of the source. The decay is particularly important in this case since it is asserted that the vibration is halted by 'pushing the cilia against or into the tectorial membrane'. Not only will this indisputably leave a decaying transient (which will admittedly decay more rapidly for the inversely tapered cilia than it would for an equivalent constant section cilium) but, the cilia giving rise to neural action by virtue of mechanical stress on the hair cells, arrest of the motion by contact with the tectorial membrane will give rise to additional stress on the hair cell over and above that caused by the initial acoustically-related vibrational stress.

The Stylis theory may be a useful package whereby it is possible to develop some qualitative comprehension of the functioning of normal and defective ears but it cannot withstand anything more than superficial investigation.

Yours faithfully,

V. Marples.

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University of Warwick,
Coventry CV4 7AL.

DEAR SIR,

Thank you for drawing my attention to the letter from Dr. Marples of the University of Warwick. So the pundits *do* object.

I recollect the essential features of my Duplex Theory of Hearing (Stylis, 1971) in which I postulate the following.

1. Two wave motions are produced by the stapes footplate.
   (a) True sound waves.
   (b) A gross pressure wave (a 'near field effect').
2. The basilar membrane is deflected by the latter wave which causes the travelling wave of von Bekesy. This carries the hair cells to and from the tectorial membrane.
3. The cilia of the hair cells are not fixed to the tectorial membrane.
4. The cilia act as tuned resonators to receive the frequency of the true sound waves.
5. Two factors are necessary for the transduction of sound vibrations to electrical energy.
   (a) Exposure of the cilia to endolymph.
   (b) Vibration of cilia.
6. The function of the tectorial membrane is that of dampener as well as a protective medium for the cilia from the hostile endolymphatic environment.
7. Loudness is a complex function of:
   (a) Amplitude of movement of the cilia;
   (b) Degree of freedom from the gelatinous layer of the tectorial membrane.
   (c) The various relationships revolving about the type of movement of the basilar membrane and the rods of Corti, and the relative exposure of the various rows of hair cells.

The primary aim of my theory is to improve comprehension of clinical symptoms and signs by providing a physiological basis. A further aim is to throw doubt on the presently accepted view on the fundamental set-up in the cochlea upon which all subsequent thinking has been based.

Either cilia are firmly attached to the tectorial membrane or they are not. Scientists have committed themselves entirely to the former on scant evidence only (discussed later) and have also ignored the presence of true sound waves in the cochlea since the conception of the non-resonance theories on hearing.

Should these basic premises be incorrect then physicists will have to re-orientate their thinking and apply their talents to a different set of fundamentals, such as my proposals.

Wever (1949) must also have felt uneasy about such fundamentals when he expressed his learned views on the use of models in research which I now quote. 'I have given little attention to the observations made on mechanical models of the cochlea, a very great many of which have been devised and manipulated in support of particular theories.'

'It has seemed to me that each model only reflects the properties built into it, and its performance testifies rather to the mechanical ingenuity of its inventor than to the correctness of his views about the ear.

'Likewise, and for much the same reason, I have given little credence to the mathematical formulations of the theories. They are only models of a formal, symbolic sort, and they, given an illusion of precision to ideas that on account of the limitations in our knowledge, cannot really be precise. Like the mechanical models, they appear at the present stage of our theorising quite as likely to confuse and mislead as to inform.'

Such remarks detract from the evidence based on model experiments that Marples calls upon to refute my claim that sound waves could be reflected in a straight cochlea.

On studying the reference cited, it is quite obvious that Cannell's (1969) attention was entirely on the travelling wave on the cochlear model partition. As I have pointed out, this wave is caused by only one of the two wave actions initiated by movement of the footplate. Marples has failed to distinguish between the two.

That two waves must exist is borne out by Harris and Bergeijk (1962) who studied sound propagation under water. They also showed that the 'far-field effect' (sound pressure propagation or 'true' sound waves) decreased only linearly with distance; whereas the 'near-field effect' (a movement of water particles or the gross-pressure wave) rapidly decays with distance to the second or third power. So for a given sound, true sound waves travel further than the near-field effect, and whilst the travelling wave may not be reflected, this has nothing to do with what is simultaneously happening to the true sound waves.

For should any wave motion reach the limit of the cochlea, basic laws of physics insist that they are reflected. Cannell was not looking for true sound waves and in his experiments they have gone undetected.

Marples refutes my thought relating to the dimensions of the basilar membrane by saying, 'a series of model tests has proved that the width and thickness of a membrane at the distal end of the type of model referred to above are

closely related to the frequency of the pressure wave to which the membrane responds at this location'. This theme dominates current literature.

To me this means that, if you like playing with models, you can make the distal end of a membrane wiggle to a given frequency by altering its width and thickness. But this is no indication of the *natural* role of the *basilar* membrane nor does it disprove my proposals.

Indeed, such vibration, whether coincidental or by design, would not influence my theory adversely at all, but actually assists by exposing the cilia in this region to enable them to receive the true sound waves to which they are 'tuned.'

In answer to Marples' remarks on vibrations and hair cell action in his second last paragraph I summarize further thoughts.

When cilia are withdrawn (by basilar membrane defection) from the protection of the tectorial membrane, they are exposed to an enormous gradient across the unit membrane in the form of chloride ions and also of electric potential. I postulate that vibratory movement of the elctro-negative cilia -70 mV) in this hostile electro-positive endolymphatic environment ($+80$ mV) forms the basis of a triggering mechanism for discharge potentials.

I emphasize the following:

(i) The movement need not reach its full vibration; in other words, it suffices that the vibration is initiated in the specific tuned elements (cilia), build-up and decay become unimportant. (I presume polarity of movement together with spatial, temporal and neural relationships between hair cells are important factors).

(ii) When the cilia are opposed to the tectorial membrane and surrounded by its gelatinous envelope, not only are vibrations impeded but also the cilia are protected from the hostile endolymph so necessary in transduction.

(iii) Rise and decay of vibrations, one way or another, affects all theories, but mine overcomes time taken in build-up, and provides a mechanism for more rapid decay, before the vibration even gets into full swing and whilst the cilium is in a non-communicative state, so to speak.

My concept of energy transduction has been ignored by Marples as he lapses into preconceived ideas, (such as cilia causing 'mechanical stress on the hair *cells* . . .') which he attempts to superimpose on my theory but which are rightly incompatible with it.

At first sight, the calculations on the natural resonance frequency of cilia seem a serious objection, but these again are based on assumptions; the simple fact is, that we just do not know enough detail on the composition and structure of these elements, or what subtle differences exist in chemical and physical properties of cilia at either end of the cochlea.

It is possible that cilia are bound together in groups by an interciliar extracellular substance. Such a substance, normally invisible, has been shown to exist by special staining methods and is discussed by Spoendlin (1968). It even has a certain periodicity indicating a regular molecular arrangement (Christiansen, 1964).

This would vary diameter to length relationships and with the physical qualities of the connective substance would alter drastically the assumptions on which Marples has based his calculations.

Correspondence

Kimura (1966) studied the dimensions of cilia using the electron microscope. In the guinea pig the length of the cilia of the outer hair cell varied from the shortest in the basal end ($1 \cdot 7\mu$) to the longest in the apex ($5 \cdot 7\mu$). He did not compare diameter here, but in the monkey the inner hair cell had cilia which at their tip measured 5,200 Å as compared with 3,000 Å of the basal outer hair cell.

Engstrom *et al.* (1970) also present similar findings and the electron micrographs are so clear and instructive that any interested reader ought to inspect them for himself.

So differences in dimension do exist; why not further differences in chemical and physical properties? Marples' arguments are no more proven than mine.

Finally, the question regarding the attachment or not of cilia to the tectorial membrane. The literature is very scant on this subject. Kimura says that the cilia of the inner hair cells are definitely not attached to the tectorial membrane. Because some condensation of cytoplasm occurred at the tips of the cilia and the odd tip was broken off and remained attached to the tectorial membrane, he claims that the cilia of the outer hair cell are firmly attached to this membrane. My proposition has been that the cilia enter the gelatinous layer of the tectorial membrane, and are in this regard 'embedded.' In histologically prepared material the hairs may leave 'impressions' in this layer. Nowhere in Kimura's micrographs did I gain the impression of firm fixation. The fibrillary markings in the tectorial membrane are not orientated in the lines of stress as expected if the cilia were fixed'. Iurato (1961) and Engstrom *et al.* (1962) state that the sensory hairs and filaments of the tectorial membrane are not structurally continuous.

In surface preparations of the organ of Corti the tectorial membrane is lifted off to reveal intact cilia. Even if there is some sensation of attachment the cohesion between fluid or gelatinous substance could account for this.

In electron micrographs the cilia appear as stiff, rod-like structures, a little club-shaped in that they narrow near their attachment to the cell. Such structures do not seem amenable to stretching in a rapidly repeated fashion (at the frequency of sound waves) as would occur if they were fixed, and indeed what structure could withstand this form of stress?

In Ménière's disease, the basilar membrane is deflected so far from the tectorial membrane by the hydropic distension that if the cilia were attached they would be torn apart. Should cilia depend on fixation to the tectorial membrane for their action then no recovery at all would be possible; yet clinically, at least some recovery is the rule.

The above factors are very strong arguments against firm fixation of cilia to the tectorial membrane and thus cast a serious doubt on currently accepted views.

It also casts doubt on the theory of Tiedemann (1970) who, contrary to Marples' innuendoes, offers no proof that cilia are attached to the tectorial membrane. His theory is a resonance one, postulating the fibres of the tectorial membrane as the tuned elements; shades of Hasse 1867, Ewald, 1898 and Shambaugh, 1907 whose tectorial membrane theories are discussed by Wever (1949). Tiedemann's work constitutes no threat to my theory, which is the first one to postulate free cilia as tuned resonators. It is surprising that Marples

is prepared to accept one resonance theory, yet will give no second thought to the possibility of cilia being the tuned elements. After all, these stiff, tuning-fork-like rods, free from lateral attachment are ideal to act as resonators. Fibres in the tectorial membrane are completely enclosed in connective substance and interlaced with fibrils, factors such as those which brought about the downfall of the earliest resonance theory of Helmholtz involving the basilar membrane.

Summary

1. The currently accepted view on the fundamental structure and relations of the hair cells has never been clarified sufficiently to warrant the faith it has received as a basis of acoustic research.

2. Models and mathematical formulations can give most erroneous information and, worse still, create false aura of accuracy.

3. Specific answers are presented to the questions raised by Dr. Marples. The discussion has reaffirmed my faith in the Duplex Theory, which is simple and logical and which continues to offer more to the clinician than any other.

Acknowledgements

In conclusion, I truly am sincerely grateful to Dr. Marples for giving some thought to my article and for taking the trouble to publish his views. I hope that I have convinced him that, contrary to his closing remarks on superficial investigations, his is merely a superficial rebuttal which fails to confront the fundamental issues raised. I'd rather he reconsider the broad concept of my theory with a mind uncommitted to prior ones.

Yours faithfully,

S. C. Stylis, F.R.C.S.

Mail Service No. 2692/3,
Bank of N.S.W.
Sackville Street,
London W.1.

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