The Elastic Structure of the Cochlear Partition

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1. Introduction

In most theoretical models, each resonant section of the cochlear partition (CP) is considered unconnected to its nearest neighbours. The motivation to describe various otoacoustic, perceptual, and pathological auditory phenomena has spurred the modification of this simple model to include a variety of non-linear dynamics. In this paper, we examine one further modification; the inclusion of elastic tissue that longitudinally couple one resonant region on the CP to another. One immediate implication is the production of the realistic $2f_1 - f_2$ distortion product otoacoustic emission (DPOAE). Thus, such structural mechanics may provide an answer to the seeming paradox of DPOAE production in dead ears.

2. Background and Motivation

The human ear is capable of transducing sound vibrations of the eardrum with amplitudes smaller than the diameter of a water molecule over a frequency range spanning 3 orders of magnitude. To account for this remarkable acuity, Thomas Gold suggested that the ear filters sound vibrations actively, and as a consequence, should produce sounds of its own [1]. Known as Otoacoustic Emissions (OAE's), these actively produced sounds were first recorded by David Kemp in 1978 [2].

Today, it is well established that motile hair cells in the cochlear sensory epithelium are the source of this activity. Their damage or necrosis have been linked to hearing pathology. Consequently, clinically monitoring the loss of OAE's is thought to provide an objective diagnosis of hearing dysfunction. In the clinic today, DPOAE's are used to screen newborn infants for hearing loss. Probing the ear with two simultaneous tones at frequencies f_1 and f_2 produces DPOAE's, which are the non-linear harmonic and intermodulation distortions produced by the active motion of the sensory hair cells. Oddly, the most prominent distortion occurs at a frequency $2f_1 - f_2$ for all mammals, reptiles, and birds. For this reason, the $2f_1 - f_2$ DPOAE is used extensively in clinical applications.

3. Hypothesis

There does exist some controversy over the clinical suitability of DPOAE based hearing assessment. Active mechanisms are not the only source of cubic distortions in the cochlea [3]. Skeptical clinicians have always been quick to point out that DPOAE's are recorded in dead ears where active cochlear mechanisms are no longer physiologically viable [4]. Dead hair cells, by definition, are no longer motile. How is it possible to record DPOAE's many hours after death? So there exists an air of paradox surrounding DPOAE utility; on one hand they are related to the healthy function of living cochleae, on the other, they are present in dead cochleae. How is it possible to elicit a response associated with a healthy ear from a dead ear?

We feel that the answer to this paradox lies in the structure of the sensory epithelium that houses the sensory hair cells. One common feature of living and dead cochlea is their structure. Exclude the activity of the outer hair cells and what remains is the sensory epithelium within which all the sensory cells reside. This sensory epithelium is known as the cochlear partition and can be likened to a piano keyboard - every section along its length is tuned for a particular frequency [5]. Popular cochlear models treat each piano key as a driven damped oscillator uncoupled to its nearest neighbours. The simplicity of this model is appealing and qualitatively describes a number of linear auditory phenomena.

In reality, the cochlear partition is a contiguous cellular scaffold spanning the length of the cochlea. Every resonant section is coupled structurally to its nearest neighbours through tight cellular junctions, adherens junctions, gap junctions, and desmosomes [6]. These points of attachment function to provide structural integrity, to separate ions within the cochlear fluids, and to maintain channels of chemical communication between cells. Mechanically, we suggest that such coupling is inherently elastic and can account for a number of observed non-linear cochlear phenomena including DPOAE's. Such elasticity is present in both live and dead cochleae, although its effects appear to be more pronounced in

a dead ear [7,8]. Since this elasticity is heightened in dead cochleae, we hypothesize that the longitudinal cellular elasticity should be responsible for DPOAE production in dead ears.

4. Results and Discussion

In our model, we take structural elastic coupling to be a critical feature of the cochlear system and responsible for cubic DPOAE production in dead cochleae. Since the cellular coupling is similar to that of Reisnner's membrane or that of healthy vascular tissue, we assume that the longitudinal elasticity in our model is also similar. Using experimental data from dead cochleae [9], our numerical solutions of the non-linear partial differential equations that describe the cochlear system demonstrate the production of cubic DPOAE's (see Figure 1). The technique to solve the non-linear cochlear system of equations is that suggested by Diependaal [10].

The non-linear effects are many and mimic aspects of DPOAE production in humans. For instance, the largest DPOAE produced in our numerical solutions was 7 dB (SPL) which is well within clinical limits. The level of the distortion rises in proportion to the stimulus with a slope of 3. Also, the $2f_1 - f_2$ distortion product is prominent over a wide range of longitudinal elasticity. The distortion level also increases as the tissue elasticity is increased, however, significant chaotic behaviour results for elasticity approaching that of pathologically stiff vascular tissue until they disappear altogether. Furthermore, the DPOAE's are not produced at the f_1, f_2 , or $2f_1 - f_2$ locations, but in between these locations.

5. Summary

In this paper we have examined the inclusion of structural elasticity amongst the resonant sections of the standard cochlear model. In most theoretical models, each resonant section is considered unconnected to its nearest neighbours. Histologically, this is not true, as there are many cellular junctions between the cells in the sensory epithelium of the cochlea. An immediate implication of adding such mechanics is the production of realistic 2f1-f2 distortion product otoacoustic emissions with properties similar to those found in clinical recordings. Since the structural coupling is not dependent on any active mechanism (which presumably is present only in living ears), our simulation provides an answer as to how dead ears produce cubic DPOAE's.

6. References

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Acknowledgements

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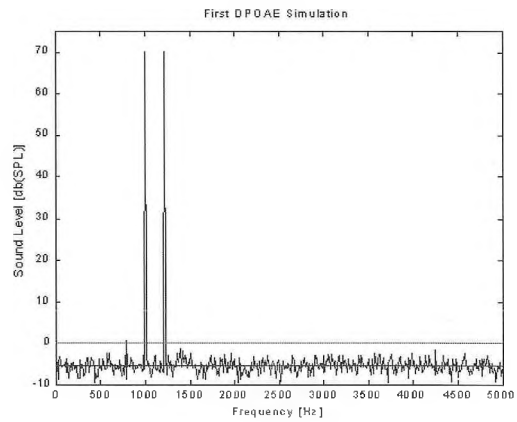

Figure 1. Distortion Product Otoacoustic Emission from Numerical Model. This figure depicts the cubic DP produced when the model was presented two tones at 1000 Hz and 1210 kHz at 70 dB (SPL). The bottom level represents the noise floor whereas the upper line show the five standard deviation from the noise floor. Clearly, a $2f_1 - f_2$ distortion is seen at 790 Hz.