

On chirp stimuli and neural synchrony in the suprathreshold auditory brainstem response

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The chirp-evoked ABR has been regarded as a more *synchronous* response than the click-evoked ABR, referring to the belief that the chirp stimulates lower-, mid-, and higher-frequency regions of the cochlea simultaneously. In this study a variety of tools were used to analyze the synchronicity of ABRs evoked by chirp- and click-stimuli at 40 dB HL in 32 normal hearing subjects aged 18 to 55 years (mean=24.8 years, SD=7.1 years). Compared to the click-evoked ABRs, the chirp-evoked ABRs showed larger wave V amplitudes, but an absence of earlier waves in the grand averages, larger wave V latency variance, smaller FFT magnitudes at the higher component frequencies, and larger phase variance at the higher component frequencies. These results strongly suggest that the chirp-evoked ABRs exhibited less synchrony than the click-evoked ABRs in this study. It is proposed that the temporal compensation offered by chirp stimuli is sufficient to increase neural recruitment (as measured by wave V amplitude), but that destructive phase interactions still exist along the cochlea partition, particularly in the low frequency portions of the cochlea where more latency jitter is expected. The clinical implications of these findings are discussed. © 2010 Acoustical Society of America. [DOI: 10.1121/1.3436527]

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I. INTRODUCTION

The auditory brainstem response (ABR) is conventionally elicited using short-pulsed ‘clicks’. Although these ‘click’ stimuli are considered broadband, they do not obtain a response from the entire cochlea. This is because click stimuli do not account for temporal dispersion of displacement maxima along the cochlea partition. Therefore the basilar membrane’s (BM) response to the lower frequency components of the click occurs too late to contribute significantly to the ABR (Pantev *et al.*, 1985).

To include this missing contribution from the lower frequency regions of the cochlea, recent studies have suggested that ‘click’ stimuli be replaced by rising-frequency ‘chirp’ stimuli. Rising-frequency chirp stimuli compensate for the temporal dispersion by delaying the higher frequency content of the stimulus until the lower frequency traveling waves are closer to the apex of the cochlea. This compensation theoretically results in simultaneous displacement maxima along the entire length of the basilar membrane within the inner ear, allowing all regions to contribute to the ABR. The result is an ABR waveform that is larger, can be recorded in less time and potentially has more diagnostic power (Dau *et al.*, 2000).

Previous reports have regarded the chirp-evoked ABR as being a more synchronous response, referring to the fact that lower-, mid-, and higher-frequency regions of the cochlea are stimulated synchronously and so all contribute to the compound ABR (Dau *et al.*, 2000; Fobel and Dau, 2004). If this were true, then it would be reasonable to expect that the

chirp-evoked ABR would evoke larger amplitudes, have better morphology and be more repeatable. Larger wave V amplitudes have been reported when comparing chirp-evoked ABRs to click-evoked ABRs (e.g., Bell *et al.*, 2002a; Dau *et al.*, 2000; Fobel and Dau, 2004), but little data exists to quantify improvements to morphology and repeatability. Indeed, one of the few studies to quantify latency variance reported that wave V latencies were more variable for chirps than for clicks (Elberling and Don, 2008). The presence (or absence) of earlier waves in the morphology of chirp-evoked ABR has been shown to be dependent on the spectral ‘flatness’ of the chirp (Dau *et al.*, 2000), the inclusion of higher frequencies in the chirp (Wegner and Dau, 2002) and the suitability of the underlying model (Fobel and Dau, 2004).

Improvements in neural synchrony with the chirp-evoked response have been largely quantified using observed improvements in wave V amplitude. Deficiencies in the morphology of other waves in the ABR, as well as impaired repeatability of latency measurements, would suggest that measuring wave V amplitude alone is not necessarily a robust measure of neural synchrony. Larger ABR peak amplitudes may be the result of larger contributions from individual neurons, contributions from more neurons and/or better neural synchrony (Don *et al.*, 1994). A stimulus that causes synchronicity in the generators of wave V does not necessarily imply that the same stimulus also causes maximum synchronicity at the BM level (Fobel and Dau, 2004).

Further analysis of chirp-evoked ABR is required before we can confirm that the entire response is more or less synchronous than click-evoked ABR.

Other tools to quantify neural synchrony also exist, each with their own set of limitations. For example, we can obtain an impression of neural synchrony from repeated wave V latency measures. We expect that highly synchronous responses would have less 'jitter' in the locations of ABR peaks and therefore exhibit less variance in repeated latency measures. Unfortunately, this method of measuring synchrony is particularly prone to additional sources of latency jitter, such as artifacts of muscle movement and background physiological noise (Lightfoot, 1993; Sanchez and Gans, 2006).

Another way to determine the level of neural synchrony is by analyzing information given by the fast Fourier transform (FFT) of the ABR. It is known that ABRs with greater jitter, such as those acquired using low frequency tone bursts, have broader peaks in the averaged ABR (Hall, 1992). Conversely, when neural synchrony is high, the averaged ABR has peaks that are narrower and higher—more nearly approximating the response of a single neuron (Shore and Nuttall, 1985). Narrower peaks have higher frequency components, and therefore the FFT magnitudes of the higher frequencies may reflect the level of neural synchrony. A limitation of the FFT, however, is that it is affected by subject age and gender (Wilson and Aghdasi, 1999) and there is again a large contribution from background physiological noise (Kavanagh *et al.*, 1988; Laukli and Mair, 1981).

One further technique that may determine the level of neural synchrony is the 'synchrony measure' (SM), originally described by Fridman *et al.* (1984) and further experimented with by Chen *et al.* (1988) and Sakai *et al.* (1994). The synchrony measure represents the degree of reproducibility for group averages of the evoked response by examining the phase variance in particular Fourier components (Fridman *et al.*, 1984). Smaller phase variances indicate greater synchronization in the origin of the ABR (Fridman *et al.*, 1982) and that the particular Fourier component is time-locked with a stimulus onset. Components that are time-locked with stimulus onset are more likely to reflect auditory-evoked potentials rather than background physiological noise (Fridman *et al.*, 1984).

If we apply multiple measures of synchrony to chirp-evoked ABRs we can provide a good indication of whether chirp stimuli evoke more synchronous responses. The aim of this study was therefore to use wave V amplitude, wave V latency variance, ABR frequency content (as shown by the FFT) and the SM to investigate whether chirp-evoked ABRs exhibit greater or less neural synchrony than click-evoked ABRs.

II. METHODS

A. Data acquisition

1. Subjects

Thirty-two subjects ranging in age from 18 to 55 years (mean=24.8 years, SD=7.1) voluntarily served as participants—15 females (mean age=22.5, SD=2.7) and 17

males (mean age=26.8, SD=9.1). All participants had audiometric thresholds of 15 dB HL or better for the frequencies 250, 500, 1000, 2000, 4000 and 6000 Hz, type A tympanograms (Jerger, 1970), and presented with no self-reported otological concerns.

2. Equipment

We used a low-noise CED 1902 bio-amplifier and a CED Power 1401 data acquisition system, both from Cambridge Electronic Design (CED) to acquire ABR waveforms. The 1902 bio-amplifier contained onboard filters and could provide up to 120 dB of gain. We presented auditory stimuli to test subjects via Etymotic 300 Ω ER-3A insert earphones, driven by a Tucker-Davis HB7 headphone buffer. We used a custom-coded Mathworks MATLAB[™] interface to drive the data acquisition and analysis.

3. Stimuli

We included two click stimuli in this study, and two rising-frequency sweeps (chirps). The first click was 100 μ s wide (commonly used in audiology) and the second was 80 μ s wide. Previous researchers suggest this second click mimics the frequency content of the chirp stimuli more precisely (Fobel and Dau, 2004). However, this may only apply to the extended frequency range of the ER-2 research earphones that they used in their study. Regardless, the inclusion of the 80 μ s click in this study gives further validation that any observed differences between chirp-evoked ABR and click-evoked ABR are not random effects.

The two chirp stimuli we included were the 'model-based' chirp, 'M-Chirp' (Dau *et al.*, 2000) and the 'ABR-based' chirp, 'A-Chirp' (Fobel and Dau, 2004). The M-Chirp is based on a linear cochlea model, obtained from cadavers, which describes the expected temporal delays down the basilar membrane (de Boer, 1980). To compensate for these delays, the M-Chirp increases exponentially in frequency (0.1 to 10.4 kHz) over a duration of 10.32 ms. The A-Chirp differs in that it is based on wave V latencies from tone bursts at various intensities (Gorga *et al.*, 1988; Neely *et al.*, 1988), and therefore its sweep rate is intensity-dependent. The A-Chirp in the present study is 7.87 ms long, corresponding to the modeled wave V latency difference between the highest (10.4 kHz) and lowest frequency (0.1 kHz) components at our chosen intensity (40 dB HL). We adjusted the temporal envelope of both chirps to weight all frequencies equally in the power spectrum. This effectively meant that the chirps had the same frequency spectra as the click stimuli (Dau *et al.*, 2000).

Additionally, we included two 'reverse chirps' which were temporally reversed A- and M-Chirps. Falling-frequency reverse chirps exaggerate the basilar membrane traveling wave delay and should theoretically evoke a highly dissynchronous response (Dau *et al.*, 2000; Shore and Nuttall, 1985). Although the focus of this study is a comparison between rising-frequency chirps and clicks, the inclusion of these reverse chirps provides some verification of the synchrony measures used in the methods.

All stimuli were presented at approximately 40 dB HL, corresponding to a peak-equivalent sound pressure level (Burkard, 1984) of 87 dB peSPL for the click and 80 dB peSPL for the chirp stimuli. Lower intensities would provide more accurate estimates of frequency-specific contributions to the ABR, but a compromise of 40 dB HL is considered optimal as the response is easily measurable (Klein and Teas, 1978). The 7 dB difference between click and chirp sound pressure level “reflects temporal integration of signal energy involved in behavioral threshold measures that probably occurs at more central stages of auditory processing and is most likely not reflected in ABR” (Wegner and Dau, 2002)—a theory supported by Nábelek (1978). A similar calibration offset, 6.7 dB, between clicks and chirps was used by Fobel and Dau (2004), however, other studies have reported that threshold differences between clicks and chirps may be as little as 2.5 dB (Agung *et al.*, 2005).

Almost all chirp studies to-date have been performed in research environments where insert earphones with an extended high-frequency response, such as Etymotic ER-2 earphones, have been available. The present study investigated the chirp-evoked ABR using ER-3A earphones, as these transducers are more prevalent (American Speech-Language Hearing Association, 2004) and are a likely target for the introduction of new stimuli into clinical practice. Although these earphones have a reduced frequency response, we fixed the start and end frequencies of our chirps to match those used in previous studies (Dau *et al.*, 2000). Differences in behavioral thresholds between the ER-2 earphones used in previous studies and the ER-3A earphones used in this current study are reported to be negligible for both the click and the chirp stimuli (Agung *et al.*, 2005).

The relationship between the custom built ABR unit’s variable gain and output dB SPL for a 1000 Hz tone was determined using an Aurical system (EN60645-1, -2 type: 2, A; monitor version 1.00; program version 2.40; DSP program version 2.40) running Aurical REM module version 2.50 and this system’s 2 cc coupler. All stimuli produced by the unit were calibrated before each ABR waveform acquisition using a loopback method whereby the output to the headphone amplifier was simultaneously monitored by the analog-to-digital converter. The gain required to achieve the required dB peSPL was determined by successive approximation, using the previously calibrated 1000 Hz tone value for dB SPL.

The pre-transducer time courses of our stimuli, excluding the reverse chirps, are shown as gray lines in Fig. 1 (arbitrary vertical scale). We recorded the acoustic time course of our stimuli using a 2 cc coupler and a Brüel & Kjær 2250 hand-held analyzer (solid lines, Fig. 1). Additionally, we recorded the spectra of our stimuli using a 2-cc coupler and the aforementioned Aurical REM module (Fig. 2).

4. Recordings

Testing took place in a sound-treated and electrically-shielded room. The test subjects lay down on a clinic bed and the lights were turned out to assist the subject to sleep if

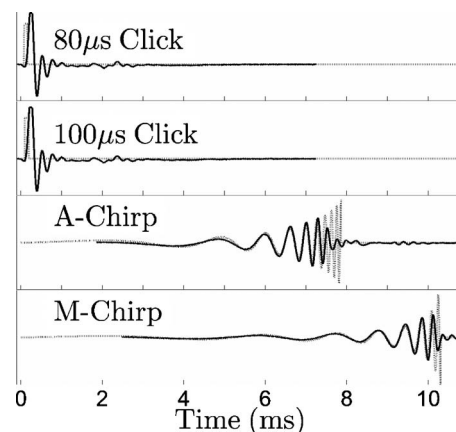


FIG. 1. The stimuli used in the current study are (top-to-bottom): an 80 μ s click, a 100 μ s click, a rising-frequency ‘A-Chirp’ (compensating for BM traveling wave delays calculated from tone burst-evoked ABR), and a rising-frequency ‘M-Chirp’ (compensating for BM traveling wave delays calculated from modeled data). Bold traces indicate the acoustic time course as measured in a 2 cc coupler. Lighter traces indicate the pre-transducer time course. The ‘reverse chirps’ of the current study are not shown, but are temporal reversals of the A-Chirp and M-Chirp.

possible. Electro-encephalic activity was recorded from the scalp via disposable silver/silver chloride electrodes.

The electrodes were placed in a vertical montage such that one electrode was attached to the forehead (active, non-inverting), one on the nape of the neck (reference), and one below the middle of the left clavicle (ground). We used the vertical electrode montage as its electrode positions do not change with test ear, and because of previous reports that it highlights wave V (Beattie and Taggart, 1989; King and Singer, 1992). Inter-electrode impedance was maintained below 5 k Ω , and in most cases was below 2 k Ω .

The test ear was randomly chosen and maintained for the entire duration of the acquisition. Responses were recorded for 45.3 ms following stimulus onset, giving a stimulus repetition rate of 22.1 stimuli per second. The stimuli were presented with alternating polarity [to minimize stimulus artifacts and the cochlear microphonic (Burkard and Don,

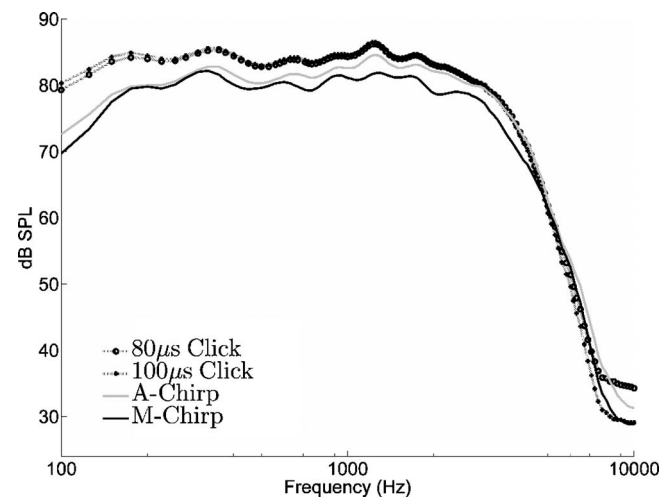


FIG. 2. The frequency spectra of the click and chirp stimuli were obtained using a 2 cc coupler and an Aurical sound level meter. All stimuli were presented at 40 dB HL, corresponding to 87 dB peSPL for the click and 80 dB peSPL for the chirp stimuli.

2006)] and were digitally generated with a reconstruction frequency of 263.2 kHz. Responses were amplified (89.5 dB) and filtered (50 to 5000 Hz Butterworth band-pass, 12 dB/oct roll-off) before being digitized by the Power 1401 ADC unit (set to 32.9 kHz, 16 bit resolution). The order of stimulus presentation was randomized and responses were recorded for 2000 epochs per stimulus.

We acquired three separate recordings for the chirps and the clicks, and one recording for the reverse chirps. ABR acquisitions took approximately 35 min per test subject.

B. Data analysis

1. Data conditioning

Recorded data was analyzed *post-hoc* using a custom-coded Mathworks MATLAB™ interface. Artifact rejection was performed, such that epochs with data that exceeded a threshold of 19 μV within a window of interest were excluded from further analysis. The window of interest for artifact rejection was 0 to 12 ms following stimulus offset for the chirps, and 0 to 12 ms following stimulus onset for the clicks and the reverse-chirps, accounting for the 0.83 ms tubing delay of the ER-3A earphones. Rejecting epochs with data that exceed a threshold has been shown to be the most straight-forward and effective in artifact rejection (Pantev and Khvoles, 1984). Following artifact rejection, the raw data was filtered using a 100 Hz–3 kHz software band pass filter.

We sought to keep the signal-to-noise ratio (SNR) of each ABR at a similar level across all stimuli and test subjects. To achieve this, we made use of the single point variance statistic, F_{SP} (Elberling and Don, 1984). When constructing the ensemble average for each ABR, we only summated as many epochs as were required to reach an F_{SP} value of 3.1. This criterion infers that the probability of an ABR being present is at least 99% (Don *et al.*, 1984; Elberling and Don, 1984, 1987). In this manner, all ensemble averages had a similar SNR; 1.6 dB, using the approximation $\text{SNR} = 10 \log_{10}(\sqrt{F_{\text{SP}} - 1})$ (Burkard and Don, 2006).

The analysis window used when calculating the F_{SP} statistic was the same as for the artifact rejection, and the ‘single point’ for each F_{SP} calculation was automatically chosen to be the strongest peak in the overall ensemble average (within the analysis window). The window parameters were appropriate to resolve low frequency noise contributions to the ABR, avoid any contribution from stimulus artifact (Sininger, 1993) and include the entire ABR signal (Don *et al.*, 1984; Elberling and Don, 1984).

The characteristics of physiological noise are such that the noise falls in and out of phase with the acquisition window and stimulation rate. This “noise memory bias” can cause oscillations in SNR estimations (Özdamar and Delgado, 1996). For this reason, we determined the number of epochs required to reach our F_{SP} threshold from a fitted slope of the increase in F_{SP} over time. A robust linear regression function (Holland and Welsch, 1977) iteratively removed outliers from the F_{SP} estimates, ensuring that oscillations above threshold did not cause the F_{SP} to converge prematurely.

We constructed grand averages for our overall population data, following artifact rejection, from the individual ensemble averages at the $F_{\text{SP}}=3.1$ detection level. Each ensemble average was first time-shifted to align wave V with the average wave V latency of the overall population, in order to minimize any phase-cancellations due to inter-subject latency variations (Don *et al.*, 1997, 1994).

2. Analysis of wave V amplitude and latency

We determined the location of the peak of wave V visually, and used a graphical interface to measure the amplitude from the peak of wave V to the next major trough.

The click-evoked wave V latencies are reported here relative to stimulus onset since the ABR is known to be an onset-driven response (Coath and Denham, 2007; Van Campen *et al.*, 1997). The treatment for the reversed chirps is the same, as they start with high frequencies and therefore have a similarly rapid onset. However, in the case of the forward chirps, the stimuli have been designed to give maximum basilar membrane deflection at stimulus *offset*. Therefore, we subtracted the stimulus duration from these ABRs to give ‘adjusted latencies’ relative to stimulus *offset*. This reflects the notion that displacement maxima on the BM should occur in all frequency channels at the same time, and thus, the latencies for the chirp and the click should be similar if the chirp latencies were expressed relative to stimulus offset (Fobel and Dau, 2004).

The duration of the M-Chirp was 10.32 ms and the duration of the A-Chirp was 7.87 ms—so the offset latencies for the chirps are measured relative to these durations. It should be noted from Fig. 1 that the acoustic durations of the chirps are less than the pre-transducer durations, due to frequency response of our earphones rolling off at 4 kHz. High-pass masking of the chirp has been shown previously to not affect wave V latency (Wegner and Dau, 2002), so we feel it is appropriate to measure offset from the end of the 10.4 kHz portion of the chirp as there is still a low frequency traveling-wave delay to be compensated for. All graphed and quoted latencies are corrected for the theoretical 0.83 ms acoustic delay along the 282 mm of ER-3A earphone tubing (Beauchaine *et al.*, 1987).

3. FFT spectrum analyses

To minimize artifacts when calculating the FFT we applied a Tukey cosine-tapered window (Geckinli and Yavuz, 1978), aligned relative to the location of the wave V peak. The window began 5.6 ms prior to wave V and ended 3.6 ms after; similar to the window parameters used by Fridman *et al.* (1984) to calculate synchrony. The window length was 301 samples and had a ratio of tapered sections to constant sections of 0.15. Pre-FFT windowing is important to increase side-lobe attenuation and reduce high-frequency artifacts that appear when start or end points are nonzero (Wilson and Aghdasi, 2001).

We zero-padded the data to extend the duration to 2048 samples before calculating the FFT. Zero-padding the ABR in this way increases the resolution of the FFT to approximately 16 Hz (Elberling, 1979).

We calculated the FFT across frequencies for each subject and analyzed the locations and magnitudes of the three major peaks that appeared in the FFT spectrum. Overall FFT spectra were generated by averaging the FFT for each test subject, as opposed to calculating the FFT of the grand average, as individual FFT spectra were required for statistical testing.

4. Component synchrony measures (CSMs) and SM

The first step to obtaining the SM is to calculate CSMs at each frequency. The CSM is a measure of phase variance of a given component frequency over a number of sub-averages of the evoked response. The CSM ranges from 0 (for large variances and apparent dissynchrony) to 1 (for responses that are perfectly synchronized).

To calculate the CSMs for each evoked response we followed the methods of [Fridman et al. \(1984\)](#); separating all available data, post artifact-rejection, into N sub-averages of 100 repetitions. Sub-averages with less than 100 repetitions were excluded. After removing any dc offset from each sub-average, we applied the same zero-padding and Tukey window as for the previous FFT spectrum analyses. We calculated the FFT of each windowed sub-average and measured the phase of each component frequency. The variances of all phase measurements were calculated as per [Mardia \(1972\)](#);

$$\text{var}\{\varphi(m)\} = 1 - \left[\frac{1}{N} \sum_N \cos \varphi_i(m) \right]^2 - \left[\frac{1}{N} \sum_N \sin \varphi_i(m) \right]^2,$$

where N is the number of sub-averages, and $\varphi_i(m)$ is the phase of the m th Fourier component of the i th sub-average.

The CSM was then taken as

$$\text{CSM}(m) = 1 - \text{var}\{\varphi(m)\}.$$

We report only three representative CSMs, the frequencies of which were chosen to correspond to the three major peaks of the FFT. However, the SM was calculated to be the average of CSMs from every available frequency up to the 3 kHz limit of our low pass filter;

$$\text{SM} = \frac{1}{M} \sum_M \text{CSM}(m),$$

where M is the number of Fourier components included in the calculation.

CSM measurements above 3 kHz can be considered noise, as they are in the stop-band of our low pass filter, and are therefore not relevant to our comparisons.

If the SM calculated for test data significantly exceeds the SM value which is expected for noise one can assume the presence of a signal. For click-evoked ABR, [Fridman et al. \(1984\)](#) prescribed a SM threshold of 0.22, at which the probability that an evoked response is present is very high (99.9%).

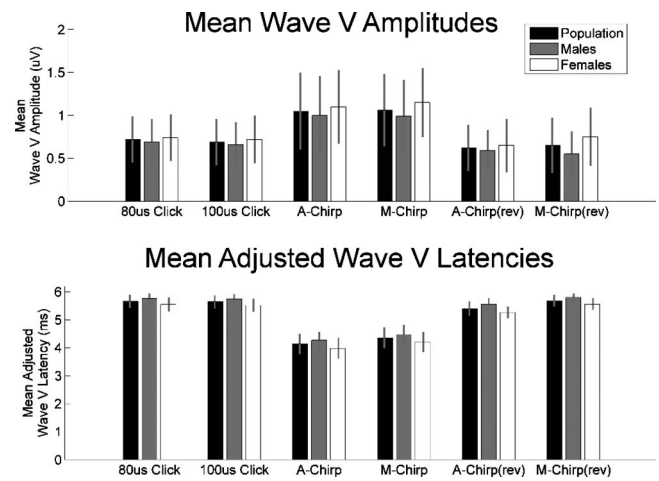


FIG. 3. Mean wave V amplitudes (top) and mean adjusted wave V latencies (bottom) for our test populations. Latencies were adjusted for the ER-3A tubing delay and, for rising-frequency chirps, the duration of the chirp was subtracted to give latency relative to stimulus offset. Vertical lines on each bar indicate one standard deviation.

5. Statistical testing

For the wave V latency variable only, we used Bartlett's test ([McDonald, 2008](#)) to test for equality of variances across stimulus groups against the alternative that variances are unequal for at least two stimulus groups. If the chirps evoke more synchronized ABRs, we would expect their variance to be less, despite their absolute latency being greater.

For all measured variables, we used Friedman tests ([Sheldon et al., 1996](#)) and Mann-Whitney ranked sum analysis tests to test for differences across stimulus groups. We used non-parametric tests since not all data sets satisfied the normality and homoscedasticity assumptions required for the use of parametric analyses. To compensate for the exclusion of ensemble averages that did not reach our F_{SP} threshold we utilized an extension of the Friedman test that allowed for unequal numbers of observations per test subject ([Kroon and Laan, 1983](#)). Whenever the Friedman result was significant we followed with multiple Mann-Whitney ranked sum analyses on samples evoked by each of the stimuli. We included the Bonferroni correction for multiple comparisons but, to maintain statistical power, we limited our planned comparisons to only those that directly involved each of the chirps. Our comparisons were therefore; each chirp to each click, each reverse-chirp to each click, and each chirp to its corresponding reverse-chirp. This gave ten permutations and a Bonferroni corrected significance level of $0.05/10=0.005$ ([McDonald, 2008](#)).

In all our measurements and comparisons, we report the result for the overall population and then separate the results for each gender to determine if the observed effects were specific to physiological differences between genders, such as cochlear length ([Don et al., 1993](#)).

III. RESULTS

Figure 3 shows the mean wave V amplitudes (top) and adjusted latencies (bottom) for males, females, and the over-

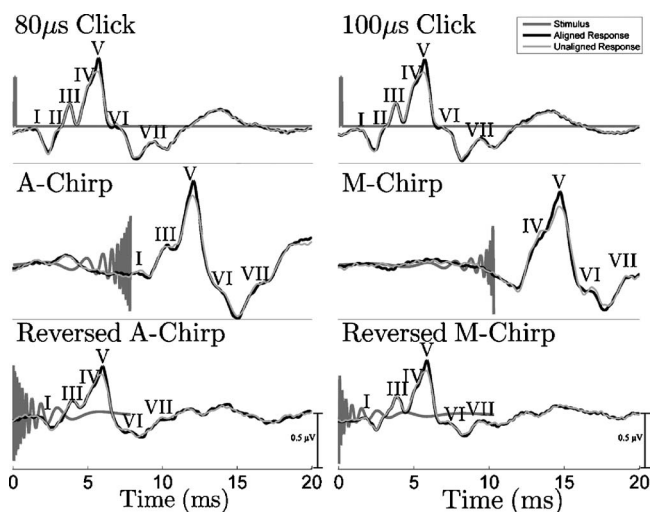


FIG. 4. Grand average waveforms (in bold) were assembled from all waveforms at a F_{SP} threshold of 3.1, after first aligning wave V locations to the group average. Vertical axis is microvolts, horizontal axis is milliseconds following stimulus onset (corrected for ER-3A tubing delay). The time course of each evoking stimulus is shown in gray (not to scale).

all test population. Figure 4 shows grand averages for the overall population; before (gray) and after (black) wave V alignment.

We expect that, if wave V is enhanced due to increased synchrony, then the earlier waves would also be enhanced. However, the grand averages (Fig. 4) suggest that the earlier waves were noticeably reduced for the chirps. Wave I is barely visible for the A-Chirp and its precise location is difficult to identify. Similarly for the M-Chirp, there is no pronounced trough following wave I that allows for accurate determination of the location of the wave I peak. Wave II for the clicks is present on the left-shoulder of wave III, but for the chirps wave II is absent. Wave III is also absent for the M-Chirp.

The grand averages for the ABRs evoked by reversed-chirps are similar to that of the clicks, but the amplitudes are smaller and some peaks are missing. In particular, wave II is barely visible as an inflection on the slope preceding wave III.

The difference between pre-aligned (Fig. 4, gray traces) and post-aligned (Fig. 4, black traces) morphologies is slight, except for a predictable increase in wave V amplitudes. It could be argued that the grand averaging process itself may have degraded wave morphologies for the chirps due to varying inter-peak latencies among subjects. To investigate if this is the case we show chirp-evoked ABRs for three representative subjects in Fig. 5. The amplitude scale here is twice that for the grand averages, and there are three repeat traces per plot. Although not shown, the click-evoked ABRs for these subjects were unexceptional and had similar morphologies to those of the grand averages. The first row in Fig. 5, for subject 18, shows chirp-evoked morphologies that allow identification of almost all the waves, although there are diminished wave I amplitudes for the M-Chirp. The second row, subject 31, shows a clear morphology for the A-Chirp, but a merging of waves III and V for the M-Chirp and again a diminished wave I. Finally, subject 3 in the last row shows

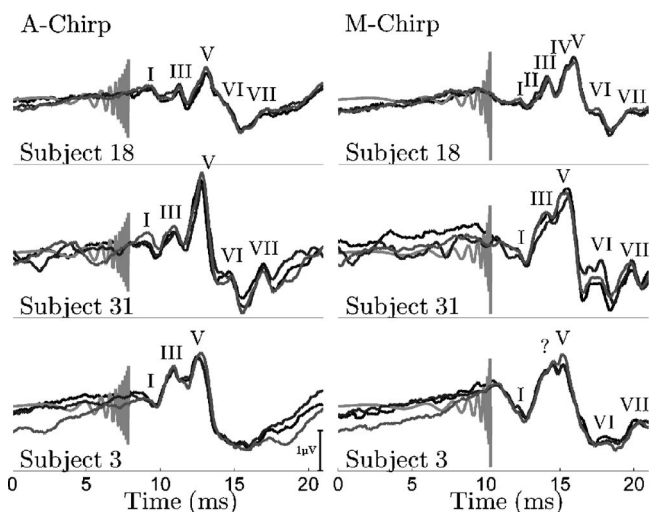


FIG. 5. Chirp-evoked ABR waveforms for three representative subjects. The left column are ABRs evoked by the A-Chirp, and the right column are ABRs evoked by the M-Chirp. Three repeat traces are shown to indicate repeatability. Morphology differences exist across the subjects shown. For the M-Chirp of subject 3, the “?” indicates an uncertainty in identifying peak III and IV. Vertical axis is microvolts, horizontal axis is milliseconds following stimulus onset (corrected for ER-3A tubing delay). The time course of each evoking stimulus is shown in gray (not to scale).

a distinctly different morphology—wave VI has disappeared for the A-Chirp, and wave III for the M-Chirp is now indistinguishable from what appears to be a wave IV+V complex.

In our measurements of wave V amplitudes, the chirps evoked larger amplitudes than the clicks and reverse chirps. The Mann-Whitney comparisons found this result to be significant ($p < 0.05$) for males, females, and the overall population—consistent with previous reports (e.g., Bell *et al.*, 2002a; Dau *et al.*, 2000; Elberling and Don, 2008; Fobel and Dau, 2004).

The effect of stimulus choice on adjusted wave V latencies, according to the Friedman analysis, was very highly significant ($p < 0.001$) for all groups. Subsequent Mann-Whitney comparisons confirmed that the adjusted location of wave V for the A-Chirps was smaller ($p < 0.05$) than for reverse A-Chirps, which in turn had smaller ($p < 0.05$) adjusted wave V latencies than the click stimuli. Adjusted wave V latencies for the M-Chirp were significantly smaller ($p < 0.05$) than those of the reverse M-Chirp and the clicks. We note for the reverse chirps that their electrical onset did not necessarily match their acoustic onset. If compensation is made for this, then the adjusted wave V latencies for the reverse M-Chirp (only) match those of the clicks. Otherwise, the reverse A- and M-Chirps have wave V latencies that are significantly longer ($p < 0.05$) than for the clicks.

For Bartlett’s test of difference in latency variances, we rejected the null hypothesis that all stimuli groups had equal variance, at the very highly significant level ($p < 0.001$) for the overall population and the male population. The pairwise F-test found the A-Chirps had significantly greater ($p < 0.05$) wave V latency variance than the clicks, for the overall population and the male subgroup. Similarly, the M-Chirps had significantly greater ($p < 0.05$) wave V latency variance than the clicks and the reverse M-Chirps. For the

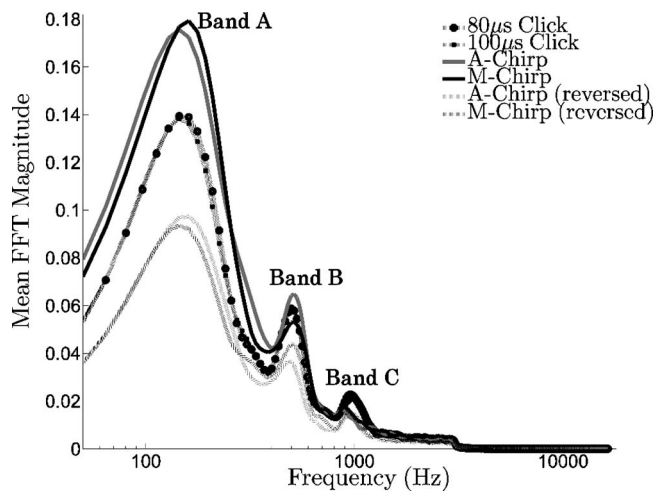


FIG. 6. Fast Fourier transforms demonstrate the differing frequency compositions of ABRs evoked by the stimuli in this study. The ranking of FFT magnitudes below 200 Hz is almost in tandem with the results for wave V amplitudes.

female subgroup, Bartlett's test indicated that stimulus choice had a highly significant ($p < 0.01$) effect on latency variance. For the paired F-tests on the female data, however, we were unable to reject the null hypotheses at the Bonferroni-corrected levels.

In the FFT plots (Fig. 6), the three predominant peaks at the center of each 'band'—A, B and C—had frequencies and relative magnitudes consistent with previous reports (Elberling, 1979; Suzuki *et al.*, 1982; Wilson and Aghdasi, 1999). In the CSM versus component frequency plots (Fig. 7), the three predominant peaks were at the same frequencies as for the FFT spectra, but the magnitude of each peak now reflected the level of synchrony at that particular frequency.

In band A, the frequencies of which are typically associated with wave V (Suzuki *et al.*, 1982), the FFT magnitudes were significantly larger ($p < 0.05$) for chirps.

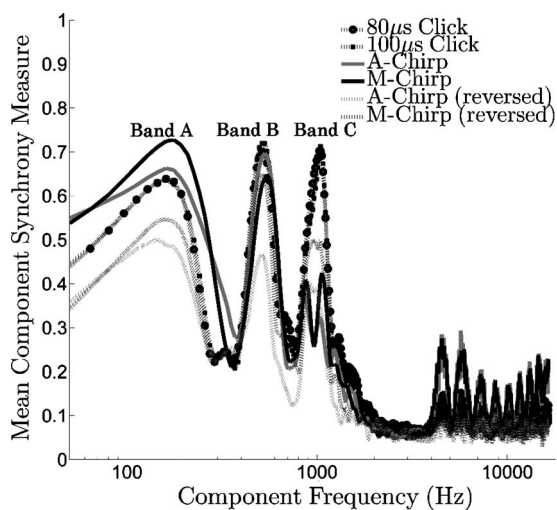


FIG. 7. The component synchrony measure, calculated at each component frequency in the FFT, indicates the phase variance across multiple ABR sub-averages. A component frequency with a CSM close to 1 is highly synchronized with the stimulus onset, whereas a component frequency with a CSM close to 0 is unsynchronized—possibly as a result of neural jitter or interference from background physiological noise.

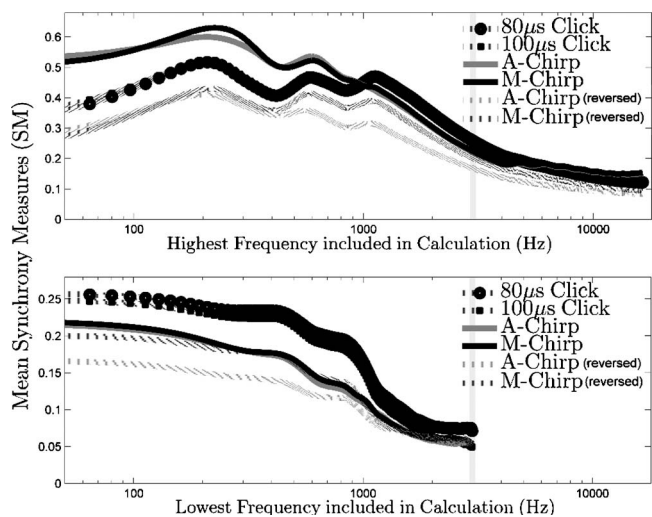


FIG. 8. (Top) The overall SMs in this study were calculated to be the average of all CSMs up to our low pass filter cut off of 3 kHz (vertical line). The component synchrony measures for A- and M-Chirps at frequencies above 1000 Hz were lower than those of the other stimuli in this study, resulting in an impaired SM. (Bottom—note the change in ordinate scale.) Here we demonstrate the effect of limiting low frequency contributions to the SM calculation. The upper frequency limit is fixed at 3 kHz while the high-pass cut-off is varied from 0 to 3 kHz. The click-evoked ABRs exhibit greater synchrony measures irrespective of high-pass cut-off.

In band B, the reverse A-Chirp had significantly smaller CSM values and FFT magnitudes than the other stimuli, for the overall population and the male subgroup.

In band C, the FFT magnitudes for the chirp- and reverse chirp-evoked ABRs were significantly smaller ($p < 0.05$), and the phase variances were greater and therefore the CSM values were significantly smaller ($p < 0.05$), than for clicks.

When calculating the SM, we summed the CSMs up to our low-pass filter cutoff frequency of 3 kHz. The effect of artificially limiting frequency contributions on the mean synchrony measure was investigated for the overall population and is shown in Fig. 8. In a low-pass analysis (Fig. 8, top), chirps have a larger SM value only if the summation is limited to contributions below approximately 600 Hz. In a high-pass analysis (Fig. 8, bottom), with the upper frequency fixed at 3 kHz, it is clear that the chirps have a smaller SM value than the clicks no matter where the lower bound is placed. Therefore, in our summation from 0 to 3 kHz, the SM values for chirps were significantly smaller ($p < 0.05$) than for the clicks, in spite of the larger observed wave V amplitudes. The smallest values of SM were for the reverse chirps, consistent with the notion that a reverse chirp should evoke a dissynchronous response (Dau *et al.*, 2000; Shore and Nuttall, 1985).

IV. DISCUSSION

A. Synchrony of chirp-evoked ABRs

1. Evidence for reduced synchrony

In this study we analyzed chirp- and click-evoked ABRs, using a variety of tools to measure synchrony in the response. Our results strongly suggest that the chirp-evoked ABRs in this study exhibited less synchrony than the click-

evoked ABRs. This was evidenced by the chirp-evoked ABRs showing an absence of earlier waves in the grand averages (Fig. 4), larger wave V latency variance, smaller FFT magnitudes at the higher component frequencies (band C) and larger phase variance at the higher component frequencies (band C), compared to the click-evoked ABRs.

This evidence for poorer synchrony in the chirp-evoked ABRs was present despite the rising chirp-evoked ABRs showing larger wave V amplitudes than the click-evoked ABRs, with these larger wave V amplitudes being consistent with several previous reports (e.g., Bell *et al.*, 2002a; Dau *et al.*, 2000; Elberling and Don, 2008; Fobel and Dau, 2004).

Investigation of the band A (the lower frequency band) FFT findings showed the significantly larger wave V amplitudes elicited by the chirp stimuli were consistent with the significantly larger FFT magnitudes elicited in band A by these same stimuli, with this band known to contribute the most to wave V amplitude (Suzuki *et al.*, 1982). It also showed that although the FFT magnitudes in band A were larger for the chirps, there were no significant differences in the CSM in band A between A-Chirps and clicks, suggesting that synchrony was similar in this band for those stimuli. Therefore, our result that the FFT magnitude was significantly greater for the chirps would suggest that increased neural recruitment, rather than increased neural synchrony, was the greater contributor to wave V for the chirps.

For the band B (the mid frequency band) FFT findings, the significantly smaller CSM values and FFT magnitudes elicited by the reverse A-Chirp were consistent with the morphology of the grand average, with band B known to contribute to waves I, III, V, VI and VII (Yokoyama *et al.*, 1994). In the grand average for the reverse A-Chirp (Fig. 4) we see that the trough following wave VI is apparently smaller than for any of the other stimuli, and the trough following wave III is smaller than for the clicks.

For the band C (the higher frequency band) FFT findings, the significantly smaller CSM and FFT magnitudes elicited by the chirp- and reverse chirps (compared to clicks) was again consistent with observations in the grand averages, with band C known to contribute to waves I, II, III, IV and V (Yokoyama *et al.*, 1994). We noted that the absence of the earlier waves was particularly evident for the A- and M-Chirps. The reverse chirps also appear to have comparatively diminished wave I amplitudes and poorer definition at wave II.

The absence of the earlier waves in the A- and M-Chirp grand averages also has a contribution from morphological variation across our test population prior to averaging. The responses in Fig. 5 demonstrate the differences in chirp-evoked morphologies that we observed for three representative subjects. The III-V inter-peak delay for the M-Chirp ABRs in Fig. 5 (right column) appears to vary across subjects, and is therefore likely to be the cause of a lack of wave III definition in the grand average. There also appears to be a trend for the M-Chirp data whereby the ABRs with shorter wave V latencies have poorer definition of individual peaks. These shorter latencies could indicate an insensitive response, where the low frequency signal components in the chirp may have been intense enough to stimulate higher

characteristic frequency (CF) portions of the cochlea. Dau *et al.* (2000) showed click-evoked ABRs that maintained a fairly consistent morphology across a range of intensities, whereas the morphologies of their chirp-evoked ABRs (when no spectral-weighting was applied to the stimulus) were strongly affected by intensity (see Dau *et al.*, 2000; Fig. 2). Although we applied a flat spectral weighting to our chirps, the reduced frequency response of the ER-3A earphones may have placed more emphasis on the contributions from the lower signal frequencies. A similar effect can also be seen in studies that use high-pass masking to limit high frequency contributions (e.g., Wegner and Dau, 2002).

2. Phase dispersion within frequency channels and reduced synchrony

We propose that the chirps in our study were able to increase neural recruitment by more synchronous displacement of the basilar membrane, but that the chirp-evoked ABRs contained more phase interactions, and were therefore less synchronous on the whole, than the click-evoked ABRs. To explore this hypothesis we can examine the process of mechanical to neural conversion that occurs on the basilar membrane. When stapes vibrations at the oval window set the cochlear fluid into motion, the response of the BM is a traveling wave propagating from the base to the apex of the cochlea. The stiffness gradient of the basilar membrane causes an intrinsic asymmetry whereby the traveling wave envelope has a shallower roll-off basally than apically (Burkard and Don, 2006). For a pure-tone stimulus the traveling wave amplitude comes to a maximum when it reaches its place of CF. The cochlear transport time (the delay from cochlea excitation to neural excitation) is therefore frequency-dependent and determined largely by passive BM properties.

Basilar membrane motion has been further described as a transmission line filter-bank that models spatial dispersion along coupled sections of the cochlear partition (e.g., Giguère and Woodland, 1994). When a portion of the BM is displaced, the delay until eighth nerve firing is determined by the cochlear filter build-up time, which is the filter impulse response time at the cochlear site of activation (Burkard and Don, 2006). Transfer functions of the cochlear filters exhibit phase dispersion in the form of frequency ‘glides’ (de Boer and Nuttall, 1997), which have been reported in BM vibrations and auditory nerve (AN) firing rates (Tan and Carney, 2003). These glides have complex behavior; increasing or decreasing in instantaneous frequency dependent on stimulus frequency (Carney *et al.*, 1999; Tan and Carney, 2003).

Rising-frequency chirps are designed to compensate for cochlear transport times (delays between frequency channels) but do not compensate for within-channel responses (glides). In fact, a direct consequence of the coupling between channels is that the impulse responses within channels are much longer for rising-frequency chirps than for clicks (Uppenkamp *et al.*, 2001). The asymmetry of the mechanical response imposes a physical limit to which one can simultaneously decrease the temporal dispersion of the impulse responses and the time delays between frequency channels (Uppenkamp *et al.*, 2001). Therefore, it is not possible to

create a stimulus that compensates both for cochlear transport time differences across frequency and for the phase curvature in the individual auditory filters (Rupp *et al.*, 2008).

Although compensation for time delays across frequency channels enhances wave V, the effect of within-channel phase dispersion is to decrease the amplitudes of earlier ABR waves. Auditory nerve fibers (the generators of waves I and II) exhibit ‘phase locking’, whereby an accurate reproduction of BM motion can be reconstructed from the timing of action potentials. Phase locking becomes less prevalent further up the auditory pathway with each additional synapse increasing the temporal jitter, and most cells beyond the auditory nerve showing indications of temporal or spatial integration (Palmer, 2006). In far-field recordings this means that within-channel dispersion is likely to cause destructive interference in the ABR of earlier ‘fast’ waves (i.e., waves I and II), whereas the temporal integration at higher levels enhances the amplitudes of later ‘slow’ waves (i.e., wave V). This is confirmed in frequency spectrum analysis of the ABR: Wave V is a low frequency wave (with a long integration window) whereas waves I and II are integrated over a much shorter window (Kevanishvili and Aphonchenko, 1979). The greater sensitivity of wave I, as compared with wave V, to phase dispersion is also explained by the differences in the mechanism of their generation. It is considered, in particular, that neural elements of wave V are of a dendritic type and therefore, are slower and monophasic, while those of wave I are of an axon type being, therefore, faster and diphasic (Kevanishvili and Lagidze, 1979).

The synchrony measures used in the present study are likely to reflect the phase dispersion of these impulse responses at each local cochlea filter. For chirps, as was found in previous studies, the filter impulse responses are more complex and temporally dispersed than for clicks (Dau, 2003). The ringing of chirp-evoked impulse responses is exaggerated in the low frequency portions, due to a reduction in traveling-wave velocity (Don *et al.*, 1994) and backward-traveling waves that reflect from the apex to the base (Carney *et al.*, 1999; Nábelek, 1978; Shera *et al.*, 2004), and results in a psychoacoustic reduction in perceived ‘compactness’ for chirps (Aiba *et al.*, 2008; A. Rupp *et al.*, 2002). By compensating for traveling wave delays down the BM, and therefore including the response from the lower frequency cochlea regions, we risk introducing latency jitter and phase distortion into the evoked response.

B. Additional factors contributing to reduced synchrony

1. Stimulus intensity

The adjusted wave V latencies between click- and chirp-evoked ABRs should be similar (Fobel and Dau, 2004). However, we found that the adjusted latencies of the chirp ABRs were significantly smaller than the click ABRs. Based on the rise time of the chirp frequency sweep we calculated that, to match the wave V latencies of the click-evoked ABRs, the evoked responses to our chirp stimuli were initiated when the instantaneous frequency was still below 2 kHz.

Previous reports have also shown disparities between click- and chirp-evoked offset wave V latencies in the order of 1–1.5 ms (Bell *et al.*, 2002b; Elberling and Don, 2008; Fobel and Dau, 2004). An explanation for this is offered that at higher intensities, “the early low-frequency energy of the chirp probably stimulates basal regions of the BM due to upward spread of excitation” (Dau *et al.*, 2000). Wegner and Dau (2002) quantified this for low frequency chirps, with and without high-pass masking, and confirmed that basalward spreads were occurring at higher intensities, including at 40 dB HL used in the present study.

If the low frequency components of the chirp stimulate high CF neurons at high intensities, then their response will be inhibited when the high frequency components of the chirp eventually arrive (Nábelek, 1978; Shore and Nuttall, 1985). The basal spread of excitation of the early low-frequency signal components in the chirp superimposes with the later activity from the mid and high frequencies (Dau, 2003). Additionally, each cycle of the chirp stimuli will excite a broader area of the cochlea at higher levels, resulting in some overlap and consequent desynchronization (Elberling and Don, 2008). In contrast, click-evoked activity is still well synchronized in mid- and high-frequency regions (Dau, 2003), presumably since the lower frequency regions of the cochlea respond later (Pantev *et al.*, 1985). Therefore, the comparative advantage in evoked amplitudes given by chirps, versus clicks, reduces with increasing stimulus intensity (Elberling and Don, 2008). Previous studies have suggested that the basal spread of excitation can be reduced by applying an envelope to the chirp (Neumann *et al.*, 1994; Spankovich *et al.*, 2008), or by adding additional cycles of the end point frequencies (Shore and Nuttall, 1985).

2. Misalignment of chirp instantaneous frequency to BM characteristic frequencies

We found that the inter-subject variance in wave V latency was significantly larger for chirps than for click stimuli. This could indicate that the temporal compensation offered by the chirp was not optimal for all subjects. Previous studies of chirp-evoked responses have found that while certain sweep rates minimize the temporal delay between unit responses, slower sweep rates only partially cancel travel time; initiating a response from the lower frequency portions before the higher frequency portions (Shore *et al.*, 1987). The observed effect would be a reduction in FFT magnitudes at higher frequencies, as the higher frequency magnitudes are reduced with decreasing stimulus frequency (Suzuki *et al.*, 1982).

The sweep rate for the A-Chirps is derived from tone-evoked wave V latencies which must necessarily include measurement errors. Low-frequency tone-evoked responses are compromised between the need for rapid onset (to elicit the ABR) and the need to minimize spectral spread (Gorga *et al.*, 1988). Accurate mapping of the true response times of the apical end of the cochlea is additionally confounded by the problem of knowing from where on the rising-slope of the stimulus to measure the response latency (Brinkmann and Scherg, 1979). Some researchers have attempted to resolve these inaccuracies by instead basing a chirp on

derived-band responses, but the resulting inter-subject latency variation has still been found to be large (Elberling and Don, 2008; Elberling *et al.*, 2007). This could indicate that the required sweep rate to compensate for cochlea delays varies from subject to subject. This is somewhat evident in our representative ABRs (Fig. 5): Subject 31 has a clear morphology for the A-Chirp (a fast sweep) whereas the morphology for the M-Chirp (a slower sweep) is distinctly different.

C. Clinical implications

1. Clinical applications for chirps

That the chirp-evoked ABR amplitudes were significantly larger than the click-evoked ABR amplitudes is of great benefit for hearing screening, since test times are likely to be shorter when utilizing chirps—and false referral rates smaller. However, the reduced synchrony evident in the chirp-evoked ABRs reduces their suitability to more complicated diagnostic procedures, since a chirp based on population or modeled data does not adequately compensate for differences in individual cochlea responses.

When considering individual cochlear responses and chirp-evoked ABRs, many variables must be considered. Post-mortem analyses of human cochleae have revealed that inter-subject basilar membrane lengths are highly variable within normal populations and that the Greenwood frequency-position function (Greenwood, 1990), upon which the M-Chirp is based, has limited accuracy (Sridhar *et al.*, 2006). The stiffness gradient of the cochlea may be larger in females than in males, corresponding to a shorter cochlea length in females (Don *et al.*, 1993). A shorter cochlea leads to a faster response time, which in turn leads to better neural synchrony and larger wave V amplitudes (Don *et al.*, 1993). These gender differences were supported by the larger wave V amplitudes and smaller wave V latencies in all ABRs in our female population, and our larger wave V latency variances in all groups for our chirp-evoked ABRs compared to our click-evoked ABRs in our male and overall populations. Finally, differences in cochlear response times between frequency regions are expected between individuals due to local differences in the sharpness of tuning as well as thresholds along the cochlea (Don *et al.*, 1994). These many differences in individual cochlea responses led Elberling and Don (2008) to suggest that the “chirp ABR systematically emphasizes individual characteristics,” therefore making it more difficult to establish normative data for chirp-evoked ABRs compared to click-evoked ABRs. In this regard, it may be of greater benefit to limit the range of frequencies encompassed within the chirp sweep. If the lower frequencies are omitted, their contribution to dissynchrony is also removed. In applications where synchrony is of greater concern than wave V amplitude, clinicians may prefer to establish normative values to, for example, band-limited chirps (Bell *et al.*, 2002a; Wegner and Dau, 2002), or chirps that have been separated into low- and high-components (Cebulla *et al.*, 2007; Plotz *et al.*, 2006).

Finally, although we observed an attenuation of earlier waves in our chirp-evoked ABRs (Figs. 4 and 5), there are reports that A- and M-Chirp-evoked ABRs have a clear wave

I down to at least 30 dB SL (Fobel and Dau, 2004). Our electrode montage and earphone selection differ from these previous studies, but our click-evoked ABRs do not seem as affected by these factors. If our assumption is correct that the required compensation for cochlea delays varies between subjects, then the expected morphologies for chirp-evoked ABR are also dependent on the suitability of the chirp to the cochlea under test. This is subjectively evident in the range of morphologies exhibited for chirp-evoked ABR in Fig. 5.

2. Individualized chirps

Chirp stimuli have been based on modeled data (Dau *et al.*, 2000), previously published wave V latencies (Elberling and Don, 2008; Fobel and Dau, 2004; Lütkenhöner *et al.*, 1990), and on average latencies of the population under test (Spankovich *et al.*, 2008). As discussed above however, differences in chirp-evoked ABRs across populations are thought to reflect differences in individual cochlear responses between individual test subjects. With these differences in mind, it seems enticing to extend the utility of the chirp by matching the travel time compensation to the individual test subject (Petoe *et al.*, 2007).

This may allow physiological and neurological modeling of individual cochleae, enhancing traditional diagnostics and allowing for more complicated procedures such as determining frequency-position functions. Unfortunately, in a pathological ear, local thresholds along the basilar membrane are likely to be highly variable. This means that the amplitude envelope of the chirp may require compensation to behavioral thresholds. Previous work on chirp amplitude envelopes have suggested using the psychoacoustic Bark scale as it distributes energy uniformly over each of the critical bands (Neumann *et al.*, 1994). Additionally, it has been suggested that the envelope of the chirp needs to compensate for the attenuation of signal frequency components above and below the principal middle-ear resonant frequency (Shore and Nuttall, 1985).

D. Conclusions

Our primary conclusions may be summarized as follows.

- We confirmed earlier reports that wave V amplitude is enhanced when using a chirp as the stimulus.
- We found, using a variety of synchrony measures, that the chirp-evoked ABRs in this study exhibited greater phase variance (jitter), indicative of reduced synchrony. It is hypothesized that the chirps enhance the amplitude of wave V by recruiting a greater number of neurons, but do not comprehensively synchronize the compound response.
- The earlier waves of the ABR were diminished or absent in the grand average for our chirp-evoked ABRs. Inter-subject variation in traveling wave delay down the cochlea (Don *et al.*, 1994), as well as phase dispersion within frequency channels, is likely to have smeared the earlier waves in the chirp-evoked ABRs.
- The chirp-evoked ABRs had a larger inter-subject variation in wave V latency than the click-evoked ABRs. This is presumably due to differences in the suitability of each

chirp to the individual physiologies, and has negative repercussions for clinics wishing to establish normative latencies for chirp-evoked ABR.

Although these conclusions suggest that chirps evoke a less synchronous response than click stimuli this is unlikely to be the generalized case. The conclusions of this research are limited to the relatively high presentation intensities used (40 dB HL), the earphone selection (Etymotic ER-3As with a limited frequency response), and the suitability of the chirp stimuli to the cochleae under test. If previous studies are correct in their suggestion that a basal spread of excitation at high intensities reduces the advantage of chirps over clicks then empirical analyses of synchrony at lower intensities may yield different results.

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