Ms published in *Concepts and Challenges in the Biophysics of Hearing* (2009) ed. NP Cooper and DT Kemp, World Scientific Publishing.
Additional material: http://www.ucl.ac.uk/~ucgbarg/pubres.htm#cochlea

COCHLEAR MECHANICS: a SIDEWAYS LOOK

A. R. GARDNER-MEDWIN

Dept. Neuroscience, Physiology & Pharmacology, University College London, London WC1E 6BT, UK

This short paper sets out some simple issues and questions that arise in viewing the cochlear mechanics literature. Given the complexity of the literature and of many current models, the approaches here may risk seeming simplistic. But the challenge of trying to dismiss them or produce more clear answers may be worthwhile.

1. The extent of intrinsic damping

Gold's model [1] of viscous damping of a miniature piano string in aqueous fluid is not very appropriate for the basilar membrane (BM). Passive damping of the resonance of a small elastic region separating two aqueous chambers may actually be much more consistent with observed critical bandwidths [2]. This raises the possibility that active mechanisms, combined with what looks rather like an enhancement of viscous damping due to the structural features of the organ of Corti, may serve to modulate damping and provide a trade-off between frequency selectivity and transient resolution, rather than simply to counter viscosity. It is interesting in this context that a raised kinematic viscosity was found necessary in a physical model of the cochlea to eliminate unphysiological standing waves [3].

2. The nature of the inertial mass

The density of most BM structures cannot greatly exceed that of the fluids, so the effective inertial mass in BM resonance is substantially a function of the pattern of surrounding fluid flow. Contributions to effective mass are proportional to the square of local fluid velocity, and for divergent flow are therefore dominated by the varying size and shape of the local zones of maximum displacement around the BM and within a travelling wave [2]. This introduces non-linearities and means that local effective mass at a point on the BM is a variable depending on local wavelength. It falls (and the local maximally sensitive frequency rises) if there is a travelling wave

slowing down as it approaches resonance at a slightly more apical point. This approach suggests a possible way to handle effective mass in a 1-D model, complementary for example to the more thorough analysis in 2-D models by de la Rochefoucauld and Olson [4].

3. The consequence of Outer Hair Cell (OHC) depolarisation

Though BM displacement towards scala media is generally agreed to cause OHC depolarisation, it seems unclear whether the result of this depolarisation and consequent shortening of OHCs will shift fluid in the same or opposite direction to the stimulus. Even an attempt to answer this directly [5] seems equivocal, since imposed current across the BM into scala media will (as observed) have depolarised the apical OHC membranes, but will simultaneously have hyper-polarised the basal portions to an uncertain extent.

4. The role of active negative stiffness

It is tempting to regard negative stiffness of BM structures (in which an active internal mechanism produces force in the same direction as displacement [6,7]) as providing amplification. The internal force adds to the effect of external force at frequencies below resonance. However, without a phase shift this internal force (in phase with the displacement) does not do work and add energy to the system: the increased energy of displacement comes from greater work done by the external force. This is the opposite of the active energy feed into a travelling wave that is required to produce cochlear amplification. A phase delay is in fact inevitable as soon as such a mechanism operates at increased frequency, but with a negative active stiffness this leads to extraction of energy from the system, similar to viscous energy dissipation. To achieve negative damping and energy feed into a BM disturbance (true amplification) an active force must act with positive stiffness and a phase delay $\leq \pi$ (optimally $\pi/2$). Active negative stiffness, where it occurs, may have a different physiological role. The benefits from positive active stiffness (which - subject to the uncertainties of the previous paragraph - may be generated by OHC prestin mechanisms) can arise in two ways [8]: firstly by reducing the energy loss from a travelling wave as this passes over basal regions of the BM that are actively stiffened for frequencies below local resonance, and secondly by feeding energy into the wave as the resonant point is approached, due to an increasing phase lag.

5. The significance of OHC membrane capacitance

In considering the role of OHC membrane potential (V_m) and prestin in cochlear amplification, attenuation of the changes of V_m at audio frequencies (due to membrane capacitance) is often considered a problem. However, if the effect of OHC motility is to oppose BM displacement at low frequencies (positive stiffening), then it is this capacitance that gives the phase lag needed to add energy to the travelling wave and counter viscous damping at frequencies approaching resonance. If a local passive resonance has frequency f and intrinsic damping with rate constant k for energy dissipation (with $Q_{passive}=2\pi f/k$), then for OHCs to give rise to high gain and high Q, their ratio of active stiffness to local passive stiffness (β) at low frequencies must be at least $2/Q_{passive}$. In addition they must have a membrane capacitance sufficient that the time constant (τ_m =RC) is approximately β/k for large β. Given these conditions, less capacitance would risk spontaneous oscillation while lower membrane resistance (as may result from large stimuli or efferent activation) lowers gain and Q. The roll-off of OHC response at audio frequencies above the 3dB cut-off frequency ($1/(2\pi\tau_m)$) is not critical, since the optimal operating conditions may be well above this, by a factor $\beta Q_{passive}$.

1. Gold T (1949) Proc R Soc Lond B Biol Sci 135, 492-8
2. Gardner-Medwin AR (2006) Proc Physiol Soc 3, DC2 [also 9]
3. White RD & Grosh K (2005) PNAS 102, 1296-1301
4. de la Rochefoucauld O & Olson ES (2007) Biophys J 93,3434-50, & in this volume
5. Mammano F & Ashmore JF (1993) Nature 365, 838-41
6. Martin P, Mehta AD & Hudspeth AJ (2000) PNAS 97, 12026-31
7. Kennedy HJ, Crawford, AC & Fettiplace R (2006) Nature 433, 880-3
8. Gardner-Medwin AR (2007) Int J Audiology 46, 652 [also 9]
9. see further details at: http://www.ucl.ac.uk/~ucgbarg/pubres.htm#cochlea