Recent Advances in Interpreting Hearing Sensitivity

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The mammalian cochlea derives its fine sensitivity to very low levels of sound from an amplification process associated with a fast cellular motor located in outer hair cells. Recent discoveries about the nature of this motor, and about its regulation, are briefly sketched alongside new evidence for the motor's effectiveness.

1. INTRODUCTION: SOME EARLIER ADVANCES (1970-89)

This paper was delivered as a Presidential Address to the 1996 Congress, held in St. Petersburg, of the International Institute of Acoustics and Vibration. It aims to bring up to date the 1989 Rayleigh Lecture entitled 'Biomechanics of Hearing Sensitivity' published[1] in January 1991. Like that paper it is concerned, by no means with how a central nervous system analyses the data coming to it along auditory nerve fibres, but rather with the initial capture of those data in the cochlea - indeed, a brain can produce all its miracles of acoustic interpretation only where it works on good initial data.

The earlier paper[1] described a comprehensive revolution in the understanding of hearing sensitivity in man and other mammals which took place during the 1970s and 1980s. This became possible only after a series of new measurement techniques (involving initially the implantation of Mössbauer sources) permitted the *in vivo* measurement[2,3] of tuning curves for basilar-membrane vibrations near the base of the cochlea at sound levels over 70 dB, and demonstrated enormously sharper tuning than previous measurements[4] (made on cadavers) had indicated. It continued after notable refinements of technique made possible an extension of such measurements to sound levels of 20 dB or less[5], where yet another massive increase in the sharpness of tuning was found - making it as sharp for basilar-membrane vibrations as it was already known to be for auditory nerve fibres. Then important clues to the source of this additional sharpening at low levels were obtained from Kemp's discovery[6] of evoked otoacoustic emissions. Very briefly, the analysis[1] of all these data suggested that the cochlea's role in frequency analysis and in sensitivity to sound at low levels arises from a combination within it of two systems as follows.

1.1. A passive macromechanical system

The properties of the cochlea as a passive macromechanical system, comprising the basilar membrane with its steeply graded stiffness distribution vibrating within the cochlear fluids (which participate in the vibration), had been increasingly appreciated from 1970 onwards. They yield to a first approximation its refined frequency selectivity. Thus, in a wide frequency range, acoustic signals of different frequencies become separated from each other, with each frequency component ending up at its own characteristic 'place' on the basilar membrane, where, therefore, a local inner hair cell can generate activity in attached auditory nerve fibres, tuned rather sharply to that particular frequency.

All of this happens because differences of pressure across the basilar membrane are propagated as traveling waves, such that each frequency component starts out as a nondispersive wave (where that membrane is stiffest - with fluid motions filling the cross-section) but undergoes more and more dispersion where the membrane stiffness becomes reduced - with fluid motions increasingly confined to a near neighborhood of the vibrating membrane. The energy in that frequency component propagates at the group velocity, which falls to zero steeply at the characteristic place (different for each frequency) where accordingly the energy can 'pile up' and thus powerfully excite the inner hair cells.

1.2. An active micromechanical system

On the other hand the biomechanics of hearing sensitivity to very low levels of sound (at any particular frequency) calls also into play an active micromechanical system, which during the second half of the 1980s was progressively identified as located in the outer hair cells, and which, through a process of positive feedback, amplifies (in healthy ears) the vibrations of the membrane. This in turns offers the inner hair cells an enhanced signal at low sound levels, so that the threshold at which they can generate activity in auditory nerve fibres is, in consequence, very substantially lowered.

It was during the early evolution of mammals that the differentiation of cochlear hair cells into 'inner' and 'outer' took place - such differentiation being absent in reptiles and birds but present in all the mammalian orders. Advantageous improvements in hearing sensitivity seem to have resulted from this differentiation of function, with inner hair cells specializing in the transduction of mechanical vibration into neural activity yet lacking any of that active motility which the outer hair cells possess. This active motility moreover, includes an ability to go into continuous vibration at an acoustic frequ- ency, sustained by a physiological (that is, metabolic) power source. Excitation of such vibrations by a very low acoustical signal can (on their communication to the basilar membrane) produce a significantly enhanced stimulus to the inner hair cells.

1.3. Plan of this survey

The present paper makes no attempt to recapitulate any of the experimental evidence for, or any of the analyses underlying, those interpretations of hearing sensitivity which have just been outlined, since all that material is available in the earlier survey[1]. By contrast, the rest of this paper is