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RELATIONS AMONG MEASURES OF THE SLOW CORTICAL RESPONSE (SCR) AND ABSOLUTE MAGNITUDE ESTIMATION (AME) LOUDNESS JUDGMENTS IN YOUNG NORMAL-HEARING ADULTS

By

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This is to certify that the Audiology Doctoral Thesis prepared by Hillary Rose Janowitz, B.S., Au.D. candidate entitled Relations Among Measures of the Slow Cortical Response (SCR) and Absolute Magnitude Estimation (AME) Loudness Judgments in Young Normal-Hearing Adults has been approved by her committee as satisfactory completion of the Audiology Doctoral Thesis requirement for the degree Doctor of Audiology (Au.D.)

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ABSTRACT

RELATIONS AMONG MEASURES OF THE SLOW CORTICAL RESPONSE (SCR) AND ABSOLUTE MAGNITUDE ESTIMATION (AME) LOUDNESS JUDGMENTS IN YOUNG NORMAL-HEARING ADULTS

By, Hillary Rose Janowitz, B. S.

The goal of the current study was to determine the relation, if any, between behavioral loudness judgments, using the Absolute Magnitude Estimation (AME) protocol, and the response properties of the slow cortical response (SCR) to 2000-Hz tone-burst stimuli. A secondary goal of the study was to determine the relation, if any, between the subjects' loudness judgments and the annoyance ratings that accompanied these loudness judgments. Eleven normal-hearing young adults participated in the study. Subjects provided an AME loudness judgment rating as well as an annoyance rating to a train of seven 2000-Hz tonal stimuli that were presented from 10 to 80 dB nHL, in 10 dB increments.

Following the loudness judgment ratings, slow cortical responses were recorded for each subject at each stimulus intensity. The acoustic stimuli for the slow cortical response recordings consisted of the same 2000-Hz tone bursts. After the slow cortical response was recorded at each stimulus intensity, each subject was asked to provide judgments of both the loudness and annoyance of these 2000-Hz tonal stimuli at that stimulus intensity.

The peak-to-peak amplitude values of waves P1-N1 and N1-P2, as well as the absolute latency values of waves P1, N1, and P2, were measured to explore the possible relations, if any, of these response measurements with the behavioral growth of loudness for these tonal stimuli. Each response property of the slow cortical response was plotted

in a scatter plot as a function of stimulus intensity. Linear regression analyses were fitted to each response data set separately and Spearman's rank correlation values were calculated.

The peak-to-peak amplitude values of wave N1-P2 have the strongest correlation to the growth of loudness as a function of stimulus intensity in comparison to the response latencies of the various peaks. Future studies should continue to look into the slow cortical response as a possible objective measure to characterize loudness growth in difficult to test populations.

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LIST OF ABBREVIATIONS

Auditory Brainstem Response	ABR
Acoustic Change Complex	ACC
Auditory Evoked Potential	AEP
Absolute Magnitude Estimation	AME
Auditory Steady State Response	ASSR
Compound Action Potential	CAP
Cross Modality Matching	CMM
Electroencephaology	EEG
Event Related Potential	ERP
Forehead	Fpz
Frontal Lobe	Fz
High Pass	HP
Intelligent Hearing System	IHS
Just-Noticeable Difference	JND
Low Pass	LP
Microvolt	µV
Middle Latency Response	MLR
Milliseconds	ms
Mismatch Negativity	MMN
Parietal Lobe	Pz
Signal-to-Noise Ratio	SNR
Slow Cortical Response	SCR

Standard Deviation	SD
Uncomfortable Loudness Level	UCL
Vertex	Cz

CHAPTER 1

INTRODUCTION

Loudness growth normalization is a common prescriptive goal for fitting hearing aids. In clinical practice, most audiologists do not formally measure the patient's growth of loudness with their hearing aid(s) to check on the accuracy of the prescriptive fit. Instead, audiologists rely on subjective reports from the aided listener that the hearing-aid output is too soft or too loud and then use these reports to modify the prescriptive-gain setting of the hearing aid. This strategy may work for most hearing-impaired listeners; however, it may be problematic for hearing-aid users who cannot provide a valid and reliable judgment of loudness. These problematic hearing-aid users may include young children, cognitively or mentally challenged adults, and elderly persons. For this patient group, it would be beneficial to audiologists if there were objective tools, such as auditory evoked potentials (AEPs) to assess objectively the normal and/or abnormal growth of loudness.

Since the 1970s, several investigators have explored whether the response properties of the Auditory Brainstem Response (ABR) and/or the Middle Latency Response (MLR) to click and/or tonal stimuli are correlated with measures of loudness perception in normal-hearing adults (Darling & Price, 1990; Davidson, Wall, & Goodman, 1990; Howe & Decker, 1984; Korczak, Sherlock, Hawley, & Formby, in preparation (see reference note) ; Madell & Goldstein, 1972; Nousak, 2001 (see reference note); Pratt & Sohmer, 1977; Serpanos, 2004; Serpanos, O'Malley, & Gravel, 1997; Silva & Epstein, 2010; Wilson & Stelmack, 1982). Overall, the results from the click-evoked ABR/MLR studies suggest that the response measures for these two AEPs are not

correlated with the underlying neural processes that mediate the perception of loudness. However, there is some recent data that suggest tone-evoked ABR/MLRs may provide a better measure of neuronal processes underlying loudness perception in normal-hearing adults (Korczak et al., in preparation; Nousak, 2001; Serpanos, 2004; Silva & Epstein, 2010).

One AEP that has been explored for this purpose is the slow cortical response. An advantage of recording the slow cortical response, in comparison to the ABR, is that the former assesses the integrity of the neuronal pathways up through and including the auditory cortex. Recent evidence suggests that the response properties of waves N1 and P2 of the slow cortical response are correlated with loudness judgments in a group of adult cochlear-implant listeners (Hoppe, Rosanowski, Iro, & Eysholdt, 2001). The stimuli employed in this study were a series of electrical pulse trains delivered to one electrode. There is no corresponding research on the relation, if any, between the response properties of the slow cortical response and loudness perception for acoustic stimuli. Thus, we do not know whether the slow cortical response correlates with acoustic loudness perception. Accordingly, the goal of this research is to determine whether response amplitudes and/or latencies of waves P1, N1, and P2 of the acoustically evoked slow cortical response are correlated with loudness judgments for the same stimuli in a group of normal-hearing adults.

CHAPTER 2

LITERATURE REVIEW

Auditory Evoked Potentials

Auditory evoked potentials (AEPs), a subset of event-related potentials (ERPs), are responses that are "evoked" by auditory stimuli including clicks, pure tones, noise, and speech (Stapells, 2009). ERPs are electrical responses generated in the brain that are time-locked to a specified stimulating event. This specific event may be a physical stimulus, a change in a train of stimuli, a missing stimulus, or a stimulus that has been designated as a "target" stimulus. AEPs are typically non-invasive and can be used to test the integrity of the peripheral and/or central auditory system (Gelfand, 2001). Examples of different AEPs include: the Auditory Brainstem Response (ABR), the Middle Latency Response (MLR), the Slow Cortical Response (SCR), and Late Cortical Responses. Picton (1990) has suggested that AEPs can be classified in four different ways. These classification schemes include: the absolute latency of the response; the relation of the response to the stimulus; the presumed underlying neural generators of the response; and the functional significance of the response. Each of these classification schemes is described briefly below.

The first classification system, the absolute latency of the response, refers to the time in milliseconds (ms) that the response occurs following the onset of the stimulus (Stapells, 2009). This temporal classification scheme divides the absolute latencies of the AEPs into several categories including: first, fast, middle, slow, and late. The first response latencies occur 0-5 ms post-stimulus onset and consist of the eighth nerve compound action potential (CAP) and the ABR waves I and II. Fast response latencies

occur 2-20 ms post-stimulus onset and consist of waves III, IV, and V of the ABR. In contrast, the latencies of the MLR occur 10-100 ms post-stimulus onset and consist of MLR waves Na, Pa, and Nb. Lastly, the cortical ERPs consist of the slow cortical response and the late cortical ERPs. The slow cortical response occurs at approximately 50-300 ms post-stimulus onset and consists of waves P1, N1, and P2. In contrast, the late cortical ERPs occur at approximately 150-1000 ms post-stimulus onset and consist of the Mismatch Negativity (i.e., MMN) and waves N2b, P3b, and N4 (Stapells, 2009).

A second classification scheme of the AEPs is based on their neural generators, or the anatomical sites from which the response is presumed to originate (Stapells, 2009). The first responses are presumed to originate in the cochlear and VIII nerve and include the CAP and ABR waves I and II. The fast responses are presumed to originate in the brainstem region and include waves III, IV, and V of the ABR. Lastly, the middle, slow, and late cortical responses are all thought to have their primary origin in the primary auditory cortex, with additional contributions from other associated areas of the cortex and the sub-cortical regions of the brain. These responses include the MLR and the various cortical responses described previously (Stapells, 2009).

Another classification scheme is based on the temporal relation of the response to the stimulus. This classification scheme is broken down into three categories: transient potentials, sustained potentials, and steady-state potentials. Transient potentials represent the averaged response that results from presentation of an individual stimulus or stimuli (Hood, 1998). The eighth nerve CAP, the ABR, the MLR, and the cortical responses (i.e., waves N1-P2, P300, and MMN) are examples of transient potentials. Sustained potentials, on the other hand, reflect either “repeated or continual stimulation” (Hood,

1998, p. 5). The frequency following response and the sustained cortical potentials are examples of sustained potentials. Thirdly, the steady-state potentials are evoked by rapidly repeating stimuli (generally stimuli are presented at a rate of 40/sec or greater). In the steady-state potentials, the response to the first stimulus overlaps with the response to the next stimulus within the same post-stimulus analysis window. The auditory steady-state response (ASSR) and the cochlear microphonic are examples of steady-state potentials (Hood, 1998).

The fourth way to classify AEPs is based on their function. AEPs are categorized as either sensory potentials or processing-contingent potentials (Stapells, 2009). Sensory potentials are obligatory responses that occur in response to the presence of the acoustic energy in the stimulus. They are also referred to as exogenous potentials. Sensory potentials are sensitive to changes in physical properties of the stimulus. For example, as stimulus intensity decreases, the peak-to-peak amplitude of the response decreases and the absolute latency of the response increases. The first, fast, middle, and slow responses are all categorized as sensory potentials. In contrast, processing-contingent potentials represent additional processing within the brain beyond the obligatory sensory stage. For example, if a wave P300 has been elicited to the speech stimulus /da/ embedded in a train of /ta/ stimuli, then this finding indicates that these speech stimuli are audible at the level of the auditory cortex, and the acoustic energy present in each of these syllables is discriminable. These processing-contingent potentials have been referred to as either discriminatory potentials and/or endogenous potentials. The late cortical responses, the MMN and wave P3b, are examples of processing-contingent potentials (Stapells, 2009).

The focus of this research is the slow cortical evoked response in normal-hearing adults. Therefore, the remainder of this literature review will focus on this response. The next section of this literature review will provide a brief history of the slow cortical response, a description of the P1-N1-P2 complex and each of the individual components, including their neural generators.

Slow Cortical Response

History of the slow cortical response. The slow cortical response was first described in the literature by Dr. P.A. Davis in 1939. In her two papers, Dr. Davis conducted a series of 41 experiments on 38 adult subjects. During these experiments, she presented soft and loud tonal stimuli and observed changes in the subjects' electroencephalogram (EEG) to the sounds (Davis, 1939a; 1939b). Electrical responses to the sound stimuli were not observable in all subjects; however, when the responses were observable, an "on-effect" occurred in response to the sound, and an "off-effect" occurred when the stimulus was not present. These effects were most prominent when the active electrode was located on the vertex of the subject's scalp. Shown in Figure 1 is an example of a single subject's response to the tonal stimulus. The "on-effect" is represented by a diphasic wave with two peaks, one occurred positive relative to the baseline and the other was negative relative to the baseline (Davis, 1939a). The first component of the response was negative with a latency of approximately 30-40 ms, and the amplitude from peak to trough ranged from just visible to 100 μV. Based on these recordings, Dr. Davis concluded that evidence of an "on-effect" to the tonal stimulus in this latency region indicated that the sound was audible to the auditory cortex.

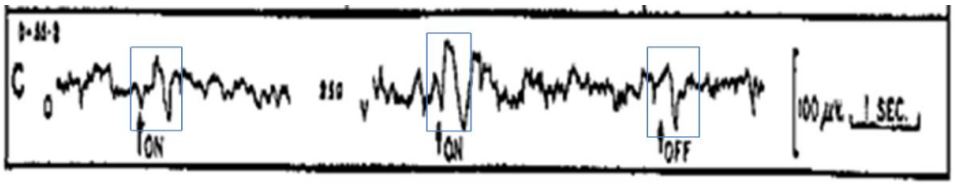

Figure 1: A single subject's response to a tonal stimulus, positivity is facing down. The first component of the response is indicated by the "on" and "off" label (approx 30-40 ms post stimulus onset) (Davis 1939a).

Description of the response. The slow cortical response to click and/or tonal stimuli in a normal-hearing adult consists of two positive peaks (labeled waves P1 & P2) and one negative peak (labeled wave N1). The use of uppercase letters "P" and "N" refer to positivity and negativity, respectively, relative to the zero-amplitude baseline (Martin, Tremblay, & Korczak, 2008). The slow cortical response, shown in Figure 2, was recorded to a click stimulus presented at a high stimulus intensity. The absolute latencies of waves P1, N1, and P2 occur at approximately 50, 80-100, and 180-200 ms post-stimulus onset, respectively. The slow cortical response has been collectively referred to as the P1-N1-P2 complex. The slow cortical response can be elicited to a brief stimulus, such as a click or a tone burst stimulus, or to more complex stimuli, such as speech syllables, words, or sentences (Martin et al., 2008). The presence of the P1-N1-P2 complex indicates that the acoustic energy present in the stimulus is audible to the listener at the level of the auditory cortex (Korczak, Kurtzberg, & Stapells, 2005). Below is a brief description of the history and response properties of each of the waves that constitute the P1-N1-P2 complex.

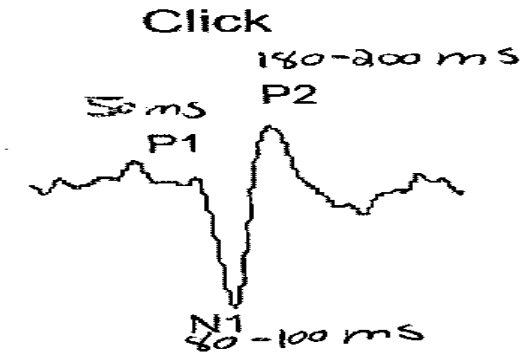

Figure 2: The P1-N1-P2 complex recorded to a click stimulus at a high stimulus intensity (Martin et al., 2008.)

P1 component. Wave P1 was first described in the AEP literature in the late 1950s and early 1960s. It was observed as a small averaged response occurring in adults with and without hearing loss, after the presentation of a click stimulus (Geissler, Frishkopf, & Rosenblith, 1958). Geissler et al. (1958) reported that P1 has an absolute latency of approximately 30 ms and is detectable near the subject's audiologic threshold. They also reported that the amplitude and latency of P1 varied with stimulus intensity. Specifically, as the intensity of the click stimulus was increased, the peak-to-peak amplitude of the response increased and the absolute latency of the response decreased. Based on these findings, Geissler and his colleagues suggested that wave P1 has cortical origins (Geissler et al., 1958).

In the early AEP literature, wave P1 was thought to be the same component as wave Pb of the MLR. Nelson, Hall, and Jacobson (1997) investigated the effects of stimulus rate on waves Na and Pa of the MLR and wave Pb/P1 of the slow cortical response. Three neurologically healthy female adults with normal-hearing sensitivity participated in the study. These two AEPs were recorded to high intensity (80 dB nHL) 500-and 4000-Hz tone burst stimuli presented at stimulus rates of 0.5, 1.1, 2.1, and 5.1

per second. Nelson and colleagues (1997) observed that as the stimulus rate increased from 0.5/sec to 5.1/sec, the detectability of wave Pb decreased. Specifically, increases in stimulus rate above 1.1/sec resulted in a decreased amplitude value and/or absence of wave Pb. In contrast, the amplitude values of waves Pa and Na of the MLR remained stable with the varying stimulus rates. Based on these findings, Nelson and colleagues concluded that wave Pb arises from neurons that have a longer refractory time in comparison to neurons that generate waves Pa and Na of the MLR. Therefore, these investigators suggested that wave Pb is similar to waves N1 and P2 of the slow cortical response (Nelson et al., 1997).

However, more recently Ponton, Eggermont, Khosla, Kwong, and Don (2002) clearly demonstrated that wave P1 of the slow cortical response and wave Pb of the MLR are two distinct AEP waveform components. Ponton and colleagues, using a brain-imaging technique known as dipole-source analysis, investigated the maturational development of these two waveform components. They recorded the MLR and slow cortical response in 118 neurologically normal-hearing subjects, who ranged in age from 5 and 20 years. Recording electrodes for this study were placed at 30 different locations on the scalp. The dipole-source analysis separated the recorded responses into two categories: 1) responses originating from sagittally-oriented neural sources; and 2) responses arising from tangentially-oriented neural sources. Responses from the dipole-source analysis revealed that the absolute latency of wave Pb of the MLR did not change as a function of age in the sagittally oriented analyses. This finding is in contrast to the various age-related changes that were observed in both the peak-to-peak amplitudes and absolute latencies of waves P1, N1, and P2 of the slow cortical responses in the tangential

analyses (Ponton et al., 2002). Based on these findings, these investigators concluded that wave Pb of the MLR and wave P1 of the slow cortical response are two distinct waveform components with differing underlying neural generators.

As previously mentioned, in normal hearing adults, wave P1 occurs approximately 50 ms after the onset of the stimulus. The amplitude of wave P1 in normal-hearing adults is typically small (typically <2 μVs) compared to the amplitude of wave P1 in children (typically ranging from 2-2.6 μVs, depending on the child's age) (Martin, Tremblay, & Stapells, 2007; Naatanen & Picton, 1987; Sharma, Kraus, McGee, & Nicol, 1997). The maximum amplitude value is expected to occur when measured by electrodes close to the vertex (i.e., when the electrode is located at the midpoint of the scalp) (Martin et al., 2007).

N1 component. Wave N1 is the most researched component of the slow cortical response. N1 is a prominent negative peak relative to the zero-amplitude baseline. It typically occurs approximately 80-100 ms following stimulus onset in normal-hearing adults. The amplitude of wave N1 is typically 2-5 μV, depending on stimulus parameters (Martin et al., 2007). Several researchers have suggested that N1 has at least three main underlying components: 1) a fronto-central negative wave referred to as wave (N1b); 2) the T-complex; and 3) a non-specific response (Naatanen & Picton, 1987; Tonnquist-Uhlen, Ponton, Eggermont, Kwong, & Don, 2003; Vaughan & Ritter, 1970; Wolpaw & Penry, 1975). Below is a brief description of each of these components of wave N1.

Frontocentral negativity (N1b). The first underlying component of N1, proposed by Vaughan and Ritter (1970), is a fronto-central negative wave, N1b, which occurs at 80-100 ms post-stimulus onset (Naatanen & Picton, 1987). This component of wave N1

is generated by bilateral vertically oriented dipoles located in/near the auditory cortex. Wave N1b is best recorded when electrodes are at or near the vertex (Cz), and this is the typical wave N1 that audiologists use in their recordings of the slow cortical response. Wave N1b inverts in polarity when electrodes are placed below the level of the Sylvian Fissure and when the reference electrode is located on the nose (Naatanen & Picton, 1987; Vaughan & Ritter, 1970). Shown in Figure 3, from Vaughan and Ritter (1970), is the representation of the scalp distribution of the slow cortical response, recorded using seven scalp electrodes and a reference electrode placed on the tip of the nose. The largest response for wave N1b occurs when the non-inverting electrode is located at the vertex, labeled Cz in Figure 3, and the response inverts with electrodes placed below the level of the Sylvain Fissure, between the 4th and 5th electrodes.

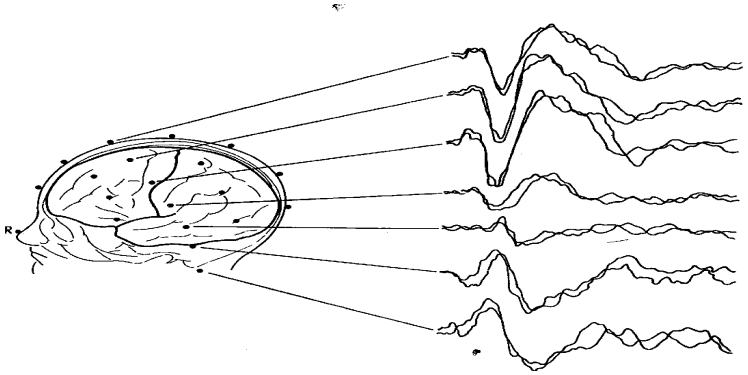

Figure 3: The scalp distribution of the slow cortical response, the largest response occurs when the non-inverting electrode is located on the vertex (Cz) and the response inverts between the 4th and 5th electrodes. The reference electrode is located on the tip of the nose (Vaughan & Ritter, 1970).

T-complex. The second component of N1, commonly referred to as the biphasic "T-complex", is a positive wave that occurs post-stimulus onset at about 100 ms and a negative wave that occurs at approximately 150 ms. (Naatanen & Picton, 1987; Wolpaw

& Penry, 1975). Wolpaw and Penry (1975) recorded N1 of the slow cortical response to a 60-dB-SL (re: threshold) click stimulus from three different locations on the scalp: the vertex (Cz), and two temporal locations: (T3, and T4). Shown in Figure 4 is an example of the N1 response recorded from the vertex (Cz) and from the two temporal lobes (T3 and T4). The response recorded from Cz displays the N1-P2 complex. Conversely, in the dotted response, a positive peak occurred at approximately 105-110 ms post-stimulus onset and was followed by a negative peak at approximately 150-160 ms post-stimulus onset (Wolpaw & Penry, 1975).

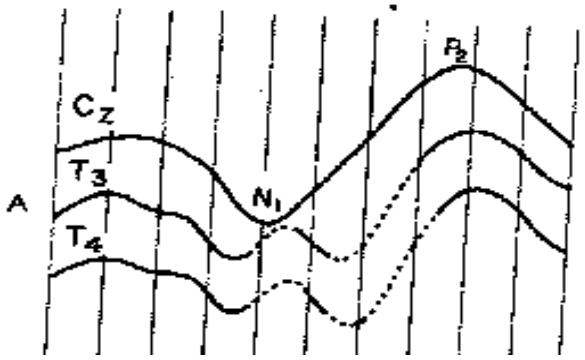

Figure 4: Wave N1 recorded from Cz and from temporal lobe locations (T3 and T4) on the scalp also known as the T-complex. The solid line representes the response recorded from the vertex (Cz) and the dotted line represents the response recorded from temporal lobe locations (T3 and T4) on the scalp (Wolpaw & Penry, 1975).

The T-complex, or the second component of wave N1, is generated in the auditory association areas within the superior temporal gyrus. It is best recorded with electrodes located at the midtemporal region of the scalp (Martin et al., 2008; Naatanen & Picton, 1987; Tonnquist-Uhlen et al., 2003). Tonnquist-Uhlen and colleagues reported that the T-complex component of N1 is largest in amplitude and present at a younger age when measured by midtemporal electrodes (i.e., T3 and T4) in comparison to the

response recorded from more central electrode locations on the scalp (i.e., C3 and C4). Based on these findings, Tonquist-Uhlen and colleagues (2003) concluded that the secondary auditory cortex contributes to the T-complex, which appears to be independent of AEPs recorded at the central electrode locations.

Nonspecific response. The third component of N1 is a negative wave with a peak latency of approximately 100 ms post-stimulus onset (Naatanen & Picton, 1987). It has its maximum amplitude at the vertex (Cz) and at lateral central electrode sites on the scalp. The generators of this component are not known, but have been suggested to originate in the frontal motor or pre-motor cortex and to be influenced by neuronal inputs from the reticular formation and the thalamus (Naatanen & Picton, 1987). This third component of N1 likely does not play a role in hearing (Naatanen & Picton, 1987).

Our recordings will focus on wave N1b of the slow cortical response. It is the primary component of wave N1 that is used for estimating behavioral thresholds and for performing supratheshold judgments of the kind of interest in the current study.

P2 component. Wave P2 is the second positive vertex peak in the P1-N1-P2 complex, and it occurs approximately 180-200 ms post-stimulus onset in normal-hearing adults (Martin et al., 2008). The amplitude of P2 is relatively large, approximately 2-5 μV or greater in adults, however, wave P2 may be absent in young children. It is best recorded using electrodes over midline central scalp regions (Martin et al., 2007). The response properties of wave P2 often co-vary with the latency and amplitude of wave N1 and, therefore, some investigators have speculated that P2 reflects the same underlying neural mechanisms as N1 (Martin et al., 2008). However, other investigators have provided evidence that maturation and various subject factors, such as attention and

sleep, have a differential effect on P2 and N1. For example, an increase in the level of attentiveness results in an increase in the amplitude value of N1 and a decrease in the amplitude of P2 (Crowley & Colrain, 2004). To date, a debate still exists in the AEP literature regarding the independence of these two waveform components. Below is a brief description of the underlying neural generators for each component of the slow cortical response and the techniques used to identify these generators.

Neural Generators of the Slow Cortical Response. Brain-imaging techniques, such as computer-generated isovoltage topographic maps, neuromagnetic mapping, and magnetic resonance imaging (MRI) techniques, have been used to identify the underlying neural generators of waves P1, N1, and P2 of the slow cortical response in human adult subjects. The underlying neural generators of the slow cortical response in humans have also been studied using intracranial recordings. Specifically, the P1-N1-P2 complex has been recorded in epileptic patients during surgery. A comparison has been made between the response properties of the intra-cranial recordings versus the same response properties obtained simultaneously with surface-electrodes recordings on the scalp. A brief description of the neural generators of each component of the slow cortical response is provided in the following section.

Neural generators of P1. Several research groups, utilizing computer-generated topographic maps and multiple recording sites on the scalp, have identified the neural generators of wave P1 in normal-hearing adults and established its origin in the primary auditory cortex, specifically within Heschl's gyrus (Huotilainen, et al., 1998; Reite, Teale, Zimmerman, Davis, & Whalen, 1988; Wood & Walpaw, 1982). Wood and Walpaw (1982) recorded the slow cortical response to high-intensity click stimuli in 11

normal-hearing adults. This response was recorded from electrodes located at 20 different scalp locations in the right hemisphere. The investigators developed isovoltage topographic maps to show those areas of the brain that were responding to the stimuli. Wood and Walpaw (1982) reported that AEPs with response latencies between 20 and 60 ms post-stimulus onset (i.e., consistent with the latency of P1) demonstrated a scalp distribution consistent with neural sources in the primary auditory cortex, on the superior temporal plane, near the temporalparietal junction.

A few years later, Reite and colleagues (1988), using neuromagnetic mapping and magnetic resonance imaging techniques, indicated that P1 was generated in the primary auditory cortex, specifically within Heschl's gyrus, as had been reported by Wood and Walpaw (1982). However, Reite et al. (1988) also reported that when the source-location estimates were transposed to magnetic resolution images of the brain, neural generators for P1 were compatible with sources in the planum temporale in both hemispheres. Thus, the neural generators of P1 may involve more components than just the primary auditory cortex (Heschl's gyrus).

In the last two decades, a series of intracranial studies were conducted with adults who suffered drug-resistant partial seizures of right or left temporal lobe origin and with epileptic adults to determine the underlying neural generators of wave P1 (Howard et al., 2000; Liegeois-Chauvel, de Graff, Laguitton, & Chauvel, 1999; Liegeois-Chauvel, Musolino, Badier, Marquis, & Chauvel, 1994). The results of these intracranial studies have suggested that the neural generators of P1 may involve structures in the brain other than the primary auditory cortex. In 1994, Liegeois-Chauvel and colleagues recorded the MLR and P1 of the slow cortical response to 1000-Hz tonal stimuli in 37 adult subjects

with drug-resistant seizures of right or left temporal lobe origin. The recordings were performed with needle electrodes located in the auditory cortex (Heschl's gyrus and planum temporale). Liegeois-Chauevel et al. (1994) concluded that P1 was generated within the primary auditory cortex and its origins are more lateral and widely distributed in the primary auditory cortex than previously thought. Specifically, they described the generators of P1 to be close to the boundaries between the primary and the secondary auditory areas.

Five years later, Liegeois-Chauvel and colleagues investigated the neural responses to speech and non-speech sounds within Heschl's gyrus, planum temporale, and the superior temporal gyrus of the auditory cortex in 17 epileptic adult patients (Liegeois-Chauvel et al., 1999). The neural generators of P1 were identified within the primary auditory cortex (Heschl's gyrus) and the planum temporale (Liegeois-Chauvel et al., 1999), confirming generators reported in earlier studies. Lastly, Howard et al. (2000) recorded the slow cortical response to click stimuli from implanted electrodes that penetrated Heschl's gyrus and the superior temporal gyrus of 18 adults undergoing surgery for intractable epilepsy. Their results suggested that the posterior lateral superior temporal area receives input either directly, or indirectly, from Heschl's gyrus. Thus, they concluded that both areas contribute to the generation of wave P1 (Howard et al., 2000). The results of these brain-imaging and intracranial recording studies suggest that the neural generators of wave P1 are complex and include the primary auditory cortex (Heschl's gyrus), hippocampus, planum temporale, and lateral temporal regions of the brain.

Neural generators of N1. Wave N1 has at least three main underlying components. However, this study will focus only on wave N1b. Accordingly, the generators of the other two components (i.e., the T-complex, and the non-specific response) will not be discussed in detail here. Numerous investigators have reported that N1b has multiple neural generators within the primary and secondary auditory cortex (Liegeois-Chauvel et al., 1999; Naatanen & Picton, 1987; Scherg, Vajsar, & Picton, 1989; Vaughan & Ritter, 1970; Wolpaw & Penry, 1975). Vaughan and Ritter (1970) were the first investigators to identify and label the N1b component of wave N1. To determine the neural generators of the N1b component, they recorded the slow cortical response from six normal-hearing adult subjects and four adult patients who had undergone carotid angiography. Vaughan and Ritter (1970) speculated that if the slow cortical response was generated within the primary auditory cortex, then the amplitude of N1 would reach a maximum near the vertex and fall to a level of zero amplitude over the Sylvian Fissure. These investigators reported that the amplitudes of waves N1, P2, and P3 all decreased in amplitude as distance from the vertex increased. However, only N1 and P2 showed a polarity inversion below the level of the Sylvian Fissure, suggesting that the neural generators of P3 must differ from the neural generators of waves N1-P2. Vaughan and Ritter (1970) concluded that the neural generators of wave N1b are within or near the primary auditory cortex in the supratemporal plane.

Scherg and colleagues (1989) used dipole-source analysis to study the neural generators of component N1b and the T-complex. One dipole recording had a vertical orientation and one had a radial orientation. Shown in Figure 5 is an example of the dipole-source analysis. The N1b response, seen in the tracings labeled number one and

two, were recorded from this vertically oriented dipole on the left and right side of the head, respectively. In contrast, the T-complex, seen in tracings number three and four, were recorded using a radial or horizontal oriented dipole. Scherg and colleagues (1989) concluded from their analyses that wave N1b is generated in or near the primary auditory cortex and the T-complex is generated from areas within the secondary auditory cortex. These sites are similar to the generator sites derived from intracranial studies discussed above. The results of these multiple studies suggest that the various components of N1 are generated from areas within the primary and secondary auditory cortex.

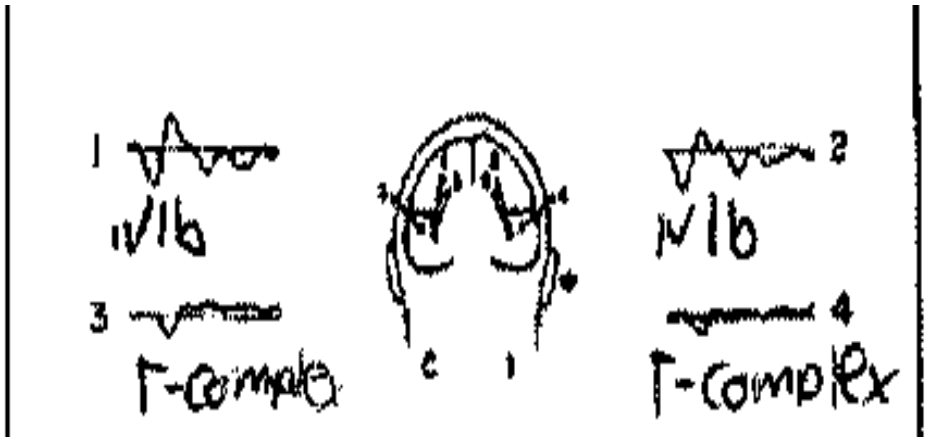

Figure 5: The dipole orientation of wave N1. Tracings number one and two represents wave N1b response underlying neural generators. Tracings number three and four represents the T-complex response underlying neural generators (Scherg et al., 1989).

Neural generators of P2. Some investigators have speculated that wave P2 reflects the same underlying neural mechanisms as wave N1b (Martin et al., 2008). However, several research groups, utilizing computer-generated topographic maps and intracranial recordings, have provided evidence that N1 and P2 are generated by different underlying neural generators (Baumann, Rogers, Papanicolaou, & Saydjari, 1990; Hari et al., 1987; Knite, Hillyard, Woods, & Neville, 1980; Rif, Hari, Hamalainen, & Sams,

1991). Collectively, the available evidence suggests that the underlying neural generators of wave P2 include: the primary auditory cortex, the secondary cortex, and the mesencephalic reticular activating system.

In 1980, Knite and colleagues recorded the slow cortical response to tonal stimuli presented at 60-dB-SL in three age-matched subject groups. A control group was comprised of 10 neurologically normal adults; the two patient groups (n=10 each) had extensive unilateral lesions of either frontal lobe or of the temporal-parietal junction. Knite et al. (1980) reported that the patients with lesions of the temporal-parietal junction had poorly developed N1 responses from all recording electrode sites; however, they had normal amplitude and latency values for wave P2, suggesting that N1 and P2 had different underlying neural generators. The overall amplitude and latency values for waves N1 and P2 were unaffected in patients with unilateral lesions of the frontal lobe (Knite et al., 1980).

Hari et al. (1987) subsequently recorded cerebral magnetic responses to noise bursts and to pauses in continuous noise to isolate the neural sources of N1 and P2 in six healthy adults. Their results indicated the underlying neural generators of P2 were separate from the neural generators for N1, confirming the findings from Knite and colleagues (1980). Specifically, P2 was stimulus related and was either absent or very small after pause onsets. The investigators also reported that the source location of P2 was more anterior than the source location of N1. Baumann et al. (1990), using dipole-source analysis, reported that the underlying source of P2 in 12 normal-hearing adults was located more anterior (.4 cm) than the underlying source of wave N1. Their results were consistent with those reported by both Hari et al. (1987) and Knite et al. (1980).

The results of these brain-imaging and intracranial studies suggest that the underlying neural generators of wave P2 differ from the underlying neural generators of wave N1. Specifically, the neural generators of P2 have been found to be more anterior than those for N1. Moreover, these generators are located within the primary auditory cortex, secondary auditory cortex, and the mesencephalic reticular activating system.

Technical Parameters

The stimulus parameters that are most critical for accurate recording of the slow cortical response are: stimulus rate, frequency, intensity, and rise/fall time. The critical recording parameters include the: electrode montage, number of recording channels, EEG analog band-pass filter setting, artifact rejection criteria, length of pre- and post-stimulus analysis window, and number of trials contributing to the average waveform. Below is a brief description of each of these stimulus and recording parameters and their effect on the slow cortical response.

Stimulus Parameters

Stimulus rate. Stimulus rate refers to the number of stimuli presented per second. A concept related to stimulus rate, which is often discussed in the AEP literature, is interstimulus interval (ISI), which refers to the silent period between stimuli. ISI is the reciprocal of stimulus rate. This reciprocal relation, however, is only true for tonal stimuli of relatively short duration, which will be employed in the current study. For example, when an investigator exports the stimulus rate as 10 stimuli per second, using a short duration tonal stimulus, then its ISI would equal 1000 ms/10, or 100 ms.

The choice of stimulus rate for recording the slow cortical response has been shown to have a considerable effect on the peak-to-peak amplitude of wave N1-P2. In the mid 1960s, Davis and colleagues conducted a series of studies to investigate the effect of stimulus rate on the amplitude of wave N1-P2 of the slow cortical response. The results of these early studies concluded that the optimal stimulus rate to use when recording the slow cortical response is approximately one stimulus per second (Davis, 1965; Davis, Mast, Yoshie, & Zerlin, 1966; Davis & Zerlin, 1966). These investigators reported that

this slow stimulus rate is optimal because of the lengthy absolute refractory period of the underlying cortical neurons. If stimuli are presented at too fast a rate (i.e., 5/sec or higher), then the cortical neurons cannot fire that quickly, resulting in a diminished amplitude for wave N1-P2 and/or complete absence of the response.

Picton, Woods, Barbeau-Barun, and Healey (1977) provided a review of the most efficient stimulus rates for recording the slow cortical evoked response. They reported that the peak-to-peak amplitude of wave N1-P2 reached its maximum at a stimulus rate between one and two stimuli per second. Therefore, Picton and colleagues reported that the most efficient stimulus rate to record the slow cortical response in adults is between one to two stimuli per second. These slow stimulus rates provide the best signal-to-noise ratio (SNR) for the response because the amplitude of wave N1-P2 is maximized. Stapells (2009) also has recommended that audiologists should use stimulus rates of one to two per second when recording the slow cortical response. The use of higher stimulus rates decreases N1-P2 amplitudes due to the absolute refractory period of cortical neurons. Therefore, in this study, the slow cortical response will be recorded at a rate of 1.1 stimuli per second.

Stimulus frequency. The peak-to-peak amplitude of wave N1-P2 decreases as stimulus frequency increases and this change in amplitude is most noticeable at the higher stimulus intensities (Antinoro, Skinner, & Jones, 1969; Evans & Deatherage, 1969; Rothman, 1970). Moreover, we now know that there is no significant change in the absolute latency of waves P1, N1, and P2 as a function of stimulus frequency. Specifically, Antinoro and colleagues (1969) investigated the relation between the peak-to-peak amplitude values for wave N1-P2 as a function of stimulus frequency in six

normal-hearing adults (23-27 years of age). They recorded the slow cortical response to tonal stimuli presented in octave steps from 125-8000 Hz at five different stimulus intensities (20, 40, 60, 80, and 100 dB SL). Antinoro et al. (1969) reported the peak-to-peak amplitude of wave N1-P2 decreased from 8.9 μV for a 500-Hz tone burst presented at 100 dB SL to 3.4 μV for an 8000-Hz tone burst presented at the same intensity. A similar pattern was seen for responses to the lower stimulus intensity signals. The magnitude/size of the in wave N1-P2 amplitude as a function of stimulus frequency was not as dramatic for the responses to the lower versus higher stimulus intensities. Specifically, as stimulus frequency increased from 500 to 8000 Hz, wave N1-P2 amplitude decreased 60% for the 100-dB-SL responses in comparison to a 40% decrease in N1-P2 amplitude for the 20-dB-SL responses.

Antinoro and colleagues (1969) speculated that the decrease in wave N1-P2 amplitude for the higher frequency responses occurs because of the pattern of the traveling wave motion on the basilar membrane. When the basilar membrane receives stimulation from a high-frequency tone (e.g., 8000 Hz), the activation is restricted to the basal end of the cochlea. In contrast, practically the entire basilar membrane is activated for a low-frequency tone (e.g., 500 Hz). As a result, a larger number of neurons discharge in response to moderate-to-high intensity tonal stimuli presented at frequencies $\leq$ 2000 Hz in comparison to the number of neurons that fire in response to high-frequency tones presented at the same levels (Antinoro et al., 1969).

Similar effects of stimulus frequency on the peak-to-peak amplitude of wave N1-P2 were reported by Evans and Deatherage (1969) and Rothman (1970). Evans and Deatherage (1969) recorded slow cortical responses to 500-4000 Hz tonal stimuli in four

adult males with normal-hearing sensitivity. The responses were recorded at 45 and 90 dB nHL. Evans and Deatherage reported an inverse relation between the peak-to-peak amplitude of the slow cortical response and stimulus frequency, such that the amplitude of the slow cortical response decreased as the stimulus frequency increased at both presentation levels. For example, Evans and Daetherage (1969) reported that when a 4000-Hz tone was presented at 90 dB nHL, the amplitude of wave N1-P2 was 45% smaller than the N1-P2 amplitude for a 500-Hz tone presented at the same intensity. A similar pattern was observed at 45 dB nHL, but the magnitude of the decrease in N1-P2 amplitude as a function of stimulus frequency was not as dramatic (Evans & Daetherage, 1969).

The following year, Rothman (1970) recorded slow cortical responses from six normal-hearing adults (16-38 years of age). The tonal stimuli ranged from 2000-8000 Hz for presentations at four sensation levels (10, 30, 50, and 70 dB). Rothman found that the peak-to-peak amplitude of wave N1-P2 decreased as stimulus frequency increased. Furthermore, Rothman reported the peak-to-peak amplitude of N1-P2 was variable between subjects, with some subjects having shallower slopes than others (especially at high frequencies). Based on this finding, Rothman (1970) suggested slow cortical responses should be recorded at frequencies $\leq$ 2000 Hz to optimize the waveform components and the SNR.

In the late 1970s, Stelmack, Achorn, & Michaud (1977) reported the absolute latency of wave N1 was longer for responses to lower frequency stimuli relative to higher frequency stimuli. Specifically, Stelmack et al. (1977) recorded slow cortical responses from 38 adult men for tonal stimuli of 500 Hz and 8000 Hz presented at 40, 55, and 80

dB nHL. They reported the absolute latency of N1 was longer for 500 Hz than for 8000 Hz for all stimulus intensities. Specifically, the latency values for N1 at 500 Hz were 137, 130, and 113 ms for the responses to the 40, 55, and 80 dB nHL stimuli, respectively. In contrast, the latency values for N1 at 8000 Hz were 108, 117, and 101 for these same presentation levels (Stelmack et al., 1977).

The collective results above indicate that as stimulus frequency increases, the N1-P2 amplitude decreases for all presentation levels. Furthermore, the latency of N1 decreases as stimulus frequency increases. In the proposed study, the slow cortical response will be recorded for a 2000-Hz tonal stimulus. This stimulus frequency was chosen for the following reasons: 1) 2000 Hz is an important frequency for speech understanding and is likely an important contributor to successful hearing-aid fittings; 2) several investigators have suggested that recording the slow cortical response at 2000 Hz will optimize the amplitude of the waveform component and the SNR; and 3) behavioral loudness judgments of 2000-Hz tonal stimuli have been reported to be reliable (Sherlock & Formby, 2005).

Stimulus intensity. In general, the results of several studies have reported that as stimulus intensity is increased, the peak-to-peak amplitude of wave N1-P2 increases and the latency of wave N1 decreases (Beagley, & Knight, 1967; Picton, Goodman, & Bryce, 1970; Picton et al., 1977; Spoor, Timmer, & Odenthal, 1969). Specifically, the N1-P2 peak-to-peak amplitude increase linearly with increasing stimulus intensity over a considerable portion of the input/output curve and saturates or decreases at high levels.

Beagley and Knight (1967) investigated the changes in the response properties of the slow cortical response as a function of stimulus intensity in eight adults with normal-

hearing sensitivity. They recorded the slow cortical response to 1000-Hz tones at 0, 10, 20, 30, 50, 60, and 70 dB nHL. Beagley and Knight (1967) reported: 1) an increase in stimulus intensity resulted in an increase N1-P2 amplitude (i.e., the mean N1-P2 amplitude value was 6.9 µV for the 20-dB-nHL responses in comparison to 13.7 µV for the 70-dB-nHL responses); 2) the absolute latency of N1 decreased as stimulus intensity increased (i.e., the mean absolute latency for wave N1 was 136 ms for the 20-dB-nHL responses versus 116 ms for the 70-dB-nHL responses); and 3) the peak-to-peak amplitude of N1-P2 saturated at stimulus intensities of approximately 60-70 dB SL and higher (Beagley & Knight, 1967).

Picton and colleagues (1970) also reported that the peak-to-peak amplitude of N1-P2 saturated at high stimulus intensities (i.e., at intensities above 70 dB ISO). An example of this saturation effect is shown in Figure 6, which reveals the peak-to-peak amplitude of N1-P2 increases linearly from 10 to 60 dB, but saturates at intensities greater than 70 dB ISO.

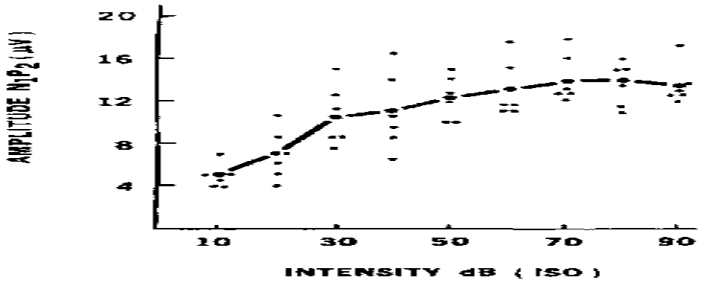

Figure 6: Peak-to-peak amplitude of wave N1-P2 increases linearly as stimulus intensities increase from 10-60 dB, then saturates above 70 dB ISO (Picton et al., 1970).

Spoor et al. (1969) recorded slow cortical responses to 1000-Hz tone bursts in three adults with normal-hearing sensitivity. The tone bursts were presented at levels ranging from 10-90 dB SL. Similar to Beagley and Knight (1969), Spoor et al. (1969)

reported the amplitude values for N1-P2 essentially doubled as the intensity increased from 10 to 90 dB SL (i.e., the mean amplitude value of wave N1-P2 was 7.2 µV for the 10-dB-SL responses in comparison to 14.3 µV for the 90-dB-SL responses). Likewise, the latency of N1 decreased from 150 ms at 10 dB SL to 89 ms at 90 dB SL. Unlike Beagley and Knight (1967), however, Spoor et al. (1969) found that the peak-to-peak amplitude of wave N1-P2 did not decrease or saturate at high stimulus intensities.

Collectively, the results indicate that as stimulus intensity increases, the peak-to-peak amplitude of N1-P2 increases in a linear fashion over the majority of the intensity range, while the latency of N1 decreases. The amplitude of wave N1-P2 tends to saturate at high intensities. The exact stimulus intensity at which response saturation occurs remains to be established. In the current study, the response of the slow cortical response will be measured over a range of stimulus intensities (10-80 dB nHL) to evaluate the influence of saturation of wave N1-P2 amplitude.

Stimulus rise/fall time. The rise time of a tonal stimulus represents "the time needed for the temporal waveform to change from 10% to 90% of its peak amplitude value (full amplitude)". In contrast, the fall time is "the time needed for the temporal waveform to change from 90% to 10% of its peak amplitude value" (Emanuel & Letowski, 2009, p. 64). The plateau is the amount of time the amplitude of the stimulus rate remains stable at its full amplitude. For auditory evoked potential stimuli, these rise and fall time values, as well as the length of the plateau, are expressed either in milliseconds and/or cycles per second. These temporal characteristics of a toneburst for AEP recordings are shown in Figure 7.

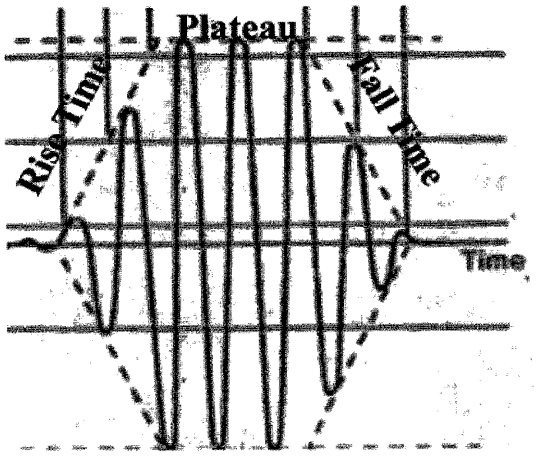

Figure 7: Temporal characteristics of rise and fall time of a tonal stimulus (P. Korczak, personal communication, November 4, 2011).

In 1968, Onishi and Davis conducted an experiment to investigate the effects of rise time on N1-P2 amplitude. These investigators presented 1000-Hz tone bursts combined with each of six rise times, 3, 10, 30, 50, 100, and 300 ms, at three different stimulus intensities, 85, 65, and 45 dB nHL. The average results for four subjects are shown in Figure 8. Peak-to-peak amplitude of wave N1-P2 was stable for rise times $\leq$ 30 ms. In contrast, N1-P2 amplitude decreased for rise times $\geq$50 ms. These findings were independent of stimulus intensity (Onishi & Davis, 1968).

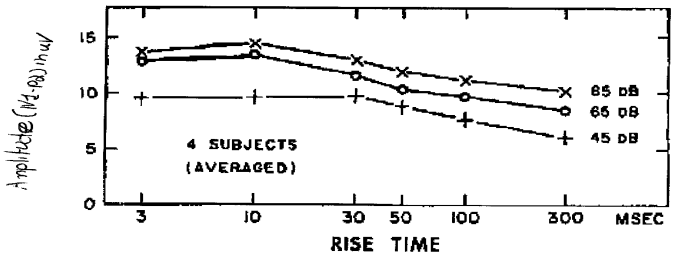

Figure 8: Effects of peak-to-peak amplitude (μV) averaged across four subjects as a function of rise time (ms). Peak-to-peak amplitude values remain essentially stable for rise times of ≤30 ms and substantially decreases for rise times ≥50 ms (Onishi et al., 1968).

Kodera, Hink, Yamada, and Suzuki (1979) recorded slow cortical responses as a function of rise time, 5, 10, and 20 ms, for 1000-Hz tone bursts presented at 60 dB SL. The results for eight normal subjects are displayed in Figure 9. Kodera and colleagues (1979) reported that longer rise times (i.e., 20 ms versus 5 ms) were associated with slightly smaller, non-significant peak-to-peak amplitude values for waves P1-N1 and N1-P2. Kodera and colleagues (1979) recommended the use of a 20-ms rise/fall time in comparison to a shorter rise/fall time to enhance frequency specificity and to improve predictions of pure-tone threshold.

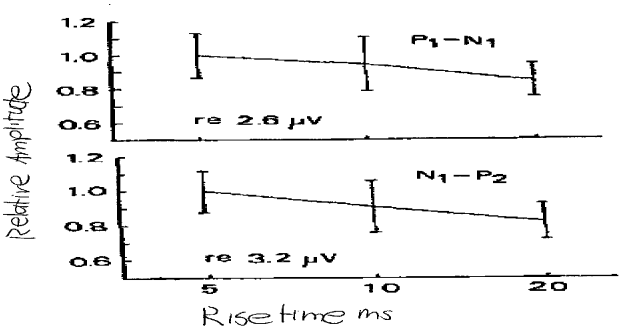

Figure 9: Effect of rise time on peak-to-peak amplitude for waves P1-N1 and N1-P2 as a function of relative amplitude. Amplitude values decreased with increasing rise times, but this finding was not statistically significant (Kodera et al., 1979).

Based on these findings, Stapells (2009) recommended that slow cortical responses be recorded using tonal stimuli with a 20-ms rise time, 20-ms plateau, and 20-ms fall time, irrespective of stimulus frequency. Therefore, we will record the slow cortical response to a 2000-Hz tone burst with 20-ms rise and fall times and a 20-ms plateau, thus yielding a total stimulus duration of 60 ms.

Recording Parameters

The slow cortical response can also be impacted by several recording parameters, including the electrode montage, the number of recording channels, the EEG analog band-pass filter setting, the artifact rejection criteria, the length of the pre- and post-stimulus analysis window, and the number of trials contributing to the average waveform. Below is a brief description of each of these recording parameters.

Electrode montage. Electrode montage defines where the electrodes are placed on the subject's scalp. Electrode sites are labeled using the International 10-20 system. According to this system, the ground electrode, located on the center portion of the forehead, is labeled Fpz, the left earlobe is labeled A1, the right earlobe is labeled A2,

and the vertex is labeled Cz (Jasper, 1958). To estimate behavioral threshold, the typical electrode montage used to record the slow cortical response is similar to that used to record the ABR. Specifically, the non-inverting, or active electrode, is located on the vertex (Cz); the ground electrode is located on the forehead (Fpz); and the inverting or reference electrode is located on the ipsilateral and contralateral mastoids or earlobes (M1 or M2; or A1 or A2) (Martin et al., 2007). In contrast, for supra-threshold recordings, the ground electrode remains on the forehead (Fpz) and the inverting/reference electrode is placed on the tip of the nose. The nose is selected as the location for the reference electrode because it is in line with the main generators of the slow cortical response in the supratemporal plane of the auditory cortex (Vaughan & Ritter, 1970). Vaughan and Ritter have also demonstrated that when the reference electrode was placed at other sites on the scalp, such as the inion or on the mastoid, the response was subjected to myogenic contamination. Therefore, these investigators have recommended that the nose be used as the reference electrode because it is aligned with the underlying neural generators of the response and is not subject to muscle contamination.

In our study, the ground electrode will be placed on the forehead (Fpz). Three non-inverting electrodes will be placed on the frontal lobe (Fz), the vertex (Cz), and the parietal lobe (Pz) of the scalp to assess the scalp distribution of the response, which is discussed in the next section of this literature review. An electrode also will be placed above and below the right eye to record contaminating artifacts from the eyes. This concept will be discussed in more detail later in this literature review. Lastly, the inverting/reference electrode will be placed on the tip of the nose as recommended by Vaughan and Ritter (1970).

Number of recording channels. The number of channels to optimize recordings of the slow cortical response differs depending on the purpose of the response. As previously mentioned, the slow cortical evoked response can be used to estimate behavioral thresholds and suprathreshold measures. If the purpose of the slow cortical response is to estimate behavioral threshold, then only one or two recording channels are needed (Martin et al., 2007). Suprathreshold recordings, on the other hand, require multiple channels to represent responses from multiple underlying neural generators based on their expected scalp topography (i.e., their amplitude distribution across the scalp) (Scherg & von Cramon 1986; Vaughan & Ritter, 1970). Several studies have demonstrated that the slow cortical response has its maximum amplitude at the fronto-central locations on the scalp (i.e., Fz and Cz) (Martin et al., 2007; Stapells, 2009).

Shown in Figure 10 is the scalp topography of the slow cortical response recorded to a 1000-Hz tone burst at a high stimulus intensity. The boxed area represents responses recorded from the central locations on the scalp. The reference electrode for these recordings was an electrode located at the tip of the nose, with the non-inverting electrodes distributed over various locations along the scalp. As can be seen in Figure 10, the amplitudes of P1-N1 and N1-P2 decrease systematically from fronto-central electrode sites (i.e., Fz, FCz, Cz) to the posterior electrode sites (i.e., Pz, and Oz).

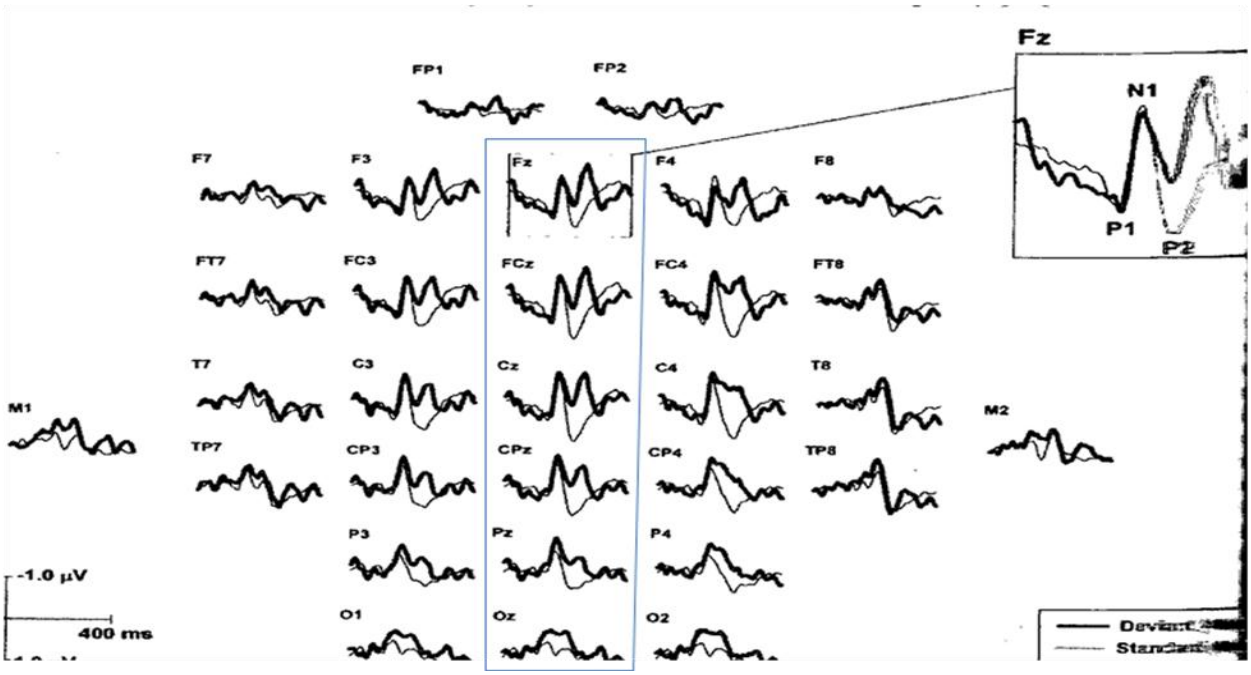

Figure 10: The expected scalp topography of the slow cortical response from multiple electrodes located on the scalp. The midline recordings are found in the inset box. The amplitude of the P1-N1 and N1-P2 responses decrease from the front of the scalp (i.e., Fz, FCz, Cz) to the back of the scalp (i.e., Pz, Oz) (Stapells, 2009).

In our research, the slow cortical response will be recorded simultaneously using a 4-channel recording system. The electrode montage on the first three channels (i.e., channel 1: Fz-nose; channel 2: Cz-nose; and channel 3: Pz-nose) will assist in clearly identifying the P1-N1-P2 response based upon scalp topography. The fourth recording channel will be used to eliminate contaminating artifacts from the eyes; one electrode will be placed above the right eye and a second electrode will be placed below the right eye (Martin et al., 2007). The contribution of any ocular artifact to the final averaged waveform will be eliminated using an artifact-rejection criteria of $\leq$100 µV, as discussed below.

EEG analog band-pass filtering. The band-pass (BP) filter settings for AEPs optimize the recording of frequencies specific to passing a desired neural signal, (i.e., the slow cortical response), while eliminating the random background electroencephaology (EEG) and myogenic activity that is not contributing to this response (Roger & Thornton, 2007). The band-pass analog filter is comprised of two sections: a high pass (HP) filter, which allows frequencies higher than the cutoff frequency to be passed, and a low pass (LP) filter, which allows frequencies lower than the cutoff frequency to be passed. A typical band-pass filter characteristic is shown in Figure 11. Frequencies within the pass-band of the filter between the HP and the LP cut-off frequencies are passed without attenuation.

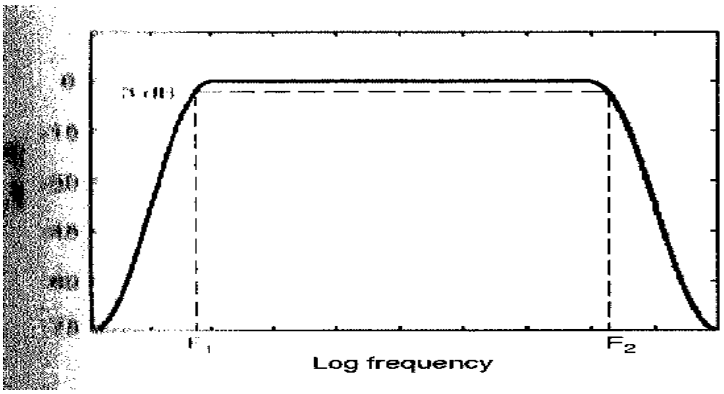

Figure 11: Band-pass filter characteristics, frequencies between F1 (LP) and F2 (HP) pass through without being attenuated (Roger & Thornton, 2007).

Fast-Fourier-Tranform (FFT) techniques demonstrate that most of the energy present in the slow cortical response to click/tonal stimuli is present at very low frequencies (Sayers, Beagley, & Henshall, 1974; Yamamoto, Sakabe, & Kaiho, 1979). Specifically, the slow cortical evoked response recorded to a moderate intensity tone burst in a normal-hearing adult has its peak energy at approximately 5-6 Hz, with little energy below 2 Hz or above 10 Hz (Hyde, 1997). Davis (1965) successfully recorded the

slow cortical response to tonal stimuli using a bandpass filter setting of 0.3 to 35 Hz to estimate behavioral thresholds. These band-pass filter parameters allowed Davis to capture the primary energy present in the response at low stimulus intensities at optimum SNRs.

Hyde (1997) and Roger and Thornton (2007) recommended analog band-pass filter settings of either 1 to 15 Hz or 1 to 30 Hz to record the slow cortical response to tonal or click stimuli in adults. The specific choice of the exact LP and HP cutoff frequencies for the analog EEG band-pass filter setting, in part, depends on the particular filter settings available on the AEP equipment. In our study, the slow cortical response will be recorded using the Intelligent Hearing System (IHS) Smart EP system. The band-pass filter will be set to pass response frequencies from 1 to 30 Hz. The default slope of the band-pass filter on the IHS system is 12 dB/octave, which has been recommended by Stapells (2009).

Artifact rejection. The purpose of employing an artifact rejection criterion, while recording an auditory evoked potential, is to minimize the EEG background noise present in the recording. A second purpose of employing an artifact rejection criterion is to maximize the SNR of the slow cortical evoked response (Martin et al., 2007). One possible confounding noise source is eye blink and/or random eye movements (Martin et al., 2007). This possible confounding effect due to ocular artifact can be assessed by placing electrodes above and below one of the subject's eyes. The artifacts generated by eye blinks and/or eye movements are often quite large in amplitude and can potentially mimic the response of interest. Therefore, Martin and colleagues have suggested two approaches to reduce this confounding effect during the recording of the cortical

potentials. The first approach is to instruct the patient to minimize eye blinking during the recording. This approach may be problematic for a passive paradigm and when recording from young children (Martin et al., 2007). The second approach is to employ an artifact rejection criterion of +/- 100 μV on all recording channels. When this artifact-rejection criterion is applied, EEG data that exceed this criterion are not allowed to contribute to the average waveform. The advantage of using this second approach is that the EEG data are preserved, and undesirable effects of eye blinks are reduced or eliminated (Martin et al., 2007).

The subjects in our study will be instructed to minimize eye blinks by fixating on a specific visual target (i.e., a smiley face on the wall). Additionally, an artifact rejection criterion of +/- 100 μV on all four recording channels will be applied to prevent ocular artifacts from contributing to the average waveform.

Pre and post-stimulus analysis window. The purpose of setting an appropriate length for the pre-and post-stimulus analysis window is to ensure the recording of the major components of the slow cortical response (i.e., waves P1, N1, and P2). The analysis window for the slow cortical response contains a pre-stimulus interval and a post-stimulus interval. According to Hall (2007), a pre-stimulus analysis window of 100 ms has been used successfully in many slow cortical response studies. A 100-ms pre-stimulus window has been suggested because it provides a stable estimate of the EEG background noise and a baseline calculation of the amplitudes for negative and positive waveform components. The post-stimulus analysis window is greater and should extend for at least 500 ms after the stimulus to ensure that all components of the waveform are captured. Hall (2007) and Martin and colleagues (2007) recommend a pre-stimulus time

of -100 ms, and a post-stimulus time of at least 700 ms, which will be used as the recording analysis window in our study.

Trials and replications contributing to the average. Trials or "sweeps" are defined as the number of times the stimulus is presented for one averaged waveform (Hyde, 1997). Replications are defined as the number times the averaged waveform is replicated (Hyde, 1997). The number of trials/replications that contribute to the average response affects the quality of the response. According to Hyde (1997), as well as Roger and Thornton (2007), the number of sweeps/trials that contributes to the total waveform average is based on clinical experience rather than any scientific rationale. Furthermore, Roger and Thornton (2007) mention that observers are able to make accurate decisions about response detection when the SNR is two or more. Both Roger and Thornton (2007) and Hyde (1997) recommend that each average of the slow cortical response should consist of at least 50 sweeps/trials, and that at least two replications should be obtained at each stimulus intensity. Therefore, we will record 50 sweeps, replicated at least twice, for a total of at least 100 sweeps at each stimulus intensity to obtain an average response.

Subject Related Parameters

There are a number of subject-related factors that can impact the recording of the slow cortical response, most notably the subject's state and age. Below is a brief description of these influences on the slow cortical response.

Subject-State

Attention. Attention to the task at hand is accomplished by brain mechanisms that act to enhance information from sound sources and to suppress information from irrelevant competing sources (Hilyard, Hink, Schwent, & Picton, 1973). The N1 amplitude is known to be larger when subjects attend to a task in comparison to passive performance. It is unclear why wave N1 is larger when the subject is attentive to the stimulus. One thought is that the larger amplitude is due to selective enhancement. Hilyard et al. (1973) conducted an experiment in which tone pips were presented at rapid stimulus rates to 10 subjects. In this experiment, a sequence of 800-Hz tone pips (50 dB above threshold) were delivered to the left ear, while an independent series of 1500-Hz tone pips (50 dB above threshold) were delivered simultaneously to the right ear. The subjects were instructed to: A) attend to the left ear, discriminate and count the number of 800-Hz signals, and report the total at the end of the recording; B) read a novel and disregard all tones; and C) attend to the right ear and count the number of 1500-Hz tone pips. Hilyard and colleagues (1973) reported that the N1 amplitude was dependent upon the type of attending condition. For example, the N1 amplitude was larger when the right ear was the attending ear compared with the response obtained when the left ear was the attending ear.

Another rationale, proposed by Naatanen (1982), is that the increased wave N1 amplitude in the attended condition is a result of a "processing negativity" superimposed on the N1 component. This means that the enhanced wave N1 amplitude is due to a negative brain potential elicited by the delivery of the stimuli, which is then added to/superimposed on the original wave N1 response. Naatanen (1982) went on to explain that the processing negativity has two components: sensory specific and frontal. The sensory-specific component reflects the identification process by which a sensory input is gradually identified as corresponding to the attentional trace. The frontal component appears to reflect further processing after the match between the sensory input and the attention trace has occurred (Naatanen, 1982). In a review of N1, Naatanen and Picton (1987) suggested that it may be possible that both hypotheses about the effects of attention on the amplitude on wave N1 are correct.

Sleep. Sleep is another factor that may influence the slow cortical response. Osterhammel, Davis, Wier, and Hirsh (1973) and Stapells (2009) reported large and complex changes in the latencies of waves P1, N1, and P2 when the subject is asleep. During sleep, a large negative component at approximately 300 ms becomes evident in the slow cortical response. This emergent wave is sometimes referred to as wave N2 (Osterhammel et al., 1973; Stapells, 2009). The slow cortical response, recorded during an awake state, light sleep stage, medium sleep stage, and deep sleep stage, is shown in Figure 12. The subject's sleep was monitored by EEG and reported as a delta index in the right column of Figure 12. The slow cortical response (N1-P2) is clearly evident in the awake stage, and is delayed and not well formed in the light, medium, and deep sleep stages. In the deepest stage of sleep (stage 4), we see the emergence of the large negative

waveform component, labeled wave N2, which does not exist when the patient is awake (Osterhammel et al., 1973). Osterhammel and colleagues reported that the absolute latencies of waves N1 and P2 occurred later as the subject's sleep stage advanced. Therefore, they suggested that slow cortical responses be recorded in an awake alert subject.

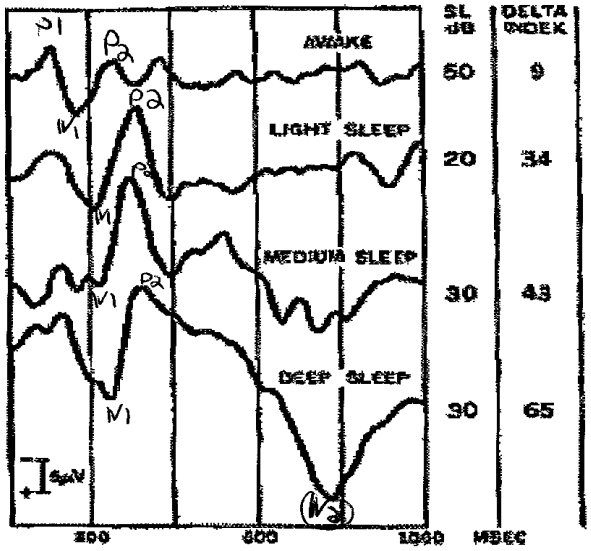

Figure 12: Various sleep-stage recordings from one subject. The N1-P2 complex is clearly evident in the awake stage, while the typical N1-P2 response is not evident in the light, medium, and deep sleep stage (Osterhammel et al., 1973).

To control for attention and sleep state, we will record the slow cortical response in awake adult subjects. The subject's state will be monitored by observing the subject throughout the test session as well as watching their EEG during the recordings. Secondly, a sequence of 2000-Hz tonal stimuli will be delivered monaurally and the subject will not be asked to attend to a stimulus in the opposite ear.

Age-Related

Maturation/age. The slow cortical response undergoes substantial maturational changes during childhood. The response continues to change throughout the teenage years (up through approximately 18 years of age) as the Central Nervous System (CNS) matures. The response changes include changes in scalp distribution, amplitude, and latency (Gomes et al., 2001; Kurtzburg, 1989; Kurtzburg, Hilpert, Kreuzer, & Vaughan, 1984; Novak, Kurtzburg, Kreuzer, & Vaughan, 1989). In addition, the waveform morphology of the slow cortical response changes quite dramatically from childhood to adulthood (Sharma et al., 1997; Stapells, 2009). The slow cortical response in adults consists of two positive peaks (labeled waves P1 & P2) and one negative peak (labeled wave N1). The absolute latencies of P1, N1, and P2 occurs at approximately 50, 80-100, and 180-200 ms post-stimulus onset, respectively. In contrast, the slow cortical response in young children is characterized by a large P1 wave at approximately 150-200 ms, which may or may not be followed by a distinguishable N1 wave.

Shown in Figure 13 are examples of slow cortical evoked responses for an adult and a seven-year-old child, each with normal-hearing sensitivity. Wave P1, with a latency of ~140 ms, is the prominent component in the child's response, followed by a broad wave N1 peak at approximately 250 ms. In contrast, wave N1 is the prominent component in the adult's response, occurring around 100 ms, proceeded by a small wave P1 at approximately 50 ms. We do not expect to see any maturational effects in this study, which will be conducted with adult subjects.

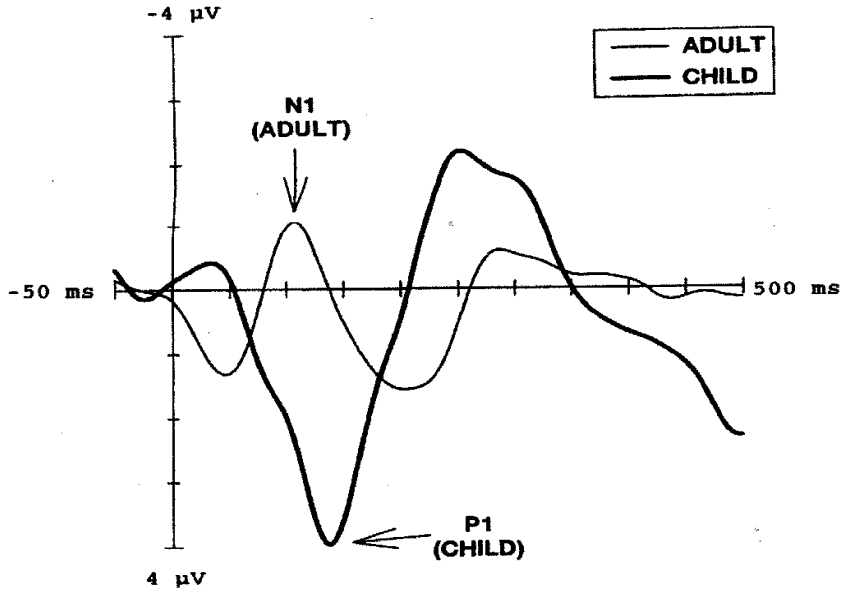

Figure 13: An adult's and seven-year old child's slow cortical response recording. In the adult's response, wave N1 is the prominent component; whereas, in the child's response, wave P1 is the prominent component. In this figure negativity is shown inversely and each segment along the X-axis represents 50 ms (Stapells, 2009).

Functional Significance

As mentioned earlier, the slow cortical response is used for two clinical purposes. The first is to estimate behavioral pure-tone thresholds in older teens and adults. The slow cortical response also can be used for suprathreshold applications to determine hearing-aid benefit and assess loudness growth. Threshold and supratheshold clinical applications of the slow cortical response are considered below.

Behavioral Threshold Estimation

Several investigators in the early 1960s speculated that the slow cortical response could be used to estimate hearing sensitivity (Appleby, McDermick, & Scott, 1963; Beagley, & Kellogg, 1969; McCandless, & Best, 1964). Specifically, Appleby and colleagues (1963) conducted a pilot study to determine if the slow cortical response to tonal stimuli could be used to assess hearing loss. Five adults with normal-hearing sensitivity and adults with hearing loss participated in the study. The investigators reported that a replicable P1-N1-P2 response was obtained approximately 15-25 dB above the behavioral pure-tone thresholds for both subject groups. Appleby et al. (1963) suggested that this response might provide a method for accurately testing infants' hearing sensitivity.

A year later, McCandless and Best (1964) recorded slow cortical responses to click stimuli in normal hearing adults, infants, and children up through five years of age. McCandless and Best (1964) reported: 1) the adult threshold values for the slow cortical response were within +/- 5 dB of their most sensitive behavioral threshold over the 250 to 4000 Hz frequency region; and 2) all children had an observable slow cortical response to clicks presented at 30 dB HL (re: audiometric zero). McCandless and Best (1964)

concluded that the slow cortical response can be reliably obtained to click stimuli within 10 dB of an individual's pure-tone audiogram. The utility of the slow cortical response in predicting behavioral pure-tone thresholds in adults with normal-hearing sensitivity and sensorinerual hearing loss was subsequently confirmed by Beagley and Kellogg (1969).

Despite the early evidence that the slow cortical response to may be used to estimate pure-tone hearing sensitivity, the ABR ultimately became the AEP of clinical choice for this purpose (Martin et al., 2008). The ABR enjoyed several advantages over the slow cortical response for behavioral threshold estimation. Namely, the ABR is not affected by the subject's attention to the task, nor by natural and/or pharmacologically induced sleep. Moreover, the ABR is highly replicable in individual subjects (Stapells, Gravel, & Martin, 1995).

Within the last two decades, the slow cortical response has enjoyed renewed interest as a tool to estimate behavioral threshold in adult compensation cases and medicolegal cases (Hyde; 1997; Hyde, Matsumoto, Alberti, & Li, 1986). The advantage of the slow cortical response to estimate behavioral pure-tone thresholds for this population is that it permits the audiologist to assess the integrity of the auditory pathway up through and including the auditory cortex. Investigators have now shown that the slow cortical response can provide accurate estimates of behavioral thresholds within 5-10 dB (Hyde, 1997; Hyde et al., 1986; Lightfoot & Kennedy, 2006; Stapells, 2009). Specifically, Lightfoot and Kennedy (2006) reported that 94% of individual threshold estimations were within 15 dB of audiometric pure-tone thresholds at 1000, 3000, and 8000 Hz. Thus, Lightfoot and Kennedy (2006) concluded that the slow cortical response

is an accurate tool to estimate pure-tone thresholds, and is within 10-15 dB of audiometric pure-tone thresholds for adults and older children.

Suprathreshold Clinical Applications of the slow cortical response

The P1-N1-P2 complex, which is also reflected in the Acoustic Change Complex (ACC), can be elicited by speech stimuli. If the P1-N1-P2 response can be successfully recorded to speech stimuli, such as /ba/ vs. /da/, then this provides objective evidence that the speech signals audible and discriminable to the cortex. Several investigators reported that one suprathreshold application of the slow cortical response is to determine hearing-aid benefit in both the adult and pediatric populations (Gravel, Kurtzberg, Stapells, Vaughan, & Wallace, 1989; Korczak et al., 2005). For instance, Korczak and colleagues (2005) conducted a study to assess the combined effects of sensorineural hearing loss and personal prescribed hearing aids on the response properties of cortical ERPs. They recorded various cortical ERPs to the speech syllables /ba/ and /da/, which were presented in soundfield at 65 and 80 dB SPL to a group of 14 adults with hearing loss. Response properties in aided versus unaided test conditions were recorded and compared. Korczak and colleagues (2005) reported that the amplitude of wave N1-P2 was substantially larger and the latency of wave N1 was substantially shorter in the aided versus unaided condition at both stimulus intensities. These investigators concluded that the presence of a larger N1-P2 amplitude and a shorter wave N1 and P2 latencies in the aided condition provided objective evidence that the aided speech signal was being processed at the cortical level in these hearing-impaired individuals. The shorter ERP latencies for aided listening indicated the brain was efficiently detecting and processing

the aided information. Similar findings were reported in a pediatric population (Gravel, 1989).

Another suprathreshold clinical application of the slow cortical response is to estimate growth of loudness. In 2001, Hoppe and colleagues conducted a study to investigate the response properties of the slow cortical response and an association with loudness perception in eight adult cochlear implant users. The stimuli in this study were trains of pulses presented electrically through the processor of the cochlear implant to a single electrode. Initially, the electrical thresholds and uncomfortable loudness levels (UCLs) were determined behaviorally. Slow cortical responses then were recorded at intensity levels ranging from each subject's threshold to his/her UCLs. These investigators reported that because the N1-P2 complex was the most stable deflection in the AEP, they evaluated the absolute latency of wave N1 and the peak-to-peak amplitude of N1-P2. Their results indicated that both the amplitudes and the latencies of the slow cortical responses depended on loudness perception. Specifically, the peak-to-peak amplitudes of N1-P2 varied between 2 μV close to threshold and 13 μV at UCL and tended to increase linearly with increasing loudness. Similarly, the absolute latency of N1 decreased with increasing stimulus loudness, such that for the lowest audible loudness category, the absolute latencies of wave N1 were between 110 and 185 ms. At the UCL, the latencies for N1 were between 78 and 105 ms. Hoppe et al. (2001) concluded that the peak-to-peak amplitudes of N1-P2 and the latency of N1 of the slow cortical response are correlated with behavioral loudness judgments for electrical stimulation in adult cochlear-implant users.

Our study will focus on the possible suprathreshold clinical application of the slow cortical response recorded to acoustic stimuli (i.e., 2000-Hz brief tone burst) in young normal-hearing adults. Specifically, we will determine the relation, if any, between the amplitudes and latencies of waves P1, N1, and P2 of the slow cortical response to 2000-Hz tonal stimuli and loudness judgments for these same stimuli in a group of young normal-hearing adults. We will use Absolute Magnitude Estimation to measure loudness perception. In our study, we will also be looking at the potential relation between loudness and the annoyance level of these same 2000-Hz tonal stimuli. The annoyance level of the tonal stimuli will be evaluated by using an annoyance rating scale adapted from Hiramatsu, Takagi, and Yamamoto (1988).

The next section of this literature review considers the behavioral perception of loudness of this 2000-Hz tone as well as annoyance level. The following topics will be discussed below: a general description of loudness, two behavioral measurements of loudness perception (i.e., category scaling and magnitude estimation), a brief description of annoyance perception and how it is measured, and a review of the literature that utilized evoked potentials to determine the relations between response properties of AEPs and loudness perception.

Loudness

The intensity of a sound refers to its physical magnitude, which may be expressed in terms as its power or pressure (Gelfand, 2010). The physical intensity of a sound can be measured using a sound level meter. In contrast, the loudness of a sound is a subjective judgment or percept of the auditory sensation that most closely corresponds to sound level. This percept may be judged or categorized on a scale extending from soft to loud (Stevens, 1936). Even though an increase in the intensity of a stimulus is associated with an increase in its loudness, a simple one-to-one correspondence between intensity and loudness does not exist. The loudness of a sound is affected by a number of additional factors such as: range of stimulus intensities presented, range of stimulus frequencies presented, instructions to the subject, and the range of permissible responses. Another possible factor that may influence loudness is annoyance of the stimulus. This factor will be explored further in this study. The influence of these additional factors on the subject's loudness judgments will be discussed later.

When measuring the relation between sound intensity and subjective loudness perception, the subject's auditory dynamic range is of interest. The dynamic range of hearing is the range of intensities between the subject's behavioral threshold and their threshold of discomfort expressed in dB (Hawkins, Walden, Montgomery, & Prosek, 1987). Therefore, determination of hearing thresholds is a central tool in determining the relation between sound intensity and loudness perception. Two general types of psychophysical scaling procedures have been proposed to measure the relation between perception and stimulus magnitude for suprathreshold stimuli. These two approaches are

just-noticeable-difference (JND) thresholds and a method utilizing more direct methods of scaling (Fechner, 1966; Stevens, 1936).

A measure of the JND threshold is one approach to obtain an estimate of the smallest detectable difference in intensity that can be discriminated 50% of the time. Gustav Fechner, in the 1800s, was the first person to suggest that the JND may be used to quantify the intensity-loudness growth transformation (Fechner, 1966). Fechner based his research on the earlier work of E. H. Weber. Weber reported that the JND increases in direct proportion to increases in stimulus intensity. As a result of this finding, Fechner proposed that the sensation of loudness increases by a constant amount each time the stimulus is increased by a JND. This relation has been described by the fraction (Δ I/I, which is known as the Weber fraction). In this Weber fraction, the numerator "ΔI" is the just noticeable physical difference in stimulus intensity and the denominator "I" refers to the base value or the smaller of the two values being discriminated (Yost & Nielson, 1977). Therefore, all JNDs could be added to estimate a scale of the total subjective magnitude of the sensation ($P=K \log S$), where K is a constant, P is the subjective loudness unit, and S is the physical intensity unit (Fechner, 1966). Fechner's two assumptions were: 1) all JND's could be summed to produce a scale of the subjective sensation; and 2) any JND is one unit along a scale of that subjective attribute. Despite the fact that these two assumptions have not held up well to experimental testing employing pure-tone stimuli (Miller, 1947; Stevens, 1936), other methods of obtaining scales relating a subjective attribute to a physical stimulus have been derived from his work (Yost & Nielsen, 1977).

In the mid 1930s, S. S. Stevens proposed another approach that utilizes more direct methods of scaling. In these scaling methods, different stimulus intensities are presented within the subject's dynamic range and subjective estimates are used directly to form a function relating the perceived magnitude of the stimulus to the physical intensity of the signal (Stevens, 1936). In these direct methods of scaling, the loudness function varies depending on which scaling method is employed. Two types of direct behavioral scaling methods are partition (Keidser, Seymour, Dillon, Grant, Byrne, 1999) and magnitude scales (Hellman & Meiselman, 1993). The most commonly used partition procedure is category scaling and the most commonly used magnitude procedure is magnitude estimation. Below is a brief description of categorical scaling. However, since magnitude estimation will be the behavioral loudness measurement employed in the proposed study, the remainder of this section of the literature review will focus on this latter technique.

Category Scaling

Category scaling is a partition technique in which subjects are asked to label their perception of loudness of the sensation according to a set of categories defined by a number and/or an adjective (i.e. 1= "soft", 5= "loud, but OK"), and these categories are outlined prior to the presentation of stimuli (Allen, Hall, & Jeng, 1990; Bentler & Pavlovic, 1989; Cox, Alexander, Talyor, & Gray, 1997; Guirao, 1991; Hawkins, et al., 1987). In this technique, subjects are required to place a range of stimuli into equally spaced or sized categories, and the data that result from these judgments are transformed to derive loudness growth functions. Factors that may affect the form of the function derived from categorical scaling are contextual effects, judgment bias, and number of

categories (Guirao, 1991; Stevens & Galanter, 1957). For instance, a small number (i. e., 1-7) of categories yields a much shallower slope for loudness growth compared to the function derived from either a partition scale which includes a larger range of categories (i. e., 1-100), or from magnitudes which allow the subject the widest possible range of choices in units (Guirao, 1991; Stevens & Galanter, 1957).

A second type of loudness scaling technique is magnitude estimation. In our study, loudness for brief tonal stimuli will be investigated using a magnitude estimation scaling procedure. Therefore, the remainder of this literature review will focus on this technique. The following topics will be discussed: a description of magnitude estimation, the Sone Scale, Steven's Power Law, Absolute Magnitude Estimation (AME), and normative magnitude estimation data.

Magnitude Estimation

According to Gescheider (1988), the most commonly used magnitude partition technique to estimate the loudness of a signal is magnitude estimation. In the mid 1950s, Stevens (1955, 1956) used this technique to make numerical estimations of the sensory magnitude produced by various stimuli. Specifically, Stevens posed the question; "How do people describe sounds when we ask them to use a numerical language instead of adjectives"? (Stevens, 1955, p. 815) In other words, Stevens (1955) asked his subjects to judge the loudness of a sound on a ratio scale. His subjects were given a reference stimulus (i.e., 10 units). Then, the subjects were asked to report their perception of how much louder or softer a second stimulus was in relation to this reference stimulus. This concept of magnitude estimation is the basis of the sone scale, which is the unit of loudness (Stevens, 1955).

According to the sone scale, one sone is equal to the loudness of a 1000-Hz tone presented at 40 dB SPL (Stevens, 1957). In Dr. Steven's (1955) experiment, a series of eight stimuli were presented to 26 subjects at intensities ranging from 40 to 110 dB in 10-dB increments. The subjects were instructed that when they heard the first tone they should assign any number they thought was appropriate to the loudness of that stimulus. Then, a second tone was presented and the subjects were instructed to give this second tone a loudness number. In other words, the subjects were judging whether the loudness of the second stimulus was twice as loud, half as loud, or whatever relation it had to the reference stimulus. The loudness judgments were then plotted to show the relation with sound intensity. Stevens (1955) reported that each 10- dB increase in the intensity of the stimulus resulted in an approximate doubling of loudness of the stimulus; thus, doubling of the value of the sone (Stevens, 1955).

The sone scale is shown below in Figure 14. One sone is shown to be equal to the loudness of a 1000-Hz tone presented at 40 dB SPL. The loudness of this 1000-Hz tone doubles when the stimulus increases by 10 dB. At 40 dB and above, the sone scale is linear when plotted as a function of sound level, revealing a power function relation of the form: L=KI^e; where the perception of loudness (L) can be expressed as a power (e) of the physical stimulus level (I), with K as a constant. This formula is known as the Steven's power law (Stevens, 1957).

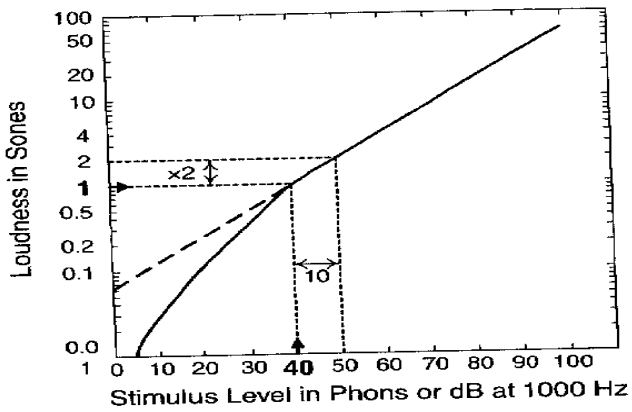

Figure 14: A graph of the sone scale. One sone is the loudness of a 40-dB tone at 1000 Hz. Loudness doubles when the simulus i.ncreases by 10 dB, at 40 dB and above (Gelfand, 2010).

Stevens power law. Steven's power law states that sensation of loudness grows as a function of stimulus level (Stevens, 1957). The exponent in the formula L=KI^e indicates the rate at which the sensation grows with stimulus magnitude. The basic assumption of magnitude estimation is that the listener is able to match his/her perception of the loudness of the stimulus using a ratio scale (Stevens, 1955, 1956, 1957). According to Stevens's definition of a ratio scale, a subject's numerical judgments should be indifferent to a mathematical transformation when the value of the reference stimulus is changed. However, this linear relation, as shown above, only applies above 40 dB. In contrast, at lower stimulus levels, the loudness function becomes steeper, indicating that loudness growth is faster at lower stimulus intensities (Hellman & Zwislocki, 1961, 1963). As a result, Hellman and Zwislocki (1961, 1963, 1964, 1968) modified Steven's magnitude estimation technique. Specifically, Hellman and Zwislocki suggested that listeners pair loudness and numbers on an absolute basis with a fixed unit rather than a ratio scale with an arbitrary unit. This modification to Dr. Steven's magnitude estimation

technique is known as Absolute Magnitude Estimation (AME) (Hellman & Zwislocki, 1961, 1963, 1964, 1968).

Absolute magnitude estimation (AME). The AME hypothesis states that subjects assign a number to a stimulus in such a way that their impression of the size of the number matches their impression of the sensation magnitude of the stimulus (Hellman & Zwislocki, 1961, 1963, 1964). Therefore, subjects are not restricted in their choice of numbers used; rather, they are encouraged to assign numbers they feel to be appropriate without any biases that may be associated with a response system devised by the experimenter. This adaptation to Stevens's magnitude estimation technique reduced biases due to self-imposed restricted response ranges. Specifically, subjects are told that any positive number that appears appropriate (i.e., whole numbers, decimals, or fractions) can be used. Furthermore, to discourage attempts to judge stimuli relative to one another, subjects are told to ignore numbers assigned to preceding stimuli when judging a particular stimulus (Gescheider, 1988). Several studies have shown consistency in the data obtained using the AME technique, which demonstrates the validity of this behavioral loudness judgment technique.

Normative magnitude estimation data. There is a large body of empirical evidence in support of the general form of the loudness function obtained with AME from normal-hearing adults (Fucci, Petrosino, Mcnoll, Wyatt, & Wilcox, 1997; Gescheider & Hughson, 1991; Hellman & Meiselman, 1988, 1990, 1993; Hellman & Zwisolocki, 1961, 1963, 1964, 1968; Zwislocki, 1983; Zwislocki & Goodman, 1980). For example, Hellman and Meiselman (1993) conducted a study to analyze the loudness growth rate of 32 normal-hearing older adults (aged 56-72) to various tone-burst stimuli

obtained utilizing the AME technique. The tone-burst stimuli employed in this study were presented at nine different sensation levels (4, 10, 15, 20, 30, 40, 50, 60, and 70 dB SL) at frequencies from 500-2000 Hz. Shown in Figure 15 are the average slopes of loudness-growth functions for a group of young normal-hearing adults and for a group of older adults (Hellman & Meiselman, 1988, 1993). The solid line represents the results from the young adults, and the dashed line represents the results from the older adults. Both curves have a clear peak in their loudness functions near 0.6 (re: sound pressure), (Hellman & Meiselman, 1993). Therefore, Hellman and Meiselman (1993) concluded that the slope of loudness-growth function obtained using AME is 0.6 (re: sound pressure), which is applicable for both young and older adults with normal-hearing sensitivity.

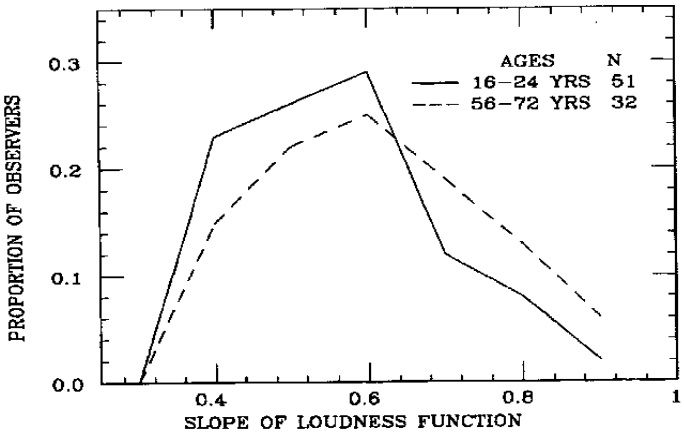

Figure 15: Loudness-growth slope values in a group of young adults and older adults. Younger adults are the solid line and older adults are the dashed-line. The loudness-growth slope value was approximately 0 .6 (re: sound pressure) in both groups (Hellman & Meiselman, 1993).

The results from the studies mentioned above were obtained from subjects tested individually in sound-attenuating rooms. Similar results have also been reported from groups of listeners tested in reverberant conditions (i.e., classrooms, and auditoriums). For example, Canevet, Hellman, and Scharf (1986) conducted a study to investigate the loudness growth function in a group of 392 adult listeners with normal-hearing sensitivity in an anechoic chamber, semi-reverberant classrooms, and auditoriums. The listeners judged the loudness of 1000-Hz tone bursts presented at 10, 20, 30, 40, 50, and 60 dB SPL using the AME technique. These results are shown in Figure 16. Shown in the left panel is the loudness-growth function for the group tested in an anechoic chamber and shown in the right panel is the loudness-growth function for the group tested in semi-reverberant rooms. Canevet and colleagues reported the slope value of the loudness growth function for the group of listeners in the anechoic chambers was 0.55. Similarly, the overall average slope of the loudness growth from the group of listeners in classrooms was 0.6 (0.3 re: sound intensity). Thus the AME technique is a reliable and valid method for measuring loudness growth in normal-hearing adult listeners.

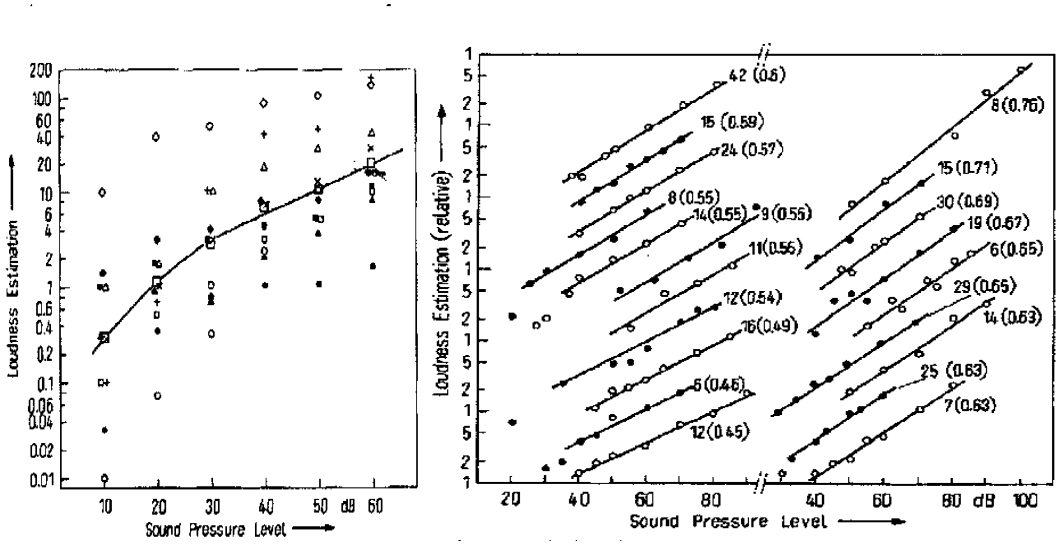

Figure 16: Left, loudness-growth function in subjects tested in an anechoic chamber room. Approximate slope value was 0 .55. Right, loudness-growth function in subjects tested in semi-reverberant rooms. Overall slope value ranged from 0.45 to 0 .76 (Canevet et al., 1986).

A related concept to loudness is annoyance. In this study, we will investigate the relation between annoyance and loudness to the 2000-Hz tone burst stimuli. A brief description of what annoyance is, factors that affect annoyance, and studies that compared annoyance and loudness ratings are briefly described below.

Annoyance

A concept that may be related to how an individual perceives loudness is annoyance level of the stimulus. Annoyance can be viewed as an indicator of negative reaction to noise (Job, 1988). A listener's annoyance perception depends on specific changes to noise conditions as well as non-acoustical factors (Job, 1988). Lazlo McRobie, Stansfeld, and Hansell (2012) conducted a 30-year review of the literature to identify acoustical as well as non-acoustic factors that influence noise annoyance ratings. These investigators found that in noisy environments the annoyance of the stimulus was

influenced by factors including: activity disturbance (i.e., window open vs. window closed); method of noise reduction (i.e., reducing the noise level at the source); prior experience of the 'before' situation (i.e., long-term exposure vs. newcomers); and media coverage of the change (i.e., availability and accessibility to information about the planned changes). Non-acoustical factors, which influence the subject's annoyance, included certain demographic, social, personal, and situational factors. Specific examples are noise sensitivity, attitude toward the noise source, and malfeasance. Additional non-acoustical factors that influence annoyance perception were time of day the noise occurred, the situation when the noise exposure occurs, and where the noise took place (i.e., outside or inside) (Laszlo et al., 2012).

Because annoyance and loudness perception are related concepts, several studies have been conducted to determine the relation/correlation, if any, between loudness and annoyance ratings to the same noise stimulus. Kuwano, Namba, & Fastl (1988) conducted a study in which 16 normal-hearing Japanese and German adults judged the loudness, noisiness and annoyance of actual and artificial aircraft and road traffic noise. Their results indicated that various factors, such as the temporal pattern of the stimulus, frequencies present in the stimulus, the duration of the stimulus, and the subjective meaning of the stimulus contributed to the concepts loudness, noisiness, and annoyance. In this study, the subjective meaning of the term annoyance had a greater effect on the judgments made by the German subjects than the Japanese subjects. Specifically, the Germans judged the stimuli on the basis of its maximum level; whereas, the Japanese subjects judged the stimuli on the basis of its energy. As a result of this finding, Kuwano

et al. (1988) suggested that the subjective meaning of the stimulus affect noisiness and annoyance judgments more than the actual loudness judgments.

Similarly, in another study conducted by Hiramatsu et al. (1988), 50 normal-hearing adults judged the loudness and annoyance on a variety of stimuli (59 environmental sounds and seven kinds of white noise). These investigators utilized a seven-step bipolar scale from "very unpleasant" to very pleasant" to measure annoyance ratings. In our study, we will be modifying this annoyance rating scale. This modification to the annoyance rating scale will be described in more detail in the methods section. The results from this study indicated that all three of these perceptual aspects of the stimulus are mutually correlated and that judgment of loudness is not independent of noisiness and/or annoyance of the sound (Hiramatsu et al., 1988). However, compared to either judgments of loudness and noisiness or noisiness and annoyance, judgments of loudness and annoyance displayed a lower correlation. The correlation coefficient between loudness and noisiness was 0.867; the correlation coefficient between noisiness and annoyance was 0.850; and the correlation coefficient between loudness and annoyance was 0.543. This finding suggests that sounds which are judged to be equally as loud may differ in their annoyance level to the subjects. In other words, some people will judge the sound as "pleasant" while other may rate the sound as "unpleasant" (Hiramatsu et al., 1988). In our study, we will measure both the loudness judgments and the annoyance judgments of 2000-Hz tonal stimuli to determine if a relation exists between the auditory percepts. The primary focus of our study is to determine if a relation exists between response properties of the slow cortical response and loudness judgments. However, we

also will investigate the relation, if any, between loudness and annoyance ratings for these tonal stimuli.

As previously mentioned, there have been a few studies that investigated the relation between the response properties (i.e., amplitudes and latencies) of various AEPs and loudness judgments. The results of these studies are discussed below.

Loudness Growths using the ABR and MLR

Beginning in the 1970s, several investigators investigated whether there was a relation/correlation between the response properties of click-evoked ABRs and/or MLRs and loudness for these same click stimuli in normal-hearing adults (Darling & Price, 1990; Davidson, et al., 1990; Howe & Decker, 1984; Madell & Goldstein, 1972; Pratt & Sohmer, 1977; Serpanos, et al., 1997; Wilson & Stelmack, 1982). These investigators concluded that there is no correlation between the response properties (i.e., amplitudes and latencies) of click-evoked ABRs/MLRs and the loudness perception for these same stimuli. Serpanos et al.(1997) speculated that "the use of more frequency-specific stimuli, such as brief tones, may provide more precise information on the nature of the relation between loudness growth and ABR wave V latency, particularly for listeners with sloping configurations of cochlear hearing loss" (p. 409).

Within the last decade, four studies have investigated whether tone-burst ABRs/MLRs provide a better understanding of the neural processes that underlie loudness (Korczak et al., in preparation; Nousak, 2001; Serpanos, 2004; Silva & Epstein, 2010). In these studies, the investigators recorded these two AEPs to tone-burst stimuli ranging in frequency from 500-4000 Hz. Judgments of loudness for these tonal stimuli was determined using a variety of techniques across these studies including: an AME

technique (Nousak, 2001), a cross-modality matching (CMM) technique (Serpanos, 2004), a combination of CMM and AME techniques (Silva & Epstein, 2010), and the Contour loudness test (Korczak et al., in preparation). Collectively, the results from these four studies suggest that tone-burst ABRs/MLRs appears to provide a better measure of the neural processes that underlie behavioral loudness perception than do click-evoked responses.

Loudness Growth using the Slow Cortical Response in Adult Cochlear-Implant users

As previously described, Hoppe et al. (2001) reported a significant relation exists between the response properties of the slow cortical response and judgments of loudness among cochlear-implant subjects. The stimuli used in this study were electrical pulse trains presented to a single electrode. Therefore, it is now important for us to investigate whether there is a relation between response properties of the slow cortical response and growth of loudness for acoustic stimuli. Accordingly, the purpose of this study is to investigate a relation, if any, between the peak-to-peak amplitude of waves P1-N1 and N1-P2 of the slow cortical response to 2000-Hz tonal stimuli and loudness perception for these same tonal stimuli in a group of young normal-hearing adults. We will also be investigating the relation, if any, between the absolute latencies of waves P1, N1, and P2 of the slow cortical response to 2000-Hz tonal stimuli and loudness perception for these same tonal stimuli in a group of young normal-hearing adults. Each of the waveform components will be evaluated separately. In our study, loudness perception will be evaluated using the AME technique.

Statement of Purpose

The goals/aim of the proposed study will be to:

1. Determine the relations, if any, between the peak-to-peak amplitudes and absolute latencies of waves P1, N1, and P2 of the slow cortical response to 2000-Hz tonal stimuli and judgments of loudness for these same tonal stimuli. An AME protocol will be used to delineate and characterize the growth of loudness in a group of normal-hearing adults.
2. If relations do exist, then the current investigators will characterize these relations.
3. Determine the relation, if any, between judgments of loudness for the 2000-Hz tonal stimuli and corresponding ratings of annoyance.

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CHAPTER 3

METHODS

Subjects

Eleven young adults, recruited via word of mouth, participated in the current study. The participants ranged in age from 23-26 years (mean age= 24.1 years SD= 0.9) and included three males and eight females. To be included in the study, participants met the following selection criteria: 1) pure-tone air-conduction thresholds $\leq$ 15 dB HL from 250-8000 Hz in both ears (Martin & Champlin, 2000); 2) normal tympanograms as defined by middle ear peak pressure ranging from +100 to -150 daPa, static admittance values ranging from .37-1.66 ml, and ear canal volumes of .63-1.46 ml (Margolis & Heller, 1987) ; 3) contralateral acoustic reflexes at 500-2000 Hz within the 90^{th} percentile range (Gelfand, Schwander, & Silman, 1990) ; 4) no self-reported otologic or neurologic history; and 5) no evidence of nor complaints of sound intolerance using a case history developed from the hyperacusis questionnaire employed at the University of Maryland Medical Center (see case history form in Appendix A). This study was approved by Towson University's Institutional Review Board (IRB).

Procedures

All testing was conducted at the Institute of Well-being (IWB) at Towson University in a double-walled sound-treated booth and took place in one test session, which lasted approximately 2 hours per participant. This test session included audiologic and electrophsyiologic testing. Each participant was asked to complete a case history form, which was sent to them prior to the test session. Each subject's responses were reviewed prior to the test session to ensure that he/she qualified for the study. During the

test session, an otoscopic examination was conducted first, followed by tympanometry and contralateral acoustic reflex testing. Next, pure-tone air-conduction testing was completed. If any participant did not meet the subject inclusion criteria, then he/she did not participate further in the study.

Each qualified subject participated in an experiment in which he/she judged the loudness of brief 2000-Hz tones using the AME protocol (to be described below) and then rated the annoyance for these same stimuli. After the AME and annoyance procedures were completed, slow cortical responses were recorded to these same stimuli. Both the behavioral and electrophysiological measures were obtained from the right ear across all subjects.

Test Protocol for the AME and Annoyance Technique

Each subject was asked to judge the loudness and annoyance of a series of brief 2000-Hz tonal stimuli. These stimuli were generated by the Intelligent Hearing System (IHS) Smart EP equipment. Each tonal stimulus had 20-ms rise and fall times, and 20-ms plateau for a total duration of 60 ms. These tonal stimuli were delivered to each subject via an ER-3A insert earphone. The tone bursts were presented at a rate of 1.1 stimuli per second.

Each subject heard a series of seven tonal stimuli presented at one stimulus intensity. Following the presentation of these seven stimuli, the subject was asked to assign a number (i.e., whole number, fraction, or decimal), providing a judgment of the magnitude/loudness of that tonal sequence. Any positive number, regardless of its magnitude was acceptable, including repeated use of the same number. Each subject was encouraged to be spontaneous about their decision, and to make his/her decision as

quickly as possible. In addition, each subject was asked to provide an annoyance rating value of 1-6 (i.e., very pleasant to very unpleasant) for each tonal sequence. This annoyance rating scale was adapted from the annoyance rating scale used by Hiramatsu et al. (1988). These investigators used a seven-step bipolar scale that ranged from "very pleasant" to "very unpleasant". The exact scale that Hiramatsu and colleagues used was not provided and was in Japanese. The annoyance rating scale used in this study is provided in Appendix B.

The level of the first train of stimuli started at a stimulus intensity between 10-80 dB nHL. The starting stimulus intensity, within this 80 dB range, was randomized across subjects. The 2000-Hz tonal stimuli were presented at levels of 10, 20, 30, 40, 50, 60, 70, and 80 dB nHL for each subject. At each of these stimulus intensities, the subject was asked to assign a positive number for the loudness, as well as an annoyance rating (both the number and corresponding descriptive term), for that train of seven stimuli. This procedure was repeated three times for each subject. The geometric mean of the last two numerical judgments of loudness and annoyance at each stimulus intensity was used for the data analysis for each subject. Each subject was given a set of written instructions for this procedure, which is provided in Appendix C.

After the AME and annoyance rating procedures were completed, the slow cortical responses to the same stimuli were recorded from each subject in the same ear used for loudness judgments. Below is a description of the test protocol for recording the slow cortical response.

Slow Cortical Response

The slow cortical responses were recorded using the Intelligent Hearing System (IHS) Smart EP system. During the recording, each subject was asked to sit quietly and relax. They also were instructed not to sleep during the recording. The subject's state was monitored by the examiner observing the subject as well as monitoring any changes in their ongoing EEG using the Smart EP software. The acoustic stimuli consisted of 2000-Hz tone bursts, ranging from 10 to 80 dB nHL in 10 dB increments. After the slow cortical response was recorded at each stimulus intensity, each subject was asked again to provide judgments of the loudness and annoyance at that stimulus intensity. This was completed one time following the recording of the slow cortical response at each stimulus intensity. These judgments were made using the same procedures described above.

Stimulus Parameters

Again, the brief 2000-Hz tone-burst signal (20-ms rise time + 20-ms plateau + 20 ms fall time) was generated by the IHS system and delivered to the right ear using an ER-3A insert earphone. The 2000-Hz tonal stimuli were presented a rate of 1.1/second and the stimulus was alternating in polarity to eliminate any possible stimulus artifact. The slow cortical responses were recorded at eight separate stimulus intensities. These stimulus intensities corresponded to the same levels that were presented for measurement of the loudness and annoyance judgments. The starting intensity for the electrophysiologic recording was 80 dB nHL and each successive run decreased in intensity by 10 dB.

Recording Parameters

The slow cortical response was recorded simultaneously from four separate recording channels. Gold-cup electrodes were placed on the following locations on the scalp: Fz, Cz, and Pz for the non-inverting electrodes; the nose for the reference or inverting electrode; and the forehead (Fpz) served as the ground electrode for all recording channels. Lastly, two electrodes were placed, one above and one below the right eye to monitor and minimize the influence of eye blinks or random eye movements. Specifically, the electrode montage for the four recording channels was as follows: channel 1: Fz to nose; channel 2: Cz-nose; channel 3: Pz-nose; and channel 4: electrodes above and below the right eye. Electrode impedances were less than or equal to 3 kOhms, and inter-electrode impedances were less than or equal to 2 kOhms for all subjects.

The following recording parameters were applied to all four recording channels. The analog EEG band-pass filter setting was 1-30 Hz and the amplifier gain was 20,000 Hz. The artifact rejection criterion was +/- 100 µV and was applied to the entire analysis window. The length of the pre- and post- stimulus analysis window was set to -100 to 700 ms, respectively for all trials. Each trial consisted of 50 sweeps, with at least two replications, yielding summed responses that consisted of at least 100 sweeps for the response at each stimulus intensity.

Identification of the Slow Cortical Response

We used the expected scalp distribution of the slow cortical response, which is a larger amplitude P1-N1-P2 response at the fronto-central electrode sites (i.e., Fz-nose, which was recorded on channel 1 and Cz-nose, which was recorded on channel 2) in comparison to the back of the head (i.e., Pz-nose, which was recorded on channel 3) to

identify the P1-N1-P2 complex. The investigator also used the recording obtained on channel 4 (i.e., above and below the right eye) to rule out influences of eye blinks or ocular movements in the expected latency range of the slow cortical response (~50-200 ms). After the P1-N1-P2 complex had been clearly identified at each stimulus intensity, a series of amplitude and latency measures were taken from the P1-N1-P2 complex. These response measurements were only taken from the response recorded at the vertex (Cz-nose or channel 2). These response measurements are described below.

Amplitude and Latency Measurements for the Slow Cortical Response

Peak-to-peak amplitudes of waves P1-N1 and N1-P2, as well as the absolute latencies of waves P1, N1, and P2, were measured from each subject's summed waveform (i.e., 100 trials) recorded at the vertex (Cz-nose) at each of the eight stimulus intensities. The peak-to-peak amplitude of wave P1-N1 was defined as the difference in the amplitude values obtained for wave P1 and wave N1. Similarly, the peak-to-peak amplitude values for wave N1-P2 was defined as the difference in the amplitude values obtained for wave N1 and wave P2.

In contrast, the absolute latency of wave P1 was defined as the first positive peak occurring between 30 and 50 ms post-stimulus onset. The absolute latency of wave N1 was defined as the largest negativity occurring between 80 and 100 ms, and the absolute latency of wave P2 was defined as the second positive peak occurring between 150 and 200 ms (Hyde, 1997; Martin et al., 2008; Naatanen & Picton, 1989).

Statistical Analyses

Descriptive statistics (i.e., mean and standard deviation (SD) values) were calculated separately for the behavioral and electrophysiological measures. Specifically, these descriptive statistics were calculated for the subjects' pre-and post-AME loudness data as well as their pre-and post- annoyance ratings for the 2000- Hz tonal stimuli obtained at each of these eight stimulus intensities. In the pre-condition, these behavioral measurements were based on the geometric mean of the subject's last two loudness judgments and last two annoyance ratings at that stimulus intensity. The behavioral measurements for the post-condition were based on the one loudness judgment and annoyance rating each subject made following the recording of the slow cortical response at each stimulus intensity. A series of dependent t-tests were completed to determine if there was a statistically significant difference between AME judgments obtained prior to the slow cortical response and AME judgments following the recording of the slow cortical response. Similarly, a series of dependent t-tests were completed to determine if there was a statistically significant difference between annoyance ratings obtained prior to the slow cortical response and annoyance ratings following the slow cortical response. The alpha level used for statistical significance was $p \leq 0.5$.

For the electrophysiologic response measurements, descriptive statistics were calculated on the subjects' peak-to-peak amplitude values for waves P1-N1 and N1-P2 at each stimulus intensity. Similarly, mean and SD values were calculated for the absolute latencies of waves P1, N1, and P2 at each stimulus intensity.

The loudness judgments for each stimulus intensity were then plotted in a scatter plot. AME loudness judgments were plotted as a function of stimulus intensity. Similarly, the

annoyance ratings for each stimulus intensity were plotted in a scatter plot. Annoyance ratings were plotted as a function of each individual's AME loudness judgments. Linear-regression functions were then fit to calculate the slope of AME behavioral judgments as a function of stimulus intensity, as well as the slope for the annoyance ratings as a function of the AME loudness judgments.

For the electrophysiologic response measurements, the peak-peak amplitude values for waves P1-N1 and N1-P2 at each stimulus intensity also were plotted in a scatter plot. Amplitude values were plotted as a function of stimulus intensity (i.e., peak-to-peak amplitude value of wave P1-N1 as a function of stimulus intensity; and peak-to-peak amplitude value of wave N1-P2 as a function of stimulus intensity). Similarly, the absolute latency values of waves P1, N1, and P2 at each stimulus intensity were plotted in a scatter plot, with latency values plotted as a function of stimulus intensity (i.e., absolute latency value of wave P1 as a function of stimulus intensity; absolute latency value of wave N1 as a function of stimulus intensity; and absolute latency value of wave P2 as a function of stimulus intensity). Linear-regression functions were fit to each scatter plot to determine the slopes of the peak-to-peak amplitude values of wave P1-N1 as a function of stimulus intensity and peak-to-peak amplitude values of wave N1-P2 as a function of stimulus intensity. Likewise, linear-regression functions were fit to each scatter plot to determine the slopes of the absolute latency of wave P1 as a function of stimulus intensity, absolute latency of wave N1 as a function of stimulus intensity, and absolute latency of wave P2 as a function of stimulus intensity.

Spearman's rank coefficient correlation values were then calculated to determine a relation between the AME loudness judgments and the response properties of the slow

cortical response and/or between loudness judgments and annoyance ratings. Each data set was evaluated independently. Specifically, the Spearman rank coefficient correlation values were calculated to determine a relation between the loudness judgments and the peak-to-peak amplitude values of wave P1-N1, and/or peak-to-peak amplitude of wave N1-P2. Similarly, the Spearman rank coefficient correlation values were calculated to determine a relation between the loudness judgments and the absolute latency values for waves P1, N1, and/or P2. Lastly, a Spearman's rank coefficient correlation value was calculated to determine a relation between the loudness judgments and annoyance ratings.

CHAPTER 4

RESULTS

In the results section, the behavioral data are presented first, followed by the measurements of the slow cortical response. Specifically, the behavioral data section includes: 1) loudness judgments obtained prior to the slow cortical response recordings; 2) annoyance ratings that accompanied the loudness judgments; and 3) comparison of the loudness judgments and annoyance ratings obtained prior to the slow cortical response versus those obtained following the slow cortical response. The response measurements for the slow cortical response section include: 1) response identification and the expected scalp distribution (i.e., decrease in amplitude going from Fz-Pz and no response in the ocular artifact channel); 2) peak-to-peak amplitude measurements of waves P1-N1 and N1-P2; and 3) absolute latency measurements of waves P1, N1, and P2. Lastly, relations between the behavioral loudness judgments and the slow cortical response measurements are considered as a possible objective tool to estimate the growth of loudness of tonal stimuli.

Behavioral Results

AME loudness judgments prior to the slow cortical response. Each subject assigned a positive number for the loudness of the 2000-Hz tonal stimuli at each stimulus intensity (ranging from 10-80 dB nHL). This rating procedure was repeated three times for each subject. The geometric mean of the last two numerical judgments of loudness at each stimulus intensity was used in the data analysis for each subject.

Shown in Table 1 are the individual and group AME data. Along the left side of the table is each individual subject's ID number and going across the top of the table are

the eight stimulus intensities, ranging in decreasing value from 80 dB nHL to 10 dB nHL. The geometric mean of each subject's loudness judgments is reported for each stimulus intensity. Shown in the last three rows at the bottom of the table are the overall arithmetic means for the loudness judgments as a function of stimulus intensity; the variability in the data as represented by the SD values the range; and the coefficient of variance (Cv) of the loudness judgments at each stimulus intensity. Cv values were calculated by dividing the SD by the mean value at each stimulus intensity. Cv is an index/measure which normalizes the variability seen in in these loudness judgments.

Table 1: AME judgments for the 2000-Hz tonal stimuli as a function of stimulus intensity for each individual subject (n=11). The descriptive statistics for the group are displayed in bold test at the bottom of each column.

Subject ID	**80 dB nHL**	**70 dB nHL**	**60 dB nHL**	**50 dB nHL**	**40 dB nHL**	**30 dB nHL**	**20 dB nHL**	**10 dB nHL**
587259	5.5	4.5	2.0	2.4	2.0	1.0	1.0	1.0
812139	7.0	6.0	3.4	4.0	3.0	3.0	2.0	2.0
944349	20.0	13.4	8.9	7.9	6.5	5.9	4.0	3.5
158829	7.0	6.0	4.5	4.0	2.4	2.9	1.4	1.0
770424	6.7	4.7	3.2	2.7	2.0	1.7	1.5	1.0
529299	7.2	4.0	3.2	2.5	1.4	1.3	1.0	0.7
163557	94.9	54.8	42.4	31.6	17.3	22.4	14.1	7.1
254354	16.0	14.5	5.0	3.9	3.0	3.0	1.0	1.0
654261	5.0	4.0	3.5	3.0	2.0	2.0	2.0	1.0
761336	6.5	4.5	3.5	2.4	2.0	1.0	1.0	1.0
817468	2.4	2.0	2.0	2.0	1.4	1.4	1.0	1.0
MEAN	**16.2**	**10.8**	**7.4**	**6.0**	**3.9**	**4.1**	**2.7**	**1.8**
SD	**26.6**	**15.1**	**11.8**	**8.6**	**4.7**	**6.2**	**3.9**	**1.9**
RANGE	**2.4-94.9**	**2.0-54.8**	**2.0-42.4**	**2.0-31.6**	**1.4-17.3**	**1.0-22.4**	**1.0-14.1**	**0.7-7.1**
Cv	**1.6**	**1.4**	**1.6**	**1.4**	**1.2**	**1.5**	**1.4**	**1.1**

There were several interesting trends seen in the AME data. First, as expected, there was a decrease in loudness judgments as a function of decreasing stimulus intensity.

This is reflected in the arithmetic mean values. Specifically, the overall arithmetic mean for the 2000-Hz tonal stimuli presented at 80 dB nHL was 16.2 compared to the mean value of 1.8 seen at 10 dB nHL. This trend was consistent across all stimulus intensities with the exception of the loudness judgments for the 30-dB-nHL stimulus. The 30-dB-nHL stimulus was judged slightly louder (4.1) than the 40-dB-nHL stimulus (3.9). Second, larger changes in mean loudness judgments occurred between the higher stimulus intensities compared with changes at lower stimulus intensities (i.e., a 5.4 change in mean loudness judgment values between 80-70 dB nHL; whereas, only a 0.9 change in mean AME values for a similar 10-dB interval at the lowest stimulus intensities 20-10 dB nHL). Third, as expected, there was a wide variability in loudness judgment data, as indicated by the SD values. This variability decreased as stimulus intensity decreased. However, when the variability was normalized via calculation of Cv values, the normalized variability was relatively consistent across stimulus intensities. Of note, the loudness judgments values for subject 163557 were much larger in comparison to the other subjects' AME data. This finding most likely contributed to this large variability seen in the SD values. Fourth, as anticipated, a wider range of AME values occurred at the high-moderate stimulus intensities (i.e., 80-50 dB nHL) in comparison to the lower stimulus intensities. For instance, the range for the 80-dB-nHL stimulus was 2.4-94.9; whereas, the range for the 10-dB-nHL stimulus was 0.7-7.1.

Each individual's AME loudness judgments are plotted as a function of stimulus intensity in Figure 17. A linear-regression function was fit to the data. The Spearman's correlation coefficient value was r=0.33, indicating a low correlation (Polite & Beck, 2012) between AME loudness judgments and stimulus intensity.

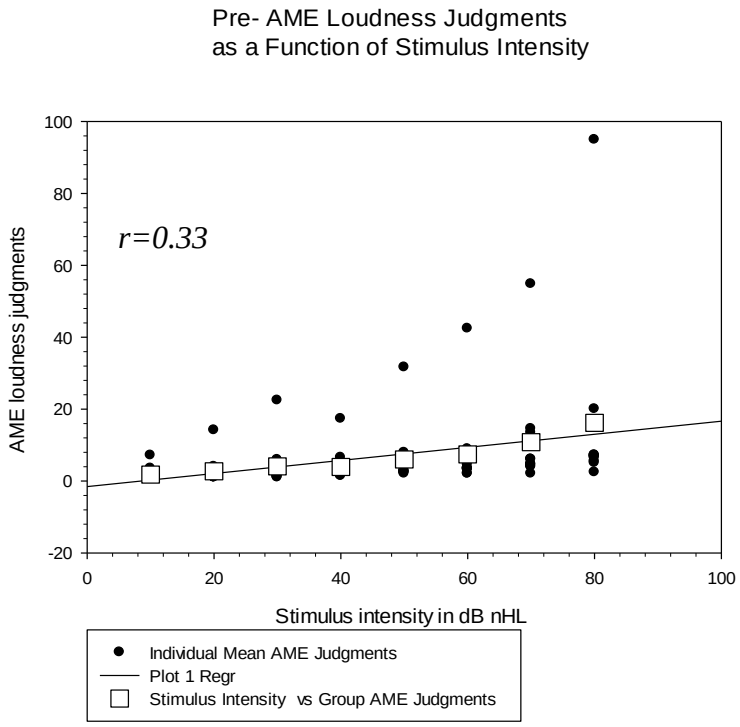


Figure 17: A scatter plot of individual subjects' (n=11) and the group's AME loudness judgments mean as a function of stimulus intensity. The filled circles represent each subject's loudness judgments and the larger open squares represent the group's mean. A linear regression function was fit through the 11 individual AME judgments and the Spearman's correlation coefficient value is r=0.33.

Annoyance judgments prior to recording of the slow cortical response. A second task was for each subject to assign an annoyance rating, using both the number and corresponding descriptive term, for the train of 2000-Hz tonal stimuli. This annoyance rating scale is shown on the bottom of Table 2. Similar to the loudness judgments, the geometric mean of the last two numerical judgments of annoyance at each stimulus intensity was used for the data analysis for each subject. Table 2, shown below, displays the geometric mean of each subject's annoyance judgments as a function of stimulus intensity. The organization of this table is the same as Table 1. The descriptive

statistics (i.e., mean, SD, range, and Cv values) for these annoyance judgments are indicated in bold at the bottom of each column. The descriptive term that correlates with its numerical number is shown at the bottom of the table.

Table 2: Annoyance ratings as a function of stimulus intensity for each individual subject (n=11). The descriptive statistics for the group's annoyance ratings are displayed in bold text at the bottom of each column.

Subject ID	**80 dB nHL**	**70 dB nHL**	**60 dB nHL**	**50 dB nHL**	**40 dB nHL**	**30 dB nHL**	**20 dB nHL**	**10 dB nHL**
587259	3.0	3.0	2.0	2.0	2.0	2.0	2.0	2.0
812139	4.0	3.0	2.4	2.0	2.0	2.0	2.0	2.0
944349	3.0	3.0	3.0	3.0	2.0	2.0	2.0	2.0
158829	4.0	3.0	3.0	2.0	1.4	1.0	1.0	1.0
770424	3.0	3.0	2.0	2.0	1.4	1.0	1.0	1.0
529299	3.5	1.4	2.0	2.0	2.0	1.4	1.4	2.0
163557	6.0	4.0	3.0	2.4	2.0	2.0	1.4	1.0
254354	6.0	5.5	2.4	2.0	2.0	2.0	1.0	1.0
654261	3.0	3.0	2.4	2.0	2.0	2.0	2.0	2.0
761336	5.5	3.0	3.5	2.4	1.0	1.0	1.0	1.0
817468	1.7	2.0	1.4	2.0	1.0	1.0	1.0	1.0
MEAN	**3.9**	**3.1**	**2.5**	**2.2**	**1.7**	**1.6**	**1.4**	**1.5**
SD	**1.4**	**1.0**	**0.6**	**0.3**	**0.4**	**0.5**	**0.5**	**0.5**
RANGE	**1.7-6.0**	**1.4-5.5**	**1.4-3.5**	**2.0-3.0**	**1.0-2.0**	**1.0-2.0**	**1.0-2.0**	**1.0-2.0**
Cv	**0.4**	**0.3**	**0.2**	**0.1**	**0.2**	**0.3**	**0.4**	**0.3**
Annoyance rating scale: **6= very unpleasant** **5= unpleasant** **4= unpleasant, but tolerable** **3= tolerable** **2= pleasant** **1= very pleasant**								

The group trends for the annoyance ratings were similar to the ones seen for the loudness judgments. First, as expected, there was a decrease in annoyance ratings for the

2000-Hz tonal stimuli as stimulus intensity decreased. Specifically, the mean annoyance rating for an 80-dB-nHL stimulus was 3.9 (unpleasant but tolerable) and decreased to 1.5 (between very pleasant and pleasant) for the 10-dB-nHL stimulus. Somewhat surprising is that stimuli presented at the two highest stimulus levels (i.e., 80 and 70 dB nHL) were judged to be tolerable (number 3) and unpleasant but tolerable (number 4). Only two subjects (163557 and 254354) judged the 80-dB-nHL stimulus to be very unpleasant (number 6). Second, the variability in these ratings, as indicated by the SD and Cv values, was quite low and was very similar across intensities. This was especially true for the moderate to low stimulus intensities (60-10 dB nHL). Third, as anticipated, a wider range of annoyance ratings occurred at high stimulus intensities (80 and 70 dB nHL) in comparison to the mid-low stimulus intensities (60-10 dB nHL). For instance, the range for the 80 and 70-dB-nHL stimulus intensity was 1.7-6.0 (pleasant to very unpleasant) and 1.4-5.5 (pleasant to unpleasant), respectively; whereas, the range for the 50 and 10-dB-nHL stimulus intensity was 2.0-3.0 (pleasant to tolerable) and 1.0-2.0 (very pleasant to pleasant), respectively.

Each individual's annoyance ratings are plotted in Figure 18 as a function of their loudness judgments. The best fitting linear regression function and Spearman's correlation coefficient are shown for this data. The correlation value was r=0.55. This finding suggests that annoyance judgments are moderately correlated (Polite & Beck, 2012) with loudness judgments.

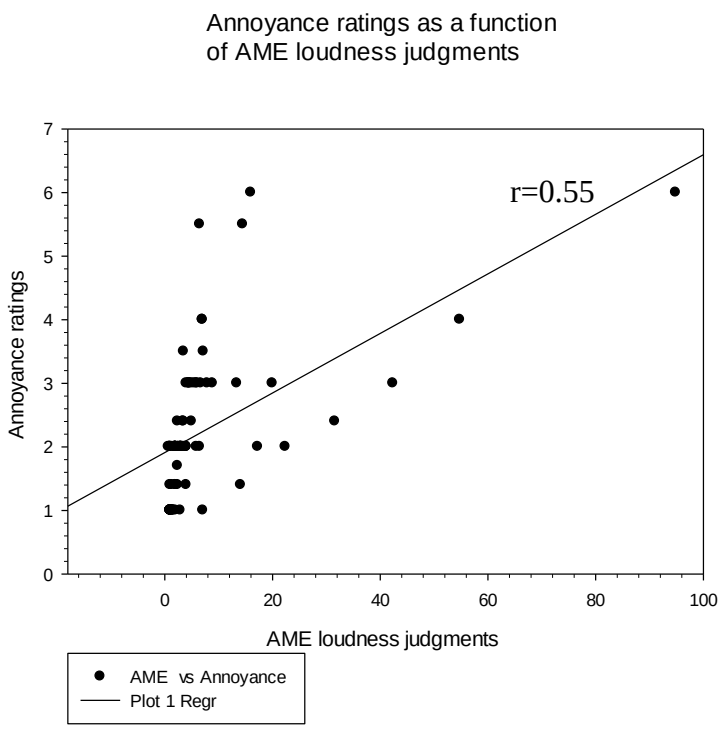


Figure 18: Scatter plot of individual subjects' (n=11) annoyance ratings as a function of their AME loudness judgments. The filled circles represent each subject's annoyance ratings. A linear regression function is fitted to the annoyance ratings. The Spearman's correlation coefficient value is r=0.55.

Comparison of AME judgments and annoyance ratings for pre AEP vs. post AEP recordings. After the recording of the slow cortical response at each intensity, subjects were asked to provide judgments of both the loudness and annoyance of the tonal stimuli at that particular stimulus intensity. The subject only provided one judgment of loudness and one judgment of annoyance. These judgments were made using the same procedures as described earlier.

Shown in Table 3 are the pre-and post-measurement arithmetic mean, SD, and range AME values as a function of stimulus intensity. The post loudness judgment values, which were measured immediately after the recording of the slow cortical response at each intensity, are shown in the lower portion of Table 3. The same

organization of the annoyance ratings, before and after the measurement of the slow cortical responses is displayed in Table 4.

The post-mean loudness judgments were lower than the pre-mean loudness judgments across all eight stimulus intensities. A series of dependent t-tests were run to determine whether these pre- vs. post-loudness judgments reached statistical significance at any of the stimulus intensities. An alpha level of $p \leq 0.05$ was used to assess significance. There were no significant differences in the pre- vs. post-loudness loudness judgments at any of the stimulus intensities. Second, the variability in these AME judgments, as indicated by the SD values, decreased in the pre- vs. post-conditions. Thirdly, the range of the AME judgment values was smaller in the post-test condition. (e.g., the range of AME values for the 60-dB-nHL stimulus intensity was 2.0-42.4 for the pre-test condition; whereas, the range of AME values for the 60-dB-nHL stimulus intensity was 1.0-10.0 for the post-test condition). This decrease in variability for the loudness judgments in the post-measurements compared to the pre-measurements suggests that the subjects may have become more comfortable with the AME loudness judgment task at post-measurement.

Table 3: The group's arithmetic mean, SD, and range of the pre-and post-AME loudness judgments as a function of stimulus intensity. The pre- AME mean, SD, and range values are presented in the top half of the table and the post-AME mean, SD, and range values are shown in the bottom half of the table.

Pre-AME Loudness Judgments								
	80 dB nHL	**70 dB Nhl**	**60 dB nHL**	**50 dB nHL**	**40 dB nHL**	**30 dB nHL**	**20 dB nHL**	**10 dB nHL**
MEAN	16.2	10.8	7.4	6.0	3.9	4.1	2.7	1.8
SD	26.6	15.1	11.8	8.6	4.7	6.2	3.9	1.9
RANGE	2.4-94.9	2.0-54.8	2.0-42.4	2.0-31.6	1.4-17.3	1.0-22.4	1.0-14.1	0.7-7.1
Post-AME Loudness Judgments								
MEAN	12.7	6.3	4.1	4.8	3.3	2.0	1.3	0.8
SD	21.1	6.4	2.5	6.8	4.0	1.3	0.7	0.5
RANGE	2.0-75.0	2-25.0	1.0-10.0	1.0-25.0	1.0-15.0	0.5-5.0	0.25-3.0	0.1-2.0

Table 4: The group's arithmetic mean, SD, and range of the pre-and post-annoyance ratings as a function of stimulus intensity. The pre-annoyance mean, SD, and range values are presented in the top half of the table and the post-annoyance mean, SD, and range values are shown in the bottom half of the table.

Pre-Annoyance Ratings								
	80 dB nHL	**70 dB nHL**	**60 dB nHL**	**50 dB nHL**	**40 dB nHL**	**30 dB nHL**	**20 dB nHL**	**10 dB nHL**
MEAN	3.9	3.1	2.5	2.2	1.7	1.6	1.4	1.5
SD	1.4	1.0	0.6	0.3	0.4	0.5	0.5	0.5
RANGE	1.7-6.0	1.4-5.5	1.4-3.5	2.0-3.0	1.0-2.0	1.0-2.0	1.0-2.0	1.0-2.0
Post-Annoyance Ratings								
MEAN	3.3	2.5	2.0	1.9	1.7	1.4	1.4	1.4
SD	1.4	0.8	0.6	0.3	0.5	0.5	0.5	0.5
RANGE	2.0-6.0	1.0-4.0	1.0-3.0	1.0-2.0	1.0-2.0	1.0-2.0	1.0-2.0	1.0-2.0

The final annoyance ratings were lower than the initial ratings at the higher-moderate stimulus intensities (80-50 dB nHL), and remained essentially stable at the lower stimulus intensities. A series of dependent t-tests were run to determine whether these pre- vs. post-annoyance rating differences reached statistical significance at any of the stimulus intensities. The pre- vs. post- annoyance rating judgments at 80, 70, and 60 dB nHL were significant. At all other stimulus intensities, the differences in the pre- vs. post-annoyance ratings did not reach statistical significance. This finding suggests that the subjects became somewhat less annoyed/bothered by the 2000-Hz tonal stimuli at louder stimulus intensities after post-measurement exposure. Second, the variability seen in the post-measurement condition, as indicated from the SD, was similar to that

measured for the pre-condition and was relatively equivalent across stimulus intensities (70-10 dB nHL). Third, the range of annoyance values remained stable between the initial and final annoyance rating. For instance, the initial pre-measurement range for the 40-dB-nHL condition was 1.0-2.0 (very pleasant to pleasant) and the final post-measurement range was1.0-2.0 (very pleasant to pleasant). This stability was especially evident at the lower stimulus intensities (40-10 dB nHL).

To summarize, the current data revealed the following trends for the behavioral AME loudness data. 1) Loudness judgments decreased as a function of decreasing stimulus intensity. 2) The largest change in mean loudness judgments occurred at the higher compared to lower stimulus intensities. 3) The Spearman correlation coefficient revealed a low correlation ($r= 0.33$) between AME loudness judgments and stimulus intensity. 4) Despite the fact the post-loudness judgments were somewhat lower than the pre-condition mean loudness judgments at all stimulus intensities, the differences in the pre-and post- measurement conditions did not reach statistical significance for any stimulus intensity.

Similarly, the current data revealed the following trends for the annoyance data. 1) The annoyance ratings for the 2000-Hz tonal stimuli decreased as stimulus intensity decreased. 2) The Spearman correlation coefficient revealed a moderate correlation ($r= 0.55$) between the annoyance and AME judgments. 3) The pre-and post- measurement annoyance ratings were statistically significant for 80, 70, and 60 dB nHL, with the post-annoyance judgments being consistently lower.

Electrophysiology Results

Response identification and expected scalp distribution. Shown in Table 5 are the number of subjects who had an identifiable wave P1, N1, and P2 at each stimulus intensity. Stimulus intensities, ranging from 80-10 dB nHL are shown at the top of the table and the waveform components (P1-N1-P2) are show in the left column. Some subjects did not have an identifiable wave P1, N1, and/or P2 component, most notably at the lower stimulus intensities (i.e., 20-10 dB nHL). Furthermore, waves N1 and P2 were more readily discerned than was wave P1. Table 6 is organized the same way as Table 5. Again, the number of subjects with measurable recordings decreased in amplitude going from the front to the back of the scalp (as well as those who had no response detected in the ocular movement channel). All subjects who had a measurable response displayed a decrease in amplitude of the P1-N1-P2 complex when going from the front to the back of the scalp (i.e., Fz-Pz). No subjects had a response in the ocular movement channel (i.e., channel 4) that occurred in the same latency range as the slow cortical response.

Table 5: The number of subjects (n=11) who had a measurable P1, N1, and P2 component for the 2000-Hz tonal stimuli at each stimulus intensity

	80 dB nHL	**70 dB nHL**	**60 dB nHL**	**50 dB nHL**	**40 dB nHL**	**30 dB nHL**	**20 dB nHL**	**10 dB nHL**
Wave P1	10/11	10/11	9/11	9/11	8/11	9/11	8/11	6/11
Wave N1	11/11	11/11	11/11	11/11	11/11	11/11	8/11	8/11
Wave P2	11/11	11/11	11/11	11/11	11/11	11/11	7/11	8/11

Table 6: Individual subjects' (n=11) response identification confirmation. The top portion of the table indicates the number of subjects (of those who had a response at that stimulus intensity) whose amplitude decreased going from the front of the scalp to the back of the scalp (Fz-Pz). The bottom portion of table indicates the number of subjects (n=11) who did not have any responses in the ocular movement channel (i.e., channel 4).

Response Identification: Amplitude decrease Fz-Pz								
	80 dB nHL	**70 dB nHL**	**60 dB nHL**	**50 dB nHL**	**40 dB nHL**	**30 dB nHL**	**20 dB nHL**	**10 dB nHL**
Wave P1	10/10	10/10	9/9	9/9	8/8	9/9	8/8	6/6
Wave N1	11/11	11/11	11/11	11/11	11/11	11/11	8/8	8/8
Wave P2	11/11	11/11	11/11	11/11	11/11	11/11	7/7	8/8
Response Identification: No Response on Ocular Channel								
	80 dB nHL	**70 dB nHL**	**60 dB nHL**	**50 dB nHL**	**40 dB nHL**	**30 dB nHL**	**20 dB nHL**	**10 dB nHL**
Wave P1	11/11	11/11	11/11	11/11	11/11	11/11	11/11	11/11
Wave N1	11/11	11/11	11/11	11/11	11/11	11/11	11/11	11/11
Wave P2	11/11	11/11	11/11	11/11	11/11	11/11	11/11	11/11

Peak-to-peak amplitude of waves P1-N1 and N1-P2. Shown in Table 7 are the mean, SD and Cv values for the amplitudes for waves P1-N1 and N1-P2 at each stimulus intensity. Similar to the previous tables, stimulus intensity is shown across the top of the table and the waveform components are shown along the left side of the table.

Table 7: The mean, SD and Cv values for the peak-to-peak amplitudes of waves P1-N1 and N1-P2 at each stimulus intensity. Wave P1-N1 amplitude values are on the top of the table and wave N1-P2 amplitude values are on the bottom of the table. These amplitude values are expressed in microvolt's μV.

Wave P1-N1	**80 dB nHL**	**70 dB nHL**	**60 dB nHL**	**50 dB nHL**	**40 dB nHL**	**30 dB nHL**	**20 dB nHL**	**10 dB nHL**
Mean	5.10	4.36	4.09	3.01	3.09	2.55	3.14	1.65
SD	2.22	2.25	1.55	1.57	2.27	1.09	1.48	1.21
Cv	0.43	0.51	0.38	0.52	0.73	0.43	0.47	0.73
Wave N1-P2								
Mean	10.65	8.96	7.67	6.99	5.36	5.68	5.13	3.77
SD	4.61	3.61	3.00	2.82	2.02	2.05	1.75	1.33
Cv	0.43	0.40	0.39	0.40	0.38	0.36	0.34	0.35

First, as expected, the mean peak-to peak amplitudes of waves P1-N1 and N1-P2 decreased as a function of decreasing stimulus intensity. Specifically, the overall mean amplitude values for waves P1-N1 and N1-P2 presented at 80-dB-nHL were 5.10 and 10.65 μV, respectively, compared to mean amplitude values of 1.65 and 3.77 μV, respectively for the 10-dB-nHL stimulus. This trend held for the responses at all stimulus intensities, with the exception of the peak-to-peak amplitudes for wave P1-N1 at 40 and 20-dB-nHL and for the amplitude value at 30-dB-nHL for wave N1-P2. Additionally, the variability in the data for both peak-to-peak amplitudes, as indicated by the SD and Cv values, was quite similar for all stimulus intensities.

Each individual's peak-to-peak P1-N1 and N1-P2 amplitude values are plotted in Figure 19 as a function of stimulus intensity. Wave P1-N1 amplitude values are plotted in the graph on the left and wave N1-P2 amplitude values are plotted in the graph on the right side; both are shown as a function of stimulus intensity. Linear-regression functions

are fitted for each scatter plot, along with Spearman's correlation coefficient values, which revealed a moderate correlation between the slow cortical response amplitudes and stimulus intensity. Specifically, the correlation values were r=0.46 and r=0.58 for waves P1-N1 and N1-P2 respectively. These correlation values suggest a somewhat stronger correlation for the amplitude of wave N1-P2 as a function of stimulus intensity than the amplitude of wave P1-N1 as a function of stimulus intensity.

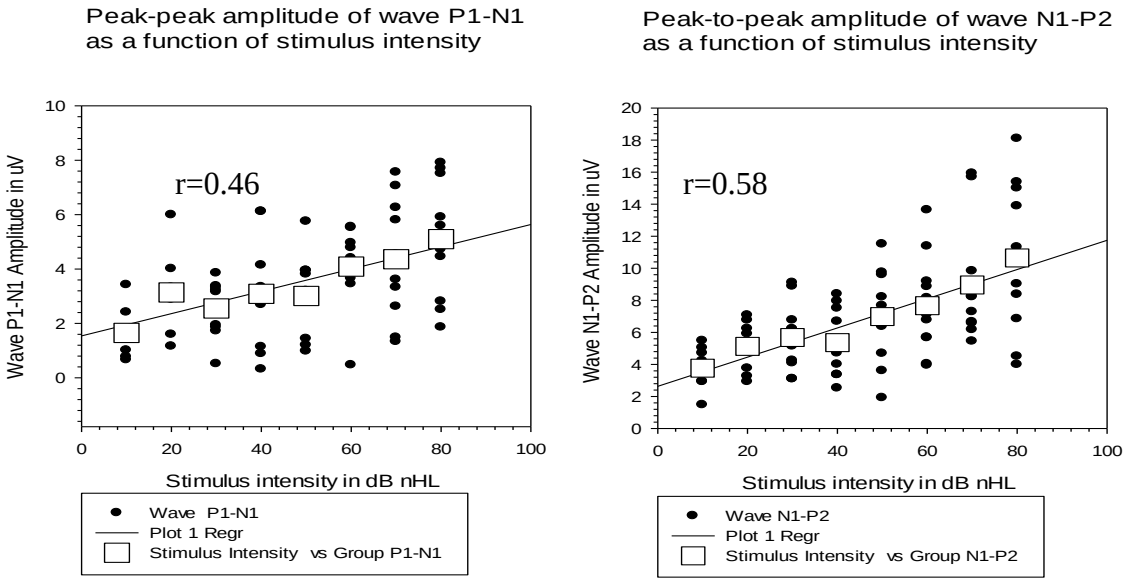


Figure 19: Waves P1-N1 and N1-P2 plotted as a function of stimulus intensity in the left and right panels, respectively. The filled circles represent each subject's amplitude values in μV and the open squares represent the group's mean. Linear-regression functions are fitted to the respective data sets and corresponding correlation coefficients are indicated.

Latency of waves P1, N1, and P2. Shown in Table 8 are the mean, SD and Cv values for the absolute latency values of waves P1, N1, and P2. The organization of the table is the same as Table 7.

Table 8: The mean, SD and Cv values of waves P1, N1, and P2 latency at each stimulus intensity. Wave P1 latency values are shown at the top, wave N1 values in the middle, and wave P2 latency values at the bottom of the table. The latency values are expressed in milliseconds.

Wave P1	**80 dB nHL**	**70 dB nHL**	**60 dB nHL**	**50 dB nHL**	**40 dB nHL**	**30 dB nHL**	**20 dB nHL**	**10 dB nHL**
Mean	49.14	49.28	50.71	52.73	51.45	53.22	54.08	57.87
SD	3.70	4.70	3.41	5.38	4.60	3.13	3.96	4.66
Cv	0.08	0.09	0.07	0.10	0.09	0.06	0.07	0.08
Wave N1								
Mean	94.06	94.57	94.69	97.24	96.98	95.84	100.28	102.38
SD	5.47	8.24	4.65	7.93	3.96	6.93	3.34	9.71
Cv	0.06	0.09	0.05	0.08	0.04	0.07	0.03	0.09
Wave P2								
Mean	163.04	164.44	164.44	164.69	169.91	173.22	179.80	182.53
SD	12.0	15.1	14.7	13.1	15.0	14.2	10.4	18.6
Cv	0.07	0.09	0.09	0.08	0.09	0.08	0.06	0.10

Mean P1, N1, and P2 latencies for the 2000-Hz stimuli increased as a function of decreasing stimulus intensity. Specifically, the overall mean latencies for waves P1, N1, and P2 presented at 80-dB-nHL were 49.14, 94.06, and 163.04 ms, respectively, compared to mean latency values of 57.87, 102.38, and 182.53 ms for the 10-dB-nHL stimulus. This trend was consistent across all stimulus intensities with the exception of the P1 latency value at 40-dB-nHL and the N1 latency value at 40 and 30-dB-nHL. Second, the largest change in latency occurred at the lower stimulus intensities (30-10 dB nHL) for waves P1, N1, and P2. Third, variability, as indicated by the SD values, was relatively low to moderate for waves P1 and N1; whereas, variability was approximately

three times larger for wave P2. The variability stayed consistent for all of the stimulus intensities. It was more difficult to identify wave P2 in several subjects' responses, which likely contributed to this higher variability. When the variability was normalized across waveform components, reflected in the Cv values, this normalized variability was relatively consistent across waves and was independent of stimulus intensity.

Each individual's P1, N1, and P2 latency values are plotted in separate panels as a function of stimulus intensity. Best-fitting linear regression functions are shown along with corresponding Spearman's correlation coefficients for each data set. The negative correlation values were r=-0.48, r=-0.32, and r=-0.41 for waves P1, N1, and P2 latencies, respectively, indicating a low to moderate negative correlation (Polite & Beck, 2012) between latency values and stimulus intensity.

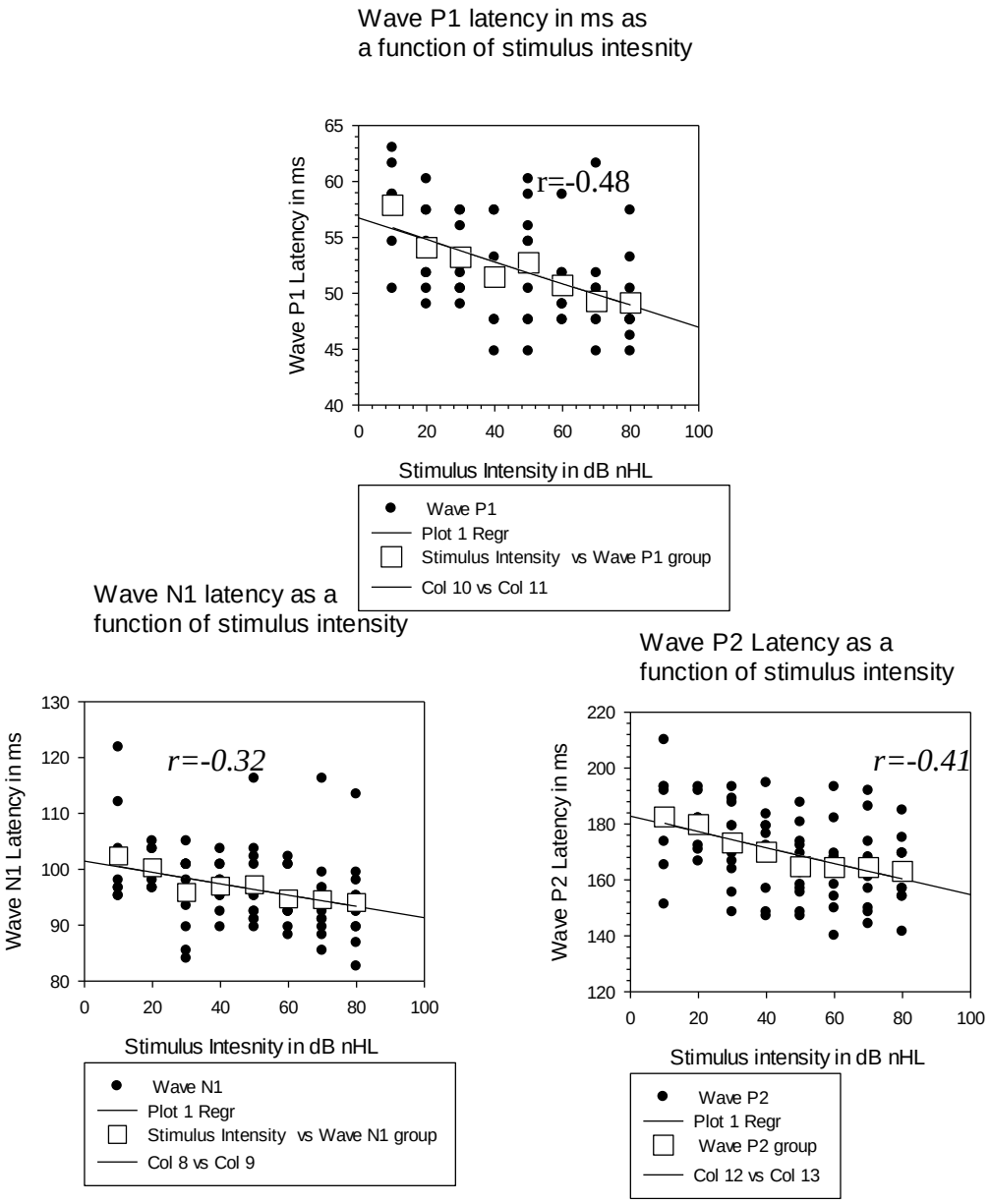


Figure 20: Wave P1, N1, and P2 latency values plotted as a function of stimulus intensity, for P1 on the top, N1 on the bottom left , and P2 s on the bottom right. Latency values are shown in ms. The filled symbols represent individual data and the open squares represent the group means. A linear-regression function has been fitted to each data set and correlation values are shown for waves P1, N1, and P2, respectively.

To summarize, the electrophysiological data revealed the following patterns for the P1-N1-P2 complex amplitude and latency values: 1) The most stable deflections were measured for waves N1 and P2. Wave N1 and P2 responses were present 63.6%-100% of the time across all eight stimulus intensities, as shown in the response identification table (Table 5); 2) The peak-to-peak amplitude values of waves P1-N1 and N1-P2 decreased and the latency values of P1, N1, and P2 increased as a function of decreasing stimulus intensity. 3) Spearman's correlation coefficient values revealed a moderate correlation between waves P1-N1 and N1-P2 amplitude values and stimulus intensity (r=0.46 and r=0.58, respectively) and low to moderate negative correlations between waves P1, N1, P2 latency values stimulus intensity (r=-0.48, r=-0.32, and r=-0.41, respectively). Thus, the peak-to-peak amplitude value for wave N1-P2 has a stronger correlation with stimulus intensity compared to the other response measurements of the slow cortical response.

Characterization Between Behavioral AME Judgments and the Response Properties of the Slow Cortical Response

The investigators of the current study calculated the relation between the behavioral loudness judgments (i.e., the slope value of AME loudness judgments) and the response growth properties of the slow cortical response (i.e., peak-peak amplitude of waves P1-N1 and N1-P2; and absolute latency of waves P1, N1, and P2). These slope values, for the behavioral and the electrophysiology responses, are displayed in Table 9. Along the left side of the table are the specific behavioral and electrophysiological measurements and the slope values for each of these measurements on the right side of the table.

Table 9: The mean slope value for the loudness judgment (AME) and mean growth values for the slow cortical response (i.e., peak-to-peak amplitude of waves P1-N1 and N1-P2; and latency vales of waves P1, N1, and P2)

AME	0.33
P1-N1 peak-to-peak amplitude	0.46
N1-P2 peak-to-peak amplitude	0.58
Wave P1 Latency	-0.48
Wave N1 Latency	-0.32
Wave P2 Latency	-0.41

There were several interesting trends seen in these slope data. First, the majority of the correlation values are larger for the response properties of the slow cortical response versus the correlation value for the loudness judgments. The only exception to this pattern was for the latency of wave N1. The negative correlation for the changes in wave N1 latency as a function of stimulus intensity (r=-0.32) is essentially the reciprocal of the correlation for AME loudness judgments as a function of stimulus intensity (r=0.33). Secondly, the positive correlation value for the peak-to-peak amplitude of wave N1-P2 was the highest value, suggesting that the growth in amplitude of this waveform component as a function of intensity may be the most sensitive indicator/predictor of the growth of loudness.

CHAPTER 5

DISCUSSION

The discussion section is organized similarly to the results section. First, the results from the behavioral data are discussed followed by the results from the response measurements of the slow cortical response. Specifically, the behavioral data section is comprised of: 1) loudness judgments obtained prior to and following the slow cortical response recordings; and 2) annoyance ratings that accompanied these AME judgments. The response measurements for the slow cortical response section are comprised of: 1) response identification and the expected scalp distribution (i.e., decrease in response amplitude going from Fz-Pz and no response in the ocular artifact channel); 2) peak-to-peak amplitude measurements of waves P1-N1 and N1-P2; and 3) absolute latency measurements of waves P1, N1, and P2. Third, a comparison between the calculated correlations of the AME behavioral loudness judgments and the response measurements of the slow cortical response are discussed. Lastly, the limitations of the current study as well as future directions are described.

Behavioral Results

AME loudness judgments. To review, the AME hypothesis states that subjects assign a number to a stimulus in such a way that their impression of the size of the number matches their impression of the sensation magnitude of the stimulus (Hellman & Zwislocki, 1961, 1963, 1964). In other words, subjects are not restricted in their choice of numbers used to judge the loudness of stimuli; rather, they are encouraged to assign numbers they feel to be appropriate without any biases that may be associated with a response system devised by the experimenter. The results of the current study indicated

that: 1) as anticipated, the mean loudness judgments decreased as stimulus intensity decreased; and 2) the largest change in mean loudness judgments occurred at the higher versus lower stimulus intensities. In the current study, the relation between subjects' AME loudness judgments for a 2000-Hz tonal stimulus as a function of stimulus intensity was explored using linear regression analysis techniques. The results of this analysis revealed a correlation value of r= 0.33, suggesting a low correlation between these two variables. In the previous literature, the average slope values for the AME loudness function for stimulus frequencies between 500-2000 Hz in young and older normal hearing adults as well as naïve listeners in diverse listening conditions was 0.6 (re: dB SPL) (Canevet et al., 1986; Hellman & Meisleman, 1988, 1993). It should be noted, however, that Nousak (2001) converted this 0.6 dB SPL value into a 0.3 dB SI value. In the current study, our correlation value is based on dB HL and thus the current finding are in excellent agreement with these past studies, indicating the validity of this loudness growth technique.

One somewhat unexpected finding for the AME loudness judgments in the current study was that the majority of the subjects' assigned low number values for their AME loudness judgments across stimulus intensities. Specifically, they chose numbers between one and seven, even for the higher stimulus intensities (80 and 70 dB nHL), as seen in Table 1. Personal communication with Dr. Nousak (April, 2013) revealed that the normal-hearing adult subjects in her study gave a wide range of AME values (i.e., approximately 1-100) for their loudness judgments of the 1000-Hz tonal stimuli. However, their slope or exponent values calculated in the individual subject's behavioral AME loudness curves ranged from 0.19 to 0.39, similar to the mean AME slope value

obtained in the current study. One possible explanation for these lower AME loudness judgments in the current study may be that many of the subjects completed this experiment following a similar experiment that utilized the Contour loudness test to characterize loudness growth. Therefore, it is possible that the current subjects came to the test session with a mindset to use numbers between one and seven regardless of the instructions provided to them. There was one subject (subject 163337) who consistently assigned larger AME judgment values compared to the rest of the subjects. This most likely contributed to the high variability, and wide range, especially for the higher stimulus intensities (80-60 dB nHL).

Lastly, one additional variable explored in this study was the reliability of the subjects' AME judgments. Specifically, the mean AME loudness obtained prior to the slow cortical response recordings were compared to the mean AME judgments obtained following these slow cortical recordings. Even though the mean AME loudness judgments obtained following the slow cortical responses were somewhat lower, there was no statistically significant difference in the mean AME values between the pre- and post- test conditions at any of the stimulus intensities. This finding suggests that the subjects' AME judgments were reliable. None of the literature addressed in literature review for this study addressed this question; therefore no comparisons can be made to the earlier AME literature. The current investigators hypothesized that the slightly lower AME values in the post- test condition may indicate that the subjects became more familiar with the AME loudness judgment task.

Annoyance ratings. The pattern of results seen for the annoyance ratings in the current study were similar to the pattern seen for the AME loudness judgments. Specifically, 1) as expected, there was an overall decrease in annoyance ratings for the 2000-Hz tonal stimuli as a function of decreasing stimulus intensity; and 2) a wider range of annoyance ratings occurred at the two highest stimulus intensities in comparison to the moderate and low stimulus intensities. We were surprised at the relatively low annoyance ratings for the high-stimulus intensities. In the current study, the relation between the subjects' annoyance judgments for the 2000-Hz tonal stimuli and their AME loudness judgments for these same stimuli was explored. A linear-regression analysis revealed a moderate correlation (r=0.55) between these two variables. Only a few studies have investigated the correlation between annoyance and loudness; annoyance and noisiness; and noisiness and loudness to environmental sounds, white noise, actual noises (i.e., aircraft and road traffic), and artificial noises (Hiramatsu et al. 1988; Kuwano, et al., 1988). Hiramatsu and colleagues reported that annoyance and loudness had a lower correlation value (r=0.54) compared to the correlation values for annoyance and noisiness and noisiness and loudness (r=0.85 and 0.87, respectively). The subjects in Hiramatsu and colleagues study used a five-step monopolar scale from "not loud" to "very loud" to judge the loudness of the various stimuli. The correlation between AME loudness judgments and annoyance reported in the current study is in excellent agreement with that reported by Hiramatsu and colleagues (1988).

There are a couple of reasons as to why there may only be a moderate correlation between annoyance and loudness. First, annoyance is a very subjective term. It is possible that the subjects in this study, like prior studies, associate loudness and annoyance as two

separate concepts. Secondly, the subjects in the current study were not limited by their response ranges (except to positive numbers) and thus, provided a relatively large range of AME values to represent loudness. However, the subjects provided a much smaller range of annoyance values across those same stimulus intensities: their response range was restricted to numbers between 1 and 6. For example, subject 812139 had AME values ranging from 7.0 for the 80-dB-nHL stimulus down to 2.0 for the 20 and 10-dB-nHL stimuli. However, this subject's annoyance rating ranged from unpleasant but tolerable to pleasant (i.e., 4-2) from 80-10 dB nHL. In future studies looking at the relation between these two subjective attributes of an acoustic signal, investigators should try to balance the accepted responses allowed on these two scales better.

Lastly, one additional variable explored in this study was the reliability of the subjects' annoyance ratings. Specifically, the mean annoyance ratings obtained prior to the slow cortical response recordings were compared to the mean annoyance ratings obtained following these slow cortical recordings. There was a statistically significant difference in the pre- vs. post- annoyance rating judgments at stimulus intensities of 80, 70, and 60 dB nHL, such that the post-annoyance ratings were always lower than the pre-annoyance ratings. At other stimulus intensities, the differences in the pre- versus post-annoyance ratings judgments did not reach statistical significance. This finding suggests that the subjects' annoyance ratings, for the most part, were reliable, providing further evidence that loudness and annoyance may be two different concepts. To date, no previous literature has addressed this question; therefore, no comparisons can be made to the earlier annoyance rating literature. We hypothesized that these statistically significant differences in annoyance ratings which occurred at the higher stimulus intensities, likely

indicate that the subjects were somewhat less annoyed/bothered by these higher intensity 2000-Hz tonal stimuli after becoming more familiar with the stimuli and task.

Electrophysiology Results

Response identification and expected scalp distribution. In the literature, several investigators have identified two important criteria for clearly identifying the presence of the slow cortical response. The first is that the amplitude of the slow cortical response should decrease as it goes from the fronto-central electrodes (i.e., Fz and Cz) to the back of the scalp (i.e., Pz) (Martin et al., 2007; Stapells, 2009). In the current study, this expected change in waveform morphology was evident in all eleven subjects' responses when they had a repeatable slow cortical response at that specific stimulus intensity. The second rule was no slow cortical response should be evident in the ocular artifact channel (i.e., channel 4), in the expected latency region of the slow cortical response, approximately 50-250 ms (Martin et al., 2007; Stapells, 2009). Again, none of the eleven subjects had a replicable response in the ocular movement channel, in the 50-250 ms time window, at any of the stimulus intensities. These findings indicate that all the measurable responses were true slow cortical responses coming from the proposed neural generators, and no ocular artifacts contributed to the responses within the expected latency range.

Peak-to-peak amplitude of waves P1-N1 and N1-P2. As anticipated, the current results indicate that as the stimulus intensity of the 2000-Hz tonal stimulus was increased from 10-80 dB nHL, the mean peak-to-peak amplitudes of waves P1-N1 and N1-P2 increased as well (i.e., 1.65-5.10 μV and 3.77-10.65 μV for waves P1-N1 and N1-P2, respectively). Beagley and Knight (1967) recorded the slow cortical response to 1000-Hz

tones at 0, 10, 20, 30, 50, 60, and 70 dB nHL and reported that the mean N1-P2 amplitude value increased from 6.9 μV for the 20-dB-nHL responses in comparison to 13.7 μV for the 70-dB-nHL responses. This study did not report any mean peak-to-peak amplitude values for wave P1-N1. In general, the current results, at least wave N1-P2 amplitude, are in agreement with the literature, which revealed larger N1-P2 amplitude values occur at higher stimulus intensities (Beagley, & Knight, 1967; Picton et al., 1970; Picton et al., 1977; Spoor et al., 1969).

In the current study, the relation between the response amplitudes for waves P1-N1 and N1-P2 and stimulus intensity was explored. The results of the linear regression analyses revealed correlation values of r= 0.46 and r= 0.58 for waves P1-N1 and N1-P2, respectively. These moderate correlations between peak-to-peak amplitude values as a function of stimulus intensity reflect a slightly stronger correlation for wave N1-P2 as a function of stimulus intensity in comparison to wave P1-N1.

In the present study, there was no sign of saturation in the amplitude growth of waves P1-N1 and/or N1-P2 at 70 and/or 80 dB nHL. This finding is in contrast to Beagley and Knight (1967), who reported a saturation effect in the amplitude of wave N1-P2 at stimulus levels of 60-70 dB SL re: threshold and higher. Likewise, Picton and colleagues (1970) reported amplitude saturation effects at intensities above 70 dB ISO. It is possible that the stimulus intensities employed in the current study were not high enough to replicate the effects of saturation reported found in these earlier studies (Beagley & Knight, 1967; Picton et al., 1970).

Latency values of waves P1, N1, and P2. In the current study, as expected, the absolute latency values of waves P1, N1, and P2 decreased as stimulus intensity increased from 10-80 dB nHL. Specifically in the current study the mean latency values for the 10-dB-nHL stimulus were 57.87, 102.38, and 182.53 for waves P1, N1, and P2, respectively, versus 49.14, 94.06, and 163.04 for waves P1, N1, and P2, respectively, for the 80-dB-nHL stimulus. These current latency findings are in agreement with Beagley and Knight (1967), who reported that the mean absolute latency for wave N1 was 136 ms for the 20-dB-nHL responses versus 116 ms for the 70-dB-nHL responses to 1000-Hz tonal stimuli. Similarly, Spoor et al. (1969) reported that the latency for wave N1 decreased from 150 ms at 10 dB SL to 89 ms at 90 dB SL to 1000-Hz tone bursts.

In the current study, the relation was explored between changes in the latencies of waves P1, N1, and P2 as a function of stimulus intensity. The results of the linear regression analyses revealed correlation values of r= -0.48, r=-0.32, and r=-0.41, for waves P1, N1, and P2, respectively, indicating a low to moderate negative correlation between absolute latency values and stimulus intensity. It appears that stimulus intensity is better correlated with the slow cortical response amplitude in comparison to response latency. This is especially true for wave N1-P2 amplitude.

Characterization between Behavioral AME Judgments and the Response Properties of the Slow Cortical Response

In the current study, we attempted to characterize the relation between the subjects' AME behavioral loudness judgments and the response properties of the slow cortical response. This was in an attempt to learn whether the amplitude and/or latency properties of this electrophysiological response could provide an objective measure of

growth of loudness for the 2000-Hz tonal stimuli. The results of the current study can be compared with the results from Nousak's (2001) dissertation and Hoppe and colleagues (2001) study, who also investigated this relation. As previously mentioned, Nousak (2001) obtained AME loudness judgments for a 1000-Hz tonal stimulus at several stimulus intensities for normal-hearing adults. She then compared the slope value obtained from the behavioral loudness growth functions to the slope values obtained from the scatter plots of the response properties of ABR wave V and MLR wave Pa as a function of stimulus intensity. More recently, Hoppe and colleagues (2001) characterized the relation of the slow cortical response measurements (i.e., latency of wave N1 and amplitude of wave N1-P2) and the subjects' perceptual growth of loudness via electrical stimulation in cochlear-implant recipients. The growth of loudness for the cochlear implant subjects was assessed by delivering electrical pulses to electrodes located in the apical, medial, and basal end of the cochlea. The correlation-coefficient data obtained in the current study and in these two previous studies are summarized in Table 10.

Table 10: Slope values from the current study and two recent studies that characterized the relations between loudness growth and response growth properties of the tonal ABR/MLR (Nousak, 2001) and the slow cortical response (Hoppe et al., 2001).

	Current Study	**Nousak (2001)**	**Hoppe et al. (2001)**
Loudness Growth	AME: 0.33	AME: 0.29	Not reported
SCR Amplitude	P1-N1: 0.46 N1-P2: 0.58	Wave V: 0.18 Na-Pa: 0.13	Basal: 0.82 Medial: 0.69 Apical: 0.83
SCR Latency	P1: -0.48 N1: -0.32 P2: -0.41	Wave V: 0.08 Wave Pa: 0.08	Not reported, fitted with an exponential decay

In the current study, in general the peak-to-peak amplitude values of the slow cortical response have a stronger correlation as a function of stimulus intensity for the 2000-Hz tonal stimulus in comparison to the absolute latency values. This finding is in agreement with Nousak (2001) who also reported that the correlation values for the amplitudes of ABR wave V and MLR wave Na-Pa were better correlated with the behavioral growth of loudness for their 1000-Hz tonal stimulus in comparison to the latency values. In the current study, the correlation values for the slow cortical response properties were greater than the correlation value for the AME loudness judgments. Nousak (2001), on the other hand, reported the exact opposite; that is, her correlation value for the AME loudness judgments was higher than the correlation values for the electrophysiology responses. Lastly, the correlation values for the response properties of the slow cortical response were considerably higher than those reported by Nousak (2001) for the ABR and MLR. The discrepancy between the current results and those of Nousak (2001) may be related to the differences in the underlying neural generators for the ABR/MLR versus the slow cortical response. The slow cortical response assesses the integrity of the entire auditory pathway up through and including the auditory cortex; whereas, the ABR and the MLR assess up the low to mid auditory brainstem region and sub-cortical areas of the brain. In the cases of the former response, loudness judgments involve activation of cortical networks and, thus, auditory evoked potentials which reflect this activation would likely result in higher correlation values.

Hoppe and colleagues (2001) reported that the correlation value of the peak-to-peak amplitude of wave N1-P2 and the growth of loudness of the electrical pulses depended on electrode positions. Specifically, correlation values of r= 0.82, r=0.69, and

r= 0.83 for the basal, medial, and apical electrodes, respectively were reported. In the current study, the correlation values of the peak-to-peak amplitude value of wave N1-P2 was not as high (r=0.58) as those values reported by Hoppe et al. (2001). It is likely that the loudness growth mediated by a highly synchronized response to electrical stimulation in cochlear-implant subjects would be processed in a different fashion than acoustical stimulation and yield a higher correlation. Hoppe and colleagues (2001) did not report a correlation value for N1 latency as a function of loudness growth.

Collectively, the results of the current study as well as those two earlier studies suggest that the peak-to-peak amplitude of wave N1-P2 demonstrates a higher correlation with the growth of loudness, as a function of stimulus intensity, compared to the peak-to-peak amplitude of wave P1-N1 and the absolute latency values of waves P1, N1, and P2.

Limitations/Future Directions

The results from the current study are encouraging and are in general agreement with the results of the studies completed within the last decade. However, there were some limitations in the current study that need to be addressed. First, the current study only included 11 normal-hearing adult subjects. Future studies should consider including more subjects as part of the sample. Additionally, subjects with sensorineural hearing loss as well as those who experience loudness recruitment should be included to compare the relations, if any, of their results with their normal-hearing peers. Second, only one stimulus frequency (2000 Hz) was tested. In order to utilize this AEP as a way to measure the growth of loudness objectively in difficult to test clinical populations, the responses should better represent the audiogram. Therefore, it would be important to consider utilizing a lower frequency stimulus tone (i.e., 500 Hz) to evaluate a relation between

loudness growth and response growth properties of the slow cortical response at multiple stimulus frequencies. Lastly, objective tests are needed most for those who cannot provide a reliable and valid response (i.e., young children, the elderly and cognitively challenged adults). Future preliminary studies should consider testing young children and cognitively challenged adults. This means taking into consideration the maturational effects and other factors that may influence the slow cortical response.

In summary, from the current study, the peak-to-peak amplitude of wave N1-P2 of the slow cortical response appears to be the most strongly correlated with increasing stimulus intensity. Because participants in this study also participated in a similar comparing study, which used a restricted categorized behavioral technique to assess loudness (Contour loudness test) (Cox et al., 1997), it will be interesting to compare the behavioral exponent for loudness in both of these studies. This finding may give more insight to the neuronal processes that underlie behavioral loudness perception growth. The slow cortical response should continue to be investigated in future studies as a potential objective measure to characterize behavioral loudness growth.

Appendix A- Case History and Hyperacusis Form

ID #: _____ AGE_____ Date of Test_____

Do you think you have a hearing problem?	YES	NO
Do you have bothersome ringing in one or both of your ears?	YES	NO
If YES, is the ringing in one, or both ears?	BOTH,	LEFT, RIGHT
Do you ever feel dizzy or lightheaded?	YES	NO
Have you ever had medical/surgical treatment for your ears?	YES	NO
Have you ever had ear aches or drainage from your ears?	YES	NO
Do you have aural fullness?	YES	NO
Do you have any tingling sensation and/or facial numbness/ paralysis of the face?	YES	NO
Do you have any health history that would prohibit participation in this study?	YES	NO
*Are you sensitive to very loud sounds? (i.e., firecrackers, loud sirens, jet engine, ect)	YES	NOT SURE NO

*** If you answer yes or not sure please complete the Sound Sensitivity/Intolerance Section on the following page.**

SOUND SENSITIVITY/INTOLERANCE

6. Write the approximate date when you began to be sensitive to moderate and/or loud sounds: _____

7. Please indicate if the onset was: ☐ gradual ☐ sudden ☐ uncertain

8. Was the onset associated with any particular event? ☐ Yes ☐ No

   If yes, please describe: _____

9. Is your sound tolerance problem usually: ☐ RIGHT ☐ LEFT ☐ BOTH

10. Have you received any treatment for your sound tolerance problem?

    ☐ Yes ☐ No

    If yes, please describe:

    _____

11. Do you use hearing protection devices to avoid loud sounds? ☐ Yes ☐ No

12. If yes, what percentage of the time (_____%) and what type?
    ☐ earplugs ☐ earmuffs ☐ both
13. Since you first noticed a problem with sound tolerance, has there been any change? ☐ Yes ☐ No Is it ☐ Better ☐ Worse ☐ Same?
14. Do you experience any ☐ pain and/or ☐ discomfort for moderate or loud sounds? ☐ Yes ☐ No
    If yes, indicate the duration of the pain or discomfort by checking one of the boxes below.
    ☐ During exposure to the sound ☐ For ___ hours
    ☐ For ___ minutes ☐ For ___ days
15. There are three rating scales below for you to describe, as of today, the severity of your sound tolerance problem, the distress this problem causes you, and its overall effect on your life.

SEVERITY:	0	1	2	3	4	5	6	7	8	9	10 (the worst)
DISTRESS:	0	1	2	3	4	5	6	7	8	9	10 (the worst)
EFFECT ON LIFE:	0	1	2	3	4	5	6	7	8	9	10 (the worst)

16. Please check below the activities that you avoid or limit due to your sound tolerance problem:

☐	concerts	☐	restaurants	☐	housekeeping
☐	church	☐	sports	☐	movies
☐	social events	☐	shopping	☐	music
☐	driving	☐	work	☐	other
☐	sports events	☐	child care		

17. Please check below any of the following situations that affect your sound tolerance:

☐ stress	☐ late in the day	☐ weather
☐ hormonal cycle	☐ barometric pressure	☐ other
☐ early in the day		

18.Please check below any sounds that you are sensitive to:

☐noise ☐music ☐talking ☐paper noises ☐clatter ☐mechanical, monotonous sounds ☐none of the above ☐other (please describe below)

_____

Appendix B- Annoyance Rating Scale

6: Very Unpleasant

5: Unpleasant, but Tolerable

4: Slightly Unpleasant

3: Tolerable

2: Pleasant

1: Very Pleasant

(Adapted from Hiramatsu et al., 1988)

Appendix C- Behavioral Absolute Magnitude Estimation Instructions

You are going to hear repeated tone bursts of different intensities in random order. Your task is to tell me how loud they are by assigning numbers to them. You may use any positive numbers that appear appropriate to you, whole numbers, decimals, or fractions. Do not worry about running out of numbers; there will always be a smaller number than the smallest you use and a larger one than the largest you use. Moreover, do not worry about consistency. Simply try to match an appropriate number to each tone regardless of what you may have called the previous stimulus. You may listen to the same tone burst sequence [seven times] before deciding your numerical estimate of loudness. However, it is best to be as spontaneous and quick in your response as possible. After you reached a decision, report your judgment to the experimenter. Do you have any questions?

(from Hellman & Meiselman, 1988)

Behavioral Annoyance Rating Instructions

An annoyance rating sheet has also been provided for you with responses that range from 1 (very pleasant) to 6 (very unpleasant). A second task that you have is to tell me how annoying the repeated tone bursts of different intensities are. You are to tell me a number 1-6 as well as the descriptive term that corresponds with that number that you feel most appropriate for each tone burst sequence. You will tell me the annoyance rating number and descriptive term that correlates with that number following your loudness judgments of the tone bursts. Do you have any questions?

APPENDIX D- IRB APPROVAL

TOWSON UNIVERSITY

APPROVAL NUMBER: 13-A025

To: Hillary Janowitz
212 Washington Avenue Apt 309
Towson MD 21204

From: Institutional Review Board for the Proctection of Human Subjects, Melissa Osborne Groves, Member

Date: Tuesday, December 04, 2012

RE: Application for Approval of Research Involving the Use of Human Participants

APPROVED DEC 4 2012 MOG/WP

Office of University Research Services

Towson University
8000 York Road
Towson, MD 21252-0001
t. 410 704-2236
f. 410 704-4494

Thank you for submitting an Application for Approval of Research Involving the Use of Human Participants to the Institutional Review Board for the Protection of Human Participants (IRB) at Towson University. The IRB hereby approves your proposal titled:

Relations Among Measures of the Slow Cortical Response and Absolute Magnitude Estimation (AME) Loudness Judgments in Young Normal-Hearing Adults

If you should encounter any new risks, reactions, or injuries while conducting your research, please notify the IRB. Should your research extend beyond one year in duration, or should there be substantive changes in your research protocol, you will need to submit another application for approval at that time.
We wish you every success in your research project. If you have any questions, please call me at (410) 704-2236.

CC: P. Korczak
File

TOWSON
UNIVERSITY

Date: Tuesday, December 04, 2012

NOTICE OF APPROVAL

TO: Hillary Janowitz **DEPT:** ASLD

PROJECT TITLE: *Relations Among Measures of the Slow Cortical Response and Absolute Magnitude Estimation (AME) Loudness Judgments in Young Normal-Hearing Adults*

SPONSORING AGENCY:

APPROVAL NUMBER: 13-A025

The Institutional Review Board for the Protection of Human Participants has approved the project described above. Approval was based on the descriptive material and procedures you submitted for review. Should any changes be made in your procedures, or if you should encounter any new risks, reactions, injuries, or deaths of persons as participants, you must notify the Board.

A consent form: [/] is [ ] is not required of each participant

Assent: [ ] is [ ] is not required of each participant

This protocol was first approved on: 04-Dec-2012
This research will be reviewed every year from the date of first approval.

Melissa Osborne Groves, Member
Towson University Institutional Review Board
WLP

APPENDIX E- Informed Consent Form

INFORMED CONSENT FORM

The Towson University Audiology Department is carrying out research on relations between the response properties of the slow cortical response and behavioral loudness judgments. We are attempting to determine a more accurate way of fitting hearing aids for infants and cognitively challenged adults. Your role in this project will consist of an approximately 3 hour experimental session.

Prior to testing, you will be asked to complete a case history form and a hyperacousis form. Additionally, a hearing test will be completed. At this experimental session, you will be asked to judge the loudness of a series of tonal stimuli of various intensities. Slow cortical response will then be recorded. The slow cortical response is a non-invasive electrophsyiologic response. This response is recorded by placing four surface electrodes on various locations on the scalp. These electrodes are non-invasive. You will be asked to stay awake for the entire test session. There are no known risks or discomforts associated with this procedure. We have reason to believe that the results of this study may be of significant value in hearing aid fitting.

Participation in this study is entirely voluntary. All information will remain strictly confidential. If the findings from this study become published at a future date, at no time will your name or identifying information be used. You are at liberty to withdraw your consent to the experiment and may discontinue participation at any time without prejudice. If you do decide to withdraw from the study, this decision will not impact any future services you would receive from the Speech Language Hearing Center at Towson University. If you have any questions after today, please feel free to call (410)704-5903 and ask for Dr. Korczak or contact Dr. Debi Gartland, Chairperson of the Institutional Review Board for the Protection of Human Participants at Towson University at (410) 704-2236.

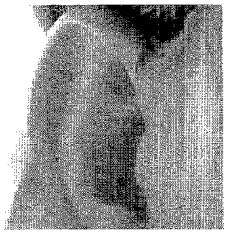
I, _____, affirm that I have read and understood the above statement and have had all of my questions answered.

Date: _____

Signature: _____

Witness: _____

Appendix F- Thesis Recruitment Flyer



Attention!

Participants Needed for Hearing Study

The Towson University Audiology Department is conducting a study in the hopes of increasing the effectiveness of hearing aid fittings for infants and the cognitively challenged. We need normal hearing young adults, ages 18-30 to participate. Free hearing screenings will be given as a part of testing. For more information about participation please contact Hillary Janowitz at hjanow1@students.towson.edu or 240-426-6280

Towson University

Audiology Department

Phone: 240-426-6280

hjanow1@students.towson.edu

Hillary Janowitz
Phone: 240-426-6280
hjanow1@students.towson.edu

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