Group Delay of Acoustic Emissions in the Ear

Tianying Ren, Wenxuan He, Matthews Scott, and Alfred L. Nuttall

J Neurophysiol 96: 2785–2791, 2006. First published August 9, 2006; doi:10.1152/jn.00374.2006. It is commonly accepted that the cochlea emits sound by a backward traveling wave along the cochlear partition. This belief is mainly based on an observation that the group delay of the otoacoustic emission measured in the ear canal is twice as long as the forward delay. In this study, the otoacoustic emission was measured in the gerbil under anesthesia not only in the ear canal but also at the stapes, eliminating measurement errors arising from unknown external- and middle-ear delays. The emission group delay measured at the stapes was compared with the group delay of basilar membrane vibration at the putative emission-generation site, the forward delay. The results show that the total intracochlear delay of the emission is equal to or smaller than the forward delay. For emissions with an f2/f1 ratio <1.2, the data indicate that the reverse propagation of the emission from its generation site to the stapes is much faster than a forward traveling wave to the f2 location. In addition, that the round-trip delays are smaller than the forward delay implies a basal shift of the emission generation site, likely explained by the basal shift of primary-tone response peaks with increasing intensity. However, for emissions with an f1 ≪ f2, the data cannot distinguish backward traveling waves from compression waves because of a very small f1 delay at the f2 site.

INTRODUCTION

The human and animal cochlea not only senses a variety of environmental sounds but also generates sounds. Ear-generated sound can be recorded in the external ear canal using a sensitive miniature microphone and is termed an otoacoustic emission (Kemp 1978). When two tones generated by two speakers at frequencies f1 and f2 (f2 > f1) are delivered to the ear canal (Fig. 1), these airborne stimuli vibrate the eardrum and the middle-ear ossicular chain and so propagate into the cochlear fluids. These sounds in the fluid initiate the vibration of the basilar membrane (BM), a spiral membrane structure along the cochlear length (Cooper and Rhode 1997; Robles et al. 1991, 1997). BM vibrations travel to their best-frequency (BF) locations, where the vibration shows the largest amplitude and nonlinearity, i.e., nonproportional growth with stimulus intensity (Rhode 1971; Robles and Ruggiero 2001) (f1 and f2 peaks on the right-side plots in Fig. 1). Distortion product otoacoustic emissions (DPOAEs) at frequencies (n + 1)f1−n f2 and (n + 1)f2−n f1 (n = 1, 2, 3, . . . ) are generated by nonlinear vibration at the f1 and f2 overlapping location (the DP site in Fig. 1) (Cooper and Rhode 1997; Dong and Olson 2005; Kim et al. 1980; Knight and Kemp 2001; Robles et al. 1991, 1997; Shera and Guinan 1999; Siegel et al. 1982; Tubis et al. 2000). The DPOAE at the frequency of 2f1−f2 has the largest amplitude among different emissions (Lonsbury-Martin et al. 1990; Probst et al. 1991) and is used to study the backward-transmission mechanism in this study.

To explain how cochlea-generated sound propagates backward from its generation site to the cochlear base, there are two different theories in the literature. The prevailing view is that the emission reaches the cochlear base by a backward-traveling wave, a slow-propagating transverse wave, along the BM (Bowman et al. 1998; Kemp 1986; Knight and Kemp 2001; Mahoney and Kemp 1995; Schoonhoven et al. 2001; Shera and Guinan 1999; Tubis et al. 2000). A different view is that the emission propagates to the cochlear base by the cochlear fluids as a longitudinal compression wave (Avan et al. 1998; Ren 2004; Robles et al. 1997; Ruggiero 2004; Siegel et al. 2005; Wilson 1980).

The round-trip delay of an emission measured as phase-frequency slope (i.e., group delay) in the human or animal ear canal is roughly twice as large as the forward delay (Kimberley et al. 1993; Mahoney and Kemp 1995; Schneider et al. 1999; Schoonhoven et al. 2001). This has been considered to be evidence of the backward-traveling wave. However, the round-trip delay reported in the literature was measured in the ear canal and includes delays arising from the external and middle ears. The aim of this study is to measure the round-trip group delay of the emission directly from the stapes and to compare the emission delay to the forward delay of a traveling wave.

METHODS

Animal preparation and general methods

Fourteen young, healthy Mongolian gerbils (40 to 80 g) were used in this study. The initial anesthesia was induced by intraperitoneal injection of ketamine (30 mg/kg) followed by intramuscular injection of xylazine (5 mg/kg). An additional half dose of the initial anesthetic was given based on the toe pinch reflex, the respiration rate, and interferometer-detected artifact signals caused by animal movement. The animal protocol was approved by the Institutional Animal Care and Use Committee of Oregon Health and Science University. Animal preparation and surgical approach were the same as in previous studies (Ren 2002, 2004). Briefly, the left auditory bulla was exposed through a ventral–lateral surgical approach. After the round window membrane was removed and a thin glass coverslip placed on the
enlarged round window, the laser beam of a heterodyne laser interferometer (OFV 3000S, Polytec, Waldbronn, Germany) was focused on the BM through an upright microscope. The voltage output of the laser interferometer was proportional to the velocity of the transverse vibration of the BM. The BF in the middle of the observed field was determined as the frequency with the maximum amplitude in a transfer function measured at 30 dB SPL (0 dB SPL = 20 µPa).

Sensitivity of the cochlea was monitored by measuring the compound action potential (CAP) using a previously described method (Ren 2002). The CAP threshold was measured before and after data collection. A cochlea with <8 dB threshold elevation at 18 kHz was considered sensitive.

Signal generation and data acquisition

A custom-written LabView-based (National Instruments, Austin, TX) program was used to control TDT hardware (System II, Tucker-Davis Technologies, Gainesville, FL) for signal generation and data acquisition. Tone bursts at f1 and f2 with 23-ms duration and 1-ms rise/fall time were generated by a D/A converter (DA3-4). The signals were connected to a dual-channel headphone buffer (HB6) through two programmable attenuators (PA4) and then used to drive two earphones (ER-2, Etymotic Research, Elk Grove Village, IL). A sensitive microphone (10 B+., Etymotic Research) was used to measure the sound pressure in the ear canal. The microphone-earphone probe was coupled into the external ear canal through a plastic coupler to form a closed sound field. Because of its length and small diameter, the coupler contributes to the measured group delay and level difference between the primary tones and the 2f1–f2 emission. Signals from the microphone and the interferometer were digitized and averaged 10 to 40 times, depending on the signal level. The magnitude and phase of the average signal at different frequencies and intensities were obtained through Fourier transform.

Group delay measurements

Sound pressure in the ear canal, the vibration of the stapes footplate, and the BM vibration at the f2 BF place were measured as a function of the frequency. Frequency of 2f1–f2 was varied by changing f1 while holding f2 constant. Magnitudes and phase of sound pressure and vibration at frequencies f1, f2, and 2f1–f2 were measured. The group delays of signals were derived from the phase-transfer functions. Phase was referred to the electrical signal from the D/A converter. The attenuator and earphone buffer introduced no significant delay. The group delay from speakers to the stapes was derived from the f1 phase-transfer function of the stapes vibration. The round-trip group delay of the emission was measured based on the phase-transfer function of the emission measured in the ear canal. The forward delay was derived from the phase-transfer function of the BM vibration measured near the f2 site. For measuring the transfer functions of the BM vibration, 23-ms tone bursts with 1-ms rise/fall time at frequencies from 500 Hz to 25 kHz in 500-Hz steps at the different intensities were presented. Magnitude and phase of the BM vibration were measured as a function of the frequency. The phase-transfer function of the BM vibration was obtained by subtracting the phase values of the stapes from those of the BM, and the corresponding magnitude transfer function was calculated by dividing the magnitudes of the BM responses by those of the stapes. To calculate group delay, the phase-transfer functions of the sound pressure in the ear canal and stapes vibration were fitted using a linear function. A third-order polynomial function was used to fit the phase-transfer function of the BM vibration. The group delays were calculated based on the fitted functions according to the equation: $D = D_{t}/\Delta\omega$, where $D$ is the group delay in seconds and $\Delta\omega$ is the phase difference in radians over the angular frequency change $\Delta\omega$.

RESULTS

All animals survived anesthesia and surgery. Six of fourteen preparations showed <8 dB hearing loss at 18 kHz when data were collected. The data presented in Figs. 2, 3, and 4 are from three of these sensitive animals. A similar example of data at a single primary level was presented at the cochlear mechanics workshop in Portland, OR (2005).

Magnitude and phase of the cubic DPOAE are presented in Fig. 2. A and B as a function of the emission frequency of 2f1–f2. Emissions were recorded at f1 levels from 45 to 75 dB SPL in 10-dB steps with f2 levels 5 dB below f1 (primary tone intensities in the following material are described using only f1 levels). At 45 dB SPL, the maximum emission occurs near 9 kHz (dash–dot line in Fig. 2A), with an f2/f1 ratio of about 1.3. The response maximum shifts to the low-frequency side and becomes broader with increasing intensity. The emission magnitude decreases as f2/f1 becomes close to 1, i.e., f1 approaches f2. Unevenly spaced magnitude curves at different intensities indicate frequency-dependent growth. For a given primary level f1, the DPOAE level at 2f1–f2 appears lower than that reported in the literature (Boettcher and Schmiedt 1995). This likely is caused by the use of a high-frequency f2 in this study. The high-frequency f2 is required here because only the high-frequency region of BM vibration could be measured using an interferometer in this study. The shape of the magnitude–frequency curve of the emission varies across animals. The emission phase curves in Fig. 2B show a negative linear relationship with the emission frequency 2f1–f2. Linear regression lines (solid lines in Fig. 2B) based on the phase curves present level-dependent phase slopes. The group delays derived from the slopes of the regression lines show that the greatest delay is 649 µs at 45 dB SPL, and the smallest is 478 µs at 65 dB SPL for this cochlea. For other animals the shortest group delays occurred at the highest intensity of 75 dB SPL.
Magnitudes and phases of the stapes vibration measured as functions of $2f_1–f_2$ and $f_1$ at 75 dB SPL are shown in Fig. 2. Stapes responses to stimuli at levels <75 dB SPL are not shown because the signal levels of the emission are close to or below the noise floor of the measurement. The frequency-dependent $f_1$ magnitude response likely results from transfer functions of the external and middle ears. The $2f_1–f_2$ magnitude curve shows a broad peak near 11 kHz ($f_2/f_1 \approx 1.21$).

Figure 2D shows that the phase of $f_1$ decreases with frequency. Although it is obvious that the slope of the $f_1$ phase demonstrates the group delay from the speaker to the stapes ($\tau_{\text{sp-mic}} \approx 223 \mu s$), this delay is not visible and is included in the emission group delay when measurements are made only in the ear canal. The phase curve of $2f_1–f_2$ indicates the emission group delay at the stapes ($\tau_{\text{2f}_1–f_2,\text{stapes}} = \tau_{\text{sp-st}} + \tau_{\text{forward}} + \tau_{\text{backward}} \approx 415 \mu s$). The cochlear round-trip group delay ($\tau_{\text{cochlear}}$) was obtained from $\tau_{\text{cochlear}} = \tau_{\text{2f}_1–f_2,\text{stables}} - \tau_{\text{sp-st}} = \tau_{\text{forward}} + \tau_{\text{backward}}$, which is 192 $\mu s$ (Fig. 2E).

Because $\tau_{\text{cochlear}} = \tau_{\text{forward}} + \tau_{\text{backward}}$, the backward group delay can be obtained by $\tau_{\text{backward}} = \tau_{\text{cochlear}} - \tau_{\text{forward}}$. The forward delay can be derived from the phase-transfer function of the BM vibration at the $f_2$ place. The magnitude and phase-transfer functions at the $f_2$ site were measured by varying the frequency of a single tone and are presented in Fig. 3. A and B. The peaks of magnitude transfer functions at low intensities in Fig. 3A indicate a BF of about 17 kHz, which is the same as $f_2$. As the stimulus intensity increases, the peak of the magnitude transfer function becomes broader and the peak-location shifts toward the low-frequency side. The response peak is at about 13 kHz at 85 dB SPL. Near the BF, the ratio of BM to stapes response magnitude decreases with the intensity. The compressive nonlinear growth, sharp tuning, and the peak shift to the low-frequency side with intensity, shown in Fig. 3A, demonstrate the healthy status of the preparation. Figure 3B shows that phase decreases with frequency and the phase slope becomes slightly flatter as intensity increases. Regression lines of the phase data (solid) and their residual values (dotted lines) were obtained using a polynomial fit, as plotted in Fig. 3C. Forward group delays calculated from Fig. 3C are plotted in Fig. 3D (solid lines). Delays increase with frequency, reaching about 320 $\mu s$ at 17 kHz, i.e., $f_2$ frequency. The forward delay also shows a slight level dependency, which decreases at frequencies >11 kHz and increases with intensity below this frequency. The cochlear round-trip delays ($\tau_{\text{cochlear}}$) at 45, 55, and 65 dB SPL were calculated as $\tau_{\text{cochlear}} = \tau_{\text{ everlasting}} - \tau_{\text{sp-st}} - \tau_{\text{sp-mic}}$ where $\tau_{\text{sp-st}}$ is 223 $\mu s$, as shown in Fig. 2D, and $\tau_{\text{sp-mic}}$ is the delay difference between $2f_1–f_2$ at the stapes (415 $\mu s$ in Fig. 2D) and $2f_1–f_2$ in the ear canal (525 $\mu s$ in Fig. 2B), i.e., 110 $\mu s$. Because of the linearity of the external- and middle-ear re-
sponses at intensities used in this study, \( \tau_{sp-st} \) and \( \tau_{sp-mic} \) should be independent of intensity. Cochlear round-trip delays at different intensities (horizontal dotted lines) were plotted together with the forward delays (solid lines) in Fig. 3D. It is evident that all horizontal dotted straight lines meet the solid lines at or below 17 kHz. This demonstrates that the roundtrip-delay of the emission in the cochlea is nearly the same as or smaller than the forward travel delay, indicating that the reverse propagation delay is very small, or the DPOAE is generated at the basal side of the best-frequency location of \( f_2 \). That the cochlear round-trip delay decreases with intensity (horizontal dotted lines Fig. 3D) indicates that the emission-generation location shifts toward the base with intensity. This interpretation is supported by the data that the response peak of the BM shifts to lower frequencies at high intensities (Fig. 3A).

To confirm the finding from the data in Figs. 2 and 3, a data set from a different sensitive cochlea is presented in Fig. 4. Data were collected at the f1 intensity of 75 dB SPL and at 14 kHz \( f_2 \). Magnitude and phase of the stapes vibration at \( 2f_1-f_2 \) (solid line) and \( f_1 \) (dotted line) are presented in A and B. To compare the stapes vibration to the emission, the DPOAE magnitude and phase (dashed lines) are plotted in Fig. 4, A and B. The stapes vibration and DPOAE amplitudes both show a similar pattern with an evident difference <10 kHz. This difference likely results from the middle- and external-ear transfer functions. The notch near 11 kHz depends on stimulus intensity and varies across animals. Group delays were calculated from the slopes of the linear regression lines of phase data and are presented in Fig. 4B. According to the emission delay at the stapes (\( \tau_{cochlear} = \tau_{2f_1-f_2stapes} = 424 \mu s \)) and the speaker-to-stapes delay (\( \tau_{sp-st} = 207 \mu s \), the cochlear round-trip delay (\( \tau_{cochlear} = \tau_{2f_1-f_2stapes} - \tau_{sp-st} = 217 \mu s \). Magnitude and phase-transfer functions of BM vibration at the \( f_2 \) place at different intensities are presented in Fig. 4, C and D. Sharp tuning and nonlinear growth indicate the healthy status of this preparation. The regression line of the phase data at 75 dB SPL (solid line) is plotted in Fig. 4E. Small residual values (dashed line) indicate a good fit of the data. Group delays derived from the fitted line in Fig. 4E are plotted as a function of frequency in Fig. 4F, which shows that the cochlear round-trip delay of \( 2f_1-f_2 \) emission (horizontal short dashes) is smaller than the forward delay (dotted horizontal line). Thus the data in Fig. 4 are consistent with the finding in Figs. 2 and 3 that the round-trip delay of the emission at the stapes is smaller than the forward delay to the \( f_2 \) site.

**DISCUSSION**

The backward-traveling-wave theory describes a transverse vibration along the cochlear partition traveling from the emission-generation site to the cochlear base at the same speed as a forward-traveling wave. Originally proposed by Kemp in 1986, this theory was based on the observation that cochlear-generated sound can be measured in the ear canal (Kemp 1978) and a mathematical demonstration that the cochlear traveling wave can travel in both directions (de Boer et al. 1986). Kemp noted that the idea of a backward-traveling-wave theory contradicts the well-known observations of Békésy that BM vibration travels only in a forward direction from base to apex (von Békésy 1960). He believed, however, that Békésy’s observation is true only for external sound-induced cochlear vibration, not for internal sound sources, such as otoacoustic emissions. The backward-traveling wave theory has been comprehensively studied mathematically and experimentally and has become widely accepted (Knight and Kemp 2001; Lukashkin et al. 2002; Schneider et al. 1999; Schoonhoven et al. 2001; Shera and Guinan 1999; Tubis et al. 2000; Withnell and McKinley 2005).

The compression-wave theory posits a longitudinal wave in the cochlear fluids, traveling at the speed of sound in water. The concept of the cochlear compression wave was first implied in a sensory outer-hair-cell swelling model by Wilson (1980), in which hair cell volume changes displace the stapes footplate and result in the emission. Although the hair cell-swelling mechanism is no longer considered likely, because of the required speed of volume changes, this theory implies that
a pressure wave in the cochlear fluids directly produces an otoacoustic emission. Compression-wave theories were subsequently advanced by a number of studies (Avan et al. 1998; Ren 2004; Robles et al. 1997; Ruggero 2004; Siegel et al. 2005).

Because the forward group delay of sound propagating from the stapes to its BF location can be measured experimentally by the phase-transfer function of BM vibration (Cooper and Rhode 1992; Nuttall and Dolan 1996; Rhode 1971; Robles and Ruggero 2001; Sellick et al. 1982), measuring the group delay of an otoacoustic emission and comparing it to the group delay of BM vibration has become an important way to distinguish backward-traveling waves from fluid-compression waves. That the emission group delay is twice the forward delay supports the backward-traveling wave theory, whereas the emission delay being equal to or smaller than the forward delay supports the compression wave theory. Narayan et al. (1998) recorded emissions in the ear canal simultaneously with BM vibration at the $f_2$ place and found that the emission group delay was similar to that of BM vibration. Narayan et al. believed that their data indicated that the emission group delay largely reflects “cochlear filter” delays at the BM (Narayan et al. 1998). One of the authors of the present work (Ren 2004) measured longitudinal patterns of the BM vibration at the emission frequency using a scanning laser interferometer and found that the phase of the BM response decreases as a function of the distance from the cochlear base. This result indicates that the BM vibration at the $2f_1–f_2$ frequency propagates in the forward direction. He also found that the emission group delay at the stapes is smaller than that of BM vibration near the emission-generation site. These findings by Narayan et al. (1998) and Ren (2004) support the compression-wave theory. The compression-wave theory is also supported by the fact that frog ears generate otoacoustic emissions even though their hearing organs do not rest on a BM (van Dijk and Manley 2001). However, Cooper and Shera (2004) found that the emission group delay is twice the forward delay based on simultaneous recordings of the emission and BM vibration in the guinea pig. Ruggero (2004) attributed the above discrepancies to different stimulus paradigms, BM-vibration measurement locations, and cochlear sensitivity.

All emission data in the literature known to us, which show that the emission round-trip delay is twice the forward delay, were measured in the ear canal. Figure 1, however, shows that the group delay of the emission in the ear canal is determined by the delay from the speaker to the stapes ($\tau_{sp-st}$), the forward delay from the stapes to emission-generation site ($\tau_{forward}$), the backward delay from this site to the stapes ($\tau_{backward}$), and the delay from the stapes to the microphone ($\tau_{sp-mic}$). It is impossible to calculate $\tau_{backward}$ unless the other delays are known. Kimberley et al. (1993) were aware that $\tau_{sp-st}$ and $\tau_{sp-mic}$ contribute to the round-trip delay of the emission measured in the ear canal. These delays, however, were believed to be insignificant and were ignored in previous studies (Kimberley et al. 1993; Mahoney and Kemp 1995; Schneider et al. 1999). Here, we measured the emission group delays in the ear canal and at the stapes and the forward group delay at the putative emission generation site on the BM. The data in Figs. 2B and 4B also show that the group delay of the $2f_1–f_2$ emission measured in the ear canal is roughly twice the forward group delay (Fig. 3D); this is consistent with previous reports (Kimberley et al. 1993; Mahoney and Kemp 1995; Schneider et al. 1999; Schoonhoven et al. 2001). The group delay of the emission at the stapes, however, is nearly equal to or smaller than the forward group delay.

Differences between the results of this study and those of previous studies are due to the delays of the external and middle ears. In contrast to previous studies, the emission was measured as stapes vibration in this study and the external and middle ear delays were not included in the emission round-trip delay. In Fig. 1, the delays of $\tau_{sp-st}$ and $\tau_{sp-mic}$ are determined by the speed of sound in air (about 0.34 mm/µs) and the distances between the speaker and the stapes and between the stapes and the microphone. Although the speed of sound in air is relatively constant, the distance can vary dramatically across different studies. The group delay of $2f_1–f_2$ emission has been measured based on the phase-frequency response (Kimberley et al. 1993; Mahoney and Kemp 1995; Schneider et al. 1999) and the signal onset time (Talmadge et al. 1999; Withnell and McKinley 2005). Either method may not accurately measure the true traveling delay because of cochlear dispersion, tuning, and nonlinearity. Limitations of the measurement of the phase-gradient delay in fixed-f1, fixed-f2, and fixed-f2/f1 paradigms were studied by Shera et al. (2000). For the cubic distortion product at frequency $2f_1–f_2$, the three measurement paradigms produce significantly different group delays. The group delay measured using the fixed-f1 method is greater than that measured using the fixed-f2 method (Bowman et al. 1997; Moulin and Kemp 1996; Schneider et al. 1999; Shera et al. 2000; Whitehead et al. 1996). These studies show that the group delay cannot be used to derive the traveling delay or the “signal front delay” of the emission in the cochlea unless the “cochlear filter delay” is known a priori (Ruggero 2004).
generation site to the stapes is much faster than a forward-traveling wave to the \( f_2 \) location. The result that the emission round-trip delays are smaller than the forward delay suggests a basal shift of the emission-generation site, likely attributable to the basal shift of primary-tone response peaks with increasing intensity (Fig. 4C). These data are consistent with the compression-wave theory rather than the backward-traveling-wave mechanism. However, for emissions with a large \( f_2/f_1 \) ratio (i.e., \( f_1 \approx f_2 \)), the results can still be interpreted using the backward-traveling-wave theory because the \( f_1 \) delay at the \( f_2 \) place can be much smaller than the \( f_2 \) delay, and the predicted emission round-trip delay based on the backward-traveling-wave mechanism is similar to that predicted according to the compression-wave mechanism.

In contrast to the frequency-dependent \( f_1 \) delay (Figs. 3D and 4F), the emission group delays (Figs. 2, B and E and 4B) are independent of the \( 2f_1–f_2 \) frequency. The mechanisms responsible for this inconsistency remain to be studied. In spite of uncertainties of interpretation, our finding that the emission round-trip group delay at the stapes is equal to or smaller than the forward delay is different from previous reports that the round-trip delay of emissions is twice as long as the forward delay.

ACKNOWLEDGMENTS

We thank E. Porsov for technical assistance. The authors also thank anonymous reviewers for critical comments and valuable suggestions on interpretation of the data.

GRANTS

This study was supported by grants from the National Institute of Deafness and Other Communications Disorders.

REFERENCES


Siegel JH, Kim DO, and Molnar CE. Effects of altering organ of Corti on cochlear distortion products \( f_2–f_1 \) and \( 2f_1–f_2 \). *J Neurophysiol* 47: 303–328, 1982.


