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4aPPa3. Fast waves, slow waves and cochlear excitation

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In idealized cochlear models intracochlear pressure is decomposed into two modes, the compression pressure (fast-mode) and the traveling wave pressure (slow-mode). Because the cochlear fluid and fluid-filled tissues are nearly incompressible, true compression accounts for only a minute component of sensory tissue motion. However, sizeable motions associated with the compression pressure occur due to experimentally induced cochleostomies, and likely due to natural anatomical asymmetries. In addition, evanescent modes exist in the region of the cochlear windows, and are also "fast". At high stimulus level fast-mode motions are able to excite hair cells and evoke auditory nerve responses. However, fast-mode motions do not seem to be amplified by the cochlear amplifier. This observation supports the concept that the amplifier relies on traveling wave curvature (wavelength), as has been proposed in cochlear models. In this paper experimental and theoretical results on the fast and slow modes are reviewed and discussed.

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INTRODUCTION

Upon acoustic stimulation, the cochlea is filled with a time and spatially varying scalar pressure field, which can be interrogated by a small hydrophone (e.g., Dancer and Franke, 1980; Nedzelnitsky, 1980; Puria et al., 1997; Olson, 1998). Spatial pressure differences produce forces that drive fluid and tissue motion. In the cochlea, the most interesting pressure difference is across the cochlear partition, the elastic tissue separating the fluid tunnels of the scalae vestibui+media and tympani. (Because of the flexibility of the membrane dividing them, scala media and scala vestibuli are mechanically continuous. Scala vestibuli = SV, scala tympani = ST) Coupling between the restoring force of the elastic tissue and the inertia of the fluid leads to the cochlear traveling wave, which transports sound energy from the base to the apex of the long, spiraling mammalian cochlea. Because of the wave, a little ways down the cochlea the pressure at the partition can be delayed by several cycles relative to the pressure at the stapes. In addition to this (i) traveling wave pressure there is (ii) a compression/rarefaction pressure mode that is in phase with the in/out plunging motion of the stapes and is approximately spatially unvarying throughout the cochlea and (iii) evanescent modes that exist in the region of the cochlear windows and decay apical of the windows (Steele et al, 2008; Watts, 2000). The evanescent modes are anti-symmetric (or nearly so) across the partition and thus drive its motion, but like the compression mode they are linked to stapes motion without delay --- thus the evanescent and compression modes are "fast" and the traveling wave mode is "slow."

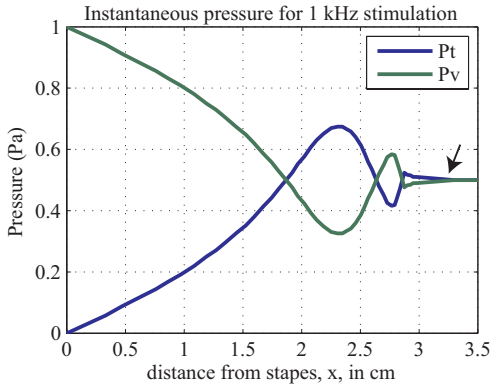

FIGURE 1. Instantaneous intracochlear pressure in scala vestibuli (Pv) and scala tympani (Pt) as a function of distance from the stapes, for 1 kHz stimulation. Arrow indicates region of fast-mode domination. Based on the 1-dimensional model of Peterson and Bogert (1950).

Compression and Traveling Wave Modes in 1-Dimensional Cochlear Theory

The best theoretical introduction to the compression mode is Peterson and Bogert (1950). They performed a 1-d analysis of cochlear mechanics, with the longitudinal dimension denoted by x. The cochlear pressures in scalae vestibuli and tympani (Pv and Pt) were written as P+ and P-, where P+ = (Pv + Pt)/2 and P- = (Pv – Pt)/2. They referred to P+ and P- as longitudinal and transverse modes. In their analysis, P+ is seen to simply be an acoustic wave in the cochlear fluid. Given the small size of the cochlea compared to sound wavelengths in water, P+ is essentially a cochlea-filling compression/decompression pressure timed with the in/out motion of the stapes. (At frequencies > 12 kHz the 3.5 cm tube length of the uncoiled human cochlea was long enough to produce longitudinal standing waves in P+; this interesting prediction was not explored further.) P- was anti-symmetric across the cochlear partition and produced a cross-partition pressure difference. In the analysis P- was governed by partition elasticity and took the form of a wave. Because the elasticity increased from base to apex, the wave-speed and wavelength decreased with distance from the stapes. The two modes were summed in order to match the boundary conditions at the stapes and round window, namely Pv(x=0) = prescribed input pressure and Pt(x=0) = 0. Figure 1 shows a "snapshot" of Pv and Pt from Peterson and Bogert's analysis for a pure tone drive at 1000 Hz, and input pressure Pv(x=0) of 1Pa. It is easy to see that P+ = 0.5 Pa at the instant under consideration, and is essentially spatially invariant. P+ would vary sinusoidally from 0.5 to -0.5 Pa as the stapes moved in and out. Following the definition P- = (Pv – Pt)/2, P- = Pv but with the P+ offset removed. In most cochlear models, only P- is considered because the compressing/rarefying motions caused by P+ would be so much smaller than the bending motions caused by P-. To see this, consider that the bending compliance of the basal cochlear partition (how much its center

moves in response to the pressure difference across it) is ~ 0.3 nm/Pa in the base, increasing to ~ 3 nm/Pa midway down cochlea (in gerbil and guinea pig, reviewed in Olson et al., 2012). The motion due to compression (how much the partition is squeezed) can be calculated by assuming that the partition's compressibility is similar to that of water. The bulk modulus, B, of water is ~ 2 x 10^9 Pa. Volume compression $\Delta V/V_o = P/B$, and linear compression $\Delta x/x_o \sim (1/3)P/B$. For $x_o = 200\mu m$ (~ the thickness of the cochlear partition), $\Delta x/P \sim 0.03$ pm/Pa. Thus, the bending compliance of the partition is 10,000 greater than its compressibility and the displacements due to P+ would be thousands of times smaller than those due to P-.

Spatial and Stimulus Level Dependence of Measured Intracochlear Pressure

Peterson and Bogert's model was 1-d, so did not explore spatial pressure variations with distance from the cochlear partition, but 2-d and 3-d models predict that P- decreases with distance from the partition (call this the z direction, with x still used for the longitudinal distance from the stapes as in Fig. 1) (e.g., Taber and Steele, 1981). Indeed, in experimental observations intracochlear pressure was easily interpretable as the sum of a P+ mode that was ~ spatially invariant and timed with the stapes motion and a P- mode that varied spatially and accumulated delay (e.g., Olson, 1999). At a fixed distance from the stapes (x), the dominance of one or the other mode depended on frequency, z location and stimulus level. Fig. 2 and 3 show ST pressure in-vivo in the gerbil cochlea, measured at the turn-one location where the best frequency is ~ 20 kHz.

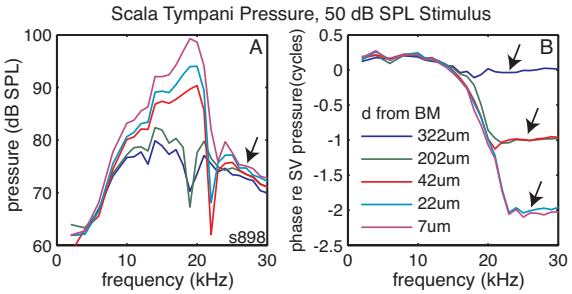

FIGURE 2. Gerbil ST pressure at various distances from the basilar membrane (BM), 50 dB SPL stimulus in the ear canal. A. Amplitude. B. Phase, referenced to the SV pressure measured near the stapes. Arrows indicates region of fast-mode domination.

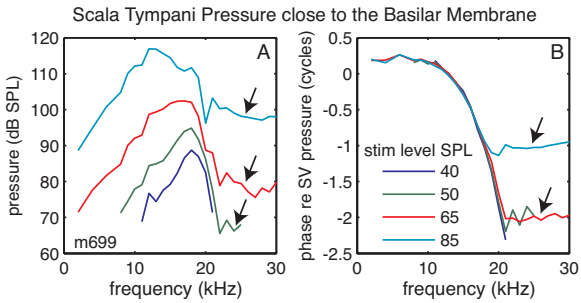

FIGURE 3. Gerbil ST pressure close to the BM (20 um), at several stimulus levels as measured in the ear canal. A. Amplitude. B. Phase, referenced to the SV pressure measured near the stapes. Arrows indicate region of fast-mode domination.

The curves in Fig. 2 are at different distances from the partition's basilar membrane (BM), all at a driving pressure in the ear canal of 50 dB SPL. The BM is a collagenous tissue that bounds the sensory tissue on the ST side. Panel A shows that the pressure amplitude was tuned when measured close to the BM. In the tuned region the pressure decreased rapidly with distance from the BM. Panel B shows the ST pressure phase referenced to the phase of the pressure in SV close to the stapes (input pressure to the cochlea). Close to the BM the phase accumulated several cycles and far from the BM the ST and SV pressures were nearly in phase. These data demonstrate the transition from pressure that, close to the partition (small z) was dominated by the slow traveling wave pressure mode, and far from the partition (large z) was dominated by the fast compression mode. At frequencies somewhat above the traveling wave peak for this x location, the pressure was fast-mode dominated even close to the partition. Notches in panel A correspond to cancellation between the fast and slow modes. Fig. 3 shows data from another

preparation, close to the BM and at various stimulus SPLs, showing that the pressure peak scaled nonlinearly – it was enhanced at low and moderate SPL. This is the cochlea's saturating nonlinearity, known as the cochlear amplifier. The fact that amplification is observed in the pressure, as well as the more thoroughly documented motion of the partition (Robles and Ruggero, 2001), evinces that amplification occurs by means of a "stimulus enhancement" as described in Kolston (2000). Fig. 3 also shows that at low SPL the slow-mode dominated the fast-mode to higher frequencies than it did at high SPL, and that the fast-mode (above the peak) is not amplified – this frequency region is linear. This makes sense since the P+ mode is not thought to cause significant motion of the BM, and the amplifier is activated by stereocilia pivoting and consequent outer hair cell electro-mechanics (Brownell et al, 1985). However, we will see below that substantial fast-mode motions do exist, and thus the observation that only the slow-wave mode is amplified is not so easily explained, and might offer a significant clue to understanding amplifier operation.

The frequency region just above the peak is interesting. Theoretically, the P+ pressure mode above the peak is equal and opposite on the two sides of the partition (locations > 3.3 cm in Fig. 1) and thus would not drive transverse (z) displacement. And based on the quick-calculation above, P+ would not lead to significant compressive motion: An 80 dB SPL (0.2 Pa) stimulus in the ear canal would cause a P+ of ~ 5 Pa (due to middle ear pressure gain) leading to motions of ~ 0.1 pm. We can look at experimental observations of BM motion to see what the motion in the fast-mode region above the peak actually is. Because it is not an interesting region for most considerations of normal hearing (since supra-peak frequencies of a given location would have their own peaks further basal), most studies do not collect data in this region. An exception is the chinchilla data of Rhode (2007).

The Fast Mode in Basilar Membrane Motion and Auditory Nerve Responses

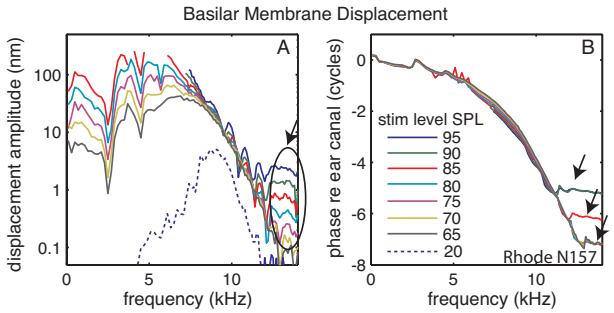

FIGURE 4. Chinchilla basilar membrane motion, at several stimulus levels as measured in the ear canal. A. Amplitude. B. Phase, referenced to the ear canal pressure. Arrows (and oval in A) indicate region of fast-mode domination.

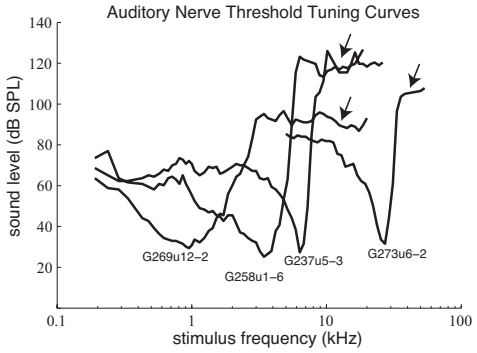

FIGURE 5. Gerbil auditory nerve threshold tuning curves. Arrows indicate region of fast-mode domination. From the doctoral study of S. Huang.

Fig. 4 shows Rhode's data collected at moderate and high SPL (65-95 dB SPL), levels at which a fast-mode motion plateau was detected. The amplitude response at 20 dB SPL is included to show that the best frequency of this location was ~ 9 kHz. A dramatic region of saturating nonlinearity existed from ~ 7 kHz to ~ 12 kHz. The high frequency limit of amplification depended on the stimulus level: as soon as the response hit the fast-mode plateau, the response was essentially linear and the response hit this plateau sooner at high SPL. The plateau was apparent in

both the amplitude and the phase, and the flat phase plateau confirmed that these motions were timed with the stapes motion, and were not the phase-accumulating wave motion of the slow-mode. The transition between slow and fast-modes occured at ~ 11 kHz at 95 dB SPL, and ~ 13 kHz at 65 dB SPL. Consider the size of the fast-mode motions: At 80 dB SPL in the ear canal (corresponding to ~ 5 Pa in the cochlea) the fast-mode motions were 0.3-0.4nm. This is a thousand times too large to be due to compression and must be due to something in the anatomy that allows the P+ pressure mode to be slightly unequal on the two sides of the partition, and thus drive bending partition motion. One possibility is the habenula perforada, small holes in the bone (for passage of auditory neurons) between the modiolus and the inner sulcus, a fluid space on the inside of the row of inner pillar cells. The modiolus runs up the middle of the cochlea but is mechanically distinct from the cochlear scalae, so the habenula perforada holes could create a pressure release that would give rise to P+ induced fluid and tissue motions. Similarly, viewing holes in the apex of the cochlea are known to release pressure unless they are completely sealed, creating fast-mode pressure differences across the partition that affect its motion (Cooper and Rhode, 1996; Dong and Cooper, 2006).

Even though significant fast-mode BM motions exist, they might not excite hair cells at all – after all, if they are produced by something like a pressure release at the habenula perforada, this might be a motion that only involves the BM and does not pivot the stereocilia. This notion was supported by the fact that auditory nerve (AN) responses seemed to not be elicited in the supra-characteristic frequency (CF) region of the fast-mode (Ruggero et al., 2000). However, a systematic study of AN responses in the supra-CF region was lacking until recently. Huang and Olson (2011) explored the supra-CF region and found that it could be excitatory, but that the stimulus level needed to be ~ 20 dB higher than what would have been predicted from matching BM responses to AN responses in the sensitive tip of the tuning curve. Four tuning curves from the study of Huang and Olson are in Fig. 5 and show these supra-CF response plateaus. These findings indicate that fast-mode motions are able to excite inner hair cells, and presumably outer hair cells, an assumption that is buttressed by the observation of a prominent plateau in the cochlear microphonic (Schmeidt and Zwislocki, 1977). The quantitative finding is worth repeating: the fast-mode motions on the BM *were* excitatory, but not equivalent to slow-mode motions in terms of hair cell excitation. Thus, the internal motions of the organ of Corti that lead to stereocilia pivoting appear to be ~ 10 times smaller for the fast-mode than for the traveling wave slow-mode (Huang and Olson, 2011; Temchin, 2011).

The Fast Mode, the Slow Mode and Cochlear Amplification

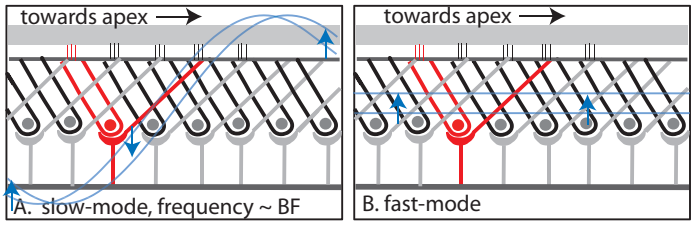

FIGURE 6. Longitudinal section of the cochlea along one of the rows of outer hair cells. Red identifies an outer hair cell supported by its Deiters' cell below. The Deiter's cell has a phalangeal process extending up to the upper boundary of the hair cells at a more apical longitudinal location. (A) Slow-mode traveling wave response. The longitudinal slant of the outer hair cell and phalangeal processes allows forces to couple apically to a region that is moving with a different phase. Since wavelength varies with frequency, this phasing is frequency dependent. (B) Fast-mode response. Here all regions move in phase, thus there is no possibility for properly phasing hair cell forces via wavelength.

Observations of the cochlear amplifier are now quite mature, in BM motion and pressure at the BM, and attention has shifted from characterizing the amplifier to understanding its operation. The cochlear amplifier is likely based in outer hair cell somatic motility, which provides sufficient wideband force (Frank et al, 1999). From the observations above, at a given location, cochlear amplification works at frequencies beginning at the passive peak and extending up in frequency to the point where the fast wave dominates. What tunes the amplifier to these location-dependent frequencies? The observation that the slow-mode is amplified but the fast-mode is not indicates that there is something special about the slow-mode motion that the fast-mode motion does not possess. We saw above that fast mode motions can excite hair cells, and so outer hair cells are presumably exerting electromotile forces – why are they not providing amplification? The most obvious difference between the two modes is the longitudinal curvature conferred by the slow-mode's traveling wave, and indeed longitudinal curvature has been employed to produce amplification in several cochlear models (Yoon et al., 2011; Geisler and Sang, 1995). As shown in Fig. 6, in these models the outer hair cell forces provides power only when the phase between the outer

hair cell and its corresponding Deiters' cell phalangeal process is correct – that is, only when the wavelength is sufficiently short. Thus, in these models amplifier tuning is based not in frequency, but in wavelength. In contrast, if the cochlear amplifier were tuned by frequency, why wouldn't the small motions of the fast-mode be amplified? On the other hand, since cochlear amplification accumulates over a longitudinal extent of ~ 1mm as the wave travels along (Cody, 1992), it could be that the fast-mode *is* amplified but its local amplification is not large enough to be detected.

How could the hypothesis of wavelength-based tuning of the amplifier be tested? If Deiters' cells phalangeal processes were removed and the amplifier still worked, that would argue against wavelength-based tuning. However, there is no clear way to remove these processes. As a more feasible alternative, one could inject air bubbles or partially drain the basal cochlea (where amplification is substantial) in order to alter the balance of fast and slow modes. However, a reduction in amplification would be hard to specifically attribute to an absence of curvature, since accumulated amplification would also be diminished. The cochlea guards its secrets.

The Fast Mode and Oto-Acoustic Emissions

An interesting final topic is whether oto-acoustic emissions travel out of the cochlea via the fast or slow mode. Explorations of the question often compare the phases of intracochlear responses and corresponding emissions, to find their relative timing. In a study of intracochlear pressure, at frequencies well below the local best frequency, the emissions followed the intracochlear response with a delay that was similar to that of a forward traveling response (Dong and Olson, 2008), evincing that the emission traveled out via a slow-mode. The same study found that at frequencies close to the local best frequency, the steep phase accumulation of emissions was ~ twice that of the forward traveling wave phase accumulation, confirming the presence of a slow reverse wave. Although this evidence favors the primacy of the slow-mode in emissions, fast-pressure modes have been found to contribute to emissions and it is hard to imagine why they would not contribute at all (Ren 2004; Dong and Olson, 2009). In summary, the mode by which emissions travel out of the cochlea is a challenging quantitative topic, important because of the clinical and scientific significance of oto-acoustic emissions.

CLOSING

Two prominent pressure modes exist in the cochlea, the compression fast-mode and the traveling slow-mode. The slow-mode leads to substantially larger BM motions than the fast-mode and is more effective at exciting hair cells, but the fast-mode also gives rise to significant motion and is able to excite hair cells. However, the cochlear amplifier does not act on the fast-mode, and this observation supports theories of amplification that rely on the slow-mode's traveling wave curvature. These theories are attractive in that they employ a robust, passive characteristic of cochlear operation – the traveling wave – to activate and tune the cochlear amplifier.

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