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Original Article

Auditory steady state responses recorded in multitalker babble

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Abstract

Objective: The primary purpose of this investigation was to determine the effect of multitalker babble on ASSRs in adult subjects with normal hearing (NH) and sensorineural hearing loss (HI). The secondary purpose was to investigate the relationships among ASSRs, word recognition in quiet, and word recognition in babble. *Design:* ASSRs were elicited by a complex mixed-modulation tonal stimulus (carrier frequencies of 500, 1500, 2500, and 4000 Hz; modulation rate of 40 or 90 Hz) presented in quiet and in babble. The level of each carrier frequency was adjusted to match the level of the multitalker babble spectrum, which was based on the long term speech spectrum average. Word recognition in noise (WIN) performance was measured and correlated to ASSR amplitude and ASSR detection rate. *Study Sample:* Nineteen normal-hearing adults and nineteen adults with sensorineural hearing loss were recruited. *Results and Conclusions:* The presence of babble significantly reduced the ASSR detection rate and ASSR amplitude for NH subjects, but had minimal effect on ASSRs for HI subjects. In addition, babble enhanced ASSR amplitude at high stimulus levels. ASSR detection rate and ASSR amplitude recorded in quiet and babble were significantly correlated with word recognition performance for NH and HI subjects.

Sumario

Objetivo: El objetivo fundamental de esta investigación fue determinar el efecto de balbuceo de hablantes múltiples en los ASSR de adultos jóvenes con audición normal (NH) y con pérdidas auditivas sensorineurales (HI). El objetivo secundario fue investigar las relaciones entre los ASSR, el reconocimiento de palabras en silencio y el reconocimiento de palabras con en medio de balbuceo. *Diseño:* Los ASSR fueron evocados por estímulo tonal de modulación mezclada compleja (frecuencias portadoras de 500, 1500, 2500 y 4000 Hz; tasa de modulación de 40 o 90 Hz) presentadas en silencio y con el balbuceo. Se ajustó el nivel de cada frecuencia portadora para emparejar el nivel del espectro del balbuceo de hablantes múltiples, el cual se basó en el promedio del espectro a largo plazo. Se midió el rendimiento para el reconocimiento de palabras en ruido (WIN) y se correlacionó con la amplitud de los ASSR y con la tasa de detección de los ASSR. *Muestra Del Estudio:* Se reclutaron diez y nueve adultos normoyentes y diez y nueve adultos con pérdida auditiva sensorineural. *Resultados Y Conclusiones:* La presencia del balbuceo reduce significativamente la tasa de detección de los ASSR y la amplitud de los ASSR en sujetos NH, pero tiene efectos mínimos en los ASSR de sujetos HI. Además, el balbuceo aumenta la amplitud de los ASSR con estímulos de niveles altos. La tasa de detección de los ASSR y la amplitud de los ASSR registrada en silencio y con balbuceo, fueron significativamente correlacionadas con el rendimiento para reconocer palabras en sujetos NH y HI.

Key Words: Auditory evoked potentials; Normal hearing; Hearing loss; Speech perception; Auditory steady-state responses

The presence of hearing loss interferes with the ability to understand speech and the effects can be disabling in the presence of background noise (Beattie, 1989; Carhart & Tillman, 1970; Divenyi & Haupt, 1997a, 1997b, 1997c; Dubno et al, 1984; Gordon-Salant, 1987; Hirsh, 1950; Olsen et al, 1975; Souza & Turner, 1994). Speech is characterized, in part, by dynamic changes in amplitude and frequency which the auditory system must resolve in order for accurate speech perception to occur. A number of important speech features (e.g. voice onset time and fundamental frequency) occur at high rates of amplitude and frequency modulation.

Hearing loss, in addition to reducing the audibility of speech signals, may interfere with the ability to process amplitude and frequency changes in the speech signal (Dimitrijevic et al, 2004; Grose et al, 1989; Harkrider et al, 2009; Horwitz et al, 2002; Yin et al, 2008). These changes in processing may occur at different levels in the auditory pathway (Eggermont, 1994; Krishna & Semple, 2000; Krishnan, 2002; Rhode, 1994; Steinhauer, 2003). It is difficult, however, to differentiate at which level(s) in the auditory system the reduction in processing occurs using behavioral measures. Behavioral measures are complicated further by the interaction of attention and cognition with audition (Fisher et al, 2000; Wingfield, 1996). In contrast, an electrophysiologic correlate of speech perception has the advantage of reducing the influence of non-auditory factors and presents the possibility of assessing the auditory system at both subcortical and cortical levels (Snell & Frisina, 2000; Tremblay et al, 2004).

The auditory steady state response (ASSR) is an auditory evoked potential (AEP) elicited by pure tones, which are modulated in amplitude and/or frequency and have been used typically as an objective estimate of behavioral pure-tone thresholds (e.g. John et al, 1998; Picton et al, 2003). The ASSR stimulus consists of a carrier

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Abbreviations

α	alpha
AEP	Auditory evoked potential
AM	Amplitude modulated
ASSR	Auditory steady-state response
FM	Frequency modulated
f_m	Modulation frequency
HI	Hearing impaired
MM	Mixed modulated
NH	Normal hearing
NU 6	Northwestern University Auditory Test No. 6
RMANOVA	Repeated measures analysis of variance
SNR	Signal-to-noise ratio
TPP	Tympanometric peak pressure
V_{ea}	Equivalent ear canal volume
WIN	Words in noise
WIQ	Words in quiet
Y_{TM}	Peak compensated static acoustic admittance

frequency which is modulated in amplitude (amplitude modulation or AM), frequency (frequency modulation or FM), or both (mixed modulation or MM). Single and multiple carrier frequencies presented either monotically or dichotically have been used to elicit the ASSR. Recently, ASSRs to complex tonal stimuli have shown significant correlations with measures of word recognition in adults and speech feature discrimination in infants (Cone & Garinis, 2009; Dimitrijevic et al, 2004, 2001; Leigh-Paffenroth & Fowler, 2006).

One advantage of the ASSR is that subcortical and cortical responses can be elicited by the same stimulus when delivered at different modulation rates, which allows potential differences in processing at subcortical and cortical levels to be evaluated. In general, high ASSR modulation frequencies (e.g. 90 Hz) evoke responses primarily from subcortical regions, and low ASSR modulation frequencies (e.g. 40 Hz) evoked responses primarily from subcortical and cortical regions of the auditory system (Herdman et al, 2002). Recording electrophysiologic responses at different levels of the auditory system is important particularly when presenting signals in noise. Subcortical and cortical levels of the auditory system may differ in their response to signals presented in the presence of noise. In a series of articles Fowler and Mikami (1992a, 1992b, 1995, & 1996) measured auditory evoked potential masking level differences (AEP-MLD) with wave V of the auditory brainstem response (ABR), Pa of the middle latency response (MLR), and P2 of the cortical evoked response. The AEP-MLD paradigm measured the effects of stimulus phase for tones in noise similar to classic psychoacoustic masking level difference paradigms (Hirsh, 1948). No MLD was recorded for the ABR (Fowler & Mikami, 1995) or for the MLR, but an AEP-MLD was recorded for P2 (Fowler & Mikami, 1992a, 1992b, 1996). Similarly, Weihing and Musiek (2008) measured auditory-evoked responses recorded in noise from brainstem and thalamocortical areas. Monaural and binaural clicks presented in white noise revealed binaural enhancements at all masking levels for the MLR, but only at low to mid levels of the ABR. Binaural interactions were present in both ABR and MLR, but were significantly reduced in the MLR at higher masking levels. These data suggest the possibility that subcortical and cortical neural generators respond differently in the presence of noise and that this effect may be dependent on stimulus level.

A second advantage is that the ASSR stimulus can be constructed to approximate the temporal and spectral characteristics of speech. Temporal characteristics of speech include fluctuations in amplitude and frequency, which are similar to the amplitude and frequency modulations in the MM ASSR stimulus. The predominant spectral content of speech includes energy from approximately 300 to 5000 Hz, which can be approximated by the choice of carrier frequencies. A third advantage of the ASSR is that it reflects neural phase-locking to the steady-state stimulus and may reflect (or be related to) auditory temporal processing (Grose et al, 2009). The use of multitalker babble in the recording of ASSRs provides a realistic competing message that is encountered frequently by individuals in common listening situations. An objective electrophysiologic correlate of speech recognition may prove useful in the evaluation of individuals who are unable to provide reliable behavioral responses (e.g. patients with cognitive deficits).

The primary purpose of this investigation, therefore, was to assess ASSRs in quiet and in the presence of multitalker babble in NH and HI subjects. The secondary purpose was to determine the extent to which ASSRs were related to word recognition performance. Specifically, the experiment addressed the following three questions: (1) Does the presence of multitalker babble influence the detectability or amplitude of speech-spectrum ASSRs? (2) Does modulation rate influence the detectability or amplitude of speech-spectrum ASSRs? and (3) Is the detectability or amplitude of speech-spectrum ASSRs correlated with performance on a word recognition-in-noise task?

Materials and Methods

Subjects

The present study was approved by the local Institutional Review Board and all participants signed an informed consent document prior to their participation in the study. Normal-hearing adults (NH) and adults with sensorineural hearing loss (HI) were recruited. The normal-hearing participants ($n = 19$, from 20 to 29 years, mean age 23 years, seven males) had air- and bone-conduction pure-tone thresholds ≤25 dB HL for the octave frequencies 250–8000 Hz. The participants with sensorineural hearing loss ($n = 19$, from 48 to 78 years, mean age 66 years, 19 males) had air- and bone-conduction thresholds restricted to a pure-tone average (500, 1000, and 2000 Hz) of ≤50 dB HL, with thresholds ≤70 dB HL at 2000 Hz and ≤100 dB HL from 3000–8000 Hz (see Figure 1 for the mean audiograms of each group). Eight participants in the hearing loss group were experienced hearing-aid users, ten were new hearing aid users, and one did not wear hearing aids. Tympanometric measures—peak compensated static acoustic admittance (Y_{tm}), equivalent ear canal volume (V_{ea}), and tympanometric peak pressure (TPP)—were within normal limits (Roup et al, 1998; Wiley et al, 1996) for all participants. Participants with conductive hearing loss, air-bone gaps >10 dB, history or presence of otologic pathology, history of possible retrocochlear pathology, history of stroke, seizure disorder, or dementia were excluded from the study. The presence of retrocochlear pathology in subjects with asymmetrical hearing loss was unlikely as previous clinical reports indicated a negative auditory brainstem response test or a positive history of significant noise exposure that was consistent with the asymmetry in pure-tone thresholds.

Speech-spectrum ASSRs

ASSRs were elicited by two complex signals each constructed of 100% amplitude modulated (AM) and 20% frequency modulated (FM) pure tones with carrier frequencies of 500, 1500, 2500, and 4000 Hz. The first stimulus was modulated at rates of less than 65 Hz and the second

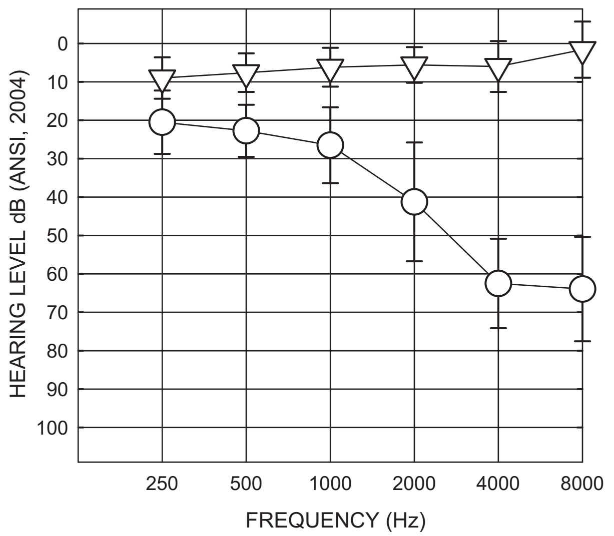

Figure 1. Mean hearing thresholds (dB HL) and standard deviations for NH subjects (n = 19, inverted open triangles) and for HI subjects (n = 19, open circles). Error bars represent one standard deviation.

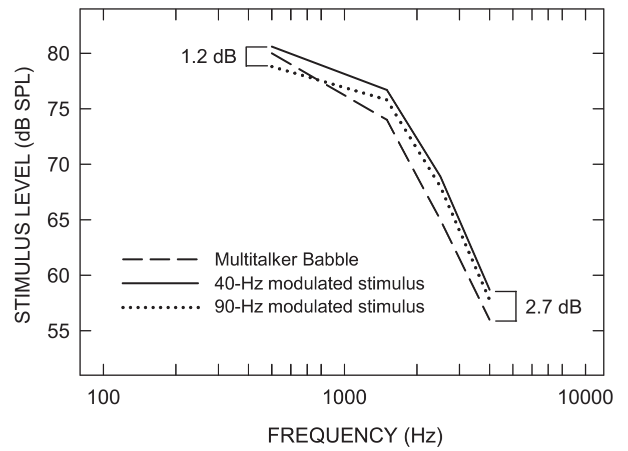

Figure 2. Stimulus spectrum for the 40-Hz ASSR stimulus (solid line), the 90-Hz ASSR stimulus (dotted line), and the multitalker babble (dashed line) showing equivalent stimulus level. The differences among the stimuli and babble were ≤2.7 dB as noted in the figure.

stimulus was modulated at rates above 75 Hz (see Table 1). The stimulus with modulation rates below 65 Hz will be referred to as the 40 Hz stimulus, and the stimulus with modulation rates above 75 Hz will be referred to as the 90 Hz stimulus. The ASSR stimuli were constructed and generated using Intelligent Hearing Systems' (IHS) SmartEP-ASSR System (version 2.21). The SmartEP ASSR Stimgen module was used to adjust the level of each carrier frequency to match the level of the multitalker babble spectrum, which was based on the long-term speech spectrum average (see Figure 2) (French & Steinberg, 1947). Stimulus level was calibrated acoustically with a Bruel & Kjær 2250 sound level meter using an ER3-A insert earphone and a Bruel & Kjær 2 cm^3 coupler (DB-0138) attached to a Bruel & Kjær 4152 artificial ear. The 1/3-octave band levels of the individual carrier frequencies were 80 dB SPL at 500 Hz, 76 dB SPL at 1500 Hz, 69 dB SPL at 2500 Hz, and 59 dB SPL at 4000 Hz (Figure 2). The combination of the individual carrier frequencies resulted in a complex signal with an overall SPL of 76 dB. The ASSR stimuli were calibrated acoustically before data collection, on a weekly basis during data collection, and after data collection.

The ASSR stimuli were presented in quiet and in the presence of multitalker babble. The multitalker babble was constructed from six speakers (three female and three male) talking about different topics and produced by Donald Causey in 1979 at the Biocommunications Laboratory at the University of Maryland (Sperry et al, 1997). The multitalker babble was reproduced on a compact disc (CD) and presented via an audiometer (Grason Stadler, Model 61) and an ER-3A insert earphone. The ASSR stimulus and the multitalker babble were combined acoustically in a custom double-barreled foam ear tip for simultaneous monaural presentation. The presentation level of the single complex ASSR stimulus was presented from 60 to 76 dB SPL in 4-dB steps and the multitalker babble was presented continuously at 60 dB SPL resulting in five signal-to-noise ratios (SNRs) from 0 to 16 dB in 4-dB increments.

Table 1. Speech-spectrum ASSR stimulus modulation rates for each carrier frequency for the 40-Hz and 90-Hz stimuli used in the experiment.

Carrier Frequency (Hz)	*~40 Hz Stimulus (Hz)*	*~90 Hz Stimulus (Hz)*
500	37.1	79.1
1500	44.9	86.9
2500	52.7	94.7
4000	60.5	102.5

ASSRs were recorded with the listener seated comfortably in a reclining chair in a double-walled sound booth. In order to optimize the signal-to-noise ratio of the responses, the 40-Hz ASSRs were recorded with the listener quietly reading to encourage wakefulness and the 90-Hz ASSRs were recorded in an environment conducive to sleep (Levi et al, 1993). During the recording of 40-Hz ASSRs, reading material was propped on the listener's lap and subjects were encouraged to turn pages with minimal movement. The EEG noise was monitored by the examiner during all ASSR recordings. The potentials were recorded from Ag/AgCl electrodes attached to the high-forehead midline (F_z; non-inverting), mastoid of the test ear (M_1 or M_2; inverting), and low-forehead midline (F_{pz}; ground). The responses were amplified (100k) and bandpass filtered (30–300 Hz). Sweep duration was 1.024 seconds and data were collected in blocks of 20 sweeps. Each block was averaged with any previous block(s) and the maximum number of blocks per stimulus level was limited to 20 (maximum of ~7 minutes averaging time). ASSRs were analysed in the frequency domain by FFT of the average time-domain waveforms. The presence of an ASSR was determined automatically by the SmartEP-ASSR detection algorithm which required that all of the following criteria were met: (1) SNR ≥6.13 dB at the modulation frequency (f_m), (2) SNR ≥6.13 dB at the side bins (f_m ±4.88 Hz), (3) absolute amplitude of the response at the f_m >12.5 nV, and (4) noise level <50 nV at the f_m ±4.88 Hz. Averaging was automatically stopped when $F_{(2,10)}$ reached $p \leq 0.05$ for five frequency bins (4.88 Hz) above and below the f_m; otherwise recording continued until the maximum number of sweeps (400) was reached.

Note that the the IHS algorithm includes statistical analysis at f_m and the surrounding spectral bins. The software uses a split-sweep technique where two buffers determine the level of the signal and the level of the noise in each recording. This is different from other manufacturers' software, which calculates an SNR ratio relative to the f_m only.

Word recognition

The words-in-noise (WIN) test provides a measure of word recognition in multitalker babble across a range of SNRs (Wilson, 2003; Wilson & McArdle, 2005, 2007). The WIN protocol includes two 25-item lists of Northwestern University List number 6 (NU6) words (Tillman & Carhart, 1966) presented in quiet at 60- and 84-dB HL, and two 35-item lists of NU6 words presented in multitalker babble. The babble level was fixed at 80 dB SPL and the level of the words varied from 104 to 80 dB SPL in 4-dB decrements (i.e. seven SNRs from 24 dB to 0 dB). The materials were reproduced by CD and presented monaurally via an audiometer (Grason Stadler, Model 61) and ER-3A insert earphone. Word recognition testing was conducted for the same ear that was used for the ASSRs. Testing was conducted in a double-walled sound booth with the verbal responses of the listener scored by the examiner.

The experimental procedures for each subject were conducted in two sessions within a four-week period with each session lasting approximately two hours. Session 1 included the audiologic evaluation, the WIN test, and one of the ASSR stimuli (40 Hz or 90 Hz) in both conditions (quiet and babble). Session 2 included the remaining ASSR stimulus in both conditions. The session in which the 90-Hz and 40-Hz ASSR stimuli were presented was counterbalanced across subjects. All stimuli were presented monaurally to the right ear ($n = 34$), or to the left ear in cases where pure-tone thresholds in the right ear did not meet the audiometric inclusion criteria ($n = 4$).

Data analysis

Speech-spectrum ASSRs were quantified by response detection and response amplitude. Separate repeated measures analysis of variance (RMANOVA) were performed for each group (NH and HI) to examine the effects of condition (quiet versus babble), carrier frequency, and modulation rate on the number of ASSRs detected. The number of ASSRs detected was summed across the five stimulus levels prior to analysis. Similarly, separate RMANOVAs were performed for each group to assess the effects of condition, carrier frequency, modulation rate, and stimulus level on ASSR amplitude. Post-hoc analyses (planned pairwise comparisons with Bonferroni adjustments for multiple comparisons) were performed for each significant main effect and interaction.

Word recognition performance was quantified by: (1) percent correct in quiet at each stimulus level, and (2) the SNR that corresponded to the 50% correct point on the performance-SNR function. The 50% correct point was calculated using the Spearman-Kärber equation (Finney, 1952) and was based on 10 words per SNR for a total of 70 words. The potential relationship between speech-spectrum ASSRs and word recognition performance was investigated using Pearson's *r*. The percent correct performance for words presented in quiet was compared to ASSRs recorded in quiet and the SNR that corresponded to the 50% correct point from the WIN test was compared to ASSRs recorded in babble. The following two sets of ASSR measures were used to test the relationships: (1) the number of ASSRs detected and (2) the ASSR amplitude; correlation analyses were conducted separately for each dependent variable (detection and amplitude) and for each group (NH and HI). For each group, the number of ASSRs detected was combined across presentation level for each carrier frequency and for each condition (quiet and babble) to form four composite variables. Similarly, the ASSR amplitude data were averaged across presentation level for each carrier frequency and for each condition to form four composite variables for each group. The formation of the composite variables was based on Cronbach's alpha, which revealed reliability estimates ≥ 0.80 for all measures (estimates > 0.70 are considered acceptable for the creation of composite variables) (Field, 2009). The reliability estimates mean that the ASSR amplitude across presentation level and condition was consistent so the composite variable is a reliable representation of the single-variable ASSR amplitudes. Specifically, the following composite variables were established for each group for detection and amplitude and were used for the correlational analyses: (1) 40-Hz ASSR recorded in quiet, (2) 40-Hz ASSR recorded in babble, (3) 90-Hz ASSR recorded in quiet, and (4) 90-Hz ASSR recorded in babble. All p-values were set at $\leq .05$.

Results

ASSR detection

The number of ASSRs detected at each carrier frequency is shown in Figure 3. The data for the NH group is illustrated in the left panel and the data for the HI group is shown in the right panel. The bars represent the total number of ASSRs detected at each carrier frequency for each modulation rate (40 Hz versus 90 Hz) and each condition

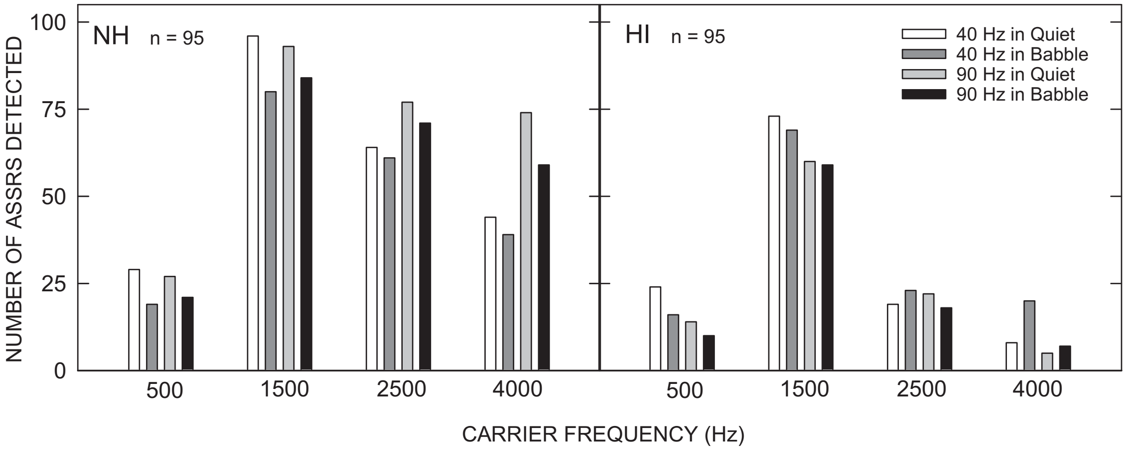

Figure 3. The number of ASSRs detected by carrier frequency is illustrated for each subject group. The bars represent the 40-Hz ASSRs recorded in quiet (white), the 40-Hz ASSRs recorded in babble (dark grey), the 90-Hz ASSRs recorded in quiet (light grey), and the 90-Hz ASSRs recorded in babble (black). The 'n' refers to the total number of stimuli presented for each condition and modulation rate.

(quiet versus babble). The total possible number of detected ASSRs for each combination of modulation rate and condition was 95 (5 presentation levels × 19 subjects). Overall, the greatest number of ASSRs was detected at 1500-Hz and a greater number of ASSRs was detected in quiet than in babble except for the HI group at 4000-Hz where the number of 40-Hz ASSRs detected in babble was greater than the number detected in quiet.

Normal hearing

A RMANOVA was performed for the NH data to determine the effects of condition (quiet versus babble), modulation rate, and carrier frequency on the number of ASSRs detected. The main effects of condition [$F(1,20) = 9.3$, $p = .006$], modulation rate [$F(1,20) = 5.2$, $p = .034$], and carrier frequency [$F(3,60) = 66.2$, $p < .001$] were significant. A greater number of ASSRs were detected in quiet than in babble and a greater number of 90-Hz ASSRs were detected than 40-Hz ASSRs. For carrier frequency, post-hoc comparisons showed a greater number of ASSRs was detected for the 1500-Hz carrier frequency than all other carrier frequencies. One three-way interaction (carrier frequency × modulation rate × condition) was significant [$F(3,60) = 2.8$, $p = .047$] and post-hoc analysis revealed the following significant comparisons: (1) a greater number of 40-Hz ASSRs was detected in quiet than in noise at 1500 Hz, (2) a greater number of 90-Hz ASSRs was detected in quiet than in noise at 4000 Hz, and (3) a greater number of 90-Hz ASSRs was detected than 40-Hz ASSRs in quiet at 4000 Hz.

Hearing impaired

A RMANOVA was performed for the HI data to determine the effects of condition, modulation rate, and carrier frequency on the number of ASSRs detected. The main effects of modulation rate [$F(1,20) = 134.2$, $p < .001$] and carrier frequency [$F(3,60) = 18.7$, $p < .001$] were significant. The main effect of condition was not significant, indicating no difference in the number of ASSRs detected in quiet compared to the number of ASSRs detected in babble. A greater number of ASSRs was detected for the 40-Hz stimulus than the 90-Hz stimulus. For carrier frequency, post-hoc analyses revealed that a greater number of ASSRs was detected for the 1500-Hz ASSR than all other carrier frequencies. One two-way interaction (carrier frequency × modulation rate) was significant [$F(3,60) = 9.2$, $p < .001$], and post-hoc analysis revealed the following significant comparisons: (1) a greater number of 40-Hz ASSRs was detected at 2500 Hz than at 500 Hz or 4000 Hz, (2) a greater number of 90-Hz ASSRs was detected at 1500 Hz than at 500, 2500, or 4000 Hz, and (3) a greater number of 40-Hz ASSRS were detected than 90-Hz ASSRs at 500 and 4000 Hz.

In summary, the greatest number of ASSRs was detected at 1500 Hz, regardless of modulation rate or subject group. A significantly greater number of ASSRs was detected in quiet than in noise for the NH group. In the HI group, however, no significant difference was found between the number of ASSRs detected in quiet and in babble. A greater number of ASSRs was detected for the 90-Hz stimulus than the 40-Hz stimulus for the NH group. In contrast, a greater number of ASSRs was detected at 40 Hz than at 90 Hz for the HI group.

ASSR amplitude

ASSR amplitude is plotted as a function of carrier frequency in Figure 4. The panels represent the data obtained in quiet and babble

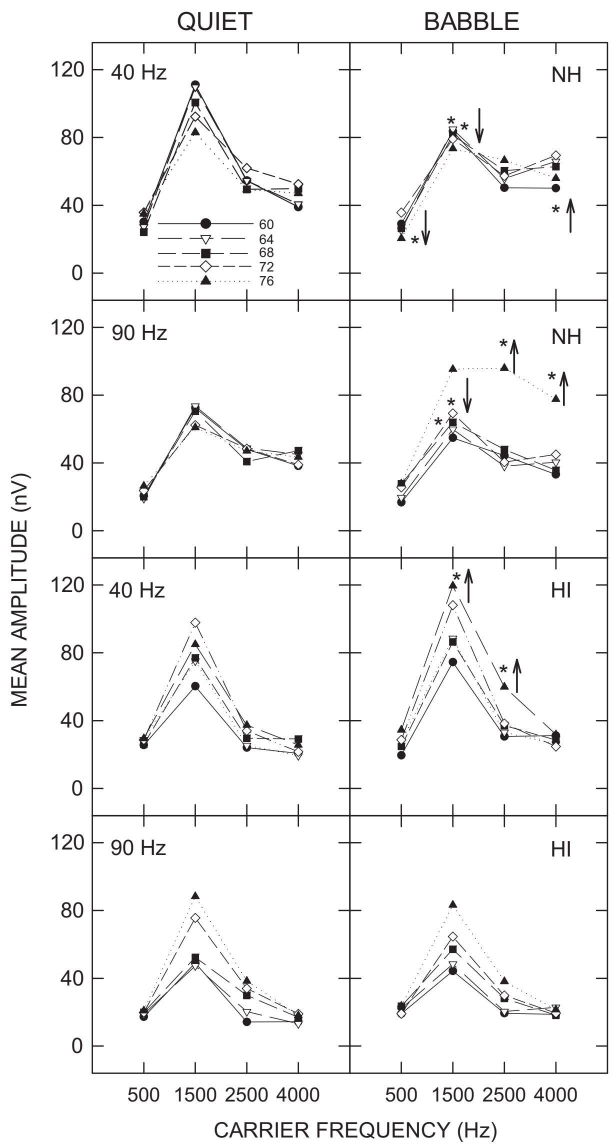

Figure 4. Mean ASSR amplitude is plotted as a function of carrier frequency and presentation level for each subject group. The left panels represent ASSRs recorded in quiet, and the right panels show mean amplitude for the ASSRs recorded in babble. The four upper graphs show data from the NH subjects and the four lower graphs show data from the HI subjects. The first and third rows represent the 40-Hz ASSR mean amplitude, and the second and fourth rows represent the 90-Hz ASSR mean amplitude. Asterisks denote significant differences in ASSR amplitude between quiet and babble conditions. The down-arrow symbols indicate a significant decrease in ASSR amplitude between quiet and babble conditions, and the up-arrow symbols indicate a significant enhancement in ASSR amplitude between quiet and babble conditions. The symbols in the legend represent stimulus level in dB SPL.

and the rows represent modulation rate (40 or 90 Hz). The data obtained from the NH group and the HI group are plotted separately. Overall, the largest amplitude was obtained at a carrier frequency of 1500 Hz and the smallest amplitudes were observed at 500- and 4000 Hz. Similarly, an increase in amplitude as a function of stimulus level was evident at 1500 Hz in the HI subjects but was less pronounced at the other carrier frequencies and in the NH subjects at all carrier frequencies. The asterisks with a down-arrow symbol indicate a significant decrease in ASSR amplitude in the babble condition compared to the corresponding quiet condition, and the asterisks with an up-arrow symbol indicate a significant increase in ASSR amplitude in the babble condition compared to the quiet condition (details are provided in the description of the statistical analyses below).

Normal hearing

A RMANOVA was performed to determine the effect of modulation rate, condition, carrier frequency, and stimulus level on ASSR amplitude for the NH group. The main effect of carrier frequency [$F(3,60) = 43.7$, $p < .001$] was significant, and pairwise comparisons revealed that the 1500-Hz amplitude was the largest and the 500-Hz amplitude was the smallest. In addition to the main effect, several two- and three-way interactions were significant. The interaction of modulation rate × stimulus level [$F(4,80) = 6.8$, $p = .003$] revealed that the 40-Hz ASSR was larger than the 90-Hz ASSR at all stimulus levels, except at 76 dB SPL. The carrier frequency × condition interaction [$F(3,60) = 4.7$, $p = .013$] revealed that (1) the 1500-Hz ASSR amplitude in quiet was larger than the 1500-Hz ASSR amplitude in babble, (2) the 1500-Hz ASSR amplitude was larger than all other carrier frequencies in quiet and in babble, and (3) the 2500- and 4000-Hz ASSR amplitudes were larger than the 500-Hz ASSR amplitude in quiet and in babble. The stimulus level × condition interaction [$F(4,80) = 5.3$, $p = .013$] showed that the ASSR amplitude for the quiet condition was larger than the ASSR amplitude for the babble condition at the lowest stimulus level (60 dB SPL); however, at the highest stimulus level (76 dB SPL) the amplitude for the babble condition was larger than the amplitude for the quiet condition.

Post-hoc tests for the three-way interaction of condition × carrier frequency × stimulus level [$F(12,240) = 2.6$, $p = .038$] revealed the following significant comparisons: (1) 1500-Hz ASSR amplitude was larger than the 500-, 2500-, and 4000-Hz ASSR amplitudes at each combination of condition and stimulus level, (2) the 500-Hz ASSR amplitude was smaller than the 1500-, 2500-, and 4000-Hz ASSR amplitudes at each combination of condition and stimulus level, (3) the 500-Hz ASSR amplitude was significantly smaller in babble than in quiet at 76 dB SPL (see asterisks with a down-arrow symbol in upper right panel of Figure 4), (4) the 1500-Hz ASSR amplitude was significantly smaller in babble than in quiet at 60 and 64 dB SPL (see asterisks with a down-arrow symbol in the two upper right panels of Figure 4), and (5) the 2500-Hz and 4000-Hz ASSR amplitudes were significantly larger in babble than in quiet at 76 dB SPL (see asterisks with an up-arrow symbol in the right panel of the top two rows of Figure 4). Post-hoc tests for the three-way interaction of condition × modulation rate × stimulus level [$F(4,80) = 4.9$, $p = .011$] revealed the following significant comparisons: (1) the amplitude of the 40-Hz ASSRs was larger than the amplitude of the 90-Hz ASSRs in babble at 64 and 68 dB SPL and in quiet at 72 dB SPL, and (2) the amplitude of the 90-Hz ASSRs recorded in babble was larger than the amplitude of the 90-Hz ASSRs recorded in quiet at 76 dB SPL (see asterisks with an up-arrow symbol in the right panel of the second row of Figure 4).

Hearing impaired

A repeated measures analysis of variance (RMANOVA) was performed to determine the effect of modulation rate, condition, carrier frequency, and stimulus level on ASSR amplitude for the HI group. The main effects of modulation rate [$F(1,20) = 11.4$, $p = .003$], carrier frequency [$F(3,60) = 24.8$, $p < .001$], and stimulus level [$F(4,80) = 12.3$, $p < .001$] were significant. The main effect of condition was not significant indicating no difference in ASSR amplitude in quiet compared to ASSR amplitude in babble. For modulation rate, the 40-Hz ASSR was larger in amplitude than the 90-Hz ASSR. For carrier frequency, post-hoc analyses revealed that the amplitude at 1500 Hz was significantly larger than the amplitude at the other carrier frequencies, and that there were no significant differences in amplitude among the 500, 2500, and 4000 Hz carrier frequencies. For stimulus level, post-hoc analyses revealed that the amplitude significantly increased from 60 dB SPL to ≥68 dB SPL, from 64 to 76 dB SPL, and from 68 to 76 dB SPL. The two-way interaction of carrier frequency × stimulus level was significant [$F(12,240) = 6.6$, $p = .001$] and revealed the following significant comparisons: (1) the amplitude of the 1500-Hz ASSR was larger than the amplitude of the 500-, 2500-, and 4000-Hz ASSRs at each stimulus level, and (2) there was a significant increase in ASSR amplitude from 60 to 76 dB SPL at 500 and 1500 Hz.

In summary, the main effect of carrier frequency was driven by the relatively large amplitude of the 1500-Hz ASSR in both subject groups at both modulation rates and across most presentation levels. The effect of adding babble to the ASSR stimulus produced either a reduction or an enhancement in the amplitude of the response. For the NH group, the babble produced a significant reduction in amplitude at the 500 and 1500 Hz for the 40 Hz ASSR, and at 1500 Hz for the 90 Hz ASSR; there was a significant enhancement in amplitude at the highest presentation level (76 dB SPL) at 4000 Hz for the 40 Hz ASSR, and at 2500 and 4000 Hz for the 90 Hz ASSR. In the HI group, there was no significant reduction in ASSR amplitude for responses recorded in babble, however, significant amplitude enhancement was observed at 1500 and 2500 Hz at 76 dB SPL for the 40-Hz ASSR.

Word recognition

Word recognition performance for the two subject groups is summarized in Table 2. Words-in-quiet (WIQ) performance for the NH group ranged from 92 to 100% at 60 dB HL, and from 88 to 100% at 84 dB HL. The scores for the HI group ranged from 20 to 100% at 60 dB HL and from 24 to 100% at 84 dB HL. The percent correct scores for the word-recognition-in-noise (WIN) test ranged from 70 to 87% for the NH group and from 6 to 70% for the HI group. The SNR that corresponded to the mean 50%-correct point on the WIN function ranged from 1.6 to 6.4 dB for the NH group and eighteen of the nineteen NH subjects had SNRs that were <6.0 dB. For the HI group, the SNR that corresponded to the mean 50%-correct point on the WIN function ranged from 6.4 to 24.4 dB.

Table 2. Mean and standard deviation word recognition performance for quiet and noise conditions for normal-hearing and hearing-impaired listeners.

	WIQ		*WIN*	
Group	*60 dB HL (% correct)*	*84 dB HL (% correct)*	*(% correct)*	*(SNR required for 50%-point)*
NH	99 (2)	98 (3)	80 (5)	3.7 (1.4)
HI	70 (23)	87 (17)	47 (17)	12.9 (4.7)

Note: WIQ = word recognition in quiet test, WIN = word recognition in noise test, NH = normal-hearing group, HI = hearing-impaired group.

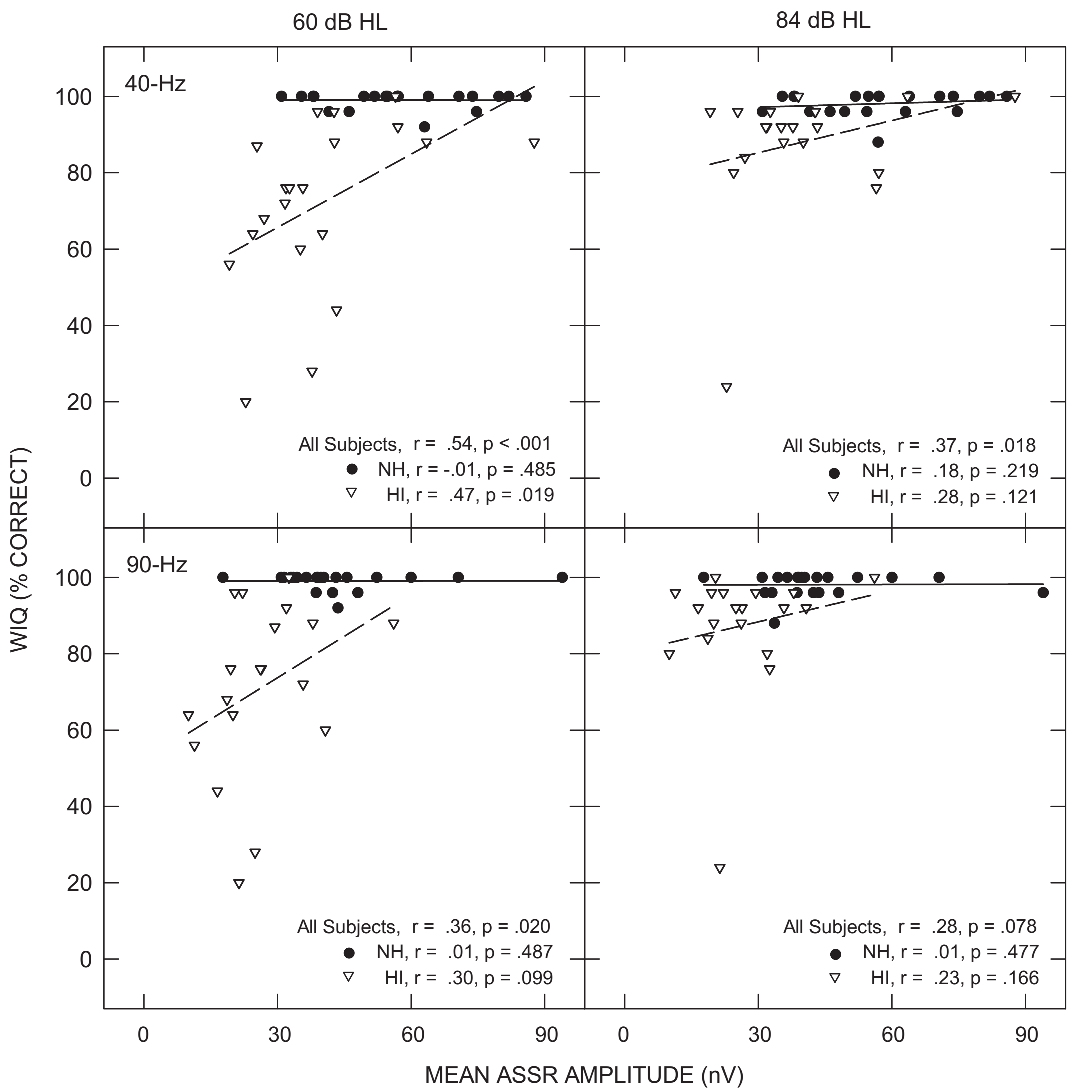

Figure 5. Scatter plot of word recognition in quiet performance (% correct) and mean ASSR amplitude (nV) for individual NH subjects (n = 19, open circles) and HI subjects (n = 19, inverted open triangles). The left panels show data from word recognition performance at 60 dB HL and the right panels show data from word recognition performance at 84 dB HL. The 40-Hz ASSR data is plotted in the upper graphs and the 90-Hz ASSR data is plotted in the lower graphs.

Speech-spectrum ASSRs and word recognition

ASSRs and WIQ

WIQ performance is plotted as a function of ASSR amplitude in Figure 5. WIQ performance at 60 dB HL and at 84 dB HL is plotted separately for the 40- and 90-Hz ASSR amplitudes. The parameter within each panel is subject group. Regression lines for each subject group are plotted for each bivariate set of variables and the resulting Pearson's r values are shown in the lower-right portion of each panel. Overall, significant correlations were found between WIQ performance at 60 dB HL and ASSR amplitude at each modulation rate (40 and 90 Hz) as well as between WIQ scores at 84 dB HL and ASSR amplitude at 40 Hz. The amplitude of 40-Hz ASSRs recorded in quiet was significantly correlated with the WIQ performance at 60 dB HL for the HI group ($r = .47$, p = .019) as seen in the upper left graph in Figure 5. As ASSR amplitude increased, WIQ performance improved up to approximately 45 nV; WIQ performance reached a plateau for ASSR amplitudes >45 nV (Figure 5). The best-fit line shows a linear relationship, which accounts for 22% of the variability in the WIQ performance. No significant correlations were identified between: (1) ASSR amplitude and WIQ at 60 dB HL for the NH group, (2) ASSR amplitude and WIQ performance at 84 dB HL for either subject group, or (3) the number of ASSRs detected in quiet and WIQ performance at 60 dB HL or 84 dB HL for either subject group.

ASSRs and WIN

The number of ASSRs detected as a function of WIN scores are presented in bivariate plots for each ASSR modulation rate (40- and 90 Hz) and each subject group (NH and HI) in Figure 6. The number of 40- and 90-Hz ASSRs detected is plotted separately for the NH subjects (closed circles) and the HI subjects (inverted open triangles). Regression lines for each subject group are plotted for each bivariate set of variables. Overall, significant correlations were found between WIN scores and the number of ASSRs detected at each modulation rate (40 and 90 Hz). The number of 40-Hz ASSRs detected in babble was significantly correlated to WIN SNRs for the NH group ($r = -.38$, p = .044), as seen in Figure 6. As the

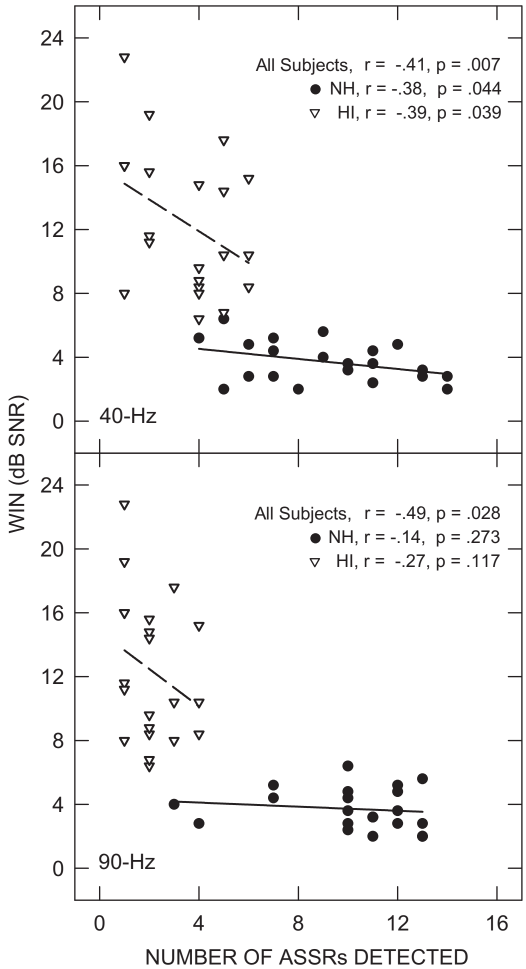

Figure 6. Scatter plot of word recognition in noise performance (dB SNR) and total number of ASSRs detected for NH subjects (n = 19, open circles) and HI subjects (n = 19, inverted open triangles). The 40-Hz ASSR data is plotted in the upper graph and the 90-Hz ASSR data is plotted in the lower graph.

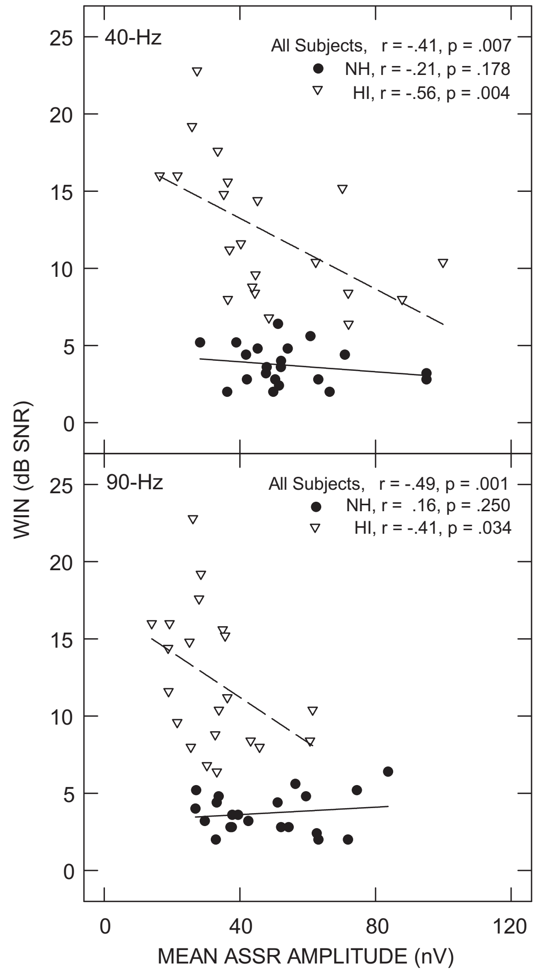

Figure 7. Scatter plot of word recognition in noise performance (dB SNR) and mean ASSR amplitude (nV) for NH subjects (n = 19, open circles) and HI subjects (n = 19, inverted open triangles). The 40-Hz ASSR data is plotted in the upper graph and the 90-Hz ASSR data is plotted in the lower graph.

number of 40-Hz ASSRs detected in babble increased, the WIN SNRs decreased (improved performance). The best-fit line shows a linear relationship, which accounts for 15% of the variability in WIN SNRs (closed circles, upper graph, Figure 6). The number of 40-Hz ASSRs detected in babble also was significantly correlated to WIN SNRs for the HI group (r = .39, p = .039). As the number of 40-Hz ASSRs increased, WIN SNRs decreased (improved performance) as seen in the inverted open triangles in the upper graph in Figure 6. The best-fit line shows a linear relationship, which accounts for 23% of the variability in WIN SNRs. In addition to response detection, the ASSR amplitude at both modulation rates was related to WIN scores for the HI group. The WIN scores were significantly correlated with the 40-Hz ASSR amplitude (r = −.56, p = .004; inverted open triangles, upper graph, Figure 7) and with the 90-Hz ASSR amplitude (r = −.41, p = .034; inverted open triangles, lower graph, Figure 7). As ASSR amplitude increased, WIN SNRs decreased (improved performance). The best-fit lines show linear relationships, which accounted for 31% and 17% of the variability in the WIN SNRs for the 40-Hz and 90-Hz ASSR amplitudes, respectively.

Discussion

The current study was designed to assess ASSRs in quiet and in the presence of multitalker babble in NH and HI subjects. Auditory processing was assessed by ASSR stimuli, which were modeled on the temporal and spectral characteristics of speech. The effects of carrier frequency, modulation rate, and stimulus level were measured. In addition, the extent to which auditory processing, as measured by ASSRs, was related to word recognition performance was examined. Subjects with reduced word recognition performance were expected to have fewer ASSRs and/or reduced ASSR amplitude compared to subjects with good word recognition performance.

ASSRs in quiet

The effect of carrier frequency showed that the greatest number of ASSRs was detected at 1500 Hz, regardless of modulation rate or subject group. The amplitude of the 1500-Hz ASSR was relatively large in both subject groups across most presentation levels and modulation rates. In contrast, the 500- and 4000-Hz carrier frequencies consistently were smaller and detected less often than the 1500-Hz carrier. The 4000-Hz component was calibrated to approximately 20 dB down from the 1500-Hz component and the 500-Hz component has been shown to be less robust than the mid-frequency ASSRs (e.g. D'Haenens et al, 2007; John et al, 2001) at least for ASSRs with modulation rates >70 Hz. Others have found no effect of carrier frequency on ASSR amplitude for carriers up to 4000 Hz (Dimitrijevic et al, 2001; John & Picton, 2000) for carrier frequencies presented at the same level.

The reduced detection rate and reduced amplitude for the 500-Hz carrier frequency in the present study was similar to 40-Hz ASSR data reported by Bhagat (2008). Multiple-stimuli ASSR detection rates and amplitudes were compared to single-stimulus ASSRs in normal-hearing subjects for the carrier frequencies of 500, 1000, 1500, and 4000 Hz. ASSR amplitudes were reduced in the multiple-stimuli versus the single-stimulus condition when a low-frequency carrier (1000 Hz) was paired with a higher-frequency carrier (2000 Hz). In comparison to the Bhagat data, the 500-Hz ASSR amplitude in the present study may have been reduced by the higher-frequency carrier (1500 Hz). In addition, the Bhagat data show an enhancement of ASSR amplitude for a higher-frequency carrier when a low-frequency carrier (500 Hz) was paired with it (1000 Hz). The 1500-Hz ASSR amplitude in the present study may have been enhanced by the presence of the 500-Hz carrier frequency. The data from the present study and the Bhagat study suggest a complex effect of carrier frequency and modulation depth on the 40-Hz ASSR amplitude at least at moderately high stimulus levels (e.g. 70 dB SPL).

The effect of carrier frequency on response detection and response amplitude may be due, in part, to the modulation rates chosen for the individual carrier frequencies. Modulation transfer functions are not flat between 40 and 60 Hz. The ASSR amplitude peak of the MTF occurs at ~40 Hz for awake adult subjects, and the ASSR declines in amplitude above ~50 Hz (Picton et al, 1987). The modulation rate chosen for the 1500-Hz carrier frequency was 44.9 Hz and may represent the best modulation frequency for a MM ASSR in an awake subject (Picton et al, 2003; Picton et al, 1987). The modulation rates chosen for the 2500- and 4000-Hz carrier frequencies were ~52 Hz and ~60 Hz, respectively, which are modulation rates that elicit less robust ASSRs.

The effect of modulation rate showed that more 40-Hz ASSRs were detected than 90-Hz ASSRs in both groups, and that the 40-Hz ASSRs were larger than 90-Hz ASSRs in the HI group. Levi et al (1993) recorded ASSRs at modulation rates from 10 to 80 Hz in normal-hearing adults and showed significantly higher coherence means for the 40-Hz ASSRs than for the 80-Hz ASSRs. In the NH group in the present study, no significant differences in ASSR amplitude were found between 40- and 90-Hz ASSRs. The modulation rate effect for ASSR amplitude in the present study was inconsistent with the data from several previous studies (e.g. Levi et al, 1993; Rees et al, 1986). One possible explanation for the data in the NH subjects is that the enhanced responses at 2500 and 4000 Hz increased the variability of the response amplitude, which resulted in a non-significant main effect for modulation rate. The interaction of carrier frequency and modulation rate showed that the 1500- and 2500-Hz carriers elicited the greatest number of responses at 40 Hz as expected, but the 1500-, 2500-, and 4000-Hz carriers elicited the greatest number of responses at 90 Hz. One possible explanation for the higher number of responses at 90 Hz for the 2500- and 4000-Hz carrier frequencies was related to the modulation rates chosen for individual carrier frequencies (see Table 1). The modulation rates of 95 and 103 Hz may produce more robust responses at higher carrier frequencies (e.g. 2500 and 4000 Hz) in the asleep state than the modulation rates of 53 and 61 Hz in the awake state (see Figure 2, Levi et al, 1993). The possibility of 60-Hz artifact contributing to the ASSR response was considered unlikely given that the 4000-Hz ASSRs had low detection rates and small response amplitudes across subject groups, modulation rates, and presentation levels.

In general, high ASSR modulation rates (e.g. 90 Hz) reflect primary contributions from subcortical regions, and low ASSR modulation rates (e.g. 40 Hz) reflect contributions from subcortical and cortical regions of the auditory system (Herdman et al, 2002). These different regions may reflect fundamentally different aspects of neural representation and coding of complex signals, such as speech. Evidence from animal studies suggests that the cochlea transmits an authentic spectral and temporal representation of the complex acoustic signal (Geisler, 1988), but that cortical representation of speech involves integration of neural coding across different cortical fields (Eggermont, 1998) that result in internal representations which form the basis of auditory perception (Wang et al, 2009). The significant differences between 40- and 90-Hz ASSRs found in the present study may reflect differences in subcortical and cortical processing of steady-state signals. Differences in ASSR detection between 40- and 90-Hz modulation rates also have been reported for older subjects (Leigh-Paffenroth & Fowler, 2006). The HI subjects in the present study were aged 48–78 years and an effect of aging on ASSRs cannot be excluded. Correlation analyses showed several significant correlations between participant age and ASSR amplitude ($r = .35$ to $.63$), and between participant age and ASSR detection ($r = .49$ to $.93$). Taken together, these results suggest that age and hearing loss may interact in a complex manner with regard to steady-state signal processing in the auditory system.

There was a significant increase in ASSR amplitude at 1500 Hz as a function of stimulus level for both modulation rates in the HI group. In contrast, there was no significant increase in amplitude as a function of stimulus level (60–76 dB SPL) for the NH group. The amplitude data from the present study were similar to the results reported by Lins et al (1995) and by Picton et al (2007). The effect of stimulus level in normal-hearing subjects appears to saturate at intensities above 70 dB SPL for single stimuli (Lins et al, 1995) or above 60 dB SPL for multiple stimuli (Picton et al, 2007).

ASSRs in babble

In the present study, the effect of babble produced either a reduction or an enhancement in the amplitude of the response. A reduction in the 500- and 1500-Hz ASSR amplitude was found for the NH group for the 40- and 90-Hz stimuli. In contrast, enhancement of the ASSR amplitude was found for the 2500- and 4000-Hz ASSRs for the 90-Hz stimulus, and for the 4000-Hz ASSR for the 40-Hz stimulus at the highest presentation level (i.e. 76 dB SPL). The 4000-Hz carrier was modulated at 60.5 Hz, but persistent interference from 60-cycle artifact was unlikely due to the overall weak 40-Hz ASSRs at 4000 Hz recorded across subject groups and presentation levels. In the HI group, there was no reduction in ASSR amplitude for responses recorded in babble, however, amplitude enhancement was observed at 1500- and 2500-Hz at the highest presentation level (76 dB SPL) for the 40-Hz stimulus. Similar to the present study, Dimitrijevic et al (2004) showed a reduction in 40-Hz and 90-Hz ASSR amplitude and detection rate in the noise condition relative to the quiet condition in normal-hearing subjects. In contrast to the results of the present study, Dimitrijevic et al also recorded a reduction in ASSR amplitude and detection rate in the noise condition for the HI subjects. One possible explanation for the observed discrepancy in the

ASSR amplitude is that the stimulus levels in the present study were higher than the stimulus levels in the Dimitrijevic study, and may have contributed to complex interactions in the auditory periphery. Significant changes in ASSR amplitude have been reported for in multiple-frequency ASSRs when carrier frequencies are less than one octave apart and presented at stimulus levels above 60 dB SPL (John et al, 1998; Picton et al, 2009). One explanation for the discrepancy in the ASSR detection rate produced by noise is due to differences in calculating detection rate. The Dimitrijevic response detection rate was a combined assessment of all 40- and 80-Hz responses for AM and FM. The present study used MM ASSRs and the detection rate was simply the number of ASSRs detected. Finally, the level of stimulus and babble relative to the pure-tone thresholds may explain the lack of a reduction in amplitude /detection rate in the masked HI data. The 2500- and 4000-Hz carrier frequencies were presented near or at pure-tone threshold levels in the HI subjects (e.g. average thresholds at 4000 Hz was 58 dB SPL and presentation level was 60 to 76 dB SPL) so no effects of babble should be expected; however, the potential for physiologic recruitment in individual HI subjects should be acknowledged (Picton et al, 2005).

Cochlear nonlinearities (e.g. suppression and distortion products) also must be considered as explanations for the effects of carrier frequency and effects of quiet versus babble found in the present study. Based on the type and degree of hearing loss in the HI subjects in the present study, it can be assumed that the compressive nonlinearities of the normal cochlea were absent in the ears of the HI subjects (Ruggero & Rich, 1991). It is possible that the multiple-frequency ASSRs in the present study produced complex interactions along the basilar membrane that likely had different effects in the normal cochleas of the NH subjects compared to the damaged cochleas of the HI subjects.

It is well known that hearing loss affects speech perception performance, especially in background noise. It is less well known how different levels of auditory processing may contribute to the complex process of speech perception in noise. The results from the present study show significant differences in the robustness of speech-like ASSRs at subcortical and cortical levels that are dependent on hearing sensitivity and the presence or absence of babble. The absence of an effect of babble on ASSRs for the HI group may be reflective of an impaired peripheral auditory system (i.e. cochlear hearing loss) that does not transmit an authentic spectral and temporal representation of the acoustic signal. The addition of babble, which should further reduce the ASSR, has no cumulative deteriorating effect on the ASSR because the hearing loss effect on the ASSR is so strong.

Enhanced perception to tones under certain stimulus conditions has been known for some time in subjects with normal hearing. Early behavioral experiments showed loudness enhancement when subjects were asked to match loudness of one tone to the loudness of another tone under a variety of tone and masker conditions (Elmasian & Galambos, 1975; Zwislocki et al, 1959). Possible physiologic mechanisms underlying these enhancement effects may be due the interaction of tones and noise (Greenwood, 1988; Henry & Price, 1992; Morse & Evans, 1996; Rhode et al, 1978; Seluakumaran et al, 2008). Greenwood and Goldberg (1970) recorded single-unit responses in the cat cochlear nucleus and showed that adding noise to a tone could cause suppression or enhancement of the physiologic response.

ASSR amplitude enhancement has been reported for multiple-stimulus ASSRs for specific stimulus levels. John et al (2002) reported small, but significant, increases in ASSR amplitude for multiple-carrier frequencies (500, 1000, 2000, and 4000 Hz) when the 500- and 4000-Hz carriers were presented 10 dB higher than the other frequencies. The ASSR amplitude at 1000 Hz was enhanced and the ASSR amplitude at 2000 Hz was reduced. In the present study, the level of the 1500-Hz carrier frequency was 7 dB higher than the level of the 2500 Hz carrier and may have decreased the response amplitude at 2500 Hz. Recently, McNerny and Burkard (2010) reported amplitude enhancement of multiple-carrier frequency ASSRs when stimulus levels were near threshold. The presentation levels used in the present study were near threshold at 2500- and 4000 Hz for the HI subjects, which may explain the enhancements in ASSR amplitude.

The enhancement results of the present study are consistent with results from behavioral loudness enhancement studies, single-unit studies in animals, and multiple-stimulus ASSR studies. The interaction of multiple-frequency stimuli and stimulus level is complex and deserves further investigation of single versus multiple-frequency ASSR stimuli recorded in quiet and in noise. John et al (1998) showed significant interactions between multiple carrier frequencies when the multiple-frequency ASSRs were modulated at 40 Hz, were presented at levels >60 dB SPL, and were separated by less than one octave. This is particularly relevant to the present study in which the ASSR stimuli were modeled after speech signals with carrier frequencies less than an octave apart and presented at suprathreshold levels.

Relationships among speech-spectrum ASSRs and word recognition

Word recognition in quiet at 60 dB HL was significantly correlated to the mean 40-Hz ASSR amplitude for HI subjects ($r = -.468$, $p = .019$). In addition, WIN SNRs were significantly correlated to the number of 40-Hz ASSRs detected ($r = -.559$, $p = .004$) and for the number of 90-Hz ASSRs detected ($r = -.407$, $p = .034$) for the HI group. Dimitrijevic and colleagues reported significant correlations between ASSR detection and word recognition in quiet performance (0.65 to .85) (Dimitrijevic et al, 2001) and word recognition in noise performance (0.64 to 0.85) (Dimitrijevic et al, 2004). Significant correlations between ASSR amplitude and word recognition in noise performance (0.39 to 0.77) were also reported by Dimitrijevic et al (2004). The significant correlations found in the present study were not as strong for NH subjects as those reported by Dimitrijevic et al (2004), possibly due to the differences in the measurement of word recognition. Dimitrijevic et al obtained percent correct word recognition performance at several stimulus levels in quiet (2001) and in speech noise (2004), providing a wide range of performance for NH subjects (~20% to 100%). In the present study, two levels of WIQ and one overall measure of word recognition performance in babble was obtained with a smaller range of performance for NH subjects (70% to 87%).

Future directions

The amplitude-intensity functions of the MM stimulus used in the present study require pairwise comparisons (single carrier frequency stimulus versus a two carrier frequency stimulus; single carrier frequency versus a three carrier frequency stimulus, etc.) of the possible interactions among carrier frequencies, especially at the stimulus levels >60 dB SPL. The stimuli were chosen to reflect some of the temporal and spectral components of speech and may involve complex frequency interactions in subjects with and without hearing loss. In addition, future studies will explore the differences in 40- vs. 90-Hz ASSRs in older subjects to investigate how age and peripheral hearing loss may alter the processing at subcortical and cortical levels. Finally, the enhancement effects found in the present study for the babble condition provided an additional level of complexity for future studies in multiple-frequency ASSRs. Specifically, the modulation rate and stimulus level at which suppression versus enhancement of a response occurs should be studied in subjects with and without hearing loss. In the present study, response enhancement was found at both modulation rates in the NH group, but only at 40 Hz in the HI group in the present study. The possibility exists that peripheral hearing loss has different effects at subcortical and cortical regions.

Summary

The purpose of this investigation was to determine the effects of multitalker babble on the ASSR in NH and HI subjects. The presence of babble significantly reduced ASSR detection for NH subjects, but had minimal effect on ASSR detection or ASSR amplitude for HI subjects. Normal-hearing subjects had a higher ASSR detection rate and larger ASSRs than HI subjects. The most robust ASSRs were found for the 1500-Hz carrier frequency for both groups and across all stimulus conditions. The 4000-Hz stimulus component was ~24 dB lower than the 500-Hz component, which may explain the weak responses at this carrier frequency. The enhancement effects found for the speech-spectrum ASSRs recorded in babble require further investigation.

Significant correlations were found between word recognition performance and ASSR detection, as well as between word recognition performance and ASSR amplitude. The speech-spectrum ASSRs used in the present study provided the first step in the development of a physiologic measure of speech processing at multiple levels of the auditory system. If the relationships among ASSRs and speech discrimination or speech perception prove to be robust, then ASSRs could be used to predict speech perception performance in patients who cannot provide reliable behavioral responses, such as infants and young children, children with learning disabilities, and older adults.

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References

Beattie R.C. 1989. Word recognition functions for the CID W-22 test in multitalker noise for normally hearing and hearing-impaired subjects. *J Speech Hear Disord*, 54, 20–32.

Bhagat S.P. 2008. The effects of monotic and dichotic interference tones on 40 Hz auditory steady-state responses in normal-hearing adults. *J Am Acad Audiol*, 19, 101–19.

Carhart R. & Tillman T.W. 1970. Interaction of competing speech signals with hearing losses. *Arch Otolaryngol*, 91, 273–9.

Cone B. & Garinis A. 2009. Auditory steady-state responses and speech feature discrimination in infants. *Journal of the Academy of Audiology*, 20, 629–43.

D'Haenens W., Dhooge I., De Vel E., Maes L., Bockstael A. et al. 2007. Auditory steady-state responses to MM and exponential envelope AM(2)/FM stimuli in normal-hearing adults. *Int J Audiol*, 46, 399–406.

Dimitrijevic A., John M.S. & Picton T.W. 2004. Auditory steady-state responses and word recognition scores in normal-hearing and hearing-impaired adults. *Ear Hear*, 25, 68–84.

Dimitrijevic A., John M.S., van Roon P. & Picton T.W. 2001. Human auditory steady-state responses to tones independently modulated in both frequency and amplitude. *Ear Hear*, 22, 100–111.

Divenyi P.L. & Haupt K.M. 1997a. Audiological correlates of speech understanding deficits in elderly listeners with mild-to-moderate hearing loss. I. Age and lateral asymmetry effects. *Ear Hear*, 18, 42–61.

Divenyi P.L. & Haupt K.M. 1997b. Audiological correlates of speech understanding deficits in elderly listeners with mild-to-moderate hearing loss. II. Correlation analysis. *Ear Hear*, 18, 100–113.

Divenyi P.L. & Haupt K.M. 1997c. Audiological correlates of speech understanding deficits in elderly listeners with mild-to-moderate hearing loss. III. Factor representation. *Ear Hear*, 18, 189–201.

Dubno J.R., Dirks D.D. & Morgan D.E. 1984. Effects of age and mild hearing loss on speech recognition in noise. *J Acoust Soc Am*, 76, 87–96.

Eggermont J.J. 1994. Temporal modulation transfer functions for AM and FM stimuli in cat auditory cortex. Effects of carrier type, modulating waveform and intensity. *Hear Res*, 74, 51–66.

Eggermont J.J. 1998. Representation of spectral and temporal sound features in three cortical fields of the cat. Similarities outweigh differences. *J Neurophysiol*, 80, 2743–64.

Elmasian R. & Galambos R. 1975. Loudness enhancement: Monaural, binaural, and dichotic. *J Acoust Soc Am*, 58, 229–34.

Field A. 2009. *Discovering Statistics Using SPSS, Third edition*. London: Sage Publications Inc.

Finney D.J. 1952. *Statistical Method in Biological Assay*. London: Charles Griffin & Co.

Fisher A.L., Hymel M.R., Cranford J.L. & DeChicchis A.R. 2000. Electrophysiologic signs of auditory distraction in elderly listeners. *J Am Acad Audiol*, 11, 36–45.

Fowler C.G. & Mikami C.M. 1992a. Effects of noise bandwidth on the late-potential masking level difference. *Electroencephalogr Clin Neurophysiol*, 84, 157–63.

Fowler C.G., Mikami C.M. 1992b. The late auditory evoked potential masking-level difference as a function of noise level. *J Speech Hear Res*, 35, 216–21.

Fowler C.G. & Mikami C.M. 1995. Binaural phase effects in the auditory brainstem response. *J Am Acad Audiol*, 6, 399–406.

Fowler C.G. & Mikami C.M. 1996. Phase effects on the middle and late auditory evoked potentials. *J Am Acad Audiol*, 7, 23–30.

French N.R. & Steinberg J.C. 1947. Factors governing the intelligibility of speech sounds. *J Acoust Soc Am*, 19, 90–119.

Geisler C.D. 1988. Representation of speech sounds in the auditory nerve. *Journal of Phonetics*, 16, 19–35.

Gordon-Salant S. 1987. Effects of acoustic modification on consonant recognition by elderly hearing-impaired subjects. *J Acoust Soc Am*, 81, 1199–202.

Greenwood D.D. 1988. Cochlear nonlinearity and gain control as determinants of the response of primary auditory neurons to harmonic complexes. *Hear Res*, 32, 207–53.

Greenwood D.D & Goldberg J.M. 1970. Response of neurons in the cochlear nuclei to variations in noise bandwidth and to tone-noise combinations. *J Acoust Soc Am*, 47, 1022–40.

Grose J.H., Eddins D.A. & Hall J.W. 3rd. 1989. Gap detection as a function of stimulus bandwidth with fixed high-frequency cutoff in normal-hearing and hearing-impaired listeners. *J Acoust Soc Am*, 86, 1747–55.

Grose J.H., Mamo S.K., Hall J.W. 3rd. 2009. Age effects in temporal envelope processing: Speech unmasking and auditory steady state responses. *Ear Hear*, 30, 1–8.

Harkrider A.W., Plyler P.N. & Hedrick M.S. 2009. Effects of hearing loss and spectral shaping on identification and neural response patterns of stop-consonant stimuli in young adults. *Ear Hear*, 30, 31–42.

Henry K.R. & Price J.M. 1992. Suprathreshold comparisons of derived and enhanced compound action potentials. *Hear Res*, 63, 90–6.

Herdman A.T., Lins O., Van Roon P., Stapells D.R., Scherg M. et al. 2002. Intracerebral sources of human auditory steady-state responses. *Brain Topogr*, 15, 69–86.

Hirsh I.J. 1948. The influence of interaural phase on interaural summation and inhibition. *Journal of the Acoustical Society of America*, 20, 536–44.

Hirsh I.J. 1950. Binaural hearing aids: a review of some experiments. *J Speech Disord*, 15, 114–23.

Horwitz A.R., Dubno J.R. & Ahlstrom J.B. 2002. Recognition of low-pass-filtered consonants in noise with normal and impaired high-frequency hearing. *J Acoust Soc Am*, 111, 409–16.

John M.S., Dimitrijevic A., van Roon P. & Picton T.W. 2001. Multiple auditory steady-state responses to AM and FM stimuli. *Audiol Neurootol*, 6, 12–27.

John M.S., Lins O.G, Boucher B.L. & Picton T.W. 1998. Multiple auditory steady-state responses (MASTER): Stimulus and recording parameters. *Audiology*, 37, 59–82.

John M.S. & Picton T.W. 2000. Human auditory steady-state responses to amplitude-modulated tones: Phase and latency measurements. *Hear Res*, 141, 57–79.

John M.S., Purcell D.W., Dimitrijevic A. & Picton T.W. 2002. Advantages and caveats when recording steady-state responses to multiple simultaneous stimuli. *J Am Acad Audiol*, 13, 246–59.

Krishna B.S. & Semple M.N. 2000. Auditory temporal processing: Responses to sinusoidally amplitude-modulated tones in the inferior colliculus. *J Neurophysiol*, 84, 255–73.

Krishnan A. 2002. Human frequency-following responses: Representation of steady-state synthetic vowels. *Hear Res*, 166, 192–201.

Leigh-Paffenroth E.D. & Fowler C.G. 2006. Amplitude-modulated auditory steady-state responses in younger and older listeners. *J Am Acad Audiol*, 17, 582–97.

Levi E.C., Folsom R.C. & Dobie R.A. 1993. Amplitude-modulation following response (AMFR): Effects of modulation rate, carrier frequency, age, and state. *Hear Res*, 68, 42–52.

Lins O.G., Picton P.E., Picton T.W., Champagne S.C. & Durieux-Smith A. 1995. Auditory steady-state responses to tones amplitude-modulated at 80–110 Hz. *J Acoust Soc Am*, 97, 3051–63.

McNerney K.M. & Burkard R.F. 2010. The effects of a second stimulus on the auditory steady state response (ASSR) from the inferior colliculus of the chinchilla. *Int J Audiol*, 1–13.

Morse R.P. & Evans E.F. 1996. Enhancement of vowel coding for cochlear implants by addition of noise. *Nat Med*, 2, 928–32.

Olsen W.O., Noffsinger D. & Kurdziel S. 1975. Speech discrimination in quiet and in white noise by patients with peripheral and central lesions. *Acta Otolaryngol*, 80, 375–382.

Picton T.W., Dimitrijevic A., Perez-Abalo M.C. & Van Roon P. 2005. Estimating audiometric thresholds using auditory steady-state responses. *J Am Acad Audiol*, 16, 140–156

Picton T.W., John M.S., Dimitrijevic A. & Purcell D. 2003. Human auditory steady-state responses. *Int J Audiol*, 42, 177–219.

Picton T.W., Skinner C.R., Champagne S.C., Kellett A.J. & Maiste A.C. 1987. Potentials evoked by the sinusoidal modulation of the amplitude or frequency of a tone. *J Acoust Soc Am*, 82, 165–78.

Picton T.W., van Roon P. & John M.S. 2007. Human auditory steady-state responses during sweeps of intensity. *Ear Hear*, 28, 542–57.

Picton T.W., van Roon P. & John M.S. 2009. Multiple auditory steady state responses (80–101 Hz): Effects of ear, gender, handedness, intensity and modulation rate. *Ear Hear*, 30, 100–9.

Rees A., Green G.G. & Kay R.H. 1986. Steady-state evoked responses to sinusoidally amplitude-modulated sounds recorded in man. *Hear Res*, 23, 123–33.

Rhode W.S. 1994. Temporal coding of 200% amplitude modulated signals in the ventral cochlear nucleus of cat. *Hear Res*, 77, 43–68.

Rhode W.S., Geisler C.D. & Kennedy D.T. 1978. Auditory nerve fiber response to wide-band noise and tone combinations. *J Neurophysiol*, 41, 692–704.

Roup C.M., Wiley T.L., Safady S.H. & Stoppenbach D.T. 1998. Tympanometric screening norms for adults. *American Journal of Audiology*, 7, 1–6.

Ruggero M.A. & Rich N.C. 1991. Furosemide alters organ of corti mechanics: Evidence for feedback of outer hair cells upon the basilar membrane. *J Neurosci*, 11, 1057–67.

Seluakumaran K., Mulders W.H. & Robertson D. 2008. Unmasking effects of olivocochlear efferent activation on responses of inferior colliculus neurons. *Hear Res*, 243, 35–46.

Snell K.B. & Frisina D.R. 2000. Relationships among age-related differences in gap detection and word recognition. *J Acoust Soc Am*, 107, 1615–26.

Souza P.E. & Turner C.W. 1994. Masking of speech in young and elderly listeners with hearing loss. *J Speech Hear Res*, 37, 655–61.

Sperry J.L., Wiley T.L. & Chial M.R. 1997. Word recognition performance in various background competitors. *J Am Acad Audiol*, 8, 71–80.

Steinhauer K. 2003. Electrophysiological correlates of prosody and punctuation. *Brain Lang*, 86, 142–64.

Tillman T.W. & Carhart R. 1966. An expanded test for speech discrimination utilizing CNC monosyllabic words. Northwestern University Auditory Test No. 6. SAM-TR-66-55. *Tech Rep SAM-TR*, 1–12.

Tremblay K.L., Billings C. & Rohila N. 2004. Speech evoked cortical potentials: Effects of age and stimulus presentation rate. *J Am Acad Audiol*, 15, 226–37; quiz 64.

Wang J., Nicol T., Skoe E., Sams M. & Kraus N. 2009. Emotion modulates early auditory response to speech. *J Cogn Neurosci*, 21, 2121–8.

Weihing J. & Musiek F.E. 2008. An electrophysiological measure of binaural hearing in noise. *J Am Acad Audiol*, 19, 481–95.

Wiley T.L., Cruickshanks K.J., Nondahl D.M., Tweed T.S., Klein R. et al. 1996. Tympanometric measures in older adults. *J Am Acad Audiol*, 7, 260–8.

Wilson R.H. 2003. Development of a speech-in-multitalker-babble paradigm to assess word-recognition performance. *J Am Acad Audiol*, 14, 453–70.

Wilson R.H. & McArdle R. 2005. Speech signals used to evaluate functional status of the auditory system. *J Rehabil Res Dev*, 42, 79–94.

Wilson R.H. & McArdle R. 2007. Intra- and inter-session test, retest reliability of the Words-in-Noise (WIN) test. *J Am Acad Audiol*, 18, 813–25.

Wingfield A. 1996. Cognitive factors in auditory performance: Context, speed of processing, and constraints of memory. *J Am Acad Audiol*, 7, 175–82.

Yin S.K., Feng Y.M., Chen Z.N. & Wang J. 2008. The effect of noise-induced sloping high-frequency hearing loss on the gap-response in the inferior colliculus and auditory cortex of guinea pigs. *Hear Res*, 239, 126–40.

Zwislocki J.J., Pirodda E., Rubin H. 1959. On some poststimulatory effects at the threshold of audibility. *J Acoust Soc Am*, 31, 9–14.