A Wave of Cochlear Bone Deformation Can Underlie Bone Conduction and Otoacoustic Emissions

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Abstract. A sound signal is transmitted to the cochlea through vibration of the middle ear that induces a pressure difference across the cochlea's elastic basilar membrane. In an alternative pathway for transmission, the basilar membrane can also be deflected by vibration of the cochlear bone, without participation of the middle ear. This second pathway, termed bone conduction, is increasingly used in commercial applications, namely in bone-conduction headphones that deliver sound through vibration of the skull. The mechanism of this transmission, however, remains unclear. Here, we study a cochlear model in which the cochlear bone is deformable. We show that deformation of the cochlear bone, such as resulting from bone stimulation, elicits a wave on the basilar membrane and can hence explain bone conduction. Interestingly, stimulation of the basilar membrane can in turn elicit a wave of deformation of the cochlear bone. We show that this has implications for the propagation of otoacoustic emissions: these can emerge from the cochlea through waves of bone deformation.

INTRODUCTION

The cochlea acts as a spacial frequency analyzer where a single-frequency tone elicits a wave on the basilar membrane that peaks at a frequency-specific position. The basilar-membrane is normally set in motion thorough vibrations of the ossicles in the middle ear, which induce a deflection of the elastic oval window at the cochlear base. A displacement of the basilar membrane can, however, also be caused by vibrations of the cochlear bone. Early experiments by von Békésy show that both transmission modes cause the same type of wave on the basilar membrane: the hearing sensation of a pure tone delivered through the middle ear can be entirely canceled by a stimulation of the bone at the same frequency but carefully chosen amplitude and phase [1]. It has been argued that, bone conduction may involve a bending deformation of the cochlear bone and deflect the basilar membrane if cross-sectional areas of the chambers above and below the basilar membrane are different [1, 6].

Here, we investigate the consequences of such cochlear-bone deformation in a computational model. We find that the cochlear-bone bending deformation can be at the core bone conduction and can evoke a traveling wave on the basilar membrane.

Another related open problem in cochlear mechanics is how distortion-product otoacoustic emissions propagate from their generation site inside the cochlea back to the ear canal. The issue is further complicated for distortion-product otoacoustic emissions that consist of two components that differ in the temporal delay between their generation and the resulting emission in the ear canal [8, 9]. One component has a long delay of a few milliseconds, whereas the delay of the other component is much shorter.

Theoretical studies have suggested that both components emerge through waves on the basilar membrane and propagate backward from their generation site to the cochlear base [7, 14, 19]. This is supported by measurements of the intracochlear pressures as well as of the cochlear microphonic potential [3, 10]. Recent experiments that have used laser interferometry to measure the membrane's motion, however, only detected forward-traveling waves [4, 5, 13].

We have recently proposed that the long-delay component of a distortion-product otoacoustic emission can arise through waves on Reissner's membrane, another elastic membrane within the cochlea that extends in parallel to the basilar membrane from the cochlear base to the apex [12]. We have performed mathematical modeling and experiments to show that short surface waves can propagate along Reissner's membrane, and that the waves can be created through the cochlear active process. Because waves on Reissner's membrane have relatively short wavelengths, below 0.5 mm for frequencies above a few kHz, such backward-propagating waves have slow speeds of a few meter per second. Distortion products emerging through those waves yield accordingly delays of a few milliseconds when propagating from their generation region to the middle ear.

It remains, however, unclear how the short-delay component of an otoacoustic emission can emerge, if backward

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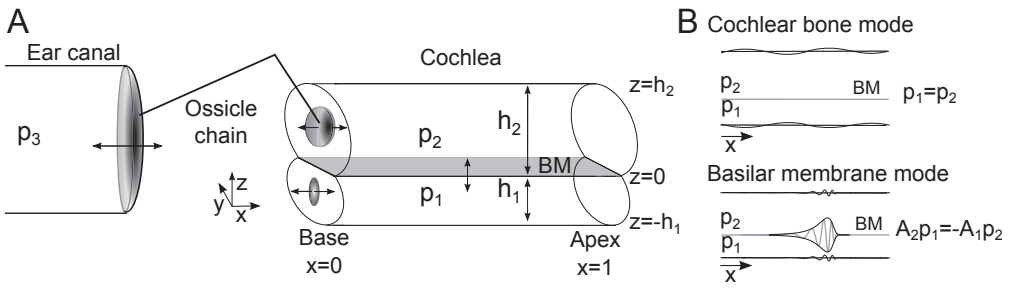

FIGURE 1. Outer, middle, and inner ear and the resulting wave modes in the cochlea. (A) Sound vibrates the ear drum, the motion of which is conveyed by the middle ear's ossicles to the inner ear, or cochlea, where it vibrates the oval window. This results in pressure changes p_1 in the scala tympani and p_2 in the combined scalae media and vestibuli. The basilar-membrane motion is proportional to the difference between p_2 and p_1. (B) The two wave modes in a cochlea with a deformable bony wall. The cochlear-bone wave (top) elicits identical pressures in both chambers and hence does not vibrate the basilar membrane. The well-known basilar-membrane wave (bottom) evokes different pressure changes p_1 and p_2 and hence basilar-membrane motion. Because of the pressure changes in each chamber, this wave also elicits a deformation of the cochlear bone [16].

waves on the basilar membrane are not involved. One possibility is that these signals are carried by a compression wave in the cochlear fluid [4, 5, 13]. Such waves involve, however, no pressure difference across the basilar membrane, and it is hence unclear how they can be generated by the membrane's nonlinear response. We show that the cochlear-bone wave described here can transport distortion products back to the ear canal, which can explain the short-delay component of otoacoustic emissions and is consistent with the simultaneous occurrence of forward-traveling waves along the basilar membrane. Details of the model and further results are given in our recent publication [16].

COMPUTATIONAL MODEL

We begin from a two-dimensional model of the cochlea (Fig. 1). The variable x describes the cochlea's longitudinal position, and the variable z the vertical deviation from the basilar membrane. We denote a pressure deviation in the scala tympani by p_1. The scala vestibuli and scala tympani are modeled as one chamber, and a pressure deviation therein is denoted by p_2. The basilar membrane separates both chambers and is located at $z = 0$. Its vertical velocity V_{bm} follows from the pressure difference across it, $(p_1 - p_2)_{|_{z=0}}$, as well as from its impedance Z_{bm}:

$$(p_1 - p_2)_{|_{z=0}} = Z_{bm} V_{bm}. \tag{1}$$

The cochlear walls are located at $z = -h_1$ and $z = h_2$. We assume that the walls are deformable under pressure, which yields a vertical velocity V_l or V_u of the lower respectively upper cochlear wall. The velocity follows from the pressure through the impedance Z_w of the cochlear wall:

$$p_1|_{z=-h_1} = -Z_w \cdot V_l, \quad p_2|_{z=h_2} = Z_w \cdot V_u. \tag{2}$$

The fluid dynamics of the cochlea is further described by linearized equations of continuity and momentum. At the basilar membrane and at the cochlear walls these take the form

$$-\rho \partial_t V_{bm} = \partial_z p_2|_{z=0} = \partial_z p_1|_{z=0}, \tag{3}$$

and

$$-\rho \partial_t V_l = \partial_z p_1|_{z=-h_1}, -\rho \partial_t V_u = \partial_z p_2|_{z=h_2}. \tag{4}$$

To simplify our calculations, we approximate the fluid as incompressible, an assumption that will be justified below. The linearized equations of continuity and momentum then yield the Laplace equations $\Delta p_1 = \Delta p_2 = 0$ which, together with the boundary conditions (1-4), describe the system.

For the solution of these equations we start from the ansatz

$$\begin{aligned} p_1 &= \tilde{p_1}(x)\,[Z_w k \cosh(k(x)(z+h_1)) - i\omega\rho \sinh(k(x)(z+h_1))] \exp(ig(x) + i\omega t - k(x)h_1), \\ p_2 &= \tilde{p_2}(x)\,[Z_w k \cosh(k(x)(z-h_2)) + i\omega\rho \sinh(k(x)(z-h_2))] \exp(ig(x) + i\omega t - k(x)h_2), \end{aligned} \tag{5}$$

in which $g(x) = \pm \int_0^x k(x')dx'$ and in which $\tilde{p_1}(x)$ and $\tilde{p_2}(x)$ are slowly-varying amplitudes. The variation of $\tilde{p_1}(x)$ and $\tilde{p_2}(x)$ can be obtained from a WKB approximation that accounts for the slow variation in the basilar-membrane impedance [15, 18]. The above ansatz (5) then fulfills the Laplace equations as well as the boundary conditions (2-4). The response of the basilar membrane, Equation (1), yields an additional constraint on the possible wave vector $k(x)$. Two possible solutions emerge which correspond to two distinct wave modes that the system can exhibit.

The first solution is given by $k_{BM}(x)^2 = -\frac{i(h_1+h_2)\rho\omega}{h_1 h_2 Z_{bm}(x)}$ and $\tilde{p_2}(x) = -\tilde{p_1}(x)\frac{h_1}{h_2}$. It involves opposite pressures on both sides of the basilar membrane and its wave vector is determined by the basilar-membrane impedance alone, not by the impedance of the cochlear walls. This wave mode hence represents the well-known basilar-membrane wave.

The second solution is $k_{BC}(x)^2 = \frac{2i\rho\omega}{(h_1+h_2)Z_w}$ and $\tilde{p_2}(x) = \tilde{p_1}(x)$. The basilar membrane is not deflected in this wave mode because the pressures below and above the membrane are equal. This wave hence travels exclusively as a deformation of the cochlear bone, and we refer to it as cochlear-bone wave. Its propagation properties follow from the impedance of the cochlear walls alone. The latter can be computed from the Young's modulus of the cochlear bone and the geometric cross sectional area change under a bending deformation [17]. We find that the cochlear-bone mode has a wavelength of a few centimeters, considerably longer than that of the basilar-membrane wave. Its wavelength remains, however, significantly shorter than that of a fluid compression wave. This shows that the acoustic impedance associated with the cochlear-bone wave is significantly lower than the acoustic impedance of the compression wave, which justifies our assumption of an incompressible fluid.

BONE CONDUCTION AND OTOACOUSTIC EMISSIONS

The basilar-membrane wave can be generated by a deformation of the cochlear bone. In a cochlea where the two chambers have different cross-sectional areas, a deformation of the upper and lower cochlear walls that is equal in magnitude but opposite in direction leads to different pressures p_1 and p_2 in the two chambers [16]. This can excite the basilar-membrane wave despite the fact that the cochlear-bone wave itself involves pressures of equal magnitude in the two chambers. Deformation of the cochlear walls can hence produce the traveling wave on the basilar membrane, providing a plausible yet novel mechanism for bone conduction. The strength of the resulting basilar-membrane vibration can be quantified in our model by considering periodic forces of equal magnitude but opposite direction applied to the top and bottom of the chambers at a certain location, and the resulting basilar-membrane wave [16]. Notably, this mode of bone conduction is not contingent on the movement of the round or oval window and hence consistent with experimental reports that bone conduction thresholds remain intact in patients and as well as animals with immobilized cochlear windows [2, 11].

Reversely, a force acting on the basilar membrane can launch a cochlear-bone wave. This occurs because a force that displaces the basilar membrane produces pressure changes on its two sides that are equal in magnitude but opposite in force. In an asymmetric cochlea with unequal height of both chambers, $h_1 \neq h_2$, the basilar-membrane wave involves pressures that differ in magnitude. A force on the basilar membrane hence produces a cochlear-bone wave, in addition to a wave on the basilar membrane.

It follows that otoacoustic emissions can emerge from the cochlea through the cochlear-bone wave. Consider the cubic distortion $2f_1 - f_2$ produced by stimulation at the primary frequencies f_1 and f_2. The distortion is created in an extended cochlear region in which the waves elicited by f_1 and f_2 peak and overlap. The resulting waves at the distortion frequencies can be computed by considering the waves elicited by pointwise stimulation and integrating over the generation region [12]. We find that the force on the basilar membrane at the distortion frequency elicits forward and backward traveling waves on the cochlear walls. The backward traveling wave elicits a pressure signal at the oval window, which vibrates the stapes and hence yields a sound signal in the ear canal. The strength of the emerging emissions can also be quantified through the computation of Green's functions and is of the observed order of magnitude [16].

The backward-propagating cochlear-bone wave can explain the short-delay component of distortion-product otoacoustic emissions. Because the cochlear-bone wave has a long wavelength of a few centimeters and hence a high propagation velocity, a distortion product propagating back from its generation region to the stapes through this mechanism experiences only a short delay of less than a millisecond.

Moreover, a part of the backward-propagating cochlear-bone wave is reflected at the stapes. The reflection contains both a forward-traveling wave on the cochlear bone as well as a forward-traveling wave on the basilar membrane. The latter can blanket the backward-traveling wave on the basilar membrane that is produced by distortion. Our computations show that it depends both on the ratio of the primary frequencies as well as on the cochlear location whether the forward- or the backward-traveling basilar-membrane wave is stronger in amplitude (Fig. 2). Consider

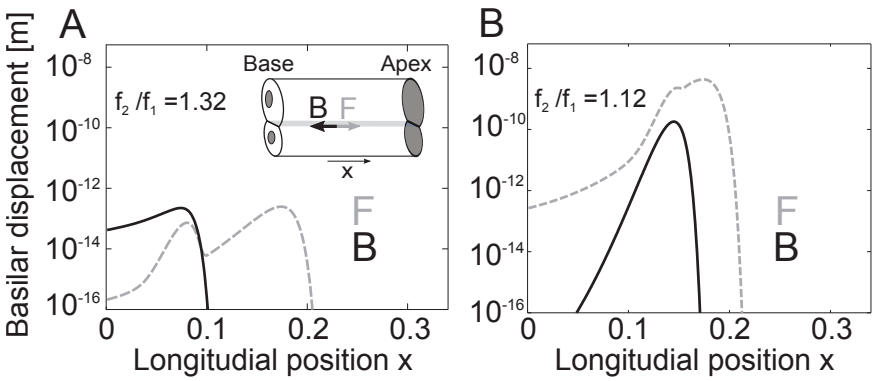

FIGURE 2. Forward- and backward traveling distortion waves on the basilar membrane. We consider the cubic distortion product $2f_1 - f_2$ as produced by the primary frequencies f_1 and f_2. Both a forward-and a backward-traveling wave on the basilar membrane emerge. (A) Amplitudes of the backward-traveling wave (B, red) and the forward-traveling wave (F, blue) for a large ratio of primaries ($f_2/f_1 = 1.32$). The backward-traveling wave dominates close to the base, but the forward-traveling wave near the best place of $2f_1 - f_2$. (B) Amplitudes of the backward- and forward traveling waves (B, red respectively F, blue) for close primaries ($f_2/f_1 = 1.12$). The forward-traveling wave dominates at all cochlear locations [16].

the distortion $2f_1 - f_2$ generated by the primary frequencies f_1 and f_2. For a relatively large ratio, $f_2/f_1 = 1.32$, the forward-traveling wave exceeds the backward-traveling wave near the base but not near the best place of $2f_1 - f_2$ (Fig. 2A). For a smaller ratio $f_2/f_1 = 1.12$, however, the forward-traveling wave blankets the backward one at every cochlear position where the corresponding waves can propagate. Such a small ratio is frequently used in experiments that aim to detect a backward-traveling basilar-membrane wave, which may explain that such experiments have detected a forward-, but not a backward-traveling basilar-membrane wave at $2f_1 - f_2$ [4, 5, 13]. Detecting the backward-traveling wave experimentally may require a comparatively large ratio of the primary frequencies. The ensuing distortion product, however, is then comparatively small which complicates its measurement.

With this study we hope to contribute to a better understanding of the mechanisms behind bone conduction as well as otoacoustic emissions and their commercial and clinical application.

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