HUMAN ACOUSTIC FINGERPRINTS

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Abstract

The subject-dependent features of transient-evoked otoacoustic emissions (TEOAE) have been compared in the case of normal and pathological subjects as well as in the case of real and simulated signals. The changes in the dynamic features of the simulated signals induced by appropriate changes in some model parameters mimate the inter-individual variability observed in real TEOAE. Differences in the middle ear anatomy may provide an important contribution to the variability of TEOAE signals in both physiological and pathological conditions.

1. Introduction

The authors' interest in acoustic fingerprints was inspired by the consideration that in order to realize an adequate mechanistic model of individual variability in a physiological function, the minimum prerequisite is to reproduce not the mean values of the function but rather the differences among normal individuals. In this respect, studying real TEOAES and trying and simulating them "in silico" provided an almost ideal benchmark to test a number of ideas and conjectures particularly concerning the non fully clarified etiology of many earing loss syndromes (Table 1).

Transient Evoked Otoacoustic Emissions (TEOAE) are signals generated by the active micromechanisms existing in Outer Hair Cells (OHC) within the Corti organ in the cochlea [see figure 1,2 and ref. (1) for a comprehensive and classical review]. In this report we briefly summarize

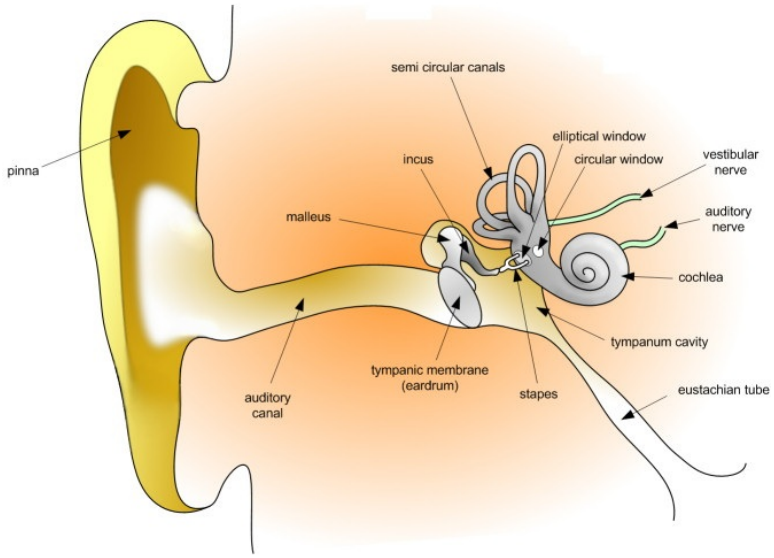

FIGURE 1. *The human auditory system.* The peripheral auditory system, including the ear canal, the tympanic membrane, the middle ear and the ossicles, as well as the inner ear, consisting in the cochlea and semicircular canals of the vestibular system, are shown. Nerves communicating with the brain are also shown. (from *Wikipedia*)

some of our results collected in studying the origin and localization of the individual features (acoustic fingerprints) observed in TEOAE (2; 3).

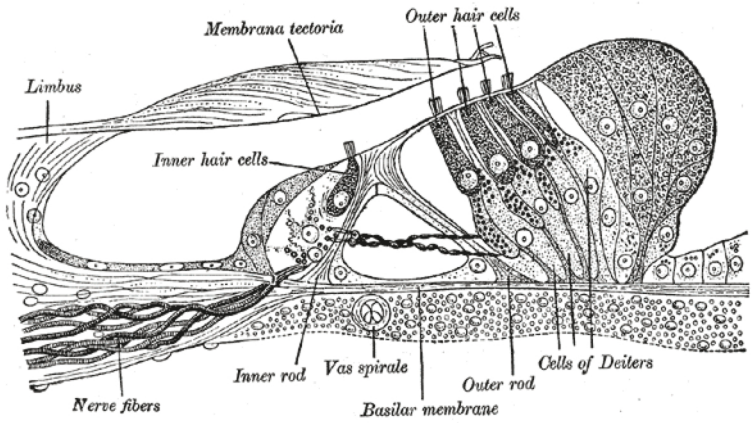

FIGURE 2. *The Corti organ.* The Corti organ is connected to the basilar membrane on the side of the aqueous fluid of the scala media. It is comprised of the supporting cells for the hair cells, the hair cells themselves, and the tectorial membrane.(from *Wikipedia*)

	Sensorineural Hearing Loss (SHL)	Conductive Hearing Loss (CHL)
Involved Anatomical Site	Inner ear cranial nerve VIII or central processing centers	Middle ear (ossicular chain), tympanic membrane or external ear
Typical cause	Noise trauma; Infections; Intrinsic abnormalities	Sound localizes to affected ear (ear with conductive loss) (deafness genes)

TABLE 1. *Sensorineural versus conductive earing loss*

2. METHODS

To provide at least an initial answer a synergic combination of Recurrence Quantification Analysis (RQA) (4) and of Principal Component Analysys (PCA) techniques was used. The former one detects fine nonlinear structures in the signals which reveal helpful in differentiating contributions to TEOAE from middle or inner ear (3); the latter one is unvaluable in providing compact, easy to understand and non-redundant representations of multivariate data-sets (9).

Real TEOAE recorded by the ILO92 system (5) allowed for two types of comparisons, namely signals from normoacousic and pathological subjects were compared among each other as well as with simulated signals.

In order to realize both comparisons, a reference set of 73 signals recorded from a homogeneous group of normoacousic subjects was built. For every signal three global RQA descriptors were calculated: %recurrence, %determinism and entropy; thus, the principal components (PCs) were reckoned from a (73*3) unit-variable matrix.

The space chosen to realize the signal comparison was the plane defined by PC1 and PC2 (explained variability: PC1=85.42%, PC2=9.23%). Over this plane every signal corresponds to a point, and the individual variability within the healthy population can be easily grasped (99% of the signals fall in the circle having centre in (0,0) and radius = 2) (figure 4).

The recurrence descriptors for each pathological and simulated signal were also calculated and successively projected by director cosines on the above reference PC1/PC2 plane (9) .

The artificial (simulated) TEOAEs were generated by the PSPICE program for the study of electronic circuits (6). The same type of stimuli used by ILO92 to elicit real TEOAEs (5) was applied to the beginning of a PSPICE model of the transmission line type (figure 3) and the output signal was recorded for different values of circuit elements corresponding to specific anatomic parts situated in the middle ear and in the cochlea ((7; 8) and Table 2). **For more details see the Supplementary Materials**.

3. RESULTS

The reproduction of the variability pattern typical of real signals (both in terms of physiological variability and of difference between normal and pathological signals) by means of the simulated ones, is the signature of the relevance of the adopted model for the study of otoacoustic emissions.

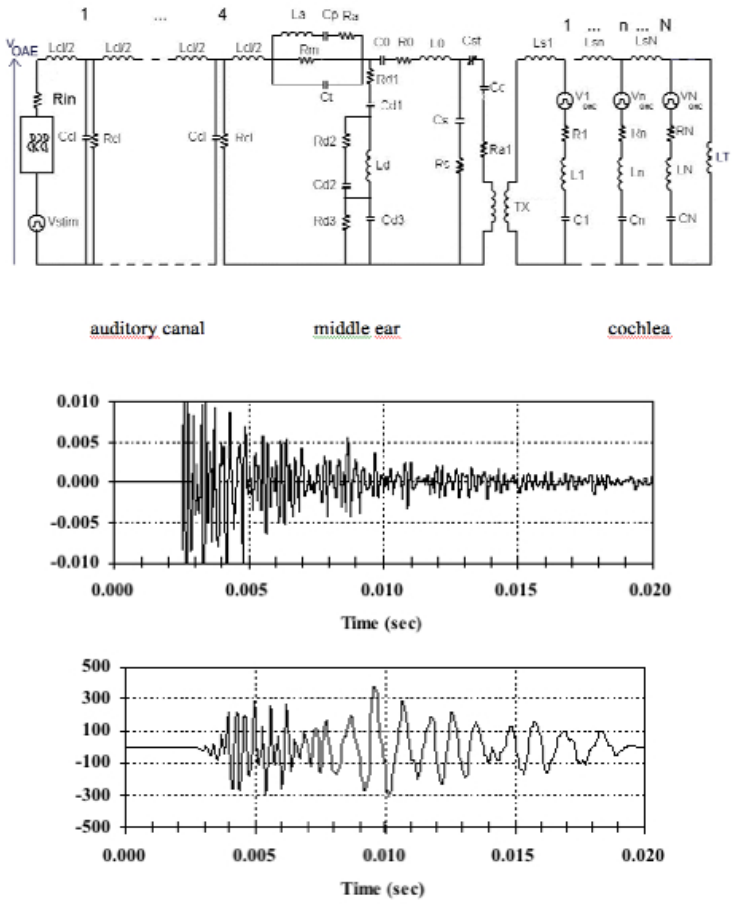

FIGURE 3. *Simulating TEOAEs signals.* Top: Electric model of the ear implemented in PSPICE (see the text). Middle: simulated TEOAE signal (V versus *time*) by the circuit in a) using 64 cochlear partitions. Bottom: measured TEOAEs signal (μPa versus *time*) from a normoacousic subject.

Circuital parameters	Values within the human physiological variability in (7)	Biological counterparts
L_0, L_d	(15-60)mH, (40-160)mH	mass of the tympanic membrane
C_{st}	(0.25-Inf) μF	stapes stiffness

TABLE 2. *Parameters [Inductance L, Capacitance C] of the PSPICE model and corresponding biological counterparts*

Plotting on the same PC1/PC2 plane points corresponding to reference signals (figure 4) and points corresponding to pathological (figure 5) and simulated (figure 6) signals, allows to draw the following considerations:

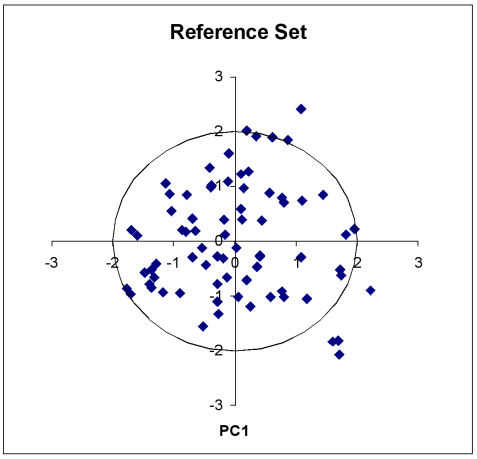

FIGURE 4. *Physiological TEOAE signals in a principal components plane.* The RQA descriptors of 73 normoacousic (NA) signals included in the reference set are %REC, %DET and ENT (signals of 512 points were analysed using: lag=1, embedding dimension=10, radius=15, line=8, first analysed point = 70). The first (PC1) and second (PC2) principal components explain 94.65% of the total variability. Since the components are standardized by construction, the circle having radius = 2 standard deviations includes the vast majority of the data set

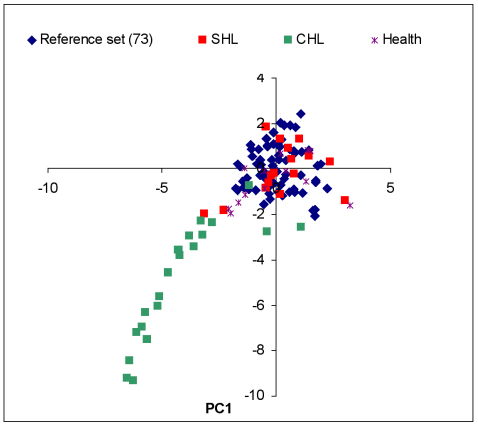

FIGURE 5. *Pathological TEOAE signals in a principal components plane.* The points corresponding to Pathological (CHL and SHL) and Normal (Health) signals are represented in the PC1-PC2 plane by projections along the score coefficients (director cosines or loadings) from the 73 reference signals in figure 4. SHL: signals from ears with sensorineural hearing losses; CHL: signals from ears with conductive hearing losses. Health: signals from normoacousic subjects recorded under identical conditions as CHL and SHL.

1. Middle-ear pathologies (CHL) fall markedly outside the region of individual variability of the normal population (normality circle). This is not the case for cochlear diseases i.e. sensorineural hearing loss (SHL).

2. As suggested by Avan and colleagues (7), small changes in the electronic model parameters associated to the middle ear produce signals falling pretty close to the reference ones, thus reproducing physiological variability in normoacousic subjects. On the other hand, the signal-points corresponding to drastic changes in the number of working cochlear sections fall far away from the normality circle. It is worth reminding that under complete destruction of the cochlear function ("dead cochlea" condition) the TEOAEs almost disappear (5).

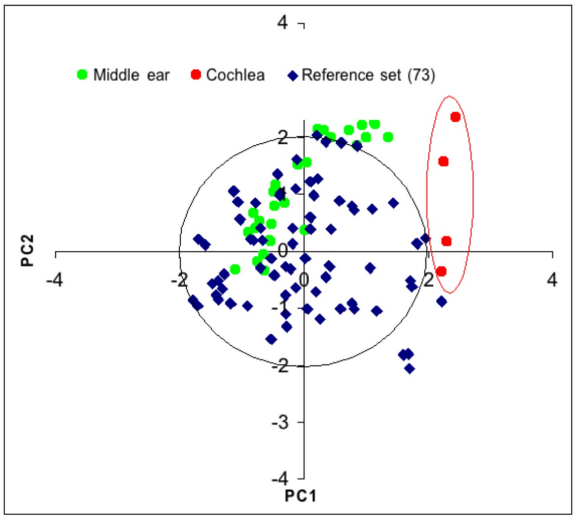

FIGURE 6. *Simulated signals in a principal components plane.* Simulated TEOAE signals were obtained using the transmission-line ear model implemented on PSPICE (see the text). "COCHLEA" points correspond to signals simulated by turning off the voltage sources in a variable number of cochlear sections. "MIDDLE EAR" points correspond to signals simulated by small changes in the circuital parameters corresponding to the middle ear (see Table 2). The points corresponding to the Reference signals in figure 4 are also included for comparison.

4. CONCLUSIONS

The middle ear contribution to TEOAE dynamic features is predominant, in particular to determine acoustic fingerprints. Three relevant facts emerge from our results:

1) A purely data-driven approach based on normal signals is able to accommodate pathological signals in a portion of the component space different from that of normal signals or, in other words, is able to "naturally" discriminate pathology.

2) The simulated signals endowed with only minor variations in the model parameters are well inside the area of physiological variability. Hence, such variability is well reproduced by the used model.

3) Also in pathological conditions real and simulated signals may be in fairly good agreement, as in the case of patients affected by inner ear pathologies and simulated signals corresponding to severe modifications of the cochlear portion of the electronic model.

All in all this work envisages a shift from the somehow rigid approach to biological phenomena widespread particularly among a considerable fraction of researchers relatively new to Biology.

Instead of an idealistic attitude towards theoretical models reproducing the ideal (average or, worse, oversimplified) case as a clean substitute of the dirty biological observations, we feel the pragmatical urgence to reproduce faithful instances from the wide spectrum of natural variability, namely the actual data in the hands of physiologists and clinicians. We hope this could be a small (but still non null) step forward in the remission of the "spherical horse syndrome" (10).

The authors are now working on the improvement of the electronic model, considered a good starting point to investigate and reproduce cochlear function, especially in the number and the amplification modality of the cochlear sections. However, because of the quite large approximations intrinsic to all the transmission line models, we foresee as necessary its evolution. Our main goal remains extending the heuristic power of cochlear models, in general, towards other applications not necessarily restricted to TEOAE, e.g. spontaneous OAEs, so to get more detailed insights into the hearing function.

5. Appendix

An electric model of the ear

The ear model being considered in the report [figure 3, top panel], inspired by the classical work in (8; 11) encompasses the human ear anatomy from the auditory canal to the Outer Hair Cells within the cochlea (14). In the model, the auditory canal is represented with a cascade of four T-sections, which corresponds to the segmented form of a uniform transmission line (12), while the middle ear is modeled as a complex electrical network based on its functional anatomy (13). An ideal transformer connects the middle ear to the cochlea, to represent the acoustic transformer ratio between the eardrum and the oval window. Finally, the cochlea is modeled as a non-uniform and non-linear transmission line. It is divided into N sections, from its base to the apex, each one consisting of a series inductor, a shunt resonant circuit (composed of a resistor, an inductor, and a capacitor), and a non-linear voltage source. In the electro-acoustic analogy, the series inductors represent the acoustic mass of cochlea fluids; the resistors, inductors and capacitors forming the shunt resonant circuits represent the acoustic resistance, mass and stiffness of the basilar membrane, respectively, while the non linear voltage sources represent the OHC active processes. Finally, the helicotrema is modeled by the inductor L_T.

The initial values of the circuit electric components are those reported in Table 1 of (8), as already used in (14). To try and reproduce the latency versus frequency behavior typical of TEOAE signals, which reflect the place-frequency dependence in the basilar membrane, a variable number of sections [up to 128] have been used to segment the cochlea. Moreover, to simulate different hearing conditions, the values of some electric components have been appropriately varied. In particular, a dead cochlea condition has been reproduced first, de-activating the voltage sources in the cochlea sections; then, to verify the finding according which TEOAE are strongly modulated by the middle ear (3), some elements in the middle ear section were varied according to the experimental study of Avan and colleagues (7). The first change considered in the middle ear section is the addition of a stapes capacitor (C_{st}) to the circuit (as already considered by (8) and (13)). When C_{st} has a large value, its impedance is small, corresponding to small tension in the stapedius muscle (C_{st} equal to infinity corresponds to no stiffness in the resting condition); conversely, when C_{st} is small, its impedance is large, corresponding to muscle high tension.

The values reported in Table 2 represent physiological conditions (7). This means that these values could correspond to different anatomical dimensions of the middle ear, as a thicker or wider tympanic membrane, but all are within the human physiological variability.Then, changes in the tympanic membrane stiffness (C_0, C_{d1}), to account for changes in the middle ear pressure,

and in the tympanic membrane mass (L_0, L_d), to simulate an additive mass, have been considered (7).

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