# Basilar Membrane Tuning in the Cat Cochlea

Abstract. *The mechanical response of the basilar membrane changes mainly in the peak region with trauma to the cochlea. Basilar membrane tuning curves measured in cochleas with reduced trauma begin to look similar to tuning curves of auditory nerve fibers.*

A number of investigators have measured the mechanical properties of the basilar membrane through the use of several different techniques and different species (*1–3*). In all of these experiments, the basilar membrane vibration amplitude, when plotted as a function of frequency for constant sound pressure level (SPL) at the tympanic membrane, shows characteristics similar to that of a low-pass filter with a sharp cutoff. Usually, a small peak is observed in the frequency region just below the cutoff. The largest peak was observed by Rhode (*4*). In contrast to the mechanical response, auditory nerve fibers are more sharply tuned and have a larger peak. Kim and Molnar (*5*) emphasized, however, that this apparent discrepancy between the mechanical and neural tuning should be interpreted cautiously because of physiological vulnerability and the nonlinearity of basilar membrane motion.

Considerable variability in the basilar membrane responses was observed by the above investigators, and damage incurred during the preparation was suspected to be the underlying cause. When measuring basilar membrane vibrations, some investigators did not monitor the condition of the cochlea (*1*). Others noted a rapid deterioration of the $N_1$ response with time (*6, 7*). Wilson and Johnstone (*6*) found no change in the shape of the basilar membrane tuning curve with a change in the $N_1$ response.

In our experiments with cats, the condition of the cochlea was evaluated both by monitoring the round window cochlear microphonic (CM) response and by histological examination of the cochleas. Initially, extensive damage to the cochleas was found by both assessment techniques. Over a period of 2 years, sources of this damage were identified and minimized (*8*). So far, however, we have not been able to prepare a cochlea that shows no signs of trauma.

The surgical approach to the basilar membrane is through the round window membrane. In order to avoid bleeding when the round window membrane is cut, capillary blood flow in the membrane is stopped by microcautery.

An optically flat, gold crystal is used as a mirror for interferometry. The mirror is dropped through the opened window onto the basilar membrane without draining the fluid from the scala tympani (*9*). The mirrors range from 50 to 120 μm in diameter; their weight is less than $10^{-8}$ g. In spite of the small weight and size, the mirrors do load the basilar membrane and produce a mismatch between the middle ear and the inner ear. Sharply tuned sound pressure drops as large as 25 dB can be seen in the ear canal. The mismatch is not observed when the cochlea is damaged. The details of these observations and a discussion of their implications for basilar membrane vibration measurements are discussed (*9*).

Light from a He-Ne laser is directed onto the mirror situated on the basilar membrane. A partially silvered reference mirror is positioned in the path of the laser beam so that the beam passes through its center. The reflections from the two mirrors are adjusted to fall onto a photodetector. The photodetector output is proportional to the square of the sum of the electric fields of the two beams. Vibration of one mirror with respect to the other produces an a-c output at the photodetector (*10*). Tones are applied at the tympanic membrane by a wide frequency range, low-distortion driver and are measured with a probe microphone (*11*). The output of the photodetector at the driving frequency is measured and utilized to obtain the vibration amplitude of the mirror on the basilar membrane.

To assess damage to the cochlea, the CM response was measured several times during the experiment. At the end of the experiment, the cochleas were prepared for histological assessment according to Epon-embedded surface preparation method (*12*). Using this histological method, we were able to determine the location of the mirror along the length of the basilar membrane.

To measure basilar membrane vibrations at low SPL's, the sound pressure at each frequency was adjusted so that the interferometer signal was about 15 dB above the noise. This signal corresponded roughly to an absolute displacement amplitude of $10^{-8}$ cm in some experiments and $10^{-9}$ cm in others, depending upon the noise level. Therefore, the SPL required to obtain a vibration amplitude of $10^{-8}$ cm can be plotted as a function of frequency (Fig. 1). The curves show a shallow minimum in the frequency region of 0.25 to 3 kHz, a relatively flat (plateau) region between 5 and 14 kHz, and a negative peak at approximately 23 kHz. These curves illustrate the variety of responses observed. The differences in these basilar membrane tuning curves are related to differences in the extent of cochlear damage as seen histologically (*13*). The changes in the basilar membrane tuning curve with increasing damage may be summarized as follows: (i) the SPL in the negative peak region is increased; (ii) the

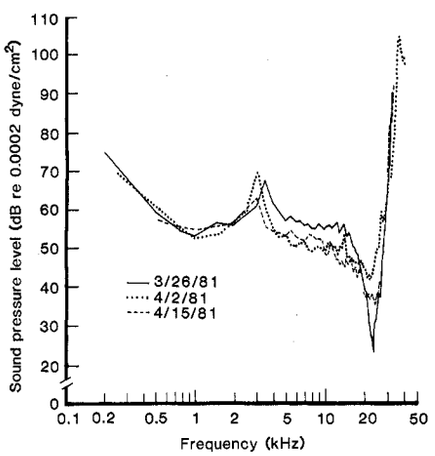


Fig. 1. Sound pressure level at the tympanic membrane required to produce a basilar membrane displacement of $10^{-8}$ cm (bulla open, septum intact). Data for 3/26/81 and 4/15/81 were obtained at $10^{-8}$ cm, and that of 4/2/81 was obtained at $10^{-9}$ cm vibration amplitudes. Since the noise levels varied slightly with frequency, the measured vibration amplitudes were higher by 5 dB at a few frequencies above 27 kHz and by 10 dB at some frequencies below 3 kHz. A linear extrapolation was used to obtain the plots shown.

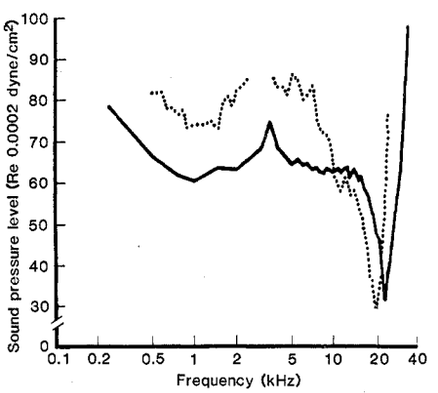


Fig. 2. (Solid line) Sound pressure level at the tympanic membrane required to produce $3 \times 10^{-8}$ cm basilar membrane vibration amplitude in cat 3/26/81. (Dotted line) Neural tuning curve based on an isorate contour of ten spikes per second above the spontaneous rate [(*18*), cat MCL 85-unit 104, bulla open, septum removed]. The two curves have very similar characteristics in the peak region (20 kHz). The main difference is in the peak-to-tail (1 kHz) ratio: 44 dB for the neural tuning curve and 30 dB for the basilar membrane tuning curve.


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low- and high-frequency slopes are reduced; (iii) the peak is broadened; (iv) the peak is shifted to a lower frequency; (v) the SPL in the plateau region below the peak frequency is decreased as much as 10 to 15 dB. The SPL in the 0.25 to 3 kHz region remains unchanged.

The basilar membrane response curve 3/26/81 (Fig. 1) is replotted in Fig. 2. The SPL required for a basilar membrane vibration amplitude of $3 \times 10^{-8}$ cm was obtained by linear extrapolation. For comparison, a neural tuning curve is shown; the threshold values for this curve are within the normal range for this cat in this frequency region (*14*). In the peak region, the low-frequency slope (86 dB per octave), the high-frequency slope (538 dB per octave), and the sharpness of resonance ($Q_{10\ dB} = 5.9$) are the same for the two curves. The principal difference is in the tip (peak) : tail (1 kHz) ratio. For the neural tuning curve, this ratio is about 44 dB, while for the basilar membrane tuning curve it is 30 dB. Tip-to-tail ratios exceeding 25 dB were observed in at least five other basilar membrane experiments. [The highest tip-to-tail ratio reported by Rhode (*4*) was 15 dB]. A second difference between our data and that of Liberman and Kiang (*15*) is that, for a given location on the basilar membrane, the frequency of the peak falls a factor of 1.4 to 1.9 below their frequency values.

These differences may be explained by the fact that the cochlea on which our measurement was made showed some high-frequency CM loss. Therefore, this basilar membrane response is not that of an entirely undamaged cochlea. Indeed, Liberman (*14, 16*) has shown that in noise-traumatized cats the tip-to-tail ratio of neural tuning curves decreases and the sensitivity in the region an octave below the peak frequency increases by 10 to 15 dB. Cody and Johnstone (*17*), recording from single auditory nerve fibers, showed that even a 1-minute exposure to a tone at 100 dB SPL results in a 30-dB loss in sensitivity in the peak region; also the peak frequency shifts down almost an octave.

The mechanical frequency response of the basilar membrane is susceptible to trauma, which affects the basilar membrane frequency response mainly in the peak region. This leads to the flat frequency response observed by most investigators, and quite often by us. However, when exceptional precautions are exercised to minimize cochlear damage, preparations are obtained in which the peak of the basilar membrane response is larger than previously seen. The basilar membrane frequency response under these conditions is closer in shape to neural tuning curves. In view of the above observations, the need for a second filter (*3*) must be reevaluated.

SHYAM M. KHANNA
DEBRA G. B. LEONARD
*Department of Otolaryngology, Columbia University, New York 10032*

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## Ethanol in Low Doses Augments Calcium-Mediated Mechanisms Measured Intracellularly in Hippocampal Neurons

Abstract. *The electrophysiological effects of ethanol in low doses (5 to 20 millimoles per liter or 23 to 92 milligrams per 100 milliliters) were examined intracellularly in CA1 cells of rat hippocampus in vitro. Inhibitory and excitatory postsynaptic potentials were increased when ethanol was applied to the respective synaptic terminal regions. Postsynaptically, ethanol caused a moderate hyperpolarization with increased membrane conductance, even when synaptic transmission was blocked. Ethanol augmented the hyperpolarization that followed repetitive firing or that followed the eliciting of calcium spikes in the presence of tetrodotoxin, but not the rapid afterhyperpolarization in calcium-free medium. Ethanol appears to augment calcium-mediated mechanisms both pre- and postsynaptically.*

Ethanol has variable effects on central mammalian neuronal activity and transmitter release (*1*). Both increased and decreased rates of spontaneous firing of single units of rat hippocampus were observed with ethanol concentrations less than 80 mg/100 ml (*2*). In the hippocampus, extracellular measurements of evoked field potentials indicated that ethanol in low doses increased excitation (*3*) and inhibition (*3, 4*). We studied the cellular mechanisms of mildly intoxicating concentrations of ethanol using the mammalian hippocampal slice preparation (*5*), which is well suited for intracellular recordings.

Male Sprague-Dawley rats (250 to 350 g) were lightly anesthetized with ether, then decapitated. The hippocampal slices were prepared (*5*) and maintained on a mesh for recording at 32° to 35°C. Ethanol, dissolved in control medium, was perfused (four cells) or focally ejected onto the slice with a micropipette (66 cells). Standard intracellular electrophysiological recording techniques were used (*6*). Only cells with spikes greater than 75 mV were studied. The mean ± standard deviation (S.D.) for the resting potentials, when measured, was 55.4 ± 4.5 mV ($N = 18$).

After a characteristic delay of 0.5 to 3 minutes, ethanol caused a moderate hyperpolarization (Fig. 1A) of 0.5 to 8 mV (mean ± S.D., 2.7 ± 1.8 mV) in 78 percent of the cells and a moderate conductance increase (Fig. 1C) of 10 to 40 percent in 74 percent of the cells. All but one cell showed either or both of these effects. The reversal potential for the increased conductance, estimated from the intersection of the current-voltage curves (*7*) in control solution and after ethanol application was between −0.5 and −6.0 mV (mean ± S.D., −3.2 ± 2.6 mV) ($N = 5$) below the resting potential, suggesting increased $Cl^-$ or $K^+$ conductance. When spontaneous spiking was present, a decreased frequency occurred in 10 of 12 cells (Fig. 1A). The amplitude of the injected depolarizing current pulse necessary to trigger a spike was usually increased after ethanol application, as expected from both the hyperpolariza-


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