EFFECTS OF SENSORINEURAL HEARING LOSS AND PERSONAL HEARING AIDS ON CORTICAL EVENT-RELATED POTENTIAL AND BEHAVIORAL MEASURES OF SPEECH-SOUND PROCESSING

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ABSTRACT

Objective: To systematically investigate the combined effects of sensorineural hearing loss and prescribed personal hearing aid(s) on cortical event-related potentials (ERPs) (waves N1, MMN, N2b and P3b) and their related behavioral measures of discrimination (d-prime sensitivity and reaction time) to the speech sounds /ba/ and /da/ presented at 65 and 80 dB ppe SPL.

Design: Cortical ERPs were recorded to /ba/ and /da/ speech stimuli presented at 65 and 80 dB ppe SPL from 20 normal-hearing adults and 14 adults with sensorineural hearing losses. The degree of sensorineural impairment at 1000 to 2000 Hz ranged from moderate losses (50-74 dB HL) to severe-profound losses (75-120 dB HL). The speech stimuli were presented in an oddball paradigm and cortical ERPs were recorded in both active and passive listening conditions at both stimulus intensities. The adults with hearing impairments were tested in the unaided and aided conditions at each stimulus intensity. Electroacoustic and real-ear testing was performed on each subject’s hearing aid(s) prior to electrophysiology testing to ensure that the hearing aids were functioning at the time of testing.

Results: The use of personal hearing aids substantially improved the detectability of all the cortical ERPs and behavioral d-prime performance scores at both stimulus intensities. This was especially true for individuals with severe-profound hearing losses. At 65 dB SPL, mean ERP amplitudes and d-prime sensitivity scores were all significantly higher or better in the aided versus unaided condition. At 80 dB SPL, only the N1 amplitudes and d-prime
sensitivity scores were significantly better in the aided condition. Even though the majority of the hearing-impaired subjects showed increased amplitudes, decreased latencies, and better waveform morphology in the aided condition, the amount of response change (improvements) seen in these measures showed considerable variability across subjects. When compared to the responses obtained from the normal-hearing subjects, both hearing-impaired groups had significantly prolonged aided RT latencies at both stimulus intensities and N2b latencies at the higher stimulus intensities.

**Conclusions:** These results suggest that hearing-impaired individuals’ brains process speech stimuli with greater accuracy and in a more effective manner when these individuals use their personal hearing aids. This is especially true at the lower stimulus intensity. The effects of sensorineural hearing loss and personal hearing aids on cortical ERPs and behavioral measures of discrimination are dependent upon the degree of sensorineural loss, the intensity of the stimuli and the level of cortical auditory processing that the response measure is assessing. The possible clinical significance of these cortical ERP and behavioral findings is discussed.
INTRODUCTION

Cortical event-related potentials (ERPs), together with behavioural measures (d-prime sensitivity and reaction time), have been successfully used to assess the cognitive processes involved in the detection and discrimination of complex stimuli, including speech sounds, in normal-hearing subjects (e.g., Cheour-Luhtanen et al., 1995; Kraus, McGee, Micco, Sharma, Carrel and Nicol, 1993; Kraus, McGee, Sharma, Carrel and Nicol, 1992; Martin, Kurtzberg and Stapells, 1999; Martin, Sigal, Kurtzberg and Stapells, 1997; Sams, Aulanko, Aaltonen and Näätänen, 1990; Whiting, Martin and Stapells, 1998). Recent evidence has shown that cortical ERPs and their related behavioral measures may be used to assess the speech detection and discrimination abilities of adults with varying degrees of sensorineural hearing loss (Oates, Kurtzberg and Stapells, 2002). Previously, we have demonstrated that as degree of hearing loss increased, latencies of ERP components and reaction time measures are prolonged and the ERP amplitudes and d-prime sensitivity scores are reduced or absent (Oates et al., 2002). It was also shown that sensorineural hearing loss has a more detrimental impact on the later stages of auditory processing compared to the earlier stages. Collectively, these electrophysiological and behavioral measures provide a window into how sensorineural hearing loss alters the timing, strength and location of the cortical brain processes associated with auditory perception. As such, they may be valuable tools to assess the speech perception benefits that individuals with sensorineural hearing loss derive from their personal hearing aids.

To date, only a few studies have investigated the combined effects of sensorineural hearing loss and personal hearing aids on cortical ERPs. In general, these have been individual case studies or studies with a small number of subjects and have reported somewhat different results. In a very early study, Rapin and Grazianni (1967) found that the majority (5/8) of their 5-to-24-month-old infants with severe-to-profound sensorineural impairments had aided cortical
ERP thresholds to click and tonal stimuli that were at least 20 dB better (lower) in comparison to their unaided thresholds. However, two of the infants had no change in their cortical ERP response thresholds for the aided versus unaided condition. Kurtzberg and colleagues compared cortical ERPs to speech stimuli in the aided versus unaided condition for four children (ages 7-27 months) with moderate to profound sensorineural hearing losses (Gravel, Kurtzberg, Stapells, Vaughan and Wallace, 1989, Kurtzberg, 1989; Stapells and Kurtzberg, 1991). These investigators reported that three of these children showed clear obligatory P1 responses which were followed by a prominent negativity. These obligatory responses, however, were only present in the aided condition. In contrast, one child with a progressive hearing loss, initially demonstrated a large P1 and the following negative obligatory responses in the aided condition when her hearing loss was in the severe range. These obligatory responses subsequently disappeared when she had no repeatable behavioral responses to air or bone conducted stimuli. An MRI was performed on this child following the loss of the cortical responses, and it was revealed that she had areas of demyelination involving the auditory radiation fibers bilaterally, which likely contributed to these results (Gravel et al., 1989). The effects of sensorineural hearing loss and personal hearing aids on the mismatch negativity (MMN) and P300 were investigated in two hearing-impaired individuals by Kraus and McGee (1994). One listener had “good” behavioral discrimination of the /ta-da/ speech contrast while wearing his hearing aid and showed a present MMN and P300, while the other had “poor” behavioral discrimination of the contrast and had an absent MMN and a present P300. An important factor that could account for some of the results in these earlier studies may have been the functional status of the hearing aid(s) at the time of testing. In these previous studies, hearing aid function was not analyzed by either electroacoustic analyses or real ear
measurements. Both of these procedures provide an objective assessment of the gain, frequency response characteristics and output limiting systems of the hearing aid(s). (For an excellent description of these two assessment techniques, see Dillon, 2001). Therefore, it is possible that the reduced or absent aided cortical ERPs reported in the previous studies may have been due, at least in part, to the subject’s hearing aid(s) not functioning properly at the time of testing or not being optimally fitted to the degree and configuration of the hearing loss. Other possibilities that may account for these earlier results include differences in methodology employed across studies (e.g., number of total trials used to collect the cortical ERPs, differences in calibration of the stimuli, and differences in how the hearing aids were adjusted at time of testing) as well as differences in the etiology of the hearing loss across subjects.

The functional effects of sensorineural hearing loss and hearing aids on speech perception may also be assessed using behavioral measures of discrimination, such as reaction time and d-prime sensitivity scores. Reaction time is defined as the time from the onset of the stimulus to the initiation of a motor response (e.g., button press), and thus provides a measure of the speed of signal processing (Gatehouse and Gordon, 1990; Wright, Spanner and Martin, 1981). In contrast, the d-prime sensitivity score is defined as a criterion-free measure of percent correct that takes into account false alarm rate (Swets, 1973; Yanz, 1984).

Given the increased emphasis on early identification and remediation of hearing loss, considerable interest exists in utilizing cortical ERPs to assess clinical populations where behavioral measures of speech detection and discrimination are difficult to obtain (e.g., infants, children and difficult-to-test patients). Cortical ERPs might provide audioligists with several additional pieces of diagnostic information in comparison to the earlier auditory evoked potentials (AEPs) (electrocochleography, auditory brainstem responses and middle latency
responses) that likely will assist in managing hearing losses in these clinical populations (for a review, see Stapells, 2002). First, cortical ERPs allow the investigator to assess the integrity of the entire auditory system, including the cochlea, brainstem, auditory cortex and associated cortical areas. Secondly, cortical ERPs can be recorded to a variety of auditory stimuli ranging from simple tonal stimuli to complex speech stimuli, such as consonant-vowel syllables, words and even full sentences. The recording of cortical ERPs to speech stimuli provide insight into the early and later cognitive processes that underlie the detection and discrimination of speech. The earlier AEPs, in contrast, are best recorded to transient stimuli, such as clicks and/or tonebursts, and thus provide no information regarding speech processing.

Several investigators have demonstrated that cortical ERPs provide very useful information regarding higher-level (cortical) responsiveness to complex speech sounds in cases where lower-level testing (e.g., auditory brainstem responses, otoacoustic emissions, and acoustic reflex testing) suggests a peripheral problem, such as auditory neuropathy (Gravel and Stapells, 1993; Starr, Picton, Sininger, Hood and Berlin, 1996). In contrast, the absence of cortical ERPs or their abnormal waveform morphology, amplitudes and/or latencies in the presence of intact earlier responses can suggest the presence of a higher-level (central) dysfunction (Gravel et al., 1989; Klein, Kurtzberg, Brattson, Kreuzer, Stapells, Dunn, Rapin and Vaughan, 1995; Stapells and Kurtzberg, 1991; Stapells, 2002). This type of objective information regarding the brain’s processing and discrimination of speech, can be invaluable when planning an appropriate remediation program for difficult-to-test clients, including hearing-impaired infants and children. The slow cortical ERPs (waves P1-N1-P2) may be used to assess the capacity of the auditory cortex to detect acoustic changes within speech stimuli (Martin and Boothroyd, 1999)
and they may also be used to provide a functional measure of the benefit provided by personal hearing aids (Gravel et al., 1989; Kurtzberg, 1989; Oates, Kurtzberg and Stapells, Reference Note 1; Purdy, Sharma, Katsch, Storey, Dillon and Ching, Reference Note 2; Rapin and Graziani, 1967). In contrast, the later cognitive ERPs and their related behavioral measures can provide audiologists with information regarding whether the brain can discriminate the acoustic differences between speech stimuli (e.g., /ba/ versus /da/). This information is indexed by the MMN on a pre-attentive level and by waves N2b, P3b and behavioral d-prime sensitivity scores on an attentive or conscious level (see Stapells, 2002, for a review). The term “pre-attentive” refers to a response that can be elicited independent of a subject’s attention to the task. Kane and colleagues have demonstrated that the MMN can be elicited in unconscious coma patients (Kane, Curry, Rowlands, Manara, Lewis, Moss, Cummins and Butler, 1996). In contrast, conscious perception of an acoustic change present in the stimuli, such as that required to elicit an N2b or P3b response, does require activation of attention triggering mechanisms within the cortex (Näätänen and Alho, 1997).

Collectively, these behavioral and electrophysiological measures also provide insight into how well or effectively the brain is perceiving the differences between these speech stimuli, as reflected in the ERP and reaction time latencies. If it is shown that these cortical ERPs and behavioral measures of discrimination provide audiologists with reliable insight into the accuracy and effectiveness with which hearing-impaired individuals detect and discriminate speech with their personal hearing aids, then these measures may play a crucial role in the future selection and fitting of these instruments.

It may be that these cortical ERPs might assist audiologists in: (1) assessing the aided speech perception abilities of the younger hearing-impaired population, (2) initially fitting
hearing aids to this clinical population, and (3) monitoring the aided benefits derived from these instruments. However, before these measures can be applied to this younger hearing-impaired clinical population, it is necessary for us to have a better understanding of the combined effects of sensorineural hearing loss and personal hearing aids on cortical ERPs in adults with varying degrees of sensorineural hearing loss.

The purposes of the present study were to: (1) assess the combined effects of sensorineural hearing loss and personal prescribed hearing aids on the detectability of cortical ERPs to /ba-da/ speech sounds in adults with sensorineural hearing loss; (2) assess the combined effects of sensorineural hearing loss and hearing aids on the amplitudes and latencies of cortical ERPs across groups with differing degrees of hearing impairment and (3) investigate whether changes in amplitude and latency of these ERPs occurring with sensorineural hearing loss and hearing aids differ for responses reflecting different stages of auditory processing.

METHODS

Subjects

Two groups of subjects participated in the study: (1) twenty adults (12 female and 8 male) with normal hearing sensitivity (mean age = 30.3 years, S.D. = 6.01) and (2) fourteen adults (6 female and 8 male) with sensorineural hearing impairments (mean age = 29.3 years, S.D. = 16.2). Of these twenty normal-hearing subjects, 10 were tested in the active listening condition and a separate 10 individuals were tested in the passive listening conditions. The 10 normal-hearing subjects who were tested in the active listening conditions and all of the hearing-impaired subjects also participated in our earlier study (Oates et al., 2002).
The normal-hearing subjects had bilateral pure-tone thresholds of 15 dB HL (ANSI, 1996) or better from 250-8000 Hz. For our analyses, the adults with sensorineural hearing impairments were divided into two groups, based on the average of their best 1000- and 2000-Hz pure-tone thresholds: 50-74 dB HL “moderate” hearing loss; and 75-120 dB HL “severe/profound” hearing loss. Table 1 presents the mean behavioral thresholds for each hearing-loss group as well as for the normal-hearing group. All participants in the study had normal tympanograms (admittance curve with a single peak between ± 50 daPa to a 226-Hz probe tone) and no self-reported history of recurrent middle-ear or neurologic problems. Acoustic immitance testing was conducted prior to each test session to ensure that there were no changes in middle-ear function. In the present study, the normal-hearing subjects were tested only in the unaided condition and the hearing-impaired subjects were tested in both the unaided and aided conditions (while wearing their prescribed personal hearing aids). Ten of the hearing-impaired subjects wore binaural hearing aids and four wore monaural instruments.

Stimuli

The consonant-vowel speech stimuli /ba/ and /da/ employed in the current study were the same stimuli used in our previous study (Oates et al., 2002). These stimuli were selected because they differ in place of articulation, an articulatory feature of speech that is particularly susceptible to the effects of peripheral hearing impairment. Information regarding the generation, presentation and acoustic characteristics of these stimuli were presented in greater detail in our
earlier paper and are presented only briefly below. The /ba-da/ stimuli were natural digitized speech tokens recorded by the STIM portion of the Neuroscan system using a 30,000 Hz sampling rate (10,000 Hz anti-aliasing filter, 12 dB/octave). These tokens were edited to 150 ms by deleting the final portion of the steady-state vowel and windowing the vowel offset.

The stimuli were presented to the subjects at 65 and 80 dB peak-to-peak equivalent (ppe) SPL in the soundfield through a loudspeaker located one meter in front of the subjects. These levels correspond to approximately 70 and 85 dB HL based on ANSI (1996) standards and measures of binaural free field (0 degree azimuth) sensitivity (Durrant and Lovrinic, 1995). The stimuli were presented at two intensities to accommodate the various degrees of sensorineural hearing loss present in the study.

The stimuli were presented in active and passive oddball paradigms. In the active listening paradigm, subjects were asked to carefully listen for the deviant stimulus and to press a button when they hear this stimulus, placing equal emphasis on accuracy and speed (Martin et al., 1997). In contrast, for the passive listening task, subjects were instructed to sit quietly, ignore the stimuli, stay awake and read a book or magazine of their choice.

The probability of occurrence for the stimuli was 90% for the standard stimuli and 10% for the deviant stimuli. The onset-to-onset interstimulus interval (ISI) was 1100 ms for the active condition, and 627 ms for the passive condition. Both speech sounds were presented as standards and deviants in separate runs. The order of stimulus presentation within a run was pseudo-randomized for the active condition, such that no run began with a deviant and two deviants could not occur in succession within a run. For the passive condition, stimulus presentation order was not randomized and every 10th stimulus was a deviant (e.g., Scherg, Vajsar and Picton, 1989).
For the active conditions, electrophysiologic responses to 200 stimuli were recorded in a run. Each run was replicated, yielding a maximum of 400 total trials for each stimulus at each intensity. In the passive condition, 1000-single trial recordings were obtained, and then replicated. This produced a total of 2000 trials for each stimulus at both intensities. A larger number of sweeps was needed for the passive conditions due to the small amplitude and large variability of the MMN (e.g., Kurtzberg, Vaughan, Kreuzer and Flieger, 1995; Lang, Eorola, Korpilahti, Holopainen, Salo and Aaltonen, 1995). The number of trails used for the passive condition was based upon two rationales: (1) MMN was successfully recorded to these same speech stimuli using 2000 total trials per contrast in subjects with simulated hearing losses as well as those with actual hearing losses (Martin et al., 1999, Oates et al., 2002); and (2) the current investigators wanted to keep the recording time to a reasonable level because the subjects were being evaluated with two speech stimuli at two separate intensities for each test session. The test protocol for the hearing-impaired subjects was the same for the unaided and aided conditions.

**EEG Recordings**

Recordings of the cortical ERPs were obtained using seven EEG channels, with gold cup electrodes placed at Fz, Cz, Pz, M1, M2, C3M (midway between C3 and M1) and C4M (midway between C4 and M2). The active electrodes for each channel were referenced to an electrode placed at the tip of the nose (Vaughan and Ritter, 1970). An eighth channel recorded electro-oculographic (EOG) activity from electrodes placed above and below the right eye. The EOG activity was continuously monitored on an oscilloscope throughout the testing. An electrode located on the 7th cervical vertebra (Cv7) served as the ground. The number of recording channels was limited to eight due to the capabilities of our recording system. Interelectrode
impedances were 5000 Ohms or less. The EEG channels was amplified (gain = 20,000), filtered (0.1 to 100 Hz, 6 dB/octave) and digitized (568 Hz per channel, 512 points), employing analysis times of 900 ms and 550 ms for the active and passive conditions, respectively. These analysis windows included pre-stimulus baselines of 100 ms for the active condition and 50 ms for the passive condition. The EOG was digitized and filtered as described above; however, the gain for the amplifier was set to 5000. Prior to averaging, the single-trial ERP waveforms were processed offline in several ways: baseline correction, digital filtering in the frequency domain (30-Hz low-pass filter, 12 dB/octave) and artifact rejection (± 100 μV for all channels). The ERPs were averaged separately for each stimulus at each intensity. For the hearing-impaired subjects, separate averaged ERP waveforms were created for the unaided and aided conditions.

**Behavioral Measures**

During the active condition, measures of reaction time (RT) and percent correct discrimination were obtained simultaneously with the ERP measures by having the listener press a button in response to the deviant stimuli. Measures of median reaction time and the number of hits, false alarms, correct rejections and missed trials were calculated for each active condition. Median reaction times were calculated for each subject as opposed to mean values because the distribution of reaction times is skewed for an individual subject’s data. Median values represent the central tendency of the data for that subject.

For the oddball paradigm employed in this study, a “hit” occurred when the subject correctly identified the deviant stimulus and pressed the button. A “correct rejection” occurred when the subject correctly identified the standard stimulus and did not press the button. A “false
alarm” occurred when the subject pressed the button when there was no deviant stimulus. A “miss” occurred when the subject did not press the button even though a deviant stimulus was present. During this active listening task, the stimuli were presented at a rate of 0.9/sec. The timing for presentation of the stimuli was not affected by when the subject pressed the button or whether the subject correctly identified the deviant stimulus. The d-prime sensitivity score was calculated by the formula: $d$-prime = $z($false alarms$) - z($hits$)$ (Swets, 1973; Yanz, 1984).

**Procedure**

All testing was performed in a double-walled sound attenuating booth. For the normal-hearing subjects, the active and passive conditions were typically recorded in one session, with the passive condition being recorded first. For the hearing-impaired subjects, the aided and unaided conditions were recorded over two sessions. Session one consisted of aided testing in the passive condition followed by the active condition at 65 and 80 dB SPL. Session two consisted of unaided testing in the passive and active conditions at both stimulus intensities.

For the hearing-impaired subjects, an electroacoustic analysis of the individual’s prescribed personal hearing aid(s) and real ear measurements were conducted (Audioscan, model RM-500) prior to the start of electrophysiologic testing. The electroacoustic analysis was conducted according to ANSI (1996) standards and provided an evaluation of the gain, frequency response and output limiting characteristics of the hearing aid(s). Following this analysis, each of the hearing-impaired subjects was instructed to adjust the volume control on their hearing aid(s) to their most comfortable listening (MCL) level. The stimuli for this task were the /ba-da/ speech sounds presented at 65 dB SPL through a loudspeaker located one meter in front of the subject. The softer speech stimuli was used as the criterion stimulus for the subject adjusting
their hearing aid to MCL because this intensity level more closely approximates normal conversational speech.

Once the hearing aid(s) were set to MCL, real ear measurements were conducted. This testing included obtaining a real ear unaided response as well as a real ear aided response for each hearing aid. The real ear insertion gain (equivalent to the aided gain minus the unaided gain at each frequency) provided by the subject’s hearing aid(s) at 1000 - 2000 Hz was compared to the target gain calculated by the NAL-RP formula at those same two frequencies. The Audioscan also provided an estimate of each hearing-impaired subject’s Speech Intelligibility Index (SII) expressed as a percentage value. The SII is a method used to predict the intelligibility of the aided speech signal based upon its unaided audibility (for a review, see Dillon, 1999, 2001).

Table 2 contains results of the electroacoustic analyses and real ear measurements performed on all hearing impaired subjects’ personal hearing aids. It also provides information on whether these subjects wore monaural or binaural instruments as well as the type of hearing aid circuits used (linear versus non linear). Each hearing-impaired subject was randomly assigned a letter A - N, which will be used in subsequent figures in the results section. The adequacy of the hearing aid fit was informally assessed using both the achieved insertion gain versus the target gain at 1000-2000 Hz as well as the SII value. Hearing aid gain at these two frequencies is critical because spectrographic analysis revealed the primary acoustic differences needed to differentiate between these two speech stimuli occurs in the 1300-1900 Hz frequency region (Oates et al., 2002).

For the majority (83.3%) of hearing aids, the user settings on the instruments either matched (within 5 dB) the NAL-RP target at 1000-2000 Hz or were within a close range (~ 5-10 dB) of this target and the aided SII values showed improvements ranging from 6 - 81%. For the
four hearing aids where the achieved insertion gain was at least 15 dB below the target gain at 1000-2000 Hz, no adjustments were made to the gain, frequency response curve or output limiting systems of the hearing aid. The rational for this approach was that we wanted to obtain a measure of the brain’s response to the aided speech signals at user settings that were typical for that hearing-impaired individual. All electrophysiologic testing for the aided active and passive conditions at both stimulus intensities was conducted with the hearing aid set to the subject’s MCL.

Insert Table 2 here

During the active condition, subjects were instructed to fixate on a small dot located one meter in front of them and to avoid blinking or swallowing during the recording task. Behavioral measures of reaction time (RT), percent correct discrimination, number of hits and false alarms, correct rejections, missed trials, and d-prime sensitivity scores were obtained by the STIM portion of the Neuroscan system.

**Data Analysis**

Following offline processing (baseline correction, artifact rejection and digital filtering) the single-trial responses of an individual subject were averaged so that responses to stimuli presented as standards were grouped separately from responses to the deviant stimuli. Difference waveforms, used to measure N2b and MMN, were obtained by subtracting the averaged responses to the standard stimuli from the averaged responses to the same stimuli presented as deviants. For each test condition, replications were averaged together.
The criteria used to determine response presence or absence were the same as those described in detail in our previous papers (Martin et al., 1999; 1997; Oates et al., 2002; Whiting et al., 1998) and thus will only be briefly reviewed. For a response to initially be considered present in either the active or passive conditions, two judges had to agree that the amplitude of the individual ERP peak was larger than the level of the pre-stimulus baseline and it had to meet certain rules regarding scalp topography and polarity inversion. The rules for the active conditions were: (1) N1 and N2 were required to be larger in amplitude at the fronto-central electrode sites (e.g., Fz and/or Cz) in comparison to the parietal electrode site (Pz); (2) N2b had to be negative in voltage relative to the nose, be smaller in amplitude at Pz than at Cz or Fz, and be larger in response to the deviants; and (3) P3b was required to be largest in amplitude at Pz.

The presence of MMN was determined using the difference waveforms in the passive conditions. To be judged present, MMN had to be more negative in amplitude at the fronto-central sites (e.g., Fz and Cz) than at the parietal electrode site (Pz). Response presence was confirmed by a polarity inversion at the mastoid electrode sites (e.g., M1/M2), but lack of inversion did not indicate the absence of a response.

If any peaks in either the active or passive conditions were judged to be questionable after applying the rules described above, the replicability of the peak was examined over two recording blocks. If the peak was replicable, then it was considered present. When a peak was judged to be absent, a zero-microvolt amplitude was assigned (Martin et al., 1999, 1997; Oates et al., 2002; Oates and Stapells, 1997; Whiting et al., 1998). Our previous ERP studies have used several statistical and averaging techniques to demonstrate that inserting a zero-microvolt value is a reasonable estimate of the amplitude of waves judges as “no response”.
Response Measurements

Response windows for each ERP peak, developed from the grand-mean waveforms to the /ba-da/ stimuli in our previous studies (Martin et al., 1999, 1997; Oates et al., 2002; Whiting et al., 1998), were employed to assist in measuring amplitudes and latencies from the individual subjects’ responses. The response windows for the active conditions were as follows: N1 was defined as the largest negativity occurring between 80 and 200 ms at Cz to the standard stimuli; N2b was defined as the largest negativity occurring between 200 and 420 ms at Cz in the difference waveform; and P3b was defined as the largest positivity occurring between 285 and 800 ms at Pz in response to the deviant stimuli. For the passive conditions, MMN was defined as the largest negativity occurring between 80 and 400 ms at Fz in the difference waveform.

Response measurements were taken from each subject’s grand-mean waveform for each condition. Amplitudes were measured relative to the pre-stimulus baseline and were taken at the point of the largest amplitude within the response window. Latency measures were taken at the center of the peak within the response window. In cases of a bifid wave of equal amplitude, latency was measured at the midpoint of the wave and amplitude was measured on either peak.

Consistent with our previous studies, three additional post-processing techniques guided the current response measurements. These were: (1) P3b responses were determined after digital low-pass filtering at 5 Hz (12 dB/oct) in the frequency domain to smooth the response (Martin et al., 1997; Oates et al., 2002; Picton, 1992); (2) if the MMN contained multiple peaks, inversion at the mastoids was examined (see Oates et al., 2002 for further description); and (3) in cases where it was difficult to determine MMN latency, the waveform was low-pass filtered at 15 Hz in order to smooth and clarify the response (Kurtzberg et al., 1995). All MMN
amplitude and latency measures, however, were taken from the response with the 0.1 to 30 Hz bandpass filter.

**Statistical Analyses**

The latency and amplitude measures for the cortical ERPs, as well as the behavioral measures, were each analyzed separately using descriptive statistics and two-way repeated measures analyses of variance (ANOVAs). A more-detailed description of the specific factors used in the ANOVAs is provided in the Results section. The ANOVAs were only conducted at the electrode locations that yielded the maximum responses for the various peaks (i.e., N1 at Cz, MMN at Fz, N2b at Cz, and P3b at Pz). Main effects and interactions were considered significant if $p < .01$. Greenhouse-Geisser epsilon correction factors were applied to the degrees of freedom when appropriate and adjusted probabilities reported (Greenhouse and Geisser, 1959). When significant results were found in the ANOVAs, Neuman Keuls *post hoc* tests were performed to determine the pattern of the significant differences. Results of the *post hoc* tests were considered significant if $p < .05$.

**RESULTS**

Since the results of the ANOVAs (see below) indicate that the ERP and behavioral results generally do not differ between the /ba/ and /da/ stimuli, the responses are averaged across the two stimuli. The only exceptions were N1, MMN and N2b latencies at 80 dB SPL. This results section will be divided into three sections to reflect the way the data were analyzed. This same pattern of organization will be used in the Discussion section.
The cortical ERPs recorded from one subject in the active condition to the /ba-da/ stimuli presented at 65 and 80 dB SPL are shown in Figure 1. This subject was 46 years old at the time of testing and has a moderate sensorineural hearing loss [2-frequency pure-tone average @ 1000-2000 Hz (PTA²) = 55 dB HL]. He has successfully worn a monaural in-the-ear hearing aid for at least 10 years. The results of the electroacoustic and real ear measurements performed on his hearing aid are found in Table 2, subject A. The top set of waveforms was recorded in the unaided condition; the bottom set in the aided condition. ERP waves N1, N2b and P3b are labeled, and behavioral reaction time (RT) latencies and d-prime sensitivity scores are indicated. In the present study, a perfect d-prime sensitivity score is 4.65. This subject’s data is representative of the trends found in the group data.

The use of a personal hearing aid substantially increased the amplitude and decreased the latency of all of this subject’s ERP components. These response changes are particularly evident at the lower stimulus intensity, where, for example, the latencies are approximately 30 ms shorter for N1 and 90 ms shorter for P3b in the aided versus unaided condition. The amplitudes of N1 and P3b are approximately 50% greater in the aided condition.

The subject’s reaction time latencies are also considerably shorter for the aided condition, especially at the lower stimulus intensity. For example, his aided RT at 65 dB SPL is 405 ms,
compared to his unaided RT of 562 ms. A much smaller improvement in reaction time latencies in the aided versus unaided condition is evident in the 80 dB responses. Despite the improvements in ERP latencies and RT measures provided by the hearing aid at both stimulus intensities, these indices are prolonged in comparison to the mean latencies obtained for these same stimuli in the normal-hearing subjects (Oates et al., 2002). This finding suggests that despite the benefits provided by his hearing aid, this subject is processing these speech stimuli in a less effective manner compared to his normal-hearing peers at both stimulus intensities.

At 65 dB SPL, this subject’s behavioral d-prime sensitivity scores also showed improvement from the unaided to aided condition; however, he was still experiencing some difficulty accurately discriminating the acoustic differences between the stimuli in the aided condition. In contrast, at 80 dB SPL, this individual had a perfect aided d-prime sensitivity score and his aided P3b and reaction time latencies (377 and 349 ms, respectively) were within the latency ranges obtained for these two measures in our normal-hearing subjects (Oates et al., 2002). The latency ranges obtained from our normal-hearing subjects to these same 80 dB SPL stimuli were: 339 - 464 ms for P3b and 283 - 410 ms for RT latencies.

Figure 2 displays waveforms obtained from the same hearing-impaired subject recorded in the passive condition to the /ba-da/ stimuli presented at 65 and 80 dB SPL. The top set of waveforms were recorded in the unaided condition, the bottom set in the aided condition. In each waveform set, the standard and deviant waveforms are on top and the difference waveform is on the bottom, with mismatch negativity labeled when present.

Insert Figure 2 about here
This subject’s waveforms to the 65 dB SPL stimuli do not show a MMN in the unaided condition. This finding is somewhat unexpected given the subject’s behavioral and N2b-P3b responses at 65 dB SPL, which show clearly present, albeit delayed and degraded, responses (see Figure 1). A small amplitude MMN, however, is present in the aided condition.

At the higher stimulus intensity, there is a questionable MMN present in the unaided condition. If it is indeed present, its amplitude is considerably smaller compared to the mean responses seen in our normal-hearing subjects (Oates et al., 2002). The bifid peak that occurred in the unaided response made exact measurement of peak latency questionable. The MMN recorded in the 80 dB SPL aided condition, however, closely resembles the amplitude, latency and morphology characteristics of the responses recorded in subjects with normal-hearing sensitivity. The intrasubject variability that occurred in the amplitude and latency measurements for the MMN as well as the possible clinical utility of recording this response in individual subjects will be discussed further in the Discussion section.

Table 3 presents the effects of sensorineural hearing loss and hearing aids on the presence of the behavioral and electrophysiological measures at both stimulus intensities for all subjects. The use of personal hearing aids had a substantial impact on response presence at the lower stimulus intensity for individuals in the moderate and severe-profoundly hearing-impaired groups. In contrast, for the higher stimulus intensity, the use of hearing aids only produced a large improvement in response presence for individuals in the severe-profound hearing loss group.

_________

Insert Table 3 about here

_________
The effects of sensorineural hearing loss and personal hearing aids on the amplitudes and latencies of the cortical ERPs to the /ba-da/ stimuli presented in the active condition are clearly evident in the grand-mean waveforms for each group. These waveforms are shown in Figure 3.

The grand-mean waveforms for the 65 dB SPL stimuli indicate that the use of hearing aids substantially improves the detectability of waves N1, N2b and P3b for individuals with moderate or severe-profound hearing impairments. For individuals with moderate hearing losses, the amplitudes of all these ERPs are substantially larger and the latencies shorter in the aided versus unaided condition. An even larger change is evident in the response for individuals with severe-profound losses. There is little or no response present in the unaided condition; however, a well-defined response is clearly present in the aided condition, albeit somewhat smaller in amplitude and later in latency.

The grand mean waveforms for the 80 dB SPL stimuli show a similar pattern. At this higher stimulus intensity, the largest response changes with hearing aids occurred for individuals in the severe-profound group. For this group, no response was present in the unaided condition; however, a response with good morphology is seen in the aided condition. Small improvements in the amplitudes and latencies of the ERP components can be seen in the aided versus unaided condition for the moderately-impaired group.

Despite the improvements provided by hearing aids, the mean aided latencies of the cortical ERPs for the moderate and severe-profound hearing loss groups are delayed relative to
the mean latencies for the normal-hearing subjects. This effect is particularly evident for the responses at the lower stimulus intensity.

The grand-mean waveforms in the passive condition for each group, shown in Figure 4, demonstrate the effects of sensorineural hearing loss and hearing aids on the amplitudes and latencies of the cortical ERPs to the /ba-da/ stimuli. For stimuli presented at 65 dB SPL, the use of hearing aids improved the detectability of MMN for individuals in the moderately hearing-impaired group. In contrast, no clear MMN was evident in the unaided or aided conditions for the severe-profound group.

At 80 dB SPL, MMN was clearly present for the normal and moderately hearing-impaired groups in the unaided condition. Two somewhat surprising results, however, were found for the aided MMN responses at this intensity: (1) for the moderately-impaired group, the aided MMN is smaller in amplitude than the unaided response; and (2) no clear aided MMN response is evident in the grand-mean waveform for the severe-profound group, despite the fact that it was judged to be present in 87.5% of the individual cases (see Table 3). It is likely that these unexpected findings reflect the poor signal-to-noise ratio of the MMN and the variability of latency across individual subject’s MMN responses which resulted in cancellation of the waveform in the grand-mean waves.

Table 4 displays the mean and standard deviation values for the hearing-impaired subjects’ aided and unaided measures of response strength (ERP amplitudes and d-prime sensitivity scores) and response timing (ERP and RT latencies) at both stimulus intensities. Table
5 provides a summary of the results of the two-way ANOVAs conducted on the amplitudes and latencies of these unaided and aided cortical ERP components and behavioral measures of discrimination for the hearing-impaired subjects. The two within-subject factors assessed were: (1) type of stimulus (ba versus da) and (2) test condition (unaided versus aided). The responses for each stimulus intensity were evaluated independently because there were some profoundly hearing-impaired subjects who were not tested at 65 dB SPL.

The results of the ANOVAs revealed that, as expected, all measures of response strength (ERP amplitudes and d-prime sensitivity scores) were significantly larger in the aided versus unaided conditions for the lower stimulus intensity. This effect can clearly be seen in Figure 3. There was also a clear trend (p < .05) for all mean ERP and RT latencies at 65 dB SPL to be shorter in the aided versus unaided condition (see Tables 4 and 5). The only exception to this pattern was MMN latency.

A somewhat different pattern of results emerged for the responses to the 80 dB SPL stimuli. Only N1 amplitudes and d-prime sensitivity scores were significantly larger/better in the aided versus unaided conditions. There was a trend for P3b amplitudes to be larger in the aided condition. All mean aided ERP and RT latencies, with the exception of MMN, were shorter in comparison to the unaided responses (see Table 4), however, none of these differences reached statistical significance (see Table 5)

Insert Tables 4 and 5 about here
Comparison of Aided ERP and Behavioral Measures to Those Obtained from Normal-Hearing Subjects

Because sensorineural hearing loss involves factors other than a reduction in the audibility of the signal, additional two-way ANOVAs were calculated to determine if the brain’s ability to process the speech stimuli is similar in individuals with normal-hearing sensitivity versus those with sensorineural hearing impairments who are fit with personal prescribed hearing aid(s).

Table 6 displays the mean and standard deviation values for the measures of response strength (ERP amplitudes and d-prime sensitivity scores) and response timing (ERP and RT latencies) in these three subject groups. Table 7 provides a summary of the results of the ANOVAs calculated on the amplitudes and latencies of the cortical ERPs and the behavioral measures of speech-sound processing in these three groups of subjects. The between-subject factor for these analyses was subject group (responses from normal-hearing group; aided response from moderately-impaired group and aided responses from severe-profound group); the within-subject factor was type of stimulus (ba versus da). The results for each stimulus intensity were evaluated independently.

At both stimulus intensities, the mean aided ERP amplitudes for the moderate hearing impaired subjects were larger than those for the normal control subjects (Table 6); however, none of these differences reached statistical significance (Table 7). There was a trend for the severe-profound hearing impaired subjects to have poorer aided d-prime scores at 80 dB SPL in comparison to their normal age-matched peers.

The mean aided ERP and RT latencies for the two groups of hearing-impaired subjects were prolonged in comparison to individuals with normal-hearing sensitivity for both stimulus intensities (see Table 6). Results of the ANOVAs revealed that only the hearing-impaired subjects’ aided RT latencies at 65 and 80 dB SPL and aided N2b latencies at 80 dB SPL reached
statistical significance (see Table 7). A clear trend, however, existed for both hearing-impaired
groups to have prolonged \emph{aided} N1, MMN and N2b latencies compared to their normal-hearing
peers at the lower stimulus intensity.

Variability in the Amount of Response Change (Improvements) Seen for the Cortical ERP
and Behavioral Measures of Speech-Sound Processing in the Aided Condition.

Figure 5 displays a comparison of each individual subject’s unaided and aided amplitudes
and latencies for the ERP components and behavioral measures in response to the 65 dB SPL
stimuli. The ERP components and behavioral measures displayed are: MMN, P3b, d-prime
sensitivity scores and reaction time latencies. Each subject’s data is represented by a letter (a-n),
which are the same as those used in Table 2. A similar pattern of results was seen for the
individual subjects’ responses to the 80 dB SPL stimuli, which will be discussed in detail below.

As expected, the vast majority of the subjects demonstrated considerable improvements
in the aided \emph{versus} unaided condition for both the behavioral and electrophysiological measures
at 65 dB SPL. There were a few subjects, however, who demonstrated the opposite effects for
some measures. For example, three subjects (“h”, “k” and “m”) had longer MMN latencies in the
aided versus unaided conditions; similarly, one subject (“b”) had longer P3b latencies in the aided versus unaided conditions. Interestingly, there were no instances where an individual had this opposite effect consistently across all of these behavioral and electrophysiological measures of speech discrimination at this stimulus intensity.

This figure also demonstrates that the majority of subjects’ MMN, P3b and RT aided latencies at 65 dB SPL are delayed relative to the mean latencies for the normal-hearing subjects. Similarly, at least 60% of the hearing-impaired subjects’ aided amplitudes and d-prime sensitivity scores were below the means of their normal-hearing peers.

A third quite interesting finding evident in this figure is that even though the majority of hearing-impaired subjects showed increased ERP amplitudes, decreased latencies and improved behavioral discrimination performance to the 65 dB SPL stimuli while wearing their hearing aids, the amount of response change was quite variable. The amount of response change for each hearing-impaired subject at each stimulus intensity was calculated by subtracting their aided amplitude or latency for each ERP component or behavioral measure from their unaided response measure for that same component at that same intensity. The range of response changes seen across subjects was then calculated for the MMN, P3b and RT latencies. The range of latency changes or improvements seen for these measures at 65 dB SPL were from: (1) 39.74 - 243.77 ms for MMN; (2) 52.74 - 249.9 ms for P3b; and (3) 34 - 160 ms for RTs. Similarly, the range of latency changes seen for these measures at 80 dB SPL were from: (1) 16 - 202.26 ms for MMN; (2) 22.85 - 256.15 ms for P3b; and (3) 10 - 184.25 ms for RT.

Considerable intersubject variability also existed when the aided versus unaided measures of response strength were compared at each stimulus intensity. The range of amplitude changes or improvements in the MMN, P3b and d-prime scores for the 65 dB SPL responses were from:
(1) 0.46 - 4.22 µV for MMN; (2) 3.52 - 18.41 µV for P3b; and (3) 0.30 - 4.23 for the d-prime scores. A similar range of amplitude changes was seen for the 80 dB SPL responses: (1) 0.06 - 2.29 λV for the MMN; (2) 0.65 - 21.64 λV for P3b; and (3) 3.45 - 4.43 for d-prime scores.

DISCUSSION

Effect of Hearing Loss and Hearing Aids on Cortical ERPs and Behavioral Measures of Speech-Sound Processing

The results of the present study demonstrate that sensorineural hearing loss and the use of personal hearing aids have a significant impact on the timing and strength of the brain processes involved in the detection and discrimination of complex speech stimuli. This effect is dependent, in part, upon the degree of sensorineural hearing loss, the intensity of the stimuli and the level of brain processing (i.e., early stages of processing versus later stages of processing) reflected by the particular ERP component and/or behavioral measure.

Results of the present study also show that for the vast majority of the hearing-impaired subjects, all cortical ERPs and their associated behavioral measures could be consistently recorded when the subjects wore their prescribed personal hearing aids. The use of amplification substantially increased the detectability of all the cortical ERPs and behavioral measures, as shown in Table 3. As would be expected, the largest improvements in detectability occurred for individuals in the severe-to-profound hearing-impaired group.

At the lower stimulus intensity, the use of personal hearing aids resulted in significant improvements in the amplitudes of all ERP components as well as significant improvements in behavioral auditory discrimination (d-prime) performance. The mean aided latencies of waves N1,
N2b and P3b and the *aided* reaction time latencies at 65 dB SPL were considerably shorter than the latencies measured in the unaided condition. At the higher stimulus intensity, the use of personal amplification only resulted in significant improvements in N1 amplitudes and d-prime discrimination performance. Thus, the maximum aided improvements in the cortical ERPs and their associated behavioral measures occurred at the lower stimulus intensity.

Collectively, these findings suggest that the hearing-impaired subjects’ use of hearing aids likely activated a larger pool of cortical neurons which contributed to the large response changes seen in the aided 65 dB SPL condition. The improved audibility of the speech signal at both stimulus intensities resulted in improved d-prime performance scores. There are at least two possible explanations for why the largest response changes occurred at 65 dB SPL: (1) the reduced audibility of the unaided speech signal for the moderate and severe-profoundly hearing-impaired individuals at the lower stimulus intensity, and (2) the 80 dB SPL signal may have activated the output limiting system of the hearing aid(s), especially for the non-linear instruments, thus creating a ceiling effect for the *aided* response changes seen at this stimulus intensity.

**Comparison of Aided ERP and Behavioral Measures to Those Obtained from Normal-Hearing Subjects**

The *aided* reaction times at both stimulus intensities and the N2b latencies at 80 dB SPL were significantly prolonged for the two hearing-impaired groups in comparison to those measures obtained from the normal-hearing subjects. There was also a clear trend for the *aided* latencies of waves N1, MMN and N2b to be prolonged for the moderately and severe-profoundly hearing-impaired groups in comparison to their normal-hearing peers at 65 dB SPL.
At 80 dB SPL, there was a trend for the *aided* d-prime sensitivity scores from the severe-profound group to be poorer than those obtained from the normal-hearing subjects. These prolongations in latency and poorer behavioral d-prime performance scores for the hearing-impaired subjects in the *aided* condition suggest that despite the improvements provided by personal hearing aids, the brain is not processing the speech signals with the same degree of accuracy and effectiveness as it is in individuals with normal-hearing sensitivity. This is especially true for subjects with severe-profound losses.

**Variability in the Amount of Response Change (Improvements) Seen for the Cortical ERPs and Associated Behavioral Measures in the Aided Condition**

Even though the majority of hearing-impaired subjects showed increased ERP amplitudes, decreased latencies and improved behavioral discrimination performance while wearing their hearing aids, the amount of response change was quite variable for both stimulus intensities. Some subjects showed quite large changes, while others showed minimal or no improvements.

The large intersubject variability that existed in these aided and unaided response measurements at both stimulus intensities is likely due, in part, to the varied degree of functional gain improvements and speech perception benefits that individuals with sensorineural hearing loss derive from their personal hearing aids, as shown for our subjects in Table 2. A second possible factor that might have contributed to this large variability is differences in the etiology of the hearing loss across subjects.

In the current study, when this opposite or worsening pattern occurred, it was most often seen for the MMN response. Recording the MMN in individual subjects can pose a number of challenges for the clinician including: (1) MMN is smaller in amplitude, generally < 2 : V, in
comparison to the majority of the other cortical ERPs; thus creating a poorer SNR for this response; (2) MMN is best visualized by calculating the difference waveform, which adds additional residual noise (1.4 X) into the response, thus degrading the SNR of the response; (3) a large number of stimulus trials are required to achieve an adequate SNR leading to long test sessions. This raises the question of the practicality of recording the MMN in a clinical setting with individual patients; (4) MMN is not always reliably elicited in either individual subjects with normal hearing sensitivity or those with varying degrees of sensorineural hearing loss (Kurtzberg et al., 1995; Lang et al., 1995; Oates et al., 2002); (5) MMN amplitude can vary substantially depending upon the level of alertness of the subject (Lang et al., 1995), and (6) the MMN is less reliably elicited in children compared to adults (Morr, Schafer, Kreuzer and Kurtzberg, 2002).

It is possible that several of these factors may have contributed to the somewhat poorer detectability of the MMN that occurred in the current study.

The Possible Use of Cortical ERPs and Behavioral Measures of Discrimination to Assess Hearing Aid Benefit

Cortical ERPs and their associated behavioral measures of speech discrimination provide a unique window into the speech perception benefits that hearing-impaired individuals derive from their personal hearing aids. These electrophysiologic and behavioral measures are closely linked to the perceptual processes, such as detection and discrimination, that underlie speech perception (for a review, see Stapells, 2002). As such, they can provide audiologist with several valuable pieces of information regarding the potential benefits that hearing aids provide at various levels of cognitive processing involved in auditory perception. This type of information is not available from the more traditional measures of hearing aid benefit including real-ear
and/or functional gain measurements, aided speech discrimination scores, and self-reported questionnaires.

For example, if certain response changes or improvements occur in wave N1, such as the presence or absence of the response in the aided versus unaided conditions respectively, these changes provide physiological evidence that the speech signal has arrived at the cortex and is potentially audible to the individual while wearing their hearing aids (see Näätänen and Picton, 1987, for a review). The reductions in latencies and increases in amplitudes for N1 in the aided versus unaided conditions, seen in the present study, suggest that the speech signal delivered by the hearing aid is arriving at the auditory cortex in a faster, more effective manner and that the aided signal is likely activating a larger pool of cortical neurons which are contributing to the response. The presence of N1 in the aided condition might also signal the arrival of potentially discriminable information to the auditory cortex (Martin and Boothroyd, 1999), however, its presence cannot be used to infer that the speech contrast was discriminable (Martin, Reference Note 3; Martin et al., 1997, 1999).

Response changes in the later cortical ERPs can provide audiologists with information regarding whether the brain can discriminate the acoustic differences between the speech stimuli better with or without a hearing aid. This information is indexed by the MMN on a pre-attentive level and by waves N2b, P3b and behavioral d-prime performance scores on an attentive or conscious level (see Stapells, 2002, for a review).

The pattern of findings that occur between the aided behavioral and cortical ERP results might also provide audiologists with valuable insight into possible speech perception difficulties a hearing-impaired listener may be experiencing while wearing their hearing aids. The pattern of test results might also assist audiologists and speech language pathologists in devising strategies
to optimize the auditory processing capabilities that hearing-impaired individuals are able to achieve with their hearing aids. For example, what is the significance of a pattern where a hearing-impaired individual’s aided RT latencies and d-prime sensitivity scores to speech stimuli are within the normal range but their cortical ERP amplitudes are reduced and/or ERP latencies are prolonged? One possible explanation might be that the ERP findings are a signal of an auditory processing problem that may only manifest itself in more difficult listening environments or with more complex stimuli.

A second pattern of findings could be absent behavioral responses to speech stimuli but present obligatory and pre-attentive cortical ERPs, such as N1 and MMN. This specific pattern of electrophysiologic findings provide evidence that the speech signal has been neurally coded at the level of the cortex and the brain is able to discriminate the acoustic changes present in the signal on a pre-attentive level. However, further auditory training and/or practice might be necessary for the brain to consciously make the discrimination between the /ba/ and /da/ stimuli as reflected in improved behavioral measures of discrimination and the presence of the later cortical responses (waves N2b and P3b).

Recent unpublished data indicate that obligatory cortical ERPs might also be useful in assessing the aided benefit that hearing-impaired infants and children derive from their personal hearing aids (Purdy et al., Reference Note 2). We have also been successful in recording cortical ERPs to speech stimuli in a small group (n=5) of school-aged children with sensorineural hearing losses while wearing their personal hearing aids (e.g., see case study in Stapells, 2002). Collectively, these preliminary findings suggest that cortical ERPs might be a useful tool for assessing hearing aid benefit in the younger hearing-impaired population and may be of
assistance to audiologists in initially fitting and adjusting these instruments in this clinical
population.

**Summary and Future Directions**

The results of this study have demonstrated that cortical ERPs and their related behavioral
measures may be used to assess the benefit that individuals with sensorineural hearing loss are
deriving from their prescribed personal hearing aid(s). The use of hearing aids generally resulted
in shorter latencies, larger amplitudes and better waveform morphologies of the cortical ERP
components and improved behavioral discrimination (d-prime) scores and RT latencies in the
aided condition. The amount of response change (improvement) seen for the individual ERP
peaks in the aided versus unaided conditions, however, was quite variable among individual
subjects at both stimulus intensities. This large variability in response measures may be due to
differences in the degree of functional gain benefit and improvement in speech intelligibility that
individual subjects received from their hearing aid(s) as well as differences in the etiology of the
hearing loss across subjects.

This study also demonstrated that even though the use of hearing aids resulted in
improved aided response measures for both the behavioral and electrophysiological indices of
speech perception, these hearing-impaired individuals process speech in a less effective manner
and with less accuracy while wearing their hearing aids in comparison to their normal hearing
counterparts. This finding was especially true at the lower stimulus intensity.

The results of the present study in conjunction with our previous study demonstrate that
the presence of these obligatory cortical responses (N1 and MMN) in either the aided or unaided
conditions provide an indication that the auditory cortex is not only responsive to the speech
stimuli but can also detect the acoustic changes present within these stimuli, even in the presence of severe-profound sensorineural impairment (Oates et al., 2002). The clinical feasibility of the MMN as a diagnostic tool for the hearing impaired, however, may be limited due to its lower detection rates in comparison to waves N1, N2b and P3b. Lastly, recording the later cortical ERPs (N2b and P3b) and their associated behavioral measures of discrimination (d-prime sensitivity scores and RT latencies) can provide audiologists with a quantitative measure of the impact that degree of sensorineural hearing impairment has on the brain’s ability to accurately and effectively process speech stimuli (Oates et al., 2002), as well a direct assessment of the speech-perception benefits that hearing-impaired individuals derive from their personal hearing aids.

Preliminary (unpublished) results in hearing-impaired infants and young children also suggest that cortical ERPs may hold promise in evaluating the speech perception benefits that this clinical population derive from their personal hearing aids. In the future, these responses may help audiologists fine tune the frequency, gain, and/or output characteristics of these instruments in difficult-to-test populations. Additional studies are needed to determine which cortical ERPs components (P1-N1-P2, MMN, N2b, or P3b) are most beneficial in assessing the speech detection and discrimination abilities of this younger hearing-impaired population. Secondly, further studies are needed to determine if cortical ERPs and their related behavioral measures may be used to monitor the ongoing improvements in auditory perception that these hearing-impaired children derive from audiologic-rehabilitation therapy. Lastly, because many elderly individuals experience difficulty with speech perception even with the use of hearing aids, future cortical ERP studies with this clinical population might shed some light on the possible interaction between hearing loss and underlying cognitive processes that may be altering the perception of the aided speech signal for these individuals.
FOOTNOTE

1 In our earlier noise-masking studies (Martin et al., 1999; 1997; Oates and Stapells, 1997), the data for each wave was re-analyzed in several ways: (1) leaving out results for those conditions judged as “no response”, (2) replacing the zero-microvolt values with estimates of the waveform’s residual noise levels, and (3) using a mean MMN amplitude measure that did not contain any zero-microvolt values. The results of these additional statistical analyses were consistent with those obtained when a zero-microvolt value was inserted for no response, indicating that this is a valid procedure.
ACKNOWLEDGEMENTS

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REFERENCES


REFERENCE NOTES


Table 1. Descriptive statistics on the pure-tone audiometric thresholds (in dB HL) for the normal-hearing subjects and the two hearing-loss groups.

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<th>Hearing</th>
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<th>2000 Hz</th>
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<td>Mean</td>
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<td><strong>Severe/Profound (N= 9)</strong></td>
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<td>Mean</td>
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<td>88.2</td>
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Table 2. Results of Electroacoustic Analyses and Real Ear Measurements Performed on All Hearing-Impaired Subjects’ Personal Hearing Aids

<table>
<thead>
<tr>
<th>Subject</th>
<th>PTA @ 1-2 kHz (dB HL)</th>
<th>Monaural/ Binaural HAAs</th>
<th>Type of HA Circuit (Linear/NonLinear)</th>
<th>HF Average Gain (dB SPL)</th>
<th>Maximum SSPL 90 (dB SPL)</th>
<th>Insertion Gain vs Target Gain*</th>
<th>Speech Intelligibility Index (SII)** at 65 dB SPL</th>
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<tr>
<td></td>
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<td>Unaided</td>
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<td>A</td>
<td>55</td>
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<td>Linear</td>
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<td>118</td>
<td>matched***</td>
<td>1%</td>
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<td>B</td>
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<td>Bin (RE)</td>
<td>Non Linear</td>
<td>58</td>
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<td>Mon (LE)</td>
<td>Linear</td>
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<td>131</td>
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<tr>
<td>D</td>
<td>80</td>
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<td>54</td>
<td>129</td>
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<td>E</td>
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<td>F</td>
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<td>134</td>
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<td>matched***</td>
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<td>K</td>
<td>75</td>
<td>Bin (RE)</td>
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<td>22</td>
<td>102</td>
<td>matched***</td>
<td>29%</td>
</tr>
<tr>
<td>L</td>
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<td>Bin (RE)</td>
<td>Linear</td>
<td>30</td>
<td>112</td>
<td>~ 10 dB low</td>
<td>0%</td>
</tr>
<tr>
<td>M</td>
<td>80</td>
<td>Bin (RE)</td>
<td>Non Linear</td>
<td>46</td>
<td>129</td>
<td>matched***</td>
<td>0%</td>
</tr>
<tr>
<td>N</td>
<td>55</td>
<td>Bin (RE)</td>
<td>Linear</td>
<td>30</td>
<td>117</td>
<td>matched***</td>
<td>11%</td>
</tr>
</tbody>
</table>

* Target Gain was assessed at 1-2k Hz using the NAL-RP formula
** Speech Intelligibility Index was calculated by Audioscan, Model RM-500
*** Matched = Achieved Insertion Gain was within 5 dB of NAL-RP Target Gain at 1-2 kHz
RE = Right Ear; LE = Left Ear
Table 3. Effects of average hearing sensitivity @ 1000-2000 Hz (i.e., 2 frequency pure-tone average, PTA²) and personal hearing aids on presence (in percent) of the behavioral and electrophysiological measures to the speech stimuli /ba/ and /da/.

<table>
<thead>
<tr>
<th>Cortical ERPs</th>
<th>PTA² = 0-24 dB (N = 10)</th>
<th>PTA² = 50 - 74 dB (N = 5)</th>
<th>PTA² = 75-120 dB (N = 9)</th>
</tr>
</thead>
<tbody>
<tr>
<td>N1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>65 dB Unaided</td>
<td>100.0</td>
<td>50.0</td>
<td>14.3</td>
</tr>
<tr>
<td>Aided</td>
<td>DNA</td>
<td>100.0</td>
<td>100.0</td>
</tr>
<tr>
<td>80 dB Unaided</td>
<td>100.0</td>
<td>100.0</td>
<td>11.1</td>
</tr>
<tr>
<td>Aided</td>
<td>DNA</td>
<td>100.0</td>
<td>100.0</td>
</tr>
<tr>
<td>MMN</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>65 dB Unaided</td>
<td>100.0</td>
<td>50.0</td>
<td>11.1</td>
</tr>
<tr>
<td>Aided</td>
<td>DNA</td>
<td>100.0</td>
<td>78.0</td>
</tr>
<tr>
<td>80 dB Unaided</td>
<td>100.0</td>
<td>100.0</td>
<td>11.1</td>
</tr>
<tr>
<td>Aided</td>
<td>DNA</td>
<td>100.0</td>
<td>87.5</td>
</tr>
<tr>
<td>N2b</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>65 dB Unaided</td>
<td>100.0</td>
<td>50.0</td>
<td>0.0</td>
</tr>
<tr>
<td>Aided</td>
<td>DNA</td>
<td>100.0</td>
<td>88.9</td>
</tr>
<tr>
<td>80 dB Unaided</td>
<td>100.0</td>
<td>100.0</td>
<td>33.3</td>
</tr>
<tr>
<td>Aided</td>
<td>DNA</td>
<td>100.0</td>
<td>100.0</td>
</tr>
<tr>
<td>P3b</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>65 dB Unaided</td>
<td>100.0</td>
<td>50.0</td>
<td>14.3</td>
</tr>
<tr>
<td>Aided</td>
<td>DNA</td>
<td>100.0</td>
<td>77.8</td>
</tr>
<tr>
<td>80 dB Unaided</td>
<td>100.0</td>
<td>100.0</td>
<td>11.1</td>
</tr>
<tr>
<td>Aided</td>
<td>DNA</td>
<td>100.0</td>
<td>100.0</td>
</tr>
<tr>
<td>BEHAVIORAL (d-prime &gt; 1)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>65 dB Unaided</td>
<td>100.0</td>
<td>50.0</td>
<td>0.0</td>
</tr>
<tr>
<td>Aided</td>
<td>DNA</td>
<td>100.0</td>
<td>88.9</td>
</tr>
<tr>
<td>80 dB Unaided</td>
<td>100.0</td>
<td>100.0</td>
<td>0.0</td>
</tr>
<tr>
<td>Aided</td>
<td>DNA</td>
<td>100.0</td>
<td>100.0</td>
</tr>
</tbody>
</table>

DNA = did not assess
Table 4. Mean and standard deviation values for the hearing-impaired subjects’ unaided and aided cortical ERP components and behavioral measures of discrimination to the /ba-da/ stimuli presented at 65 dB SPL (top) and 80 dB SPL (bottom).

<table>
<thead>
<tr>
<th>Name of Measure</th>
<th>Amplitudes (V)</th>
<th>Latencies (ms)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N1^1</td>
<td>MMN^2</td>
</tr>
<tr>
<td><strong>65 dB SPL</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Unaided</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>-1.11</td>
<td>-0.48</td>
</tr>
<tr>
<td>S.D.</td>
<td>2.01</td>
<td>1.09</td>
</tr>
<tr>
<td><strong>Aided</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>-3.79</td>
<td>-1.63</td>
</tr>
<tr>
<td>S.D.</td>
<td>2.99</td>
<td>1.94</td>
</tr>
<tr>
<td><strong>80 dB SPL</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Unaided</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>-1.84</td>
<td>-1.02</td>
</tr>
<tr>
<td>S.D.</td>
<td>2.59</td>
<td>1.76</td>
</tr>
<tr>
<td><strong>Aided</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>-4.26</td>
<td>-1.46</td>
</tr>
<tr>
<td>S.D.</td>
<td>3.11</td>
<td>1.59</td>
</tr>
</tbody>
</table>

1 N1 measured in standard waveform at Cz
2 MMN measured in difference waveform at Fz
3 N2b measured in difference waveform at Cz
4 P3b measured in deviant waveform at Pz
Table 5. Results of the 2-way ANOVAs on aided and unaided cortical ERP amplitudes, latencies and behavioral results for the moderate and severe-profound hearing-impaired groups at each stimulus intensity: 65 dB SPL (top) and 80 dB SPL (bottom).

<table>
<thead>
<tr>
<th>Name of Measure</th>
<th>Test Condition (Unaided vs Aided)</th>
<th>Speech Stimulus (ba vs da)</th>
<th>TC x SS&lt;sup&gt;1&lt;/sup&gt;</th>
<th>F</th>
<th>df</th>
<th>p</th>
<th>F</th>
<th>df</th>
<th>p</th>
<th>F</th>
<th>df</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Amplitudes</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>N1</td>
<td>16.08 1,11 .002*</td>
<td>.143 1,11 .713</td>
<td>.168 1,11 .689</td>
<td>N1</td>
<td>26.85 1,3 .014</td>
<td>5.97 1,3 .092</td>
<td>3.23 1,3 .170</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>MMN</td>
<td>14.72 1,13 .002*</td>
<td>.939 1,13 .350</td>
<td>.053 1,13 .821</td>
<td>MMN</td>
<td>.523 1,2 .545</td>
<td>5.88 1,2 .136</td>
<td>1.42 1,2 .356</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>N2b</td>
<td>14.73 1,11 .003*</td>
<td>.162 1,11 .695</td>
<td>.010 1,11 .922</td>
<td>N2b</td>
<td>30.97 1,2 .031</td>
<td>.244 1,2 .670</td>
<td>3.92 1,2 .186</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>P3b</td>
<td>21.22 1,11 .001*</td>
<td>.085 1,11 .777</td>
<td>1.26 1,11 .285</td>
<td>P3b</td>
<td>23.71 1,3 .017</td>
<td>.128 1,3 .744</td>
<td>5.94 1,3 .093</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>d-prime</td>
<td>24.98 1,11 .000*</td>
<td>.443 1,11 .519</td>
<td>1.21 1,11 .296</td>
<td>d-prime</td>
<td>20.22 1,3 .021</td>
<td>.054 1,3 .830</td>
<td>.193 1,3 .690</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>Latencies</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>N1</td>
<td>12.27 1,10 .006*</td>
<td>3.04 1,10 .112</td>
<td>.591 1,10 .460</td>
<td>N1</td>
<td>.132 1,4 .735</td>
<td>42.91 1,4 .003*</td>
<td>.380 1,4 .571</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>MMN</td>
<td>1.84 1,11 .191</td>
<td>.181 1,11 .679</td>
<td>.503 1,11 .493</td>
<td>MMN</td>
<td>.019 1,2 .903</td>
<td>2.09 1,2 .285</td>
<td>.128 1,2 .755</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>N2b</td>
<td>2.42 1,10 .151</td>
<td>1.97 1,10 .190</td>
<td>14.26 1,10 .004*</td>
<td>N2b</td>
<td>12.98 1,4 .023</td>
<td>6.30 1,4 .066</td>
<td>21.00 1,4 .010</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>P3b</td>
<td>8.20 1,10 .017</td>
<td>.496 1,10 .497</td>
<td>.019 1,10 .894</td>
<td>P3b</td>
<td>4.04 1,4 .115</td>
<td>1.45 1,4 .295</td>
<td>.226 1,4 .659</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>d-prime</td>
<td>25.88 1,12 .000*</td>
<td>4.61 1,12 .053</td>
<td>2.36 1,12 .150</td>
<td>d-prime</td>
<td>11.94 1,3 .041</td>
<td>3.56 1,3 .156</td>
<td>.929 1,3 .406</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* Indicates statistical significance (p < .01)

<sup>1</sup>TC x SS = Test Condition x Speech Stimulus
Table 6. Means and standard deviation values for ERP components and behavioral measures to the /ba-da/ stimuli presented at both stimulus intensities for the normal-hearing subjects and the aided responses for the moderate and severe-profoundly impaired groups

<table>
<thead>
<tr>
<th>Name of Measure</th>
<th>Amplitudes (V)</th>
<th>Latencies (ms)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N1</td>
<td>MMN</td>
</tr>
<tr>
<td><strong>65 dB SPL</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal-Hearing Subjects</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>-3.27</td>
<td>-2.58</td>
</tr>
<tr>
<td>SD</td>
<td>2.39</td>
<td>1.95</td>
</tr>
<tr>
<td>Aided Responses for Moderate HL</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SD</td>
<td>2.46</td>
<td>1.47</td>
</tr>
<tr>
<td>Aided Responses for Severe-Profound HL</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>-2.28</td>
<td>-1.20</td>
</tr>
<tr>
<td>SD</td>
<td>2.46</td>
<td>1.76</td>
</tr>
<tr>
<td><strong>80 dB SPL</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normal-Hearing Subjects</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>-3.19</td>
<td>-1.89</td>
</tr>
<tr>
<td>SD</td>
<td>1.66</td>
<td>1.41</td>
</tr>
<tr>
<td>Aided Responses for Moderate HL</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>-6.43</td>
<td>-2.06</td>
</tr>
<tr>
<td>SD</td>
<td>2.79</td>
<td>1.72</td>
</tr>
<tr>
<td>Aided Responses for Severe-Profound HL</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>-3.47</td>
<td>-1.16</td>
</tr>
<tr>
<td>SD</td>
<td>2.81</td>
<td>1.52</td>
</tr>
</tbody>
</table>

1 N1 measured in standard waveform at Cz; 2 MMN measured in difference waveform at Fz; 3 N2b measured in difference waveform at Cz, and 4 P3b measured in deviant waveform at Pz
Table 7. Results of 2-way ANOVAs on cortical ERP amplitudes, latencies and behavioral measures for the normal-hearing subjects and the *aided* responses for the moderately and severe-profoundly hearing-impaired groups at both stimulus intensities: 65 dB SPL (top); 80 dB SPL (bottom).

<table>
<thead>
<tr>
<th>Name of Measure</th>
<th>Subject Group</th>
<th>Speech Stimulus (ba vs da)</th>
<th>SG X SS²</th>
<th>Name of Measure</th>
<th>Subject Group</th>
<th>Speech Stimulus (ba vs da)</th>
<th>SG X SS²</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>F</td>
<td>df</td>
<td>p</td>
<td>F</td>
<td>df</td>
<td>p</td>
<td>F</td>
</tr>
<tr>
<td>65 dB SPL</td>
<td>N1</td>
<td>3.77</td>
<td>2,19</td>
<td>.042</td>
<td>.521</td>
<td>1,19</td>
<td>.479</td>
</tr>
<tr>
<td></td>
<td>MMN</td>
<td>2.76</td>
<td>2,2</td>
<td>.086</td>
<td>1.15</td>
<td>1,21</td>
<td>.295</td>
</tr>
<tr>
<td></td>
<td>N2b</td>
<td>1.28</td>
<td>2,21</td>
<td>.298</td>
<td>1.18</td>
<td>1,21</td>
<td>.290</td>
</tr>
<tr>
<td></td>
<td>P3b</td>
<td>1.45</td>
<td>2,19</td>
<td>.259</td>
<td>.134</td>
<td>1,19</td>
<td>.718</td>
</tr>
<tr>
<td>80 dB SPL</td>
<td>N1</td>
<td>3.08</td>
<td>2,19</td>
<td>.069</td>
<td>1.99</td>
<td>1,19</td>
<td>.174</td>
</tr>
<tr>
<td></td>
<td>MMN</td>
<td>.948</td>
<td>2,19</td>
<td>.405</td>
<td>.204</td>
<td>1,19</td>
<td>.656</td>
</tr>
<tr>
<td></td>
<td>P3b</td>
<td>1.12</td>
<td>2,18</td>
<td>.349</td>
<td>.041</td>
<td>1,18</td>
<td>.842</td>
</tr>
</tbody>
</table>

1 Three Subject Groups = Responses from Normal-Hearing Group and *Aided* Responses from Moderately- and Severe-Profoundly -Impaired Groups  
2 TC x SS = Test Condition x Speech Stimulus  
* Indicates Statistical Significance (p < .01)

FIGURE LEGENDS
Figure 1. Waveforms recorded from one subject to the /ba-da/ stimuli presented at 65 and 80 dB ppe SPL in the active condition and their associated behavioral measures (d-prime sensitivity scores and RT latencies). The top row of waveforms are in the unaided condition, the bottom row of waveforms are in the aided condition. Each waveform set contains the responses to stimuli presented as standards recorded at Cz (lighter lines) and responses to stimuli presented as deviants recorded at Pz (darker lines). Waves N1, N2b and P3b are labeled. In this and subsequent figures, positivity is represented as an upward deflection.

Figure 2. Waveforms recorded from the same hearing-impaired individual as shown in Figure 1 to the /ba-da/ stimuli presented at 65 and 80 dB ppe SPL in the passive condition. The top row of waveforms are in the unaided condition; the bottom row of waveforms are in the aided condition. Within each waveform set, the top row of waveforms are responses to stimuli presented as standards (lighter lines) and as deviants (darker lines). The bottom row of waveforms are difference responses calculated by subtracting the responses to stimuli presented as standards from the responses to the same stimuli presented as deviants. The data shown are recorded from an electrode located at Fz. MMN is labeled when present. The amplitude scale for all of these responses is ± 3.5 : V.

Figure 3. The grand-mean waveforms to the /ba-da/ stimuli in the active condition are displayed for the subjects with normal-hearing sensitivity (0-24 dB HL) and for the hearing-impaired subjects with moderate (50-74 dB HL) and severe-profound (75-120 dB HL) sensorineural impairments. The top half of the figure are the responses to the 65 dB ppe SPL stimuli; the bottom half of the figure are the responses to the 80 dB ppe SPL stimuli. Within the set of waveforms for
each intensity, the top row are responses in the unaided condition, the bottom row are the responses in the aided condition. Each set of waveforms contains responses to stimuli presented as standards recorded at Cz (lighter lines) and responses to stimuli presented as deviants recorded at Pz (darker lines). Waves N1, N2b and P3b are labeled.

Figure 4. The grand-mean difference waveforms to the /ba-da/ stimuli in the passive condition for subjects with normal-hearing sensitivity (0-24 dB HL) and for subjects with moderate (50-74 dB HL) and severe-profound (75-120 dB HL) sensorineural hearing impairments are displayed. The responses to the 65 dB ppe SPL stimuli are located in the top half of the figure, the responses to the 80 dB ppe SPL stimuli are located in the lower half of the figure. Each set of waveforms at each intensity contains the responses recorded in the unaided condition (top row) and the responses recorded in the aided condition (bottom row). The difference waveforms were calculated by subtracting the responses to stimuli presented as standards from the responses to those same stimuli presented as deviants. The difference waveforms were recorded from an electrode located at Fz, and MMN is labeled.

Figure 5. Each plot represents a comparison of each hearing-impaired subject’s unaided and aided responses to the 65 dB SPL behavioral and electrophysiologic measures. Measures representing response strength (d-prime scores and MMN - P3b amplitudes) are displayed in the top row and measures reflecting the effectiveness of processing (RT latencies and MMN - P3b latencies) are displayed in the bottom row. Each subject’s data are represented by a separate letter (a-n). Each plot contains the mean amplitude or latency value for that response in normal-hearing subjects, as shown by a dotted line. The amplitude and/or latency values that correspond to “no response” for each response measure are indicated by the hatched regions.
Figure 1

65 dB SPL

UNAIDED

Amplitude (µV)

-20
-10
0
10
20

Latency (ms)

0
200
400
600
800

N1

d' = 2.71
RT = 562 ms

P3b

N2b

d' = 3.18
RT = 405 ms

AIDED

Amplitude (µV)

-20
-10
0
10
20

Latency (ms)

0
200
400
600
800

Hearing-Impaired
Subject A
PTA2 = 55 dB HL

80 dB SPL

Amplitude (µV)

-20
-10
0
10
20

Latency (ms)

0
200
400
600
800

d' = 4.65
RT = 374 ms

d' = 4.65
RT = 349 ms

Standard @ Cz
Deviant @ Pz
FIGURE 2

65 dB SPL

UNAIDED

STD DEV

DIFF

MMN?

AIDED

STD DEV

DIFF

MMN

Latency (ms)

0 200 400

0 200 400

Hearing-Impaired
Subject A
PTA2 = 55 dB HL

---

Standard @ Fz

Deviant @ Fz