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EFFECT OF UNILATERAL HEARING LOSS ON THE SPEECH-EVOKED AUDITORY BRAINSTEM RESPONSE IN THE PRESENCE OF NOISE

by

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submitted in partial fulfillment of the
requirements for the degree of:

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Approved by:
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Abstract: Speech-evoked auditory brainstem responses (ABRs) were acquired in quiet and in the presence of noise at two study sessions to investigate 1) test-retest variability and 2) subcortical representation of speech stimuli. Participants were adults with normal hearing in both ears who listened monaurally and adults with unilateral deafness. Results indicate consistency in responses across sessions and several differences between hearing groups for magnitudes of discrete components.

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ABBREVIATIONS

AAO	Age at Onset (of Hearing Loss)
AAOSPHL	Age at Onset of Severe-to-Profound Hearing Loss
AAT	Age at Test
ANOVA	Analysis of Variance
ABR	Auditory Brainstem Response
AEP	Auditory Evoked Potential
CV	Consonant-Vowel
CROS	Contralateral Routing of Signal
dB HL	Decibels (Hearing Level)
dB SPL	Decibels (Sound Pressure Level)
Hz	Hertz
kHz	Kilohertz
kΩ	Kilohms
LOD	Length of Deafness
µs	Microseconds
µV	Microvolts
ms	Milliseconds
NH	Normal Hearing
PTA	Pure Tone Average
SD	Standard Deviation
UHL	Unilateral Hearing Loss
Cz	Vertex

“Our verbal capability is often taken for granted; so seamlessly does it function under virtually all conditions encountered. The intensity of the acoustic background hardly matters – from the hubbub of a cocktail party to the roar of a waterfall’s descent, humans maintain their ability to interact verbally in a remarkably diverse range of acoustic environments. Only when our sense of hearing falters does the auditory system’s masterful role become truly apparent.”

~ S. Greenberg, A. Popper, and R. Fay (2004)

INTRODUCTION

Adults with normal hearing in one ear and severe-to-profound hearing loss in the opposite ear are a unique clinical population. Individuals who rely on single-ear input due to severe-to-profound unilateral hearing loss (UHL) often report listening differences compared to adults with normal hearing (NH) in both ears. These differences reflect a number of binaural processing mechanisms that are absent with UHL. First, high frequency sounds presented near the ear with hearing loss are a challenge to perceive due to the "head shadow effect" for frequencies above 1000 Hz. Although low frequency sounds wrap around the head to the good ear, higher frequency sounds are blocked by the physical presence of the head when sound comes from the side affected by hearing loss. Second, two normal hearing ears provide a binaural summation effect, or increased loudness perception compared to listening with one ear alone. With bilateral normal hearing, loudness is perceived to increase by 3 dB at the threshold for hearing and 6 dB for sounds at 35 dB above threshold (Causse & Chavasse, 1942; Shaw, Newman, & Hirsch, 1947). Individuals with UHL do not benefit from summation effects, as they rely on one ear with significantly better hearing than the other. Normal hearing in both ears also allows for the reduction of the negative effects of background noise and reverberation on speech recognition, known as binaural squelch (Giolas & Wark, 1967; Koenig, 1950). The ability to improve sound detection and comprehension in noise through the effect of binaural squelch is not achieved in cases of unilateral hearing loss (Valente, Valente, Enrietto, & Layton, 2002). Finally, individuals with UHL have difficulty localizing sound due to the loss of interaural time, intensity, and phase cues, which help to organize sound as auditory pathways cross en route to the central auditory cortex.

Functionally, many adults with UHL report difficulty in hearing from the affected side, hearing at a distance, communicating in noisy environments, and localizing sound (Andersen, Schroder, & Bonding, 2006; McLeod, Upfold, & Taylor, 2008). Andersen, Schroder, and Bonding (2006) studied the subjective hearing handicaps reported by adults with unilateral deafness following acoustic neuroma excision through a questionnaire addressing problems in various everyday situations. Despite having normal hearing thresholds in the contralateral ear, all participants (N=53) reported a hearing handicap when in noisy surroundings.

As conventional hearing handicap inventories do not fully evaluate functional listening abilities associated with unilateral deafness, McLeod, Upfold, & Taylor (2008) created an inventory to assess a wide range of listening conditions for adults with UHL. Compared to normal hearing participants, subjects with unilateral deafness reported decreased hearing in all conditions examined by the inventory, including face-to-face communication and direct listening at the normal-hearing ear. The most significant handicaps were reported in situations involving background noise and hearing at the side affected by hearing loss.

Audiologic rehabilitation does not address all of the aforementioned issues associated with UHL. If an individual has normal hearing in the better-hearing ear, conventional amplification is not an option. Fitting a traditional hearing aid on the side with hearing loss is not recommended, as nerve deafness, poor word recognition, and lack of aided benefit typically contraindicate the use of a standard hearing aid. Currently, rehabilitative recommendations for UHL include the fitting of 1) a system to route sound from the poor side to the good side (e.g., CROS, Bi-CROS), 2) an osseointegrated bone-conduction implant (e.g., Baha™), 3) a high-powered hearing aid in the ear with hearing loss (i.e., transcranial CROS), or 4) a deep-canal-insertion hearing aid with a bone conduction transducer (i.e., TransEAR™). The philosophy underlying each device is to route the signal to the better-hearing cochlea. While the head shadow effect is minimized by using an assistive device, hearing handicaps related to lack of binaural summation, binaural squelch effect, and localization still exist.

Research suggests that stimulating the normal-hearing cochlea of adults with UHL does not result in the same auditory pathway stimulation as found in adults with NH in both ears (J.B. Firszt, Ulmer, & Gaggl, 2006; Ponton, et al., 2001; Vasama & Makela, 1995). These studies, utilizing functional magnetic resonance imaging (fMRI) responses and cortical auditory evoked potentials (AEPs), are objective measures of central auditory function and provide physiologic information of how sound is encoded at the auditory cortex and other processing centers of the brain.

The auditory system can be assessed at several levels, not only the cortex. Auditory signals travel from the cochlea of the inner ear to multiple relay centers in the brainstem prior to being processed at the level of the auditory cortex. In addition to cortical measures, the auditory brainstem response (ABR) is another example of a physiologic response evoked by sound. More specifically, the ABR represents the synchronous firing of neurons of the peripheral and central auditory nervous system in response to an acoustic stimulus (Hood, 1998), reflecting the integrity of the neural pathway extending from the eighth nerve to the lateral lemniscus on the contralateral side of stimulation (Moller, Jho, Yokota, & Jannetta, 1995; Ponton, Moore, & Eggermont, 1996). This measure is used as the gold standard to diagnose the presence and degree of hearing impairment for children and difficult-to-test populations in the clinical setting. The most common stimulus used to obtain the ABR is a click or tone, both of which elicit established responses in the form of peaks and troughs over a 10 millisecond recording time period (Gorga, Kaminski, Beauchaine, & Jesteadt, 1988). Despite its clinical utility, the ABR in response to clicks or tones does not provide information about the ability of the individual to discriminate more complex acoustic events.

Recent work has established that in addition to conventional stimuli, speech can elicit an auditory brainstem response (Akhoun, Gallego, et al., 2008; Johnson, Nicol, & Kraus, 2005; Skoe & Kraus, 2010). This physiologic response largely mimics the acoustic features of the speech stimuli, a characteristic not evident when using conventional ABR stimuli. Research has suggested that the speech-evoked ABR may provide a physiologic representation of poor speech encoding in some populations, such as children with learning impairments (Cunningham, Nicol, Zecker, Bradlow, & Kraus, 2001; Wible, Nicol, & Kraus, 2004). Similar to the click- and tone-evoked responses, the speech-evoked ABR is thought to be generated by the superior olivary nuclei, lateral lemniscus, and possibly the inferior colliculus. While its recording window overlaps with some later AEPs, the frequency content of the speech-evoked ABR is higher than would be seen in a cortical or middle latency response, signifying brainstem origin (Johnson, et al., 2005). In general, the conventional ABR is considered an exogenous response because it is characterized by the stimulus used to elicit the response (e.g., intensity, duration, and polarity). Alternatively, cortical responses are considered endogenous, as they are primarily shaped by internal cognitive processes (Hood, 1998). The observation that the speech-evoked ABR mimics its acoustic stimulus with good fidelity is further support of its formation in the brainstem.

The speech-evoked ABR uses an acoustic stimulus (e.g., /da/) to elicit a response that consists of a series of peaks and troughs corresponding to the speech sound. These waves represent the acoustic features of the consonant (i.e., the transient component) and vowel (i.e., the periodic component) if a consonant-vowel (CV) stimulus is utilized. Similar to other subcortical AEPs, the latency and amplitude of the recorded components provide estimates of the timing and magnitude, respectively, of neuronal responses (Johnson, et al., 2005). Clinical software recently became available for the measurement of the speech-evoked ABR using standard AEP hardware (BioMARK™, Bio-logic – a division of Natus Medical, Inc.).

Recent studies have investigated the speech-evoked ABR in quiet and in the presence of white noise (N. Russo, Nicol, Musacchia, & Kraus, 2004; N. Russo, Nicol, Trommer, Zecker, & Kraus, 2009; N. M. Russo, Nicol, Zecker, Hayes, & Kraus, 2005). All previous studies of the response in noise showed a significant degradation relative to the response in quiet. These studies are limited to using Gaussian white noise at a signal-to-noise ratio of +5 dB with a signal intensity of 80 dB SPL. Gaussian white noise is commonly used in auditory work, and it exhibits a flat spectrum across all frequencies, while instantaneously, its value follows a "normal" or bell-shaped probability density function (Durrant & Boston, 2007). The clinical population of interest in previous studies of the speech-evoked ABR in noise has primarily been children with normal hearing in both ears, with or without learning disorders.

Akhoun et al. (2008) included adult subjects with UHL in a study of the speech-evoked ABR using the CV stimulus /ba/. Speech-evoked ABRs were recorded separately from the better-hearing ear and the ear with hearing loss of 6 subjects. The ear with hearing loss (termed as the "non-functional auditory pathway") was stimulated to confirm that the speech-evoked ABR was a true auditory response rather than an artifact-generated response. The root-mean-square (RMS) value of responses from the better-hearing ear of UHL participants approximated responses from adults with NH in both ears (N=6). Individual response components and timing were not examined, and no analyses were conducted to correlate the stimulus with the response. The main purpose of including adults with UHL in the study was to authenticate the neural origins of the speech-evoked ABR.

Few studies have examined the neural processing of speech sounds in individuals with UHL in their normal-hearing ears compared to those with NH in both ears. Given the common complaints of individuals with UHL, knowing how speech sounds are neurally processed in both quiet and in the presence of background noise might provide insight about the physiologic origin of functional difficulties in this group. While cortical studies have identified differences between UHL and NH groups, it is unknown whether differences exist at the brainstem level between groups.

The primary aim of this study was to provide new information about how speech is neurally encoded at the level of the brainstem when the auditory system relies on single ear input. Investigating the speech-evoked ABR in the presence of noise may provide insight toward rehabilitation philosophies for overcoming difficulties associated with UHL, as well as shaping counseling and intervention approaches by audiologists who work with this population.

METHODS

Study Objectives

The study objectives were to 1) characterize speech-evoked ABR measures for two groups of adult participants, those with normal hearing (NH) in both ears but stimulated in one ear, and those with unilateral hearing loss (UHL), and 2) determine the test-retest variability of speech-evoked ABR measures for these two groups.

Participants

The study protocol was approved by the Human Research Protection Office at Washington University School of Medicine (study #08-0103). Participants were recruited and tested at Washington University School of Medicine in St. Louis, Missouri using both patient referrals and the Volunteer for Health database. All participants were at least 18 years of age at the time of testing and denied having any diagnosed learning disabilities. Subjects with diagnosed learning impairments were excluded from this study, as the speech-evoked ABR demonstrates sensitivity to learning disorders (Wible, et al., 2004).

Normal Hearing (NH) Participants

Twelve adults (four males and eight females, ages 28-64 years, mean 45.1, SD 10.6) served as normal hearing controls for adults with UHL. Normal hearing (NH) was defined as air conduction hearing thresholds of 25 dB HL or better across the standard audiometric frequencies (250-8000 Hz) in both ears. The mean 3-frequency pure tone average (PTA = mean of thresholds at 500, 1000, and 2000 Hz) of the NH group was 12.1 dB HL (SD 6.4) for the right ear and 11.4 dB HL (SD 5.9) for the left ear. Average audiometric thresholds for the NH group are shown in Figure 1 (left).

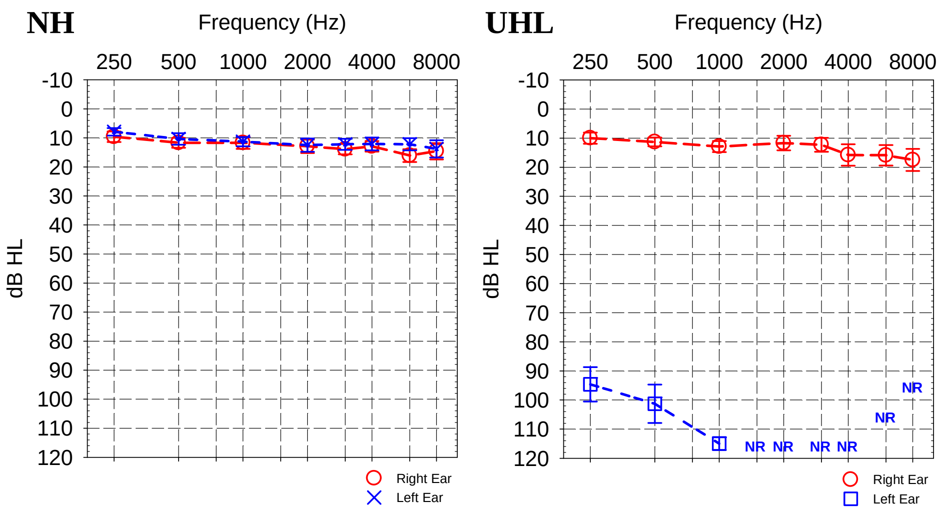


Figure 1. *Average audiometric thresholds for each ear for the normal hearing (NH) and unilateral hearing loss (UHL) participants.*

Unilateral Hearing Loss (UHL) Participants

Twelve adults (four males and eight females, ages 32-62 years, mean 47.2, SD 10.0) with severe-to-profound hearing loss in the left ear and normal hearing in the right ear participated in the study. Unilateral hearing loss (UHL) was defined as air conduction hearing thresholds of 70 dB HL or worse in one ear and normal hearing (25 dB HL or better) in the contralateral ear across the standard audiometric frequencies (250-8000 Hz). Average audiometric thresholds for the UHL group are displayed in Figure 1 (right).

The mean PTA of the good (right) ear for the UHL group was 11.9 dB HL (SD 5.5). Nine UHL participants had thresholds which were beyond the limits of the audiometer. For the other three participants, the poor (left) ear PTA for the UHL group was 101.1 dB HL (SD 15.5). One UHL participant had notably better low-frequency hearing thresholds in the left ear (poor ear) at 250 and 500 Hz (40 and 35 dB HL, respectively), steeply sloping to a profound sensorineural hearing loss at 1000 Hz. The individual's left ear PTA was in the severe range (83 dB HL), allowing for inclusion in the study.

Demographic information is summarized in Table 1. The etiology of UHL varied across the group: surgical removal of acoustic neuroma (3), meningitis (1), genetic (1), unknown/idiopathic (7). Five subjects had a congenital/early childhood onset of UHL, and 7 subjects had adult-onset UHL. The mean age at onset of hearing loss (AAO) was 23.4 years (SD 21.1, range 0 – 57). The mean age at onset of severe-to-profound hearing loss (AAOSPHL) was 24.4 years (SD 21.1, range 0 - 57). Length of deafness was defined as the age at time of testing minus the age of onset of severe-profound UHL (LOD = AAT-AAOSPHL). The mean LOD was 22.8 years (SD 20.7, range 2 – 59).

Five of the 12 participants used some form of amplification in everyday listening: CROS (2), transcranial CROS (1), and BAHA (2). One additional participant had been approved for a BAHA but had yet to undergo surgery. None of the participants wore their amplification devices during the study procedures.

UHLs	Gender	AAT (years)	AAOHL (years)	AAOPHL (years)	LOD (years)	Etiology	Onset	HA use (current)
U01	M	32	29	29	3	Unknown	Sudden	None
U02	F	35	3	4	31	Unknown	Progressive	None
U03	M	37	3	3	34	Unknown	Unknown	None
U04	F	42	37	37	5	Meningitis	Sudden	T-CROS
U05	M	42	38	38	4	AN removal	Sudden	CROS
U06	F	46	30	30	16	Unknown	Sudden	None
U07	F	49	0	0	49	Unknown	Congenital	None
U08	F	49	0	0	49	Genetic	Congenital	None
U09	F	54	46	46	8	Unknown	Sudden	None
U10	F	59	46	46	13	AN removal	Sudden	BAHA
U11	M	59	57	57	2	AN removal	Sudden	BAHA
U12	F	62	3	3	59	Unknown	Sudden	CROS
Mean		47.2	24.3	24.4	22.8			
SD		10.0	21.2	21.1	20.7			

Table 1. *Demographic information for each UHL participant. Age at test (AAT), age at onset of hearing loss (AAOHL), age at onset of severe-to-profound hearing loss (AAOSPHL), and length of deafness (LOD) are listed for each individual. AN represents acoustic neuroma. T-CROS represents transcranial CROS hearing aid. Group means and standard deviations are included at the bottom of the table.*

Experimental Design

Speech-evoked ABRs were recorded under 6 presentation conditions during one test session and were repeated at a second test session to obtain test-retest measures. Conditions were randomized for each participant at each test session: 80 dB SPL (Quiet, +10 SNR pink noise, +5 SNR pink noise) and 60 dB SPL (Quiet, +10 SNR pink noise, +5 SNR pink noise).

Measurements

Click-evoked ABRs were recorded at the beginning of each session. Approximately 1000 sweeps were collected and repeated to verify wave presence. Waves I, III, and V were marked on the superimposed raw waveforms by the examiner. Click-evoked ABRs were collected again at the beginning of the second test session. Latency (ms) and magnitude (μV) were analyzed. Following this, speech-evoked ABRs were recorded at each session. A recent study (Hornickel, Skoe, & Kraus, 2009) indicated a "right ear advantage" for speech-evoked ABRs measured in the right and left ears of adults with NH. In the present study, only the right ear was stimulated across all participants, as those in the UHL group were required to have hearing loss in the left ear. Two trials of 3000 sweeps were collected for each listening condition. Both trials were averaged to create a calculated wave of 6000 sweeps. Trials with more than 10% of sweeps rejected as artifact were repeated to obtain a cleaner response with less artifact contamination. A total of 6 calculated waveforms were generated at the first test session, and all conditions were repeated at the second test session. A total of 12 waveforms were analyzed for each subject. Seven prominent waves were identified for each waveform: V, A, C, D, E, F, and O. Latency (ms) and magnitude (μV) values were calculated for each wave for each condition, unless it was determined that a wave was not present.

Components of the Speech-Evoked ABR in Response to /da/

Figure 2 illustrates the similarity between the acoustic signal /da/ and the recorded speech-evoked ABR. The stimulus utilized in the BioMARK™ software is a synthetically generated stop consonant (/d/) and shortened vowel (/a/) consisting of a fundamental frequency and 5 formants. The stimulus (top) is shifted approximately 6 ms to align with the onset of the physiologic response (bottom). Components of the speech-evoked ABR include the onset (V, A), transition from consonant to vowel (C), periodic portion/frequency following response (D, E, F) and offset of the response (O). The onset of the response correlates with the presentation of the consonant, and the latter part of the response is referred to as the frequency following response (FFR). The FFR is a sustained response which exhibits periodicity mirroring the frequency information in the vowel. In a naturally-produced vowel, this would follow the rate of glottal pulsing. Following the FFR, the offset of the stimulus is coded in the response. A total of 7 waves can be marked (i.e., V, A, C, D, E, F, O), though in the presence of noise, some waves may be obliterated.

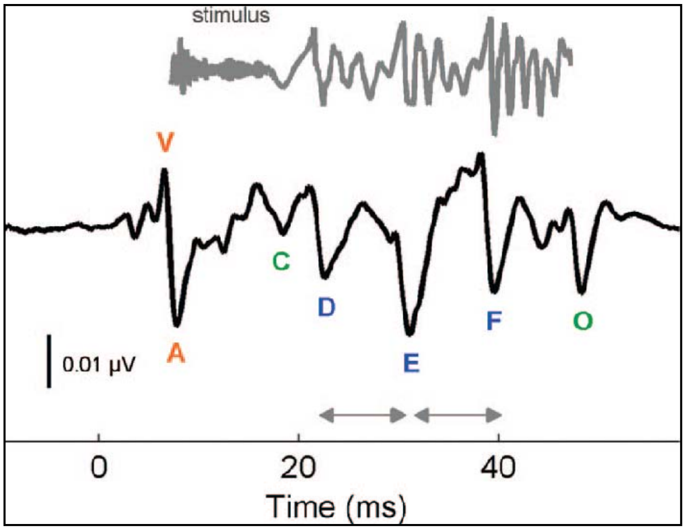


Figure 2. *The synthesized speech syllable /da/ is shown at the top of the figure. The corresponding speech-evoked ABR waveform is illustrated beneath. The stimulus has been shifted ~6 ms to align with the onset of the neural response (Skoe & Kraus, 2010).*

Equipment

The Bio-logic Auditory Evoked Potential (AEP) System v7 with BioMARK™ software v2 was used with the Bio-logic Navigator Pro unit to collect and analyze all waveforms. Reusable gold disc electrodes were placed on the scalp and earlobes. A Bio-logic insert earphone with a foam tip was used to present the stimuli to the right ear of each participant. A Grason-Stadler GSI-61 audiometer was used for determining hearing thresholds with insert earphones.

Stimulus Parameters

Click-evoked responses were elicited using a 100 μs duration click presented at 80 dB nHL at a rate of 13.3/second using rarefaction polarity. The default /da/ stimulus within the BioMARK™ software was used to elicit the speech-evoked ABR. The stimulus was 40 ms in duration with a fundamental frequency that linearly rose from 103 to 125 Hz with voicing beginning at 5 ms and an onset noise burst during the first 10 ms. The onset was followed by a formant transition between the consonant and the beginning of the vowel. While the utterance was short and there was no steady-state vowel (the first and second formant frequencies transitioned across the duration of the vowel), the stimulus was voiced and perceived as a consonant-vowel stimulus (Johnson, et al., 2005). The formant frequencies ranged as follows: F1: 220-720 Hz; F2: 1700-1240 Hz; F3: 2580-2500 Hz; F4: 3600 Hz (constant); and F5: 4500 Hz (constant) (N. Russo, et al., 2004).

Pilot data were obtained in the summer of 2009 to determine intensity levels, signal-to-noise ratios, and noise type to be used in the study. For the present study, pink noise was utilized as the ipsilateral noise masker. This stimulus was chosen because the differences in spectral characteristics between white and pink noise, which may allow for preservation of the speech components (notably high-frequency components). Pilot work for the current study suggested that a 0 dB SNR using pink noise would obliterate much of the response, and a +15 SNR showed little effect on the response relative to the quiet condition.

Based on the pilot data, the present study included stimuli presented at intensity levels of 80 and 60 dB SPL in quiet and in the presence of ipsilateral noise. The two intensity levels were selected to enable comparison to normative values in the BioMARK™ software (80 dB SPL) and to study the response when the speech stimulus was presented at a level representative of normal, conversational loudness (60 dB SPL) (J. B. Firszt, et al., 2004; Skinner, Holden, Holden, Demorest, & Fourakis, 1997). Pink noise was presented ipsilaterally with the speech stimulus at signal-to-noise ratios (SNRs) of +10 and +5 for both intensity levels. The stimuli were presented at a rate of 10.9/second with alternating polarity to minimize stimulus artifact and the cochlear microphonic. Lower-frequency components of the response are also accentuated by using an alternating polarity (Skoe & Kraus, 2010).

The sound level output of the insert receivers of the ABR system were calibrated in accordance with the American National Standards Institute (ANSI) standards as part of an annual clinical audiometric calibration. In addition, the dB linear (20 Hz to 20 kHz) SPLs of the speech, click and noise stimuli used in this study were measured and used to set testing levels.

Recording Parameters

A single-channel recording (vertex (Cz) active, right earlobe reference, left earlobe ground) was used to record the response. Impedance values were 5 kΩ or better, and all three electrodes were within 2 kΩ of one another. Impedance values were optimized at the beginning of each test session and checked periodically throughout the test session to ensure that values remained stable. In cases when impedances could not be achieved better than 5 kΩ, it was ensured that all electrodes had similar impedances. An epoch time of 85.33 ms (pre-stimulus 17.4 ms, post-stimulus 67.93 ms) and 1024 data points were used to generate and display the waveform. Gain was set to 100,000 with artifact rejection enabled for activity +/- 23.8 μV. Filters were set at 100 Hz and 2000 Hz.

Booth Setup

Testing took place in a single-walled booth while the examiner was seated outside the booth with the recording equipment. Participants were seated in an ergonomically-designed chair with a headrest and watched a closed-captioned movie on a portable DVD player, positioned approximately 1 meter away from the test chair. Participants were instructed to relax, avoid movement, and remain awake during the test session. A short break was offered halfway through each two-hour test session.

Data Processing

For click-evoked responses, latency and magnitude values were manually entered into Microsoft Excel and processed in SPSS using an Analysis of Variance (ANOVA) test.

Three individuals with experience analyzing speech-evoked ABR measures determined wave presence/absence for all recorded waveforms. Peak-picking was based on marking the peak with the largest amplitude and well-defined peak or trough near the suggested normative latency values for adults (ages 18-28) in the BioMARK™ software. Wave V was characterized by a positive peak around 6-8 ms post-stimulus. Due to differences in stimulus duration, intensity, and frequency components, Wave V of the speech-evoked ABR was expected to be later in latency than Wave V of the click-evoked ABR. For waves A, C, D, E, F, and O, the most negative trough following a positive peak was generally selected as the respective wave. Overall noise (pre-stimulus activity) and wave morphology supported or brought into question the validity of markings (e.g., if the response was noisy before the onset of the stimulus, there was a chance that artifact may have affected the response). In cases where wave selection was difficult due to small magnitude or poor morphology, the initial test session was compared to the follow-up test session. For all noise conditions, the quiet conditions from the same session were used as references. Very seldom was a peak picked in noise (e.g. Wave D for 80 dB SPL +5 SNR) if the same wave was not present in the quiet condition. In cases when a broad trough was identified, the earliest and most negative point of the broad wave was selected for analysis, unless it did not support other data, such as the quiet condition. Finally, a no-stimulus run (0 dB SPL) was occasionally acquired during a test session to aid in peak-picking. This condition allowed the examiners to rule out myogenic artifact from true neural responses to sound.

Data were converted to a text file and imported to The Brainstem Toolbox (Skoe & Kraus, 2010) using MATLAB vR2009B. Grand average waveforms were created by averaging data points from participant waveforms for each condition following text file conversion. Latency and magnitude values, as calculated in the AEP software, were analyzed in Microsoft Excel and SPSS.

RESULTS

The purpose of the present study was to characterize speech-evoked ABR measures for adult participants with either unilateral hearing loss (UHL) or normal hearing (NH) in both ears but stimulated in one ear, and to determine the test-retest variability of speech-evoked ABR measures for these two groups. Results for click-evoked ABR are presented first to determine whether responses to non-speech stimuli were similar for the hearing groups. These results are followed by descriptive information for speech-evoked ABR, including individual waveforms, grand average waveforms, and presence of waveforms under different testing conditions. The stability of waveform latency and magntiude over sessions, and the test-retest correlations, are presented next to address the major goals of this project. The influences of other testing condition factors are also presented, including stimulus level and noise, hearing group and session. Major analyses were conducted using repeated measures Analysis of Variance (ANOVA). For all analyses, effects were reported as significant at $p<0.05$.

Click-evoked ABR

Figure 3a displays individual waveforms for click-evoked ABR responses elicited at 80 dB SPL and recorded at two test sessions for a NH (top) and UHL (bottom) participant. For individual participants in both hearing groups, latencies for Waves I, III and V were within the normal range and increased from

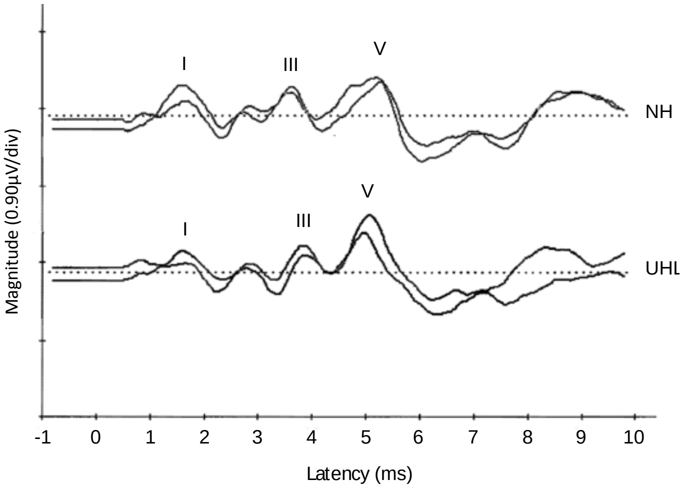


Figure 3a. *Individual waveforms are shown for click-evoked ABR responses elicited at 80 dB SPL and recorded at two test sessions for a NH and UHL participant, respectively. Waves I, III, and V are marked.*

Wave I to V. Click-evoked latency and magnitude values were consistent between Session 1 and 2. Data were analyzed using a mixed design Analysis of Variance (ANOVA) with the main factors of Group (NH, UHL) and Session with Session treated as a repeated measure (using SPSS Version 17). Each wave (I, III, V) was analyzed separately. For latency values, there was no significant main effect for Group (Wave I: F(1,22)=1.57, p=.22; Wave III: F(1,22)=0.15, p=.70; Wave V: F(1,22)=0.80, p=.38) or Session (Wave I: F(1,22)=0.76, p=.39; Wave III: F(1,22)=0.39, p=.54; Wave V: F(1,22)=0.81, p=.38), nor was there a significant interaction between Group and Session (Wave I: F(1,22)=0.41, p=.53; Wave III: F(1,22)=1.67, p=.21; Wave V: F(1,22)=1.29, p=.27). For magnitude values, there was also no significant main effect for Group (Wave I: F(1,22)=2.61, p=.12; Wave III: F(1,22)=1.15, p=.30; Wave V: F(1,22)=0.55, p=.47) or Session (Wave I: F(1,22)=0.47, p=.50; Wave III: F(1,22)=0.33, p=.57; Wave V: F(1,22)=1.15, p=.30), nor was there a significant interaction between Group and Session (Wave I: F(1,22)=1.11, p=.31; Wave III: F(1,22)=0.09, p=.77; Wave V: F(1,22)=1.10, p=.31).

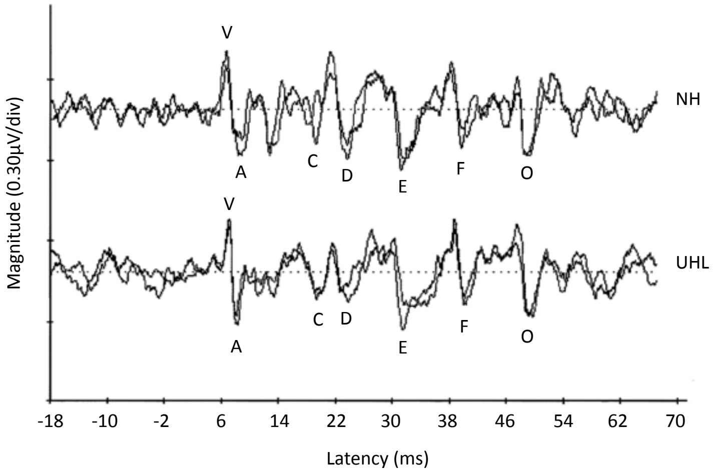


Figure 3b. *Individual waveforms are shown for speech-evoked ABR responses elicited at 80 dB SPL in quiet and recorded at two test sessions for a NH and UHL participant, respectively. Waves V, A, C, D, E, F, and O are marked.*

Speech-evoked ABR

Individual Waveforms

Speech-evoked ABR waveforms from individual subjects are shown in Figure 3b. These responses were elicited at 80 dB SPL in quiet and recorded at two test sessions for a NH (top) and UHL (bottom) participant. Waveforms from each session are superimposed. For both participants, the waveform included the transient onset components (V and A), the transition from the consonant to the vowel (C), the sustained periodic components (D, E, and F), and the transient offset component (O).

Grand Average Waveforms

Grand average waveforms were calculated for subjects in both groups and reflect the averages of all participants' waveforms for each specific condition. As shown in Figure 4, panel A displays responses at 80 dB SPL in quiet, and in noise at +10 and +5 SNR for NH and UHL groups. As previously illustrated in Figure 3b, the averaged waveform included waves V, A, C, D, E, F and O. In noise compared to quiet, the components remained distinguishable in the grand average, however the morphology of the peaks was altered, magnitudes were reduced and some latencies, primarily those for onset responses, tended to increase with increasing noise. Panel B shows a fairly similar pattern for responses evoked at 60 dB SPL in quiet, and in noise at +10 and +5 SNR.

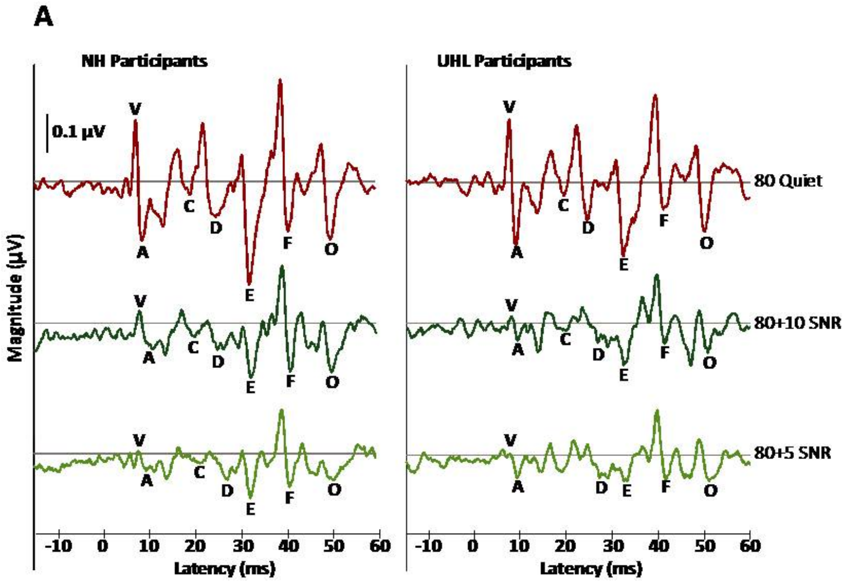
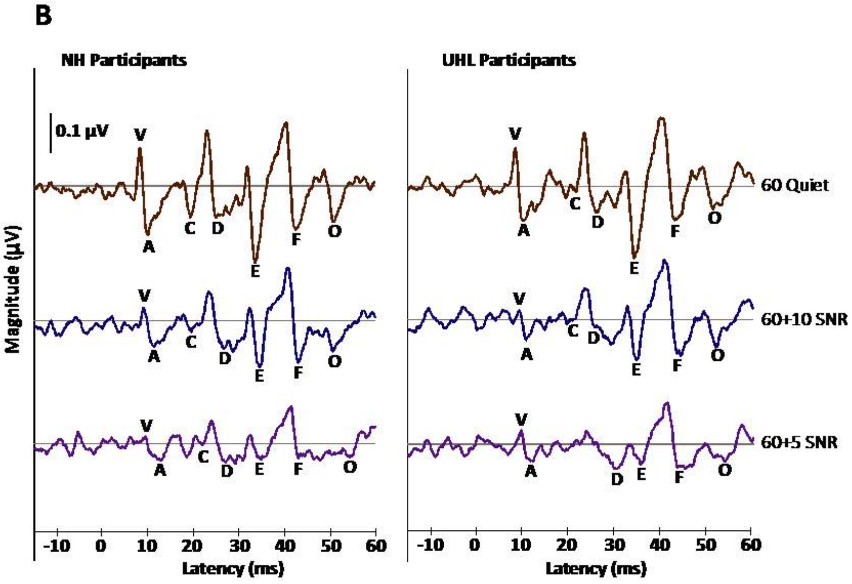


Figure 4. *Grand average waveforms are displayed for NH (n = 12) and UHL (n = 12) groups. Panel A (above) shows responses at 80 dB SPL in quiet, and in noise at +10 and +5 SNR. Panel B (below) shows responses at 60 dB SPL in quiet, and in noise at +10 and +5 SNR.*



Wave Presence across Conditions

It is not uncommon for waves to be obliterated when elicited in the presence of ipsilateral noise. Wave presence was calculated to determine if trends existed across groups or conditions for the speech-evoked ABR. Figure 5 illustrates the presence of speech-evoked ABR components across all listening conditions for the NH group (left) and the UHL group (right). In the bar graphs, conditions at 80 dB SPL precede conditions at 60 dB SPL. For NH participants in quiet at 80 dB SPL, Waves V, A, E, F and O were present 100% across both sessions, with a slightly reduced presence for C and D. As noise was added and the SNR was decreased from +10 to +5, the presence of all waves was diminished. Wave F was present most often in the presence of noise, followed by E and O. The transient Waves V and A, as well as C, were particularly affected by the presence of noise. At 60 dB SPL and in noise, Wave F showed persistence. In UHL participants, the pattern was somewhat similar with a greater reduction in the presence of Waves V and A at the +5 SNR conditions for both 80 and 60 dB SPL, compared to NH. Because the missing data would severely limit power if analyses were restricted to cases with complete data, as many subjects as possible were included for each individual analysis. Accordingly, degrees of freedom vary from analysis to analysis.

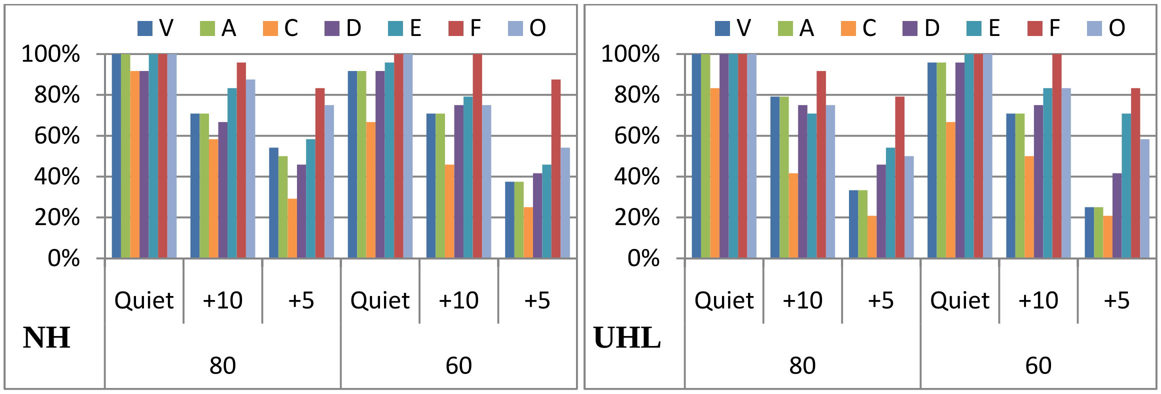


Figure 5. *Presence of speech-evoked ABR components across all listening conditions for the NH group (left) and UHL group (right). Conditions at 80 dB SPL precede conditions at 60 dB SPL.*

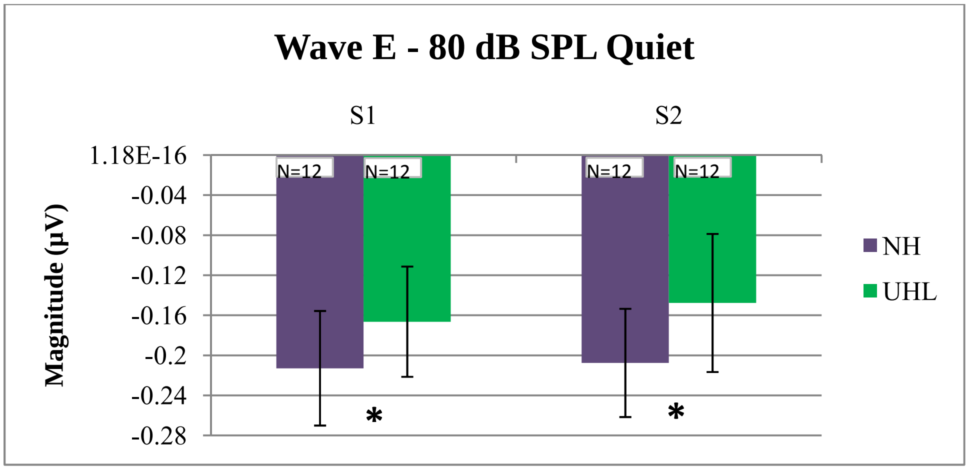
Numerical Averages across Group and Condition

Group latencies and magnitudes for the speech-evoked ABR components are organized in the Appendices. Sample sizes and standard deviations are also included in the tables. Appendix A lists the mean values for each session for the NH group and UHL groups at 80 dB SPL in quiet and noise, and Appendix B lists the mean values for each group at 60 dB SPL. If a wave was not present for one or more subjects in a given condition, fewer waves were averaged to determine the value in the table. At 80 dB SPL, waves V and A occurred before 8 ms, and Waves C through E occurred between 18 and 40 ms. The offset Wave O had a latency near 49 ms. Latencies were increased at 60 dB SPL and when noise was introduced.

Effect of Session and Hearing Group

To assess the variability in the means across sessions, a 2 (session) x 2 (group) ANOVA was performed for each waveform to determine the effect of session and hearing group on wave latency and magnitude. Session was treated as a repeated measure. Comparing Sessions 1 and 2, latency was not significantly different, with three exceptions: Wave F at 80 dB SPL in quiet [F (1,22) = 5.32,p = 0.03], Wave A at 80 dB SPL +10 SNR [F (1,12) = 5.36, p = 0.04], and Wave E at 60 dB SPL in quiet [F (1,21) = 9.53, p = 0.006]. Magnitude of waves between Sessions 1 and 2 were not significantly different, with one exception: Wave V at 80 dB SPL in quiet [F (1,22) = 7.29, p = 0.013].

Comparing NH and UHL groups, no significant differences in latency were found for measured waves. For magnitude, significant differences were noted between hearing groups for the following waves: in the 80 dB SPL in quiet condition, Wave E [F (1,22) = 5.783, p = 0.025] and Wave F [F (1,22) = 4.86, p = 0.038]; and for the 60 dB SPL +10 SNR condition, Wave A [F (1, 12) = 4.913, p = 0.047]. There were no significant Session by Group interactions. These results are shown in Figure 6. Significance is noted with an asterisk (*).



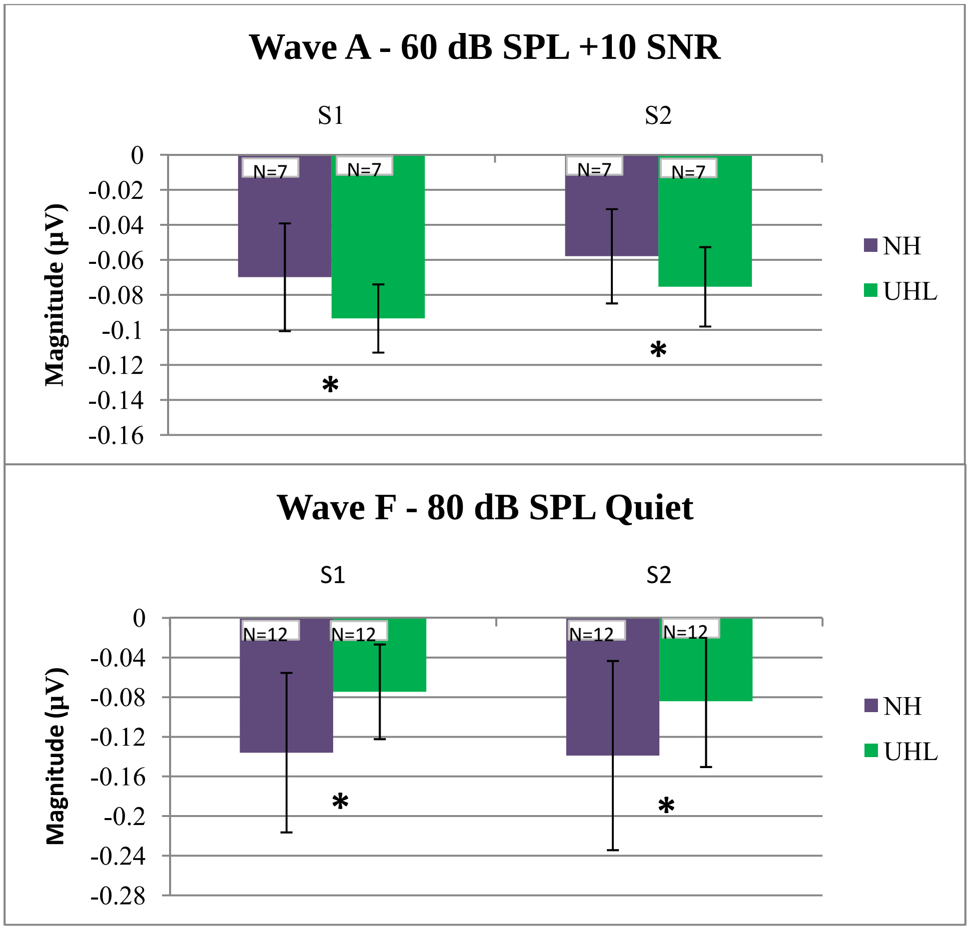


Figure 6. *Significant differences between the NH and UHL groups were found for the magnitudes of Wave E (80 dB SPL quiet), Wave F (80 dB SPL quiet), and Wave A (60 dB SPL +10 SNR) for both sessions (S1, S2). Error bars represent +/- 1 SD. The number of participants whose data was included for each analysis (N) is displayed.*

To further assess test-retest variability, the inter-session residual correlations were examined and are reported in Table 2. These correlations reflect the relative stability of rank-order of individuals over sessions independent of group. Generally, the correlations are positive and of modest magnitude, indicating consistency in performance across sessions. Nonetheless, there is considerable variability in the correlations, reflecting the small sample sizes and challenging measurement for some of the waveforms under particular testing conditions.

	LATENCY					
	80 dB SPL			60 dB SPL		
	Quiet	10 SNR	5 SNR	Quiet	10 SNR	5 SNR
V	0.920 (24)	0.876 (14)	0.766 (4)	0.791 (21)	0.478 (14)	n/a
A	0.685 (24)	0.917 (14)	0.994 (4)	0.810 (21)	0.508 (14)	n/a
C	0.721 (19)	0.641 (8)	n/a	0.441 (13)	(-0.137) (8)	n/a
D	0.430 (22)	0.561 (14)	0.982 (6)	0.579 (21)	0.690 (15)	0.501 (6)
E	0.197 (24)	0.458 (16)	0.851 (10)	0.535 (23)	0.798 (16)	0.855 (11)
F	0.585 (24)	0.273 (21)	0.314 (18)	0.548 (24)	0.590 (24)	0.533 (17)
O	0.579 (24)	0.732 (17)	0.710 (14)	0.817 (24)	0.624 (16)	0.023 (8)

	MAGNITUDE					
	80 dB SPL			60 dB SPL		
	Quiet	10 SNR	5 SNR	Quiet	10 SNR	5 SNR
V	0.705 (24)	0.114 (14)	(-0.995) (4)	0.738 (21)	0.304 (14)	n/a
A	0.587 (24)	0.396 (14)	0.897 (4)	0.035 (21)	(-0.068) (14)	n/a
C	0.572 (19)	(-0.05) (8)	n/a	0.197 (13)	0.064 (8)	n/a
D	0.369 (22)	0.438 (14)	(-0.323) (6)	0.053 (21)	0.504 (15)	0.845 (6)
E	0.683 (24)	0.365 (16)	0.790 (10)	0.819 (23)	0.632 (16)	0.353 (11)
F	0.510 (24)	0.616 (21)	0.424 (18)	0.607 (24)	0.715 (24)	0.412 (17)
O	0.304 (24)	0.250 (17)	0.103 (14)	0.664 (24)	0.384 (16)	(-0.493) (8)

Table 2. Residual correlations for latency (top) and magnitude (bottom) across sessions.

Effect of Presentation Level and Noise on the Speech-evoked ABR

A 2 (80 dB SPL, 60 dB SPL) x 2 (quiet, +10 SNR) x 2 (NH, UHL) ANOVA was performed to examine the effects of level, noise, and group on wave latency and magnitude. Data from Session 1 were analyzed separately from Session 2. Data from the +5 SNR conditions were not included in these analyses due to the increased obliteration of waves in the noisiest condition. Missing data were excluded on an analysis-by-analysis basis to maximize power for each analysis.

Significant differences in latency were found between conditions at 80 dB SPL and 60 dB SPL for the Wave V, A, D, E, F and O. Significant differences are not reported for Wave C due to wave presence variability and therefore a reduced sample size. Significant differences in magnitude were found between conditions at 80 dB SPL and 60 dB SPL for Wave V and O. Wave magnitude was not significantly affected by presentation level for either session for waves A, D, E, and F. There were no significant Level by Noise by Group interactions. Results are summarized in Table 3.

		Session 1			**Session 2**		
	Wave	df	F	p	df	F	p
Latency	V	1, 12	23.912	<.001	1, 10	21.996	0.001
	A	1, 12	17.681	0.001	1, 10	13.962	0.004
	C						
	D	1, 11	0.759	0.402	1, 11	8.246	0.015
	E	1, 13	63.997	<.001	1, 14	63.606	<.001
	F	1, 21	57.080	<.001	1, 20	20.407	<.001
	O	1, 13	5.687	0.033	1, 15	2.093	0.169
Magnitude	V	1, 12	35.396	<.001	1, 10	2.594	0.138
	A	1, 12	0.484	0.500	1, 10	4.657	0.056
	C						
	D	1, 11	0.696	0.422	1, 11	0.081	0.781
	E	1, 13	0.006	0.942	1, 14	0.872	0.366
	F	1, 21	0.078	0.783	1, 20	0.909	0.352
	O	1, 13	10.327	0.007	1, 15	6.438	0.023

Table 3. *Summary of statistical significance ($p<0.05$) for the effect of presentation level.*

Significant differences in latency were found for quiet versus noise at +10 SNR for Waves V, A, D, E, F and O. Again, significant differences are not reported for Wave C due to variability in wave presence and a reduced sample size. Significant differences in magnitude were found for quiet versus noise at +10 SNR for Wave V, A, D, E, F, and O. There were no significant Level by Noise by Group interactions. Results are summarized in Table 4.

		Session 1			**Session 2**		
	Wave	df	F	p	df	F	p
Latency	V	1, 12	17.86	0.001	1, 10	22.205	0.001
	A	1, 12	31.824	<.001	1, 10	50.981	<.001
	C						
	D	1, 11	42.003	<.001	1, 11	59.402	<.001
	E	1, 13	23.141	<.001	1, 14	35.367	<.001
	F	1, 21	31.989	<.001	1, 20	29.544	<.001
	O	1, 13	58.789	<.001	1, 15	17.232	0.001
Magnitude	V	1, 12	36.323	<.001	1, 10	9.829	0.011
	A	1, 12	14.764	0.002	1, 10	28.991	<.001
	C						
	D	1, 11	7.872	0.017	1, 11	16.300	0.002
	E	1, 13	47.903	<.001	1, 14	34.674	<.001
	F	1, 21	1.089	0.308	1, 20	6.603	0.018
	O	1, 13	4.294	0.059	1, 15	17.863	0.001

Table 4. *Summary of statistical significance ($p<0.05$) for the effect of noise.*

DISCUSSION

Assessment of Test-Retest Variability

Our data showed consistency in performance from Session 1 to Session 2 for both groups of participants. Differences were noted for a few discrete waves, but overall, responses were consistent between sessions for responses to both click and speech stimuli.

Effects of Unilateral Hearing Loss

The primary differences between the NH and UHL groups were observed in wave magnitude. Between group differences were not assessed at +5 SNR, as pair-wise deletion of all conditions tested would reduce the number of waveforms on which analyses would be performed. Visually, it is evident the UHL group has poorer periodicity at the +5 SNR conditions than the NH group; future analyses will address the analysis of these conditions.

All assessments were made with adults with UHL in the left ear. Differences have been reported in speech-evoked ABRs between the left and right ears in NH adults (Hornickel, et al., 2009). Notably, more robust frequency encoding was demonstrated in the right compared to the left ear of adults. Therefore, it is plausible that adults with UHL in the right ear may have poorer encoding of speech stimuli in quiet or in noise. Because ear differences may exist, the findings of the present study cannot be generalized to all adults with UHL.

Effects of Presentation Level

While differences were noted between intensity levels (80 dB SPL versus 60 dB SPL) for latency, fewer differences were noted for magnitude. In a typical brainstem response, measuring waves I, III, and V in a 10 ms time window, a latency-intensity function would show both a delay in latency with decreasing intensity as well as a reduction in response magnitude. Previous studies have suggested that there are multiple mechanisms contributing to the speech-evoked ABR waveform, possibly one set of neurons which respond to sound onset, and another which encode the periodic elements (Chandrasekaran & Kraus, 2010). Perhaps the onset contributors are more susceptible to the effect of presentation level, while the generators of the FFR are less affected by intensity level.

All previous studies of the speech-evoked ABR using a /da/ stimulus have employed a presentation level of 80 dB SPL. Our investigation of an intensity level more representative of conversational level (60 dB SPL) has allowed us to explore more of a "real-life" stimulation of the auditory system, and the results show that while we are eliciting a brainstem response, the auditory system may behave differently in response to speech across different intensities than would be expected with conventional ABR measurement.

Effects of Ipsilateral Noise

The present results match the conclusions of previous research characterizing the speech-evoked ABR in the presence of noise: the onset response is quickly degraded in the presence of noise, but wave F of the periodic portion remains the most resilient. However, only 4 waves (V, A, C, and F) were marked in the germinal study of the speech-evoked ABR in noise (N. Russo, et al., 2004); in the present study, up to 7 waves could be marked in each response. Additionally, by using two noise levels, multiple waveform conditions were available to determine wave presence and latency.

Johnson et al. (2005) described the anticipated effects of noise on the speech-evoked ABR based on the acoustic differences between consonants and vowels. Stop consonants like /d/

are typically low in amplitude and do not provide redundant information, and are thus easily masked by noise. Vowels, as they noted, tend to be more powerful and have a longer duration, providing more resilience in noise. Our results support these effects noted in children, and provide further information to characterize the response in adults with normal hearing and unilateral hearing loss.

Implications

No differences were observed between the click-evoked ABRs for latency or magnitude between NH and UHL groups. The speech-evoked auditory brainstem response in noise suggests differences in the magnitude of certain components of the response in adults with NH versus those with UHL. Therefore, some differences may exist in the processing of complex acoustic stimuli at the subcortical level for adults with UHL. This is the first characterization of a subcortical response to speech in noise studied in adults with UHL, a population about which little is known regarding neural processing.

Future Directions

Many research questions have developed from this work. We anticipate continuing to study the speech-evoked ABR in the UHL population and determining if differences exist in right ear versus left ear deafness, both in quiet and in noise.

As the test-retest reliability of the response has been established in the present study, a longitudinal study of brainstem plasticity/compensation following sudden-onset UHL would provide a great deal of information about how the brain accommodates to hearing loss in one ear. The speech-evoked ABR has been used to track the effect of auditory training in children with learning problems and has been shown to follow changes in brainstem activity with good fidelity.

Future research in our laboratory may also include characterizing the speech-evoked ABR in children with UHL. This population tends to have a different set of etiologies than adults (e.g., children would not likely lose hearing due to acoustic neuroma excisions), and this group tends to present with congenital or progressive onset. In the present study of adults with UHL, etiology varied, as did onset of hearing loss.

The use of an assistive device such as a Baha™ could be investigated with the speech-evoked ABR. The speech stimulus could be presented directly through a bone-anchored hearing aid (on the side of hearing loss) and recorded on the normal-hearing ear of participants with UHL.

Another application of the speech-evoked ABR would be the evaluation of speech encoding in individuals with cochlear implants who receive electrical stimulation of the auditory nerve. This could be performed through an electric ABR directly through the cochlear implant using a speech-like stimulus, or using an acoustic stimulus presented through a loudspeaker and the speech processor worn by the recipient. Currently, difficulties exist with electrical artifact produced by the stimulation of the internal device, a problem that many researchers are hoping to solve in the future.

CONCLUSIONS

The present study showed that some differences exist between the speech-evoked ABR response in adults with UHL and those with NH. Magnitude differences between hearing groups were observed in quiet for Waves E and F, and in the presence of ipsilateral noise for Wave A. Across participants, noise significantly degraded the response and affected both latency and magnitude. Presentation level had the greatest effect on wave latency. Future analyses will include stimulus and response correlations and evaluation of frequency encoding in the response.

REFERENCES

Akhoun, I., Gallego, S., Moulin, A., Menard, M., Veuillet, E., Berger-Vachon, C., et al. (2008). The temporal relationship between speech auditory brainstem responses and the acoustic pattern of the phoneme /ba/ in normal-hearing adults. *Clin Neurophysiol, 119*(4), 922-933.

Akhoun, I., Moulin, A., Jeanvoine, A., Menard, M., Buret, F., Vollaire, C., et al. (2008). Speech auditory brainstem response (speech ABR) characteristics depending on recording conditions, and hearing status: an experimental parametric study. *J Neurosci Methods, 175*(2), 196-205.

Andersen, H. T., Schroder, S. A., & Bonding, P. (2006). Unilateral deafness after acoustic neuroma surgery: subjective hearing handicap and the effect of the bone-anchored hearing aid. *Otol Neurotol, 27*(6), 809-814.

Causse, R., & Chavasse, P. (1942). Différence entre l'écoute binauriculaire et monoauriculaire pour la perception des intensités supraliminaires. *C R Soc Biol (Paris), 136*, 405-406.

Chandrasekaran, B., & Kraus, N. (2010). The scalp-recorded brainstem response to speech: Neural origins and plasticity. *Psychophysiology, 47*, 236-246.

Cunningham, J., Nicol, T., Zecker, S. G., Bradlow, A., & Kraus, N. (2001). Neurobiologic responses to speech in noise in children with learning problems: deficits and strategies for improvement. *Clin Neurophysiol., 112*(5), 758-767.

Durrant, J. D., & Boston, J. R. (2007). Stimuli for auditory-evoked potential assessment. In R. Burkard, M. Don & J. Eggermont (Eds.), *Auditory Evoked Potentials: Basic Principals and Clinical Application* (pp. 42-72). Baltimore: Lippincott Williams & Wilkins.

Firszt, J. B., Holden, L. K., Skinner, M. W., Tobey, E. A., Peterson, A., Gaggl, W., et al. (2004). Recognition of speech presented at soft to loud levels by adult cochlear implant recipients of three cochlear implant systems. *Ear Hear, 25*(4), 375-387.

Firszt, J. B., Ulmer, J. L., & Gaggl, W. (2006). Differential representation of speech sounds in the human cerebral hemispheres. *The Anatomical Record Part A: Discoveries in Molecular, Cellular, and Evolutionary Biology, 288A*(4), 345-357.

Giolas, T. G., & Wark, D. J. (1967). Communication problems associated with unilateral hearing loss. *J Speech Hear Disord, 32*(4), 336-343.

Gorga, M. P., Kaminski, J. R., Beauchaine, K. A., & Jesteadt, W. (1988). Auditory brainstem responses to tone bursts in normally hearing subjects. *J Speech Hear Res, 31*(1), 87-97.

Greenberg, S., Ainsworth, W., Popper, A., & Fay, R. (2004). *Speech Processing in the Auditory System* (Vol. 18). New York, NY: Springer-Verlag.

Hood, L. J. (1998). *Clinical Applications of the Auditory Brainstem Response*. San Diego: Singular Publishing Group.

Hornickel, J., Skoe, E., & Kraus, N. (2009). Subcortical laterality of speech encoding. *Audiol Neurootol, 14*(3), 198-207.

Johnson, K. L., Nicol, T. G., & Kraus, N. (2005). Brain stem response to speech: a biological marker of auditory processing. *Ear Hear, 26*(5), 424-434.

Koenig, W. (1950). Subjective effects in binaural hearing. *J Acoust Soc Am, 20*, 61-62.

McLeod, B., Upfold, L., & Taylor, A. (2008). Self reported hearing difficulties following excision of vestibular schwannoma. *Int J Audiol, 47*(7), 420-430.

Moller, A. R., Jho, H. D., Yokota, M., & Jannetta, P. J. (1995). Contribution from crossed and uncrossed brainstem structures to the brainstem auditory evoked potentials: a study in humans. *Laryngoscope, 105*(6), 596-605.

Ponton, C. W., Moore, J. K., & Eggermont, J. J. (1996). Auditory brain stem response generation by parallel pathways: differential maturation of axonal conduction time and synaptic transmission. *Ear Hear, 17*(5), 402-410.

Ponton, C. W., Vasama, J. P., Tremblay, K., Khosla, D., Kwong, B., & Don, M. (2001). Plasticity in the adult human central auditory system: evidence from late-onset profound unilateral deafness. *Hear.Res, 154*(1-2), 32-44.

Russo, N., Nicol, T., Musacchia, G., & Kraus, N. (2004). Brainstem responses to speech syllables. *Clin Neurophysiol, 115*(9), 2021-2030.

Russo, N., Nicol, T., Trommer, B., Zecker, S., & Kraus, N. (2009). Brainstem transcription of speech is disrupted in children with autism spectrum disorders. *Dev Sci, 12*(4), 557-567.

Russo, N. M., Nicol, T. G., Zecker, S. G., Hayes, E. A., & Kraus, N. (2005). Auditory training improves neural timing in the human brainstem. *Behav Brain Res, 156*(1), 95-103.

Shaw, W. A., Newman, E. B., & Hirsch, I. J. (1947). The difference between monaural and binaural thresholds. *J Exper Psychol, 37*, 229-242.

Skinner, M. W., Holden, L. K., Holden, T. A., Demorest, M. E., & Fourakis, M. S. (1997). Speech recognition at simulated soft, conversational, and raised-to-loud vocal efforts by adults with cochlear implants. *J Acoust Soc Am, 101*(6), 3766-3782.

Skoe, E., & Kraus, N. (2010). Auditory brain stem response to complex sounds: a tutorial. *Ear Hear, 31*(3), 302-324.

Valente, M., Valente, M., Enrietto, J., & Layton, K. (2002). Fitting strategies for patients with unilateral hearing loss. In M. Valente (Ed.), *Strategies for Selecting and Verifying Hearing Aid Fittings* (2nd ed., pp. 253-271). New York: Thieme.

Vasama, J. P., & Makela, J. P. (1995). Auditory pathway plasticity in adult humans after unilateral idiopathic sudden sensorineural hearing loss. *Hear Res, 87*(1-2), 132-140.

Wible, B., Nicol, T., & Kraus, N. (2004). Atypical brainstem representation of onset and formant structure of speech sounds in children with language-based learning problems. *Biol Psychol, 67*(3), 299-317.

APPENDIX A

Latency and Magnitude Values across Groups - 80 dB SPL

NH		80 dB SPL Quiet					80 dB SPL +10 SNR					80 dB SPL +5 SNR				
		N	Latency (ms)	SD	Magnitude (uV)	SD	N	Latency (ms)	SD	Magnitude (uV)	SD	N	Latency (ms)	SD	Magnitude (uV)	SD
V	S1	12	6.605	0.331	0.1480	0.0405	10	7.79	1.157	0.07521	0.030459	8	7.881	1.089	0.06333	0.023376
	S2	12	6.637	0.381	0.1299	0.0595	7	7.389	0.536	0.08933	0.036108	5	7.604	0.475	0.08118	0.051538
A	S1	12	7.754	0.589	-0.1383	0.0440	10	9.287	1.405	-0.0748	0.044561	7	9.444	1.271	-0.05322	0.021896
	S2	12	7.649	0.475	-0.1301	0.0417	7	9.304	0.914	-0.09271	0.053252	5	9.188	1.157	-0.0919	0.032979
C	S1	12	18.12	0.879	-0.0770	0.0547	7	19.261	1.136	-0.06426	0.022531	3	19.707	1.520	-0.04534	0.030291
	S2	10	18.403	1.018	-0.0672	0.0549	7	18.791	0.951	-0.06085	0.052227	4	18.555	0.717	-0.07881	0.023317
D	S1	11	23.227	0.961	-0.0979	0.0454	8	24.519	1.374	-0.0686	0.078677	5	24.756	0.965	-0.07628	0.021526
	S2	11	22.818	0.648	-0.0967	0.0697	8	24.023	0.987	-0.05427	0.045375	6	24.025	0.877	-0.0772	0.021220
E	S1	12	31.353	0.548	-0.2129	0.0572	9	32.001	1.015	-0.11226	0.057858	6	32.442	1.343	-0.12598	0.054778
	S2	12	31.768	1.658	-0.2076	0.0540	11	32.046	1.180	-0.11962	0.069584	8	31.855	0.842	-0.12185	0.050347
F	S1	12	39.679	0.495	-0.1360	0.0805	11	40.317	0.613	-0.1102	0.068042	10	40.667	0.905	-0.11464	0.062984
	S2	12	40.392	1.558	-0.1389	0.0954	12	40.707	1.319	-0.09798	0.082014	10	40.978	2.152	-0.09857	0.077919
O	S1	12	49.167	0.911	-0.1318	0.0652	11	49.527	1.015	-0.11938	0.070498	9	49.752	1.820	-0.0927	0.047994
	S2	12	49.3	1.608	-0.1506	0.0977	10	49.874	1.629	-0.12832	0.051344	9	50.217	1.897	-0.11713	0.052592

UHL		80 dB SPL Quiet					80 dB SPL +10 SNR					80 dB SPL +5 SNR				
		N	Latency (ms)	SD	Magnitude (uV)	SD	N	Latency (ms)	SD	Magnitude (uV)	SD	N	Latency (ms)	SD	Magnitude (uV)	SD
V	S1	12	6.709	0.356	0.1586	0.062192	9	7.681	1.394	0.08381	0.032236	4	7.213	0.443	0.06247	0.021073
	S2	12	6.709	0.390	0.13013	0.054304	10	7.384	0.992	0.06793	0.018897	4	6.785	0.277	0.07416	0.037782
A	S1	12	7.803	0.480	-0.13797	0.076856	9	9.099	1.475	-0.05895	0.021984	4	8.535	0.462	-0.10538	0.026903
	S2	12	8.003	0.714	-0.14952	0.063302	10	8.81	1.094	-0.07886	0.038258	4	8.133	0.178	-0.10744	0.036223
C	S1	10	18.418	1.092	-0.08317	0.051181	5	18.684	1.405	-0.08639	0.095103	3	18.497	1.954	-0.12095	0.106947
	S2	10	18.775	0.935	-0.06526	0.027541	5	18.82	1.274	-0.05112	0.015650	2	18.26	1.117	-0.06094	0.044202
D	S1	12	23.175	0.535	-0.07857	0.062062	9	24.478	0.904	-0.07241	0.027762	5	24.22	1.602	-0.05498	0.014928
	S2	12	23.251	1.003	-0.08756	0.044386	9	24.137	1.468	-0.06508	0.055607	6	24.068	1.318	-0.06892	0.037160
E	S1	12	31.389	0.444	-0.16645	0.055045	8	32.063	0.921	-0.09532	0.044165	6	32.22	1.109	-0.0885	0.034837
	S2	12	31.526	0.642	-0.14773	0.068922	9	31.517	0.706	-0.11637	0.047178	7	31.92	0.835	-0.11123	0.051813
F	S1	12	39.824	0.540	-0.07463	0.047768	12	40.534	0.928	-0.07248	0.042665	9	40.527	0.789	-0.0802	0.065488
	S2	12	40.02	0.560	-0.08402	0.066432	10	40.361	0.687	-0.07991	0.072755	10	40.429	0.755	-0.07526	0.064093
O	S1	12	48.911	0.935	-0.13847	0.058470	8	50.018	1.430	-0.11486	0.062871	6	50.282	1.400	-0.13015	0.111794
	S2	12	49.478	1.587	-0.14161	0.046379	10	49.774	1.392	-0.09309	0.045003	6	50.192	1.696	-0.10244	0.024891

APPENDIX B

Latency and Magnitude Values across Groups - 60 dB SPL

NH		60 dB SPL Quiet					60 dB SPL +10 SNR					60 dB SPL +5 SNR				
		N	Latency (ms)	SD	Magnitude (uV)	SD	N	Latency (ms)	SD	Magnitude (uV)	SD	N	Latency (ms)	SD	Magnitude (uV)	SD
V	S1	11	7.627	0.567	0.10208	0.052001	8	8.416	0.593	0.05902	0.032465	5	8.7	0.708	0.04836	0.024391
	S2	11	7.545	0.496	0.09342	0.050655	9	7.969	0.652	0.07877	0.026341	4	8.263	0.481	0.0847	0.043426
A	S1	11	8.851	0.478	-0.10686	0.051663	8	10.104	0.893	-0.06705	0.029610	5	10.298	0.896	-0.06212	0.023016
	S2	11	8.886	0.646	-0.11079	0.040926	9	9.589	0.858	-0.05449	0.025023	4	9.763	0.308	-0.05527	0.018855
C	S1	10	18.718	1.017	-0.09155	0.034402	6	19.428	1.039	-0.07049	0.033797	2	19.345	2.185	-0.05203	0.021324
	S2	6	18.622	0.496	-0.10162	0.044781	5	18.654	1.561	-0.07043	0.018728	4	18.97	0.962	-0.08827	0.024308
D	S1	12	23.941	0.770	-0.08866	0.028393	8	24.756	0.849	-0.06066	0.037518	4	25.415	0.691	-0.04659	0.012302
	S2	10	23.944	0.976	-0.08219	0.044043	10	24.979	1.339	-0.05946	0.036625	6	25.372	0.861	-0.06216	0.026713
E	S1	12	32.352	0.497	-0.16598	0.059841	10	33.113	1.169	-0.12241	0.052211	6	33.345	1.451	-0.0827	0.034999
	S2	11	32.743	0.733	-0.16284	0.050886	9	33.026	0.955	-0.11671	0.044031	5	34.004	1.194	-0.06807	0.013584
F	S1	12	41.189	0.753	-0.10252	0.072553	12	41.871	0.957	-0.1032	0.061040	11	42.55	1.516	-0.07359	0.080929
	S2	12	41.018	0.760	-0.11393	0.067838	12	41.768	0.973	-0.09069	0.064033	10	42.78	0.844	-0.05948	0.058865
O	S1	12	50.028	1.393	-0.09769	0.040800	8	50.676	1.977	-0.07371	0.017039	6	50.343	1.267	-0.0657	0.011001
	S2	12	49.855	0.717	-0.11202	0.044430	10	50.249	1.128	-0.07404	0.042337	7	51.576	1.330	-0.07021	0.032207

UHL		60 dB SPL Quiet					60 dB SPL +10 SNR					60 dB SPL +5 SNR				
		N	Latency (ms)	SD	Magnitude (uV)	SD	N	Latency (ms)	SD	Magnitude (uV)	SD	N	Latency (ms)	SD	Magnitude (uV)	SD
V	S1	11	7.592	0.410	0.09994	0.041480	8	8.056	0.939	0.05681	0.026087	3	8.233	0.822	0.07411	0.020287
	S2	12	7.649	0.607	0.09071	0.040101	9	8.422	0.717	0.06614	0.028620	3	8.67	0.336	0.05911	0.035318
A	S1	11	8.849	0.633	-0.09762	0.046629	8	9.64	0.763	-0.08302	0.034545	3	9.77	1.253	-0.06016	0.026791
	S2	12	8.888	0.846	-0.10043	0.036964	9	10.232	1.081	-0.06714	0.026189	3	10.493	0.564	-0.08126	0.031412
C	S1	8	19.616	0.897	-0.08405	0.051829	6	19.698	0.830	-0.06381	0.019087	3	21.257	0.455	-0.05525	0.014944
	S2	8	18.956	0.968	-0.08321	0.044598	6	19.17	1.257	-0.05354	0.029317	2	19.555	0.474	-0.03372	0.023770
D	S1	12	23.916	0.914	-0.07812	0.029636	11	24.257	1.251	-0.05594	0.048671	6	25.087	1.654	-0.05749	0.024754
	S2	11	24.168	0.879	-0.08269	0.037417	7	24.299	0.766	-0.05792	0.053066	4	24.59	0.723	-0.08202	0.013688
E	S1	12	32.575	0.734	-0.15569	0.074956	9	33.537	0.542	-0.11332	0.033310	11	34.019	1.309	-0.06925	0.026074
	S2	12	32.988	0.679	-0.13743	0.072735	11	33.44	0.845	-0.10435	0.068886	6	34.015	0.460	-0.07098	0.010858
F	S1	12	41.346	0.967	-0.10393	0.072462	12	41.993	1.231	-0.08456	0.076269	11	42.741	1.456	-0.09617	0.049873
	S2	12	41.087	0.914	-0.08619	0.064738	12	41.759	1.054	-0.07899	0.075951	9	42.486	0.926	-0.07828	0.036707
O	S1	12	49.781	0.931	-0.09643	0.053456	11	50.497	1.436	-0.07927	0.040352	7	51.7	1.226	-0.08436	0.048094
	S2	12	50.135	1.056	-0.09827	0.041089	9	50.729	1.105	-0.08295	0.037722	7	50.843	1.199	-0.0669	0.018313