Brief Report: The Cochlear Microphonic as an Indication of Outer Hair Cell Function

Robert H. Withnell

The extra-cellular cochlear microphonic is believed to be generated predominantly by outer hair cells and therefore it would seem reasonable to assume that the presence of a cochlear microphonic excludes outer hair cell dysfunction. Indeed, a diagnosis of auditory neuropathy might be, and has been, made on the basis of a cochlear microphonic present with an abnormal auditory brainstem response. Animal studies, however, have shown that the cochlear microphonic recorded from the round window is dominated by cellular generators located in the base of the cochlea. Primarily on this basis, it is argued that the presence of a cochlear microphonic does not exclude outer hair cell pathology and so outer hair cell integrity should not necessarily be inferred from the presence of the cochlear microphonic alone. In contrast, the absence of an otoacoustic emission in such cases is consistent with outer hair cell dysfunction.

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The cochlear microphonic (CM) is widely believed to be generated predominantly by outer hair cells and so it would seem reasonable to use it as an indication of outer hair cell function. The inference of hair cell function being normal based on the presence of a CM has precedence in the clinical literature (e.g., Chisin, Perlman, & Sohmer, 1979; Sawada, 1979; Sohmer & Pratt, 1976). In particular, this cochlear electrical correlate of an acoustical stimulus has been used most recently in the differential diagnosis of auditory neuropathy, the presence of a microphonic provided as evidence of outer hair cell integrity.

How reasonable is the assumption that the presence of a CM excludes outer hair cell dysfunction? The electrical activity recorded from the round window, promontory, or in the ear canal near the eardrum, represents a vector sum of extra-cellular currents generated all along that part of the cochlear partition excited by the acoustical stimulus and corresponding neural activity (Dallos, 1973). The AC or time-varying cochlea-generated electrical activity, referred to as the cochlear microphonic (Dallos, 1973), is thought to arise from the extracellular correlate of both outer and inner hair cell receptor currents. The outer hair cells, by virtue of their significantly greater number, contribute most to the response (Dallos, 1983). For an electrode placed on the round window, animal studies have shown that the CM is dominated by contributions from the cochlear partition from within the first few millimeters of the round window (Dallos, 1969, 1971; Patuzzi, Yates, & Johnstone, 1989).

A significant factor influencing the extra-cellular CM recorded from the round window is the electrical length constant or space constant for the exponential decay of cochlear electrical potentials. Estimated at 1 to 2 mm (Johnstone, Johnstone, & Pugsley, 1966, Strelioff, 1973), extra-cellular currents arising from the more apical turns are significantly attenuated by the time they reach the round window. Thus for stimulus frequencies that tonotopically map to the apical turns of the cochlea, one would expect much of the round-window measured microphonic to arise from the basal turn.

To put this in perspective, consider the CM recorded at the round window in response to a 200 Hz pure tone in the guinea pig. The guinea pig cochlea uncoiled is approximately 18 to 19 mm long. Assuming that the round window corresponds to a characteristic frequency (CF) place of 25 kHz (Patuzzi et al., 1989), the 200 Hz place would be approximately 14 mm from the round window (Greenwood, 1990). Patuzzi et al. (1989) estimated that the region of the cochlea tuned to 8 kHz and below "should contribute less than 2% to the total round window microphonic" (p. 186) to a high level 200 Hz tone. While some simplifying assumptions were made in arriving at this estimate, it is evident that most of the microphonic potential recorded from the round window in response to a high level 200 Hz tone arises from the base of the cochlea.

Stimulus frequencies that are used typically in human electrophysiological recordings tend to be in the range 0.5 to 4 kHz. Such frequencies are up to a decade or so higher than a 200 Hz tone and so a greater contribution to the cochlear microphonic from near the CF place might be expected, at least for the higher stimulus frequencies. In particular for stimulus levels up to 60 or 70 dB SPL, where hair cells near CF are driven much closer to saturation than in the basal region (and so the extra-cellular CM originating from any one of these hair cells is

Auditory Physiology Laboratory, Northwestern University, Evanston, Illinois.

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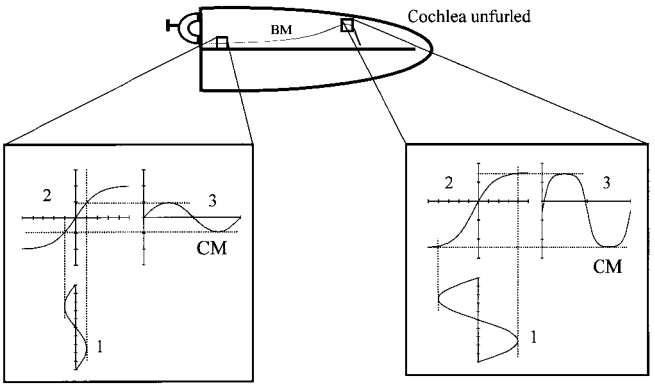

Figure 1. A schematic of the CM that, in theory, arises from one outer hair cell in the base of the cochlea versus that which arises from one outer hair cell near CF, in response to stimulation with a "low-frequency" pure tone. The cochlea is unfurled in the top part of the figure with the cochlear partition represented by a straight line or effectively the basilar membrane (BM). The BM excitation pattern is shown (toward Scala vestibuli only) that would be produced by this "low-frequency" pure tone. The figure inserts show the hypothetical CM which would arise from one outer hair cell in the base and in the apex: (1) represents basilar membrane vibration over time, which in the base is small and near CF much larger; (2) represents the receptor current (y-axis) versus basilar membrane displacement (x-axis) relationship for an outer hair cell, of which the CM is presumably the extra-cellular correlate; (3) represents the receptor current which would flow through the transduction channels of the outer hair cell over time. For small basilar membrane vibrations in the base, the stereocilia are not deflected much such that the CM generated by a single outer hair cell is small. Near CF, basilar membrane vibrations are much larger and the corresponding CM is, as a result, much larger. But, see Figure 2.

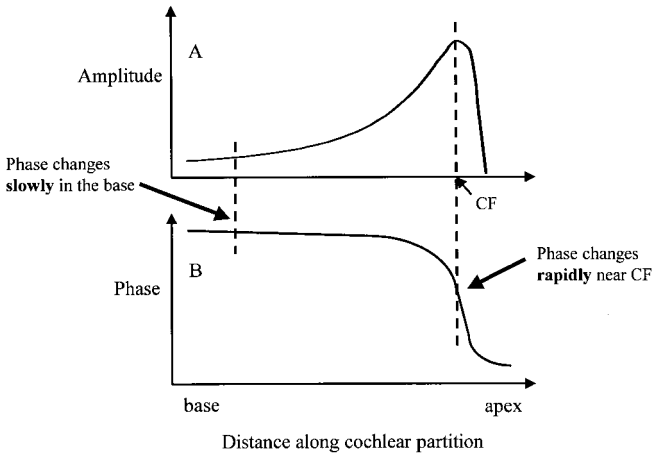

Figure 2. Figure 2A is a schematic of the hypothetical basilar membrane excitation pattern in response to the pure tone of Figure 1. Note that the amplitude of vibration is small in the base but increases significantly as one approaches CF. Figure 2B shows the corresponding phase. In the base, the basilar membrane is moving approximately in-phase, i.e., adjacent regions are moving together in the same direction. Therefore the CM generated by each hair cell in this region is summing additively. In contrast, near CF the phase of vibration of the basilar membrane is changing rapidly, i.e., adjacent regions are moving quite differently such that over a very small distance the basilar membrane will be moving in opposite directions. Therefore the CM generated by this region will not sum additively (see Pickles, 1988, pp. 68–72).

larger than that which originates from cells in the basal region), a greater contribution from these "CF" hair cells might be expected. Figure 1 is a schematic of the CM that, in theory, arises from one outer hair cell in the base of the cochlea versus that which arises from one outer hair cell near CF, in response to stimulation with a moderate level "low-frequency" pure tone. However, significant phase rotation occurs for hair cells near CF, reducing what would otherwise have been their contribution had they summed in-phase. Figure 2 is a schematic of the amplitude of vibration versus distance on the basilar membrane (from the stapes) in response to the "low-frequency" tone and the corresponding phase versus distance along the basilar membrane. It is evident that as the amplitude of vibration of the basilar membrane increases as one approaches CF, the phase of this vibration starts to change very rapidly, i.e., outer hair cells near CF are not moving in the same direction at the same time whereas in the base it is approximately true that these cells are moving in the same direction at the same time (they have similar phases). For cells near CF the extra-cellular CM does not sum additively but rather is a vector sum (see Dallos, 1973).

Further, 0.5 to 4 kHz corresponds to 26 to 13 mm from the cochlear base in humans (Greenwood, 1990) and so 0.5 to 4 kHz is presumably electrically remote from the recording site (promontory or eardrum). As such, it is to be expected that in humans the extra-cellular CM to stimuli in the 0.5 to 4 kHz frequency range would have a significant contribution from the basal turn. Sohmer, Kinarti, & Gafni (1980) supports this conjecture based on CM recordings to a 500 Hz single-cycle sinusoid.

If one assumes a dominant contribution to the extra-cellular CM from the base of the cochlea in response to a stimulus with a frequency in the range 0.5 to 4 kHz, then most of this microphonic is coming from hair cells where basilar membrane vibration is passive (the cochlear amplifier affects basilar membrane vibration only within about one half of an octave of CF [Gummer & Johnstone, 1984]). Based on the fact that the phase rotation is small in this region, the extra-cellular currents from the basal region sum almost in-phase. So if we now introduce a 25% outer hair cell loss all along the cochlear partition the CM potential to a first approximation

would be reduced by about 25%, or less than 3 dB. In contrast, such a hair cell loss may manifest something on the order of a 15 to 25 dB loss of auditory sensitivity in response to tones from 0.5 to 4 kHz (Davis, Ahroon, & Hamernik, 1989). That is, a CM may be present in ears with significant outer hair cell pathology.

With normal middle ear function, the presence of a CM in the absence of an otoacoustic emission should not be construed as indicative of normal outer hair cell function. The presence of the microphonic on its own does not mean outer hair cell function is normal; indeed, the absence of an otoacoustic emission in such a case argues for outer hair cell dysfunction as otoacoustic emissions are quite clearly inextricably linked to outer hair cell function and basilar membrane mechanics (Powers, Salvi, Wang, Spongr, & Qui, 1995).

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Address for correspondence: Robert H. Withnell, Ph.D., Department of Speech and Hearing Sciences, Indiana University, 200 South Jordan Avenue, Bloomington, Indiana, 47405.

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