importance of noise when recording CAEPs. Results are discussed in terms of the current interest in recording CAEPs in hearing aid users.

Keywords

Cortical auditory evoked potentials (CAEPs); Event-related potentials (ERPs); signals in noise; signal-to-noise ratio (SNR); N1; Auditory cortex; Hearing aids

1. Introduction

Successful communication in difficult listening environments is dependent upon how the auditory system is able to extract signals of interest from other competing information. Listening in background noise, in particular, presents a difficult challenge that often leads to communication breakdowns. Several factors contribute to the ability to hear a signal in the presence of noise including, but not limited to, reduced audibility, as well as the manner in which signals in noise are encoded throughout the peripheral and central auditory systems. For

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example, focusing on the role of the cortex, Phillips and colleagues (Phillips, 1985, 1986, 1990; Phillips & Kelly, 1992) found that it was the relation between the tone and masker level, rather than the absolute tone level that was the key contributor to the magnitude and timing of neural responses in the central auditory system (CAS). They reported that cortical neurons closely tracked the level of background noise such that noise level increments had to be matched by signal increments of the same magnitude to maintain the neural response. An earlier classic psychophysical study by Hawkins and Stevens (1950) demonstrated a similar effect. They found that masking produced by white noise was directly proportional to the level of the noise. In other words, masked thresholds closely tracked the level of background noise, and increments in masking level resulted in threshold shifts of similar magnitude.

Although this effect of signal-to-noise ratio (SNR) has been demonstrated in human psychophysical studies and in animal physiology studies, the relation between SNR and absolute tone level as measured by human cortical electrophysiology is less clear. One approach to studying how signal level and SNR are processed in the human CAS is to use electroencephalography (EEG). In particular, cortical auditory evoked potentials (CAEPs), one type of EEG, are a measure of CAS function that can provide valuable information about how large populations of neurons, recorded at the scalp, are encoding signals in noise. Traditionally, CAEP waveform morphology has been described in terms of amplitude and latency; amplitude refers to the strength of the response (measured in microvolts) and latency refers to the time after stimulus onset (measured in milliseconds). These voltage changes over time are thought to result from post-synaptic potentials within the brain and are influenced by the number of recruited neurons, extent of neuronal activation, and synchrony of the neural responses (for a review see Eggermont, 2007). The CAEP is made up of several components, including the P1-N1-P2 complex. The P1-N1-P2 complex reflects synchronous neural activity of structures in the thalamo-cortical segment of the central auditory system in response to acoustic changes (Naatanen & Picton, 1987; Vaughan & Ritter, 1970; Wolpaw & Penry, 1975). These include a change from silence to sound, sound to silence, as well as acoustic changes within a signal (for a review see Martin et al., 2008)

Human CAEP data related to encoding of signals in noise are limited. Just two studies have recorded CAEP responses to signal-in-noise stimuli while varying SNR (Kaplan-Neeman et al., 2006; Whiting et al., 1998). Both studies tested young normal-hearing individuals using speech-in-noise stimuli and found that SNR is an important variable contributing to the latency of the evoked response, while the effect of SNR on amplitude was mixed across the two studies. Three issues are important when considering the results of these studies as compared with the animal work mentioned earlier. First, while the importance of SNR in these studies was clear, the contribution of absolute signal level when presented in background noise was not directly tested. Only Whiting et al. (1998) addressed signal level; however, SNRs were not equated across signal levels. In other words, for each stimulus level, background noise levels resulted in slightly different SNRs, making comparisons across signal levels more difficult. Second, regarding the neural response to signals in noise, we are primarily interested in the obligatory encoding that is associated with the passively elicited P1-N1-P2 complex; whereas, both these studies used an oddball paradigm, optimally designed to evoke the discriminative P3 response. Participants performed a task in which they actively responded when hearing a target stimulus. It is difficult to compare those results to P1-N1-P2 responses recorded in a passive way (i.e., participants ignore a homogenous train of stimuli). Third, both experiments measured activity from a single electrode (i.e., Cz). It may be that a measure of overall scalp activity would provide another valuable perspective of neural activity.

Another motivation for this study was further application to populations with perceptual difficulties in background noise such as those with hearing impairment or older adults. CAEPs may be a measure that is sensitive to signal-in-noise difficulties experienced by these groups.

In particular, it could help to clarify previous CAEP results in hearing aid users where background noise was likely an important consideration (Billings et al., 2007).

With the existing literature in mind, we set out to determine the effect of absolute tone level and the effect of SNR on the amplitude and latency of the individual P1, N1, P2, and N2 components in order to understand how the CAS encodes signals in noise. With a long-term focus on the P1-N1-P2 complex, as opposed to later CAEPs, we used a traditional passive-listening, repeated-stimuli paradigm that minimized the contributing effects of selective attention associated with target selection in the oddball paradigm. In addition, we equated SNR across signal levels to clearly distinguish between signal level effects and SNR effects. Finally, we report results for electrode Cz and for a combination of electrodes (i.e., global field power). Based on the work of Phillips and colleagues (e.g., 1990, 1992), we hypothesized that SNR functions would reveal a main effect for SNR but no main effect for tone level. In other words, amplitudes and latencies would change (amplitudes would increase and latencies would decrease) with increasing SNR, but would be similar when compared across the two tone levels. These results improve our understanding of how the human auditory cortex encodes signals in noise and how this type of processing is reflected in human scalp-recorded evoked potentials.

2. Methods

Using a repeated measures design, participants were tested under 12 conditions: two tone levels (60 and 75 dB) and 6 SNRs (Quiet, 20, 10, 0, -10 dB). Amplitude and latency values for evoked responses P1, N1, P2, and N2 were determined and analyzed.

2.1. Participants

Fifteen young normal-hearing individuals participated in this study (mean age = 28.1 years, SD = 4.9; 7 male and 8 females; all right-handed). Participants had normal hearing from 250 to 8000 Hz (<20 dBHL) and normal tympanometric measures (single admittance peak between ± 50 daPa to a 226Hz tone). Tympanometry and air conduction testing were completed prior to each session to ensure stability of middle ear function and hearing sensitivity. All participants were in good general health with no report of significant history of otologic or neurologic disorders. All participants provided informed consent and research was completed with approval from the institutional review board at the University of Washington.

2.2. Stimuli

A signal-in-noise paradigm was used. The signal was a 1000 Hz tone with rise/fall times of 7.5 ms and duration of 756 ms. The tone was identical to the one used in our previous study (Billings et al., 2007). It was presented at two intensity levels: 60 and 75 dB SPL. Continuous white noise was added to the background to create varying SNRs. For the 60 dB SPL tone, noise levels were 40, 50, 60, 65, and 70 dBA SPL (broadband A weighted; noted as dBA). For the 75 dB SPL tone, noise levels were 55, 65, 75, 80, and 85 dBA SPL. In a 2cc coupler, the 85 dBA SPL noise corresponded to level of 71.5 dB SPL within the 1/3 octave band centered at 1000 Hz. A "Quiet" condition (i.e. no noise was added to the background) for both tone conditions was also included. The noise was prefiltered using an inverse filter to flatten the spectrum and match the bandwidth of noise that was recorded at the output of the hearing aid used previously (Billings et al., 2007). This process included recording the impulse response of the transducer using the Golay method of Zhou et al. (1992). The impulse response was then converted to the frequency domain via a fast Fourier transform, inverted, and converted back to the time domain. The real-valued result was treated as a finite impulse response filter and convolved with the white noise. This process corrected for the effects of the frequency response of the transducer. Figure 1 demonstrates the result on the spectrum of the noise; the original unfiltered noise recorded at the output of the transducer (top) is corrected to a flatter spectrum

and broader bandwidth (bottom). The resulting filtered noise had a bandwidth of approximately 5000 Hz; therefore, the 85 dBA SPL noise had a pressure spectrum level of 48 dB. Three versions of each noise level were generated and presented pseudo-randomly across the 15 subjects (i.e., each noise version was presented to five subjects). This was done to control for frozen noise effects by preventing modulations in the noise envelope from being consistent across all conditions. Eight-minute noise files were set to loop continuously in the background. The 1000 Hz tone was characteristic of the stimuli used in the decades of literature examining stimulus intensity effects on CAEPs, allowing for a better comparison of effects across studies. For all conditions, stimuli were presented using an Etymotic Research ER3A insert earphone to the right ear only. The tube of the earphone was coupled to the probe tip of the Etymotic Research ER10B+ probe tip microphone to allow for simultaneous canal recordings of the stimulus.

2.3. In-the-Canal Recordings

Online acoustic recordings were made using the ER10B+ probe tip microphone to allow for more precise stimulus intensity measurements in the ear canal. The ER10B+ probe houses a microphone and stimulus delivery tube within the same ear tip. In-the-canal acoustic recordings have been used previously to enable verification of hearing aid output in the canal (Billings et al., 2007; Caldwell et al., 2006; Souza & Tremblay, 2006; Stelmachowicz et al., 1995; Tremblay, Kalstein et al., 2006). For this study, stimuli were presented and recorded simultaneously, allowing for online verification of noise and tone levels in the ear canal at the plane of the probe tip. A Tucker-Davis Technologies real-time processor (RP2) was used with MATLAB to record and analyze the in-the-ear acoustic waveforms. Tone and noise levels of these waveforms were calculated using 1/3 octave band levels.

2.4. Electrophysiology

A PC-based system controlled the timing of stimulus presentation and delivered an external trigger to the evoked potential recording system. Each stimulus was presented in a homogeneous train with simultaneous continuous background noise. Consistent with our previous work (Billings et al., 2007, Tremblay, Billings et al., 2006), an interstimulus interval (offset to onset) of 1910 ms was used. Stimulus presentation order within tone level condition was randomized across subjects. Subjects were instructed to ignore the stimuli and watch a silent close-captioned movie of their choice. They were also asked to minimize head and body movement. A total of 400 stimulus presentations were recorded for each stimulus condition; this was done across two blocks of 200 presentations. Five-minute listening breaks were given between blocks and between recording conditions. Testing occurred over two days consisting of about three hours of testing each day.

Evoked potential activity was recorded using an Electro-Cap International, Inc. cap which housed 64 tin electrodes. The ground electrode was located on the forehead and the reference electrode was placed on the nose. Horizontal and vertical eye movement was monitored with electrodes located inferiorly and at the outer canthi of both eyes. The recording window consisted of a 100 ms pre-stimulus period and a 1400 ms post-stimulus time. Evoked responses were analog band-pass filtered on-line from 0.15 to 100 Hz (12 dB/octave roll off). Using a NeuroscanTM recording system, all channels were amplified with a gain $\times$ 500, and converted using an analog-to-digital sampling rate of 1000 Hz. Trials containing ocular artifacts exceeding $\pm$ 70 microvolts were rejected from averaging. Following ocular artifact rejection, the remaining sweeps were averaged and filtered off-line from 1 Hz (high-pass filter, 24 dB/octave) to 30 Hz (low-pass filter, 12 dB/octave).

2.5. Data Analysis and Interpretation

To compare our results with the published literature, responses were analyzed from electrode Cz (located at the vertex). In addition, global field power measures were used to quantify simultaneous activity from all electrode sites (Skrandies, 1989). Global field power is the standard deviation across channels as a function of time. Figure 2 shows the scalp topography of the P1-N1-P2 complex (top) for the Quiet (no noise) 60 dB tone condition with electrode Cz circled in the center of the head. Also shown is the global field power waveform (bottom) with peaks N1, P2, and N2 labeled. The P1 component was not included because it is not robust enough to be present in global field power measures. Peak amplitudes were calculated relative to baseline, and peak latencies were calculated relative to stimulus onset (i.e., 0 ms). Latency and amplitude values of each P1, N1, P2, and N2 response were determined by agreement of three judges. Each judge used temporal electrode inversion, global field power traces, and grand averages to determine peaks for a given condition. There were instances where the three judges could not agree on specific peak values, usually when SNR was the smallest and component peaks approached the CAEP noise floor. In these cases, the latency and amplitude value for that peak was excluded from the data. This occurred for approximately 2% of peaks for Cz and global field power waveforms.

Repeated-measures analyses of variance (ANOVA) were completed on amplitude and latency measures of each component of the evoked response (P1, N1, P2, and N2). The 2×5 analysis included the factors of tone level (60 and 75 dB) and SNR (20, 10, 0, -10 dB). Greenhouse-Geisser corrections (Greenhouse & Geisser, 1959) were used where an assumption of sphericity was not appropriate, and epsilon values are included with complete ANOVA results in Table 1.

3. Results

3.1. Effect of Signal-To-Noise Ratio

3.1.1 Results from electrode Cz—Figure 3 shows the grand mean evoked responses recorded at Cz for the 12 conditions tested. The general effect of SNR can be seen in the overall waveform morphology; that is, response amplitude increased and response latency decreased with increasing SNR. Amplitude and latency SNR growth functions clearly demonstrate the effect of SNR across CAEP components P1, N1, P2, and N2 (Figure 4). Table 1 indicates that as SNR increased, P1, N1, P2, and N2 latencies decreased significantly, and N1, P2, and N2 amplitudes increased significantly. Although there was a significant effect of SNR on P1 latency, P1 amplitude was not significantly affected by SNR.

A comparison between SNRs of 20 dB and -10 dB demonstrated average shifts in latency of 33.9 ms for P1, 59.1 ms for N1, 64.8 ms for P2, and 58.6 ms for N2. Average amplitude shifts from 20 to -10 dB were 0.26 μ V for P1, 1.75 μ V for N1, 1.73 μ V for P2, and 0.94 μ V for N2.

3.1.2. Results from Global Field Measures—Similar to the results reported for Cz, a main effect of SNR was found according to global field power measurements. As seen in grand mean global field power waveforms (Figure 5) and SNR growth functions (Figure 6), when data were reduced to a single whole-head measure there was a significant effect of SNR on latency and amplitude of the CAEP components N1, P2, and N2.

3.2. Effect of Tone Level

3.2.1. Results from electrode Cz—The effects of tone level were analyzed by comparing neural responses across signal condition (60 dB versus 75 dB). As can be seen in Figure 4 by comparing the 60 dB (dotted lines) and 75 dB (solid lines) conditions, there were no significant main effects of tone level on latency or amplitude for any of the CAEP components.

There were no significant interactions (SNR $\times$ tone) for P1, N1, P2 or N2 latency or amplitude (Table 1). Overall, the results suggest that the primary determinant of P1, N1, P2, N2, morphology is the SNR, and that these components are not strongly affected by the absolute level of the stimulus.

3.2.2. Results from Global Field Measures—When activity from all electrodes was taken into consideration, there was still no main effect of tone level. The similarities in global field power across condition can be seen in Figure 6, which shows SNR growth functions for the 60 dB tone (dotted line) and 75 dB tone (solid line). In addition, there were no interaction effects for latency or amplitude, with the one exception of P2 latency. Post hoc testing revealed a simple main effect (Kirk, 1968) of tone level on P2 latency at SNRs of -5 and -10 [SNR of -5: $F(1,80)=9.01$, $p=.004$; SNR of -10: $F(1,80)=4.70$, $p=.033$]. More specifically, the 60 dB tone latency was shorter for the SNR -5 dB condition and longer for the SNR -10 dB condition, contributing to the interaction.

3.3. Acoustic Recordings

The ER10B+ probe microphone allowed for online analysis of canal recordings of the stimuli in the ear canal of the participants. Table 2 includes means for signal and noise levels for each of the 12 recording conditions. Reported levels are 1/3 octave band measurements of the band centered at 1000 Hz. As can be seen by comparison across tone levels, SNRs were approximately equivalent. As expected, for the Quiet condition there was about a 15 dB difference between measured SNR values. Small and consistent standard deviations (indicate that the tone and noise levels were maintained such that SNRs were equivalent for both tone conditions and across all participants).

4. Discussion & Conclusion

We set out to determine the effects of tone level and SNR on CAEPs in order to further our understanding of how the CAS encodes signals in noise. Our results demonstrate a clear effect of SNR, with all CAEP subcomponents being affected by the presence of noise. This was particularly true for the component N1 as recorded at vertex (Cz) and across the scalp (global field power). The N1 component is thought to reflect a sensitivity to acoustic changes within the environment (Hyde, 1997; Naatanen & Picton, 1987). Because the N1 reflects changes in stimulus characteristics (e.g., stimulus intensity, stimulus frequency, etc.) it has been used for decades as a physiological test of hearing sensitivity (Davis, 1976; Davis et al., 1967; Perl et al., 1953) and may be of use to characterize CAS encoding in populations with signal-in-noise difficulties.

Effects of SNR were present for latency and amplitude of all components with the exception of P1 amplitude. This result is consistent with the existing data demonstrating the effects of stimulus intensity; P1 amplitude changes were not found with changes to stimulus intensity (Billings et al., 2007; Bruneau et al., 1985; McCandless & Best, 1966). The absence of a P1 amplitude effect might be related, in part, to the fact that the P1 response is small in adults, relative to N1 and P2, and thus very close to the noise floor of the EEG. Therefore, if there is an effect of SNR on P1 amplitude, a larger sample size might be required for the effect to emerge significantly.

As a side note, the presence of offset responses can be seen in Figures 3 and 5 at a latency of approximately 1000 ms. Although not a focus of this study, the general effect of SNR appears to be similar for onset and offset responses. Further study is necessary to determine whether differences across tone levels are significant and meaningful.

4.1. Signal-In-Noise Effects

Evoked response sensitivity to SNR is in agreement with several studies of cortical neurons in the cat by Phillips and colleagues (Phillips, 1985, 1986, 1990; Phillips & Kelly, 1992). Phillips and colleagues found that cortical neurons dynamically adjusted their responses to the level of the background noise. They concluded that this was evidence to support the idea that the cortical auditory system may be implicated in discriminating sounds in background noise (Phillips & Kelly, 1992). The differences in firing characteristics between cortical neurons and auditory nerve fibers demonstrate this idea. For example, auditory nerve fibers fire continuously to ongoing background noise, whereas cortical neurons fire strongly in response to the initial stimulus onset but then do not fire to continuous unchanging noise backgrounds (Gibson et al., 1985; Phillips & Hall, 1986). In other words, cortical neurons demonstrate sensitivity to *changes* in stimuli rather than just to the presence of an ongoing stimulus. As a result, cortical neurons might be more effective at encoding signals in noise because the neuron's complete firing rate dynamic range is available to encode the signal. Similarly, the results from this study demonstrate that activity from large populations of cortical neurons, recorded at the level of the scalp, is sensitive to relative stimulus levels (i.e., the difference between the signal and the continuous background noise) rather than absolute signal level at least over the tested range.

Not only is this finding in agreement with animal neurophysiological studies of cortical neurons by Phillips and colleagues (Phillips et al., 2002), it also appears to conform to aspects of the auditory image model proposed by Patterson and colleagues (e.g., Akeroyd & Patterson, 1995; Patterson, 1994). Both lines of research demonstrate adaptive threshold tracking, or adaptation to the background temporal envelope level of an ongoing stimulus. Even earlier, Hawkins & Stevens (1950) found psychoacoustic growth functions of unity at threshold (i.e., thresholds for signals closely tracked the background noise levels), and Stevens & Guirao (1967) demonstrated that thresholds for signals tracked background noise level as well. However, Stevens & Guirao (1967) also showed that suprathreshold loudness functions steepened by the presence of the masker, indicating possible effects of both absolute signal level and background noise level. Here we limited the range of absolute signal levels (i.e. tone levels of 60 and 75 dB SPL), in part because longer test sessions were not feasible. But in doing so, the range of signal levels was not as great as the range of SNR levels, and it is possible that effects of signal level might have emerged had a wider signal level function been used.

As mentioned above, the P1-N1-P2 complex is thought to arise at the level of the cortex. However, this is not to say that all levels of the human CAS encode signals in noise in this way. Burkard and Hecox (1983) examined the effect of signal and noise levels on wave V of the auditory brainstem response. They varied background broad-band noise level and click level and found that as noise levels were increased, wave V latency increased and amplitude decreased. However, there was also a robust effect of click level, leading them to conclude that there was an additive effect of signal and noise on the auditory brainstem response. In other words, they concluded that noise effects on wave V amplitude and latency depended primarily on absolute noise level and to a lesser extent on SNR. Although different stimuli and recording methodologies were employed across these studies, the differences between early and late human evoked potentials suggest possible differences in neural encoding at different levels of the CAS. However, the fact that P1, N1, P2, and N2 components are all affected by SNR, despite their distinct neural generators, raises the possibility that the effects demonstrated here are reflective of subcortical processing that is propagated to higher levels of the auditory system.

As mentioned earlier, human CAEP data related to encoding of signals in noise are limited. Just two studies have measured CAEP responses to signals in noise while varying SNR (Kaplan-Neeman et al., 2006; Whiting et al., 1998). Both studies tested young normal-hearing individuals using speech-in-noise stimuli. As with the current study, increases in SNR resulted

in monotonic decreases in N1 latency. Amplitude changes with SNR, however, were not as systematic. Whiting et al. (1999) found only small amplitude changes at larger SNRs, and not until a SNR of <5 dB did larger amplitude shifts appear. Hence, amplitude change occurred more abruptly starting at a critical level (an SNR of about 5 dB). In contrast, Kaplan-Neeman et al. (2006) found more complex, stimulus specific, nonmonotonic changes in N1 amplitude with increases in SNR. These discrepancies in amplitude effects may be due, in part, to methodological differences across the studies. For example, background noise used by Whiting et al. (1999) and that used in the current study was continuous, while Kaplan-Neeman et al. (2006) used noise that was not continuous throughout the testing block; noise duration per sweep was 1300 ms, while speech stimuli were 300 ms. In addition, an important consideration is that both previous studies elicited CAEPs using an active task with the oddball paradigm; whereas, the current study used a passive paradigm. The larger P300 response that is evoked in an oddball paradigm task often overlaps, and greatly affects, earlier components such as the N1, P2, and N2. Oddball paradigms also introduce cognitive variables such as attention and stimulus context, which are not the focus when looking at obligatory encoding of stimulus acoustics. We are not aware of any previously published data that systematically assess signal-in-noise level and absolute signal level effects on the human P1-N1-P2 complex elicited in isolation from later components.

Only Whiting et al. (1999) recorded SNR effects while varying absolute signal level. They presented speech stimuli (/da/ and /ba/) at 65 and 80 dB (peak-to-peak equivalent to 1000 Hz tone). Their data demonstrate that SNR functions across the two levels are very similar; however, SNRs were not equated across signal level (i.e., for the 65 dB stimulus, SNRs were 15, 5, and -5 dB; whereas, for the 80 dB stimulus, SNRs were 20, 10, and 0 dB). Because of this, the effect of absolute signal level while varying SNR was not clear. In contrast, the results of this experiment clearly indicate no significant effect of signal level.

4.3. Results Applied to Aided CAEPs

When addressing signal-in-noise listening, three populations with reported perception difficulties are of particular interest: (1) individuals with hearing impairment, (2) older individuals, and (3) hearing aid users. In individuals with hearing impairment, broadening of peripheral auditory filters has a direct impact on cochlear resolution. In addition, peripheral changes modify neural input to CAS resulting in deprivation-related reorganization of CAS encoding (e.g., Irvine and Rajan, 1996; Robertson and Irvine, 1989). Similarly, it is well established that age-related structural and functional changes throughout the auditory system modify how the CAS encodes incoming stimuli (for a review see Tremblay et al., 2007). In fact, abnormal CAEPs have been associated with both older individuals and hearing-impaired individuals (Oates et al., 1999; Tremblay et al., 2002; 2003; 2004), so it is possible that some of the perception-in-noise variability seen across these populations of interest might be related to each person's ability to tolerate/code signals in noise in the CAS. This point is especially important when applied to hearing aid users for at least two reasons: first, the SNR at the output of the hearing aid impacts the person's ability to perceive complex sounds, and second, there is renewed interest in recording CAEPs in individuals while wearing their hearing aids (i.e., aided CAEPs).

Our recent work in the area of aided CAEPs seems to point to the importance of SNR rather than absolute signal level in determining the morphology of the evoked response (Billings et al., 2007). We compared unaided and aided neural responses across a range of stimulus intensities and expected to find increased amplitudes and decreased latencies in the aided condition compared with the unaided condition because of the 20 dB of hearing aid gain. This assumption was based on decades of neuroscience demonstrating the effects of incremental increases in stimulus intensity (as small as 2 dB) on human electrophysiological scalp

recordings (Adler & Adler, 1989, 1991; Beagley & Knight, 1967; Butler, 1969; Martin & Boothroyd, 2000; Picton et al., 1970; Rapin et al. 1966). However, Billings et al. (2007) found no significant differences in latencies or amplitudes between the aided and unaided conditions. We hypothesized that underlying noise in the aided condition (whether amplified ambient noise or circuit noise) was an important factor. In other words maybe SNR, rather than signal level, was the key contributor to timing and magnitude of the evoked response. The results of the current study support that prediction; when SNR was maintained across two tone levels, minimal changes were seen in the amplitude and latency of the evoked response. If, in our previous studies (Billings et al., 2007; Tremblay, Billings et al., 2006), SNRs were maintained across the unaided and aided conditions, then based on the result of this experiment, differences in CAEPs across conditions would not be expected.

Taken together with our previous work, the results of this study have direct implications for aided CAEPs in general. A hallmark of the aided CAEP data to date is the large range of variability within and across studies (i.e., some individuals/studies demonstrate robust amplification effects while others do not). The importance of SNR rather than absolute signal level could explain some aspects of the varying published results (Billings et al., 2007; Gatehouse & Robinson, 1996; Gravel et al., 1989; Korczak et al., 2005; Kraus & McGee, 1994; Kurtzberg, 1989; Purdy et al., 2005; Rapin & Graziani, 1967; Sharma et al., 2004; Stapells & Kurtzberg, 1991; Tremblay, Billings et al., 2006; Tremblay, Kalstein et al., 2006). It may be that the amplification effect was absent when testing certain individuals (e.g., normal-hearing individuals) because the background noise was audible in both the unaided and aided conditions (i.e., SNR was maintained across conditions). In contrast, amplification effects may have been more readily visible in hearing-impaired individuals tested near threshold because the amplified background noise did not reach audible threshold, effectively modifying the SNR in the aided condition relative to the unaided condition. A further complication is that CAEPs were sometimes collected in the presence of background noise, provided by the sound track of the video being viewed by the subject during testing. When considering the audibility of the background noise, it is necessary to take into account the degree and configuration of hearing loss as these factors will affect what portions of the noise are audible. This point is important because it has been demonstrated that the bandwidth of the background noise affects the morphology of the evoked response (e.g., Martin & Stapells, 2005).

The current study demonstrates the importance of determining the levels of background noise in the unaided and aided conditions, and further, determining its effects on the listener. For this reason, it will also be important to determine the effects of hearing impairment and audibility of underlying noise in future aided CAEP experiments. This is especially true for experiments designed to track physiological changes over time in people who wear hearing aids. Whether the intent is to examine the effects of acclimatization or to understand brain maturation, when CAEPs are recorded from different individuals with different hearing losses and hearing aid settings, results can unknowingly be confounded by device-related variables. Although there is interest in measuring aided CAEPs in the clinic (e.g., Golding et al., 2007), it is important first to understand the sensitivity of aided CAEP to acoustic changes introduced by the hearing aid. Without a better understanding of the main variables at work when recording aided CAEPs, interpretation of the resulting neural response patterns will be difficult.

4.3. Conclusion

The results of this study extend our understanding of how signals in noise are encoded at the level of the cortex. Our results show that the CAS, as measured with scalp-recorded CAEPs, encodes signals with a strong sensitivity to SNR. Implications of these results are important for different populations with communication disorders. It may be that changes to the CAS that affect signal-in-noise listening are also reflected at the level of the cortex as measured by

CAEPs. Further studies examining cortical signal-in-noise encoding in these populations are necessary to understand how changes in neural function, at the level of the cortex, impair listening in noise.

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Abbreviations

CAS	central auditory system
SNR	signal-to-noise ratio
EEG	electroencephalography
CAEPs	cortical auditory evoked potentials
ANOVA	analysis of variance

References

- Adler G, Adler J. Influence of stimulus intensity on AEP components in the 80- to 200- millisecond latency range. *Audiology* 1989;28(6):316–324. [PubMed: 2597095]
- Akeroyd MA, Patterson RD. Discrimination of wideband noises modulated by a temporally asymmetric function. *J Acoust Soc Am* 1995;98(5):2466–2474.
- Beagley HA, Knight JJ. Changes in auditory evoked response with intensity. *J Laryngol Otol* 1967;81(8):861–873. [PubMed: 6036752]
- Billings CJ, Tremblay KL, Souza PE, Binns MA. Effects of hearing aid amplification and stimulus intensity on cortical auditory evoked potentials. *Audiology and Neuro-Otology* 2007;12(4):234–246. [PubMed: 17389790]
- Bruneau N, Roux S, Garreau B, Lelord G. Frontal auditory evoked potentials and augmenting-reducing. *Electroencephalogr Clin Neurophysiol* 1985;62(5):364–371. [PubMed: 2411518]
- Burkard R, Hecox K. The effect of broadband noise on the human brainstem auditory evoked response. I. Rate and intensity effects. *J Acoust Soc Am* 1983;74(4):1204–1213. [PubMed: 6643843]
- Butler RA, Spreng M, Keidel WD. Stimulus repetition rate factors which influence the auditory evoked potential in man. *Psychophysiology* 1969;5(6):665–672. [PubMed: 5812328]
- Caldwell M, Souza PE, Tremblay KL. Effect of probe tube insertion depth on spectral measures of speech. *Trends Amplif* 2006;10(3):145–154. [PubMed: 16959735]
- Davis H. Principles of electric response audiometry. *Ann Otol Rhinol Laryngol* 1976;28(3 Pt3):1–96. [PubMed: 779583]
- Davis H, Hirsh SK, Shelnutt J, Bowers C. Further validation of evoked response audiometry (ERA). *J Speech Hear Res* 1967;10(4):717–732. [PubMed: 5586937]
- Eggermont, JJ. Electric and magnetic fields of synchronous neural activity. In: Burkard, RF.; Don, M.; Eggermont, JJ., editors. *Auditory Evoked Potentials*. Philadelphia: Lippincott Williams & Wilkins; 2007. p. 3-21.

- Gatehouse, S.; Robinson, K. Acclimatisation to monaural hearing aid fitting - effects on loudness functions and preliminary evidence for parallel electrophysiological and behavioural effects. In: Kollmeier, B., editor. *Psychoacoustics, speech and hearing aids*. 1996. p. 319-330.
- Gibson DJ, Young ED, Costalupes JA. Similarity of dynamic range adjustment in auditory nerve and cochlear nuclei. *J Neurophysiol* 1985;53(4):940-958. [PubMed: 3998799]
- Golding M, Pearce W, Seymore J, Cooper A, Ching T, Dillon H. The relationship between obligatory cortical auditory evoked potentials (CAEPs) and functional measures in young infants. *J Am Acad Audiol* 2007;18:117-125. [PubMed: 17402298]
- Gravel J, Kurtzberg D, Stapells DR, Vaughan HG, Wallace IF. Case Studies. *Seminars in Hearing* 1989;10(3):272-287.
- Greenhouse WW, Geisser S. On methods in the analysis of profile data. *Psychometrika* 1959;24:95-112.
- Hawkins JE, Stevens SS. The masking of pure tones and of speech by white noise. *J Acoust Soc Am* 1950;22(1):6-13.
- Hyde M. The N1 response and its applications. *Audiology & neuro-otology* 1997;2(5):281-307. [PubMed: 9390837]
- Irvine DR, Rajan R. Injury- and use-related plasticity in the primary sensory cortex of adult mammals: possible relation to perceptual learning. *Clin Exp Pharmacol Physiol* 1996;23(1011):939-947. [PubMed: 8911738]
- Kaplan-Neeman R, Kishon-Rabin L, Henkin Y, Muchnik C. Identification of syllables in noise: electrophysiological and behavioral correlates. *J Acoust Soc Am* 2006;120(2):926-933. [PubMed: 16938980]
- Kirk, RE. *Experimental Design: Procedures for the Behavioral Sciences*. Vol. 1. Belmont, CA: Brooks/Cole Publishing Co.; 1968.
- Korczak PA, Kurtzberg D, Stapells DR. Effects of sensorineural hearing loss and personal hearing AIDS on cortical event-related potential and behavioral measures of speech-sound processing. *Ear and hearing* 2005;26(2):165-185. [PubMed: 15809543]
- Kraus N, McGee TJ. Mismatch negativity in the assessment of central auditory function. *American Journal of Audiology* 1994;3:39-51.
- Kurtzberg D. Cortical event-related potentials assessment of auditory system function. *Seminars in Hearing* 1989;10:252-261.
- Martin BA, Boothroyd A. Cortical, auditory, evoked potentials in response to changes of spectrum and amplitude. *J Acoust Soc Am* 2000;107(4):2155-2161. [PubMed: 10790041]
- Martin BA, Stapells DR. Effects of low-pass noise masking on auditory event-related potentials to speech. *Ear Hear* 2005;26(2):195-213. [PubMed: 15809545]
- Martin BA, Tremblay KL, Korczak P. Speech evoked potentials: from the laboratory to the clinic. *Ear Hear* 2008;29(3):285-313. [PubMed: 18453883]
- McCandless GA, Best L. Summed evoked responses using pure-tone stimuli. *J Speech Hear Res* 1966;9(2):266-272. [PubMed: 5951219]
- Naatanen R, Picton T. The N1 wave of the human electric and magnetic response to sound: a review and an analysis of the component structure. *Psychophysiology* 1987;24(4):375-425. [PubMed: 3615753]
- Oates PA, Kurtzberg D, Stapells DR. Effects of sensorineural hearing loss on cortical event-related potential and behavioral measures of speech-sound processing. *Ear Hear* 2002;23(5):399-415. [PubMed: 12411773]
- Patterson RD. The sound of a sinusoid: time-interval models. *J Acoust Soc Am* 1994;96:1419-1428.
- Perl ER, Galambos R, Glorig A. The estimation of hearing threshold by electroencephalography. *Electroencephalography and clinical neurophysiology* 1953;5(4):501-512. [PubMed: 13107596]
- Phillips D. Temporal response features of cat auditory cortex neurons contributing to sensitivity to tones delivered in the presence of continuous noise. *Hear Res* 1985;19(3):253-268. [PubMed: 4066524]
- Phillips DP, Hall SE. Spike-rate intensity functions of cat cortical neurons studied with combined tone-noise stimuli. *J Acoust Soc Am* 1986;80(1):177-187. [PubMed: 3745663]
- Phillips DP, Hall SE, Boehnke SE. Central auditory onset responses, and temporal asymmetries in auditory perception. *Hear Res* 2002;167:192-205. [PubMed: 12117542]

- Phillips DP. Neural representation of sound amplitude in the auditory cortex: effects of noise masking. *Behavioural brain research* 1990;37(3):197–214. [PubMed: 2340096]
- Phillips DP, Kelly JB. Effects of continuous noise maskers on tone-evoked potentials in cat primary auditory cortex. *Cereb Cortex* 1992;2(2):134–140. [PubMed: 1633411]
- Picton TW, Goodman WS, Bryce DP. Amplitude of evoked responses to tones of high intensity. *Acta oto-laryngologica* 1970;70(2):77–82. [PubMed: 5472109]
- Purdy, SC.; Katsch, R.; Dillon, H.; Storey, L.; Sharma, M.; Agung, K. Aided cortical auditory evoked potentials for hearing instrument evaluation in infants. In: Phonak, A., editor. *A Sound Foundation Through Early Amplification*. Chicago, Illinois: 2005. p. 115-127.
- Rapin I, Graziani LJ. Auditory-evoked responses in normal, brain-damaged, and deaf infants. *Neurology* 1967;17(9):881–894. [PubMed: 6069088]
- Rapin I, Schimmel H, Tourk LM, Krasnegor NA, Pollak C. Evoked responses to clicks and tones of varying intensity in waking adults. *Electroencephalography and clinical neurophysiology* 1966;21(4):335–344. [PubMed: 4162205]
- Robertson D, Irvine DR. Plasticity of frequency organization in auditory cortex of guinea pigs with partial unilateral deafness. *J Comp Neurol* 1989;282(3):456–71. [PubMed: 2715393]
- Sharma A, Tobey E, Dorman M, Bharadwaj S, Martin K, Gilley P, Kunkel F. Central auditory maturation and babbling development in infants with cochlear implants. *Arch Otolaryngol Head Neck Surg* 2004;130(5):511–516. [PubMed: 15148169]
- Skrandies W. Data reduction of multichannel fields: global field power and principal component analysis. *Brain Topogr* 1989;2(12):73–80. [PubMed: 2641477]
- Souza PE, Tremblay KL. New perspectives on assessing amplification effects. *Trends Amplif* 2006;10(3):119–143. [PubMed: 16959734]
- Stapells DR, Kurtzberg D. Evoked potential assessment of auditory system integrity in infants. *Clin Perinatol* 1991;18(3):497–518. [PubMed: 1934853]
- Stelmachowicz PG, Kopun J, Mace A, Lewis DE, Nittrover S. The perception of amplified speech by listeners with hearing loss: acoustic correlates. *J Acoust Soc Am* 1995;98(3):1388–1399. [PubMed: 7560508]
- Stevens SS, Guirao M. Loudness functions under inhibition. *Perception & Psychophysics* 1967;2(10):459–465.
- Tremblay, KL.; Burkard, R. Aging and auditory evoked potentials. In: Burkard, RF.; Don, M.; Eggermont, JJ., editors. *Auditory Evoked Potentials*. Philadelphia: Lippincott Williams & Wilkins; 2007. p. 403-425.
- Tremblay KL, Billings C, Rohila N. Speech evoked cortical potentials: effects of age and stimulus presentation rate. *J Am Acad Audiol* 2004;15(3):226–237. [PubMed: 15119463]quiz 264
- Tremblay KL, Billings CJ, Friesen LM, Souza PE. Neural representation of amplified speech sounds. *Ear Hear* 2006;27(2):93–103. [PubMed: 16518138]
- Tremblay KL, Kalstein L, Billings CJ, Souza PE. The neural representation of consonant-vowel transitions in adults who wear hearing AIDS. *Trends Amplif* 2006;10(3):155–162. [PubMed: 16959736]
- Tremblay KL, Piskosz M, Souza P. Aging alters the neural representation of speech cues. *Neuroreport* 2002;13(15):1865–1870. [PubMed: 12395081]
- Tremblay KL, Piskosz M, Souza P. Effects of age and age-related hearing loss on the neural representation of speech cues. *Clin Neurophysiol* 2003;114(7):1332–1343. [PubMed: 12842732]
- Vaughan HG Jr, Ritter W. The sources of auditory evoked responses recorded from the human scalp. *Electroencephalogr Clin Neurophysiol* 1970;28(4):360–367. [PubMed: 4191187]
- Whiting KA, Martin BA, Stapells DR. The effects of broadband noise masking on cortical event-related potentials to speech sounds /ba/ and /da. *Ear Hear* 1998;19(3):218–231. [PubMed: 9657596]
- Wolpaw JR, Penry JK. A temporal component of the auditory evoked response. *Electroencephalogr Clin Neurophysiol* 1975;39(6):609–620. [PubMed: 53139]
- Zhou B, Green DM, Middlebrooks JC. Characterization of external ear impulse responses using golay codes. *J Acoust Soc Am* 1992;92:1169–1171. [PubMed: 1506522]

Noise Spectrum

Unfiltered
Noise

Filtered
Noise

Figure 1.

Noise spectra of the white noise background recorded at the output of the transducer. The original unfiltered white noise (top) and filtered white noise (bottom) are shown. Golay methods resulted in filtered white noise that had a flatter spectrum, larger bandwidth (out to 5 kHz), and a steeper drop in spectrum level.

Figure 2.

Scalp topography and waveform morphology across the scalp for the Quiet, 60 dB tone condition (above). Electrode Cz is circled at the center. Below is the resulting global field power waveform demonstrating the instantaneous activity across the scalp calculated as the standard deviation of all channels as a function of time. The N1, P2, and N2 peaks are indicated.

Figure 3.

Grand mean waveforms ($n=15$) of electrode Cz for the 60dB tone (left) and the 75 dB tone (right). Waveforms are displayed for a Quiet condition and 5 SNR conditions. In general, increases in amplitude and decreases in latency occur with increasing SNR. Components P1, N1, P2, and N2 are labeled for the 60dB Quiet condition.

Figure 4.

Signal-to-noise growth functions for electrode Cz for P1, N1, P2, and N2 components. Latency (left) and amplitude (right) measures are displayed for the 60 dB tone (dotted line, open circles) and 75 dB tone (solid line, closed squares). Error bars represent 1 standard error of the mean. In general, increases in amplitude and decreases in latency occur with increasing SNR with no significant difference between tone levels.

Figure 5.

Grand mean waveforms ($n=15$) for global field power for the 60dB tone (left) and the 75 dB tone (right). Waveforms are displayed for a Quiet condition and 5 SNR conditions. In general, increases in amplitude and decreases in latency occur with increasing SNR. Components N1, P2, and N2 are labeled for the 60dB Quiet condition.

Figure 6.

Signal-to-noise growth functions for global field power for N1, P2, and N2 components. Latency (left) and amplitude (right) measures are displayed for the 60 dB tone (dotted line, open circles) and 75 dB tone (solid line, closed squares). Error bars represent 1 standard error of the mean.

Table 1

ANOVA Results

Repeated-measures analyses of variance (ANOVA) results for data collected at electrode Cz and for global field power (GFP). Results for latency and amplitude across components P1, N1, P2, N2 are included.

	Effect of SNR		Effect of Tone Level		SNR × Tone Interaction		
	F-statistic (df)	P-value	F-statistic (df)	P-value	F-statistic (df)	P-value	
Cz	Latency						
	P1	20.77 (4,48)	< 0.001	1.406 (1,12)	0.259	0.397 (4,48)	0.714
	N1	51.85 (4,44)	< 0.001	0.358 (1,11)	0.562	0.442 (4,44)	0.641
	P2	30.48 (4,44)	< 0.001	0.351 (1,11)	0.565	2.369 (4,44)	0.067
	N2	18.01 (4,48)	< 0.001	0.008 (1,11)	0.929	0.501 (4,48)	0.735
	Amplitude						
	P1	0.522 (4,40)	0.627	0.317 (1,12)	0.584	2.270 (4,48)	0.075
	N1	13.37 (4,44)	< 0.001	1.431 (1,11)	0.257	1.653 (4,44)	0.178
	P2	15.33 (4,44)	< 0.001	2.065 (1,11)	0.179	0.340 (4,44)	0.849
	N2	11.10 (4,44)	< 0.001	0.303 (1,11)	0.593	0.247 (4,48)	0.910
GFP	Latency						
	N1	60.39 (4,48)	< 0.001	3.194 (1,12)	0.099	1.745 (4,48)	0.195
	P2	71.03 (4,40)	< 0.001	1.670 (1,10)	0.225	4.878 (4,40)	0.003*
	N2	37.04 (4,36)	< 0.001	1.990 (1,9)	0.192	0.741 (4,36)	0.571
	Amplitude						
	N1	73.05 (4,48)	< 0.001	1.808 (1,12)	0.204	0.386 (4,48)	0.818
	P2	30.71 (4,40)	< 0.001	< 0.001 (1,10)	0.990	0.744 (4,40)	0.568
	N2	25.91 (4,36)	< 0.001	2.372 (1,9)	0.158	0.191 (4,36)	0.941

* simple main effect was found at SNRs of -5 and -10 (SNR of -5: $F(1,80)=9.01$, $p=.004$; SNR of -10: $F(1,80)=4.70$, $p=.033$)

Table 2

Canal Recording Results

Acoustic recordings made in the canal with the ER10B+ microphone. Values are 1/3 octave band measures centered at 1000 Hz with standard deviations shown in parentheses.

SNR Condition	60 dB Tone			75 dB Tone		
	Tone	Noise	SNR	Tone	Noise	SNR
Quiet	61.3 (2.0)	21.9 (3.0)	39.4	75.9 (2.2)	21.7 (2.6)	54.2
20	61.3 (2.0)	28.9 (1.9)	32.4	76.2 (2.0)	42.7 (1.9)	33.5
10	61.8 (1.6)	38.5 (1.5)	23.3	75.9 (2.3)	52.2 (2.4)	23.7
0	62.2 (1.6)	48.7 (1.5)	13.5	76.3 (1.7)	62.7 (1.7)	13.6
-5	62.4 (2.2)	53.7 (2.1)	8.7	76.8 (1.9)	67.8 (1.8)	9.0
-10	63.5 (2.2)	58.6 (2.3)	4.9	78.0 (1.8)	72.8 (1.7)	5.2