

## OTOACOUSTIC EMISSIONS SIMULATED IN THE TIME-DOMAIN BY A HYDRODYNAMIC MODEL OF THE HUMAN COCHLEA

R. NOBILI

*Dipartimento di Fisica "G. Galilei", Università di Padova,  
via Marzolo 8, 35131 Padova, Italy  
E-mail: rnobili@pd.infn.it*

A. VETEŠNÍK

*Department of Theoretical Physics, Palacký University,  
Svobody 26, 77146 Olomouc, Czech Republic  
E-mail: a.vetesnik@seznam.cz*

L. TURICCHIA

*Dipartimento di Matematica e Informatica, Università di Udine,  
via delle Scienze 208, 33100 Udine, Italy  
E-mail: lorenzo.turicchia@pd.infn.it*

F. MAMMANO

*Istituto Nazionale di Fisica della Materia, Scuola Internazionale Superiore di Studi  
Avanzati, via Beirut 2-4, 34014, Trieste, Italy, &  
Istituto Veneto di Medicina Molecolare, via G. Orus 2, 35131 Padova, Italy  
E-mail: mammano@sissa.it*

Time-domain simulations of the response to click of a human ear show that, if the cochlear amplifier gain (CAG) is a smooth function of basilar-membrane (BM) position, the filtering performed by a middle ear with an irregular (non-smooth) transfer function suffices to produce irregular and long-lasting residual BM oscillations at selected frequencies. Feeding back to the middle ear through hydrodynamic coupling, these oscillations are detected as otoacoustic emissions (OAEs) in the ear canal. If, in addition, also the CAG profile is irregular, residual BM oscillations are even more irregular, often ensuing to self-sustaining oscillations at CAG irregularity loci. Correspondingly, transient evoked OAE spectra exhibit sharp peaks. If both the CAG and the middle-ear transfer function are smooth, residual BM oscillations are characterized by regular waveform, extinguish rapidly and do not generate appreciable emission. Simulating localized damage to the cochlear amplifier results in spontaneous emissions and stimulus-frequency OAEs, with typical modulation patterns, for inputs near hearing threshold.

### 1 Introduction

The prevailing paradigm on transient evoked OAE generation is the transmission-line model. This is conceptually appealing, as emissions of this sort can be imagined as due to traveling wave "reflectance" at putative discontinuities of cochlear partition parameters. In transmission lines, distributed parameter discontinuities

upset the amplitude balance between progressive and regressive waves, as imposed by the continuity condition for energy-momentum local currents, and, as a by-product, generate wave reflection.

Actually, this concept does not apply to the cochlea, where energy and momentum for the BM motion are conserved globally rather than locally, for hydrodynamic coupling links distal BM sites thus overcoming possible parameter discontinuities. No continuity condition is then locally imposed to energy-momentum currents within the cochlear duct. Besides, the solutions of the BM motion equation are not of the bi-directional wave-propagation type. Rather, they are phase-delayed standing modes that in no way can be represented as the superposition of progressive and regressive wave components (as is instead, for instance, the case for guitar string standing modes). Thus, despite their name, traveling waves of a given frequency do not travel at all, and talking about wave “reflection” or “reflectance” in the cochlea is physically inappropriate and even conceptually misleading.

If no wave reflection takes place within the cochlea, how, then, are OAEs generated? Here we show how a completely different approach leads to an explanation of OAE phenomena, producing results in impressive accord with experimental data.

## 2 Methods

OAE time-domain simulations presented here are based on a published model [1-3] adapted so as to fit physical and geometrical characteristics of the human inner ear and completed with the inclusion of forward- and reverse-gain middle-ear transfer functions. The main components of the model are:

1. An array of damped oscillators coupled by long-range hydrodynamic forces representing the basilar membrane (BM) interacting with the surrounding fluid.
2. An array of damped oscillators driven by the BM acceleration representing the tectorial membrane (TM).
3. An array of nonlinear amplifiers acting as generators of forces that counteract the positional viscosity of the cochlear partition, thus boosting the oscillatory responses of the BM by 2 to 3 orders of magnitude up to 30-40 dB input sound pressure level (SPL), which corresponds to amplifier saturation.
4. Forward- and reverse-gain transfer functions, representing the effect of the middle-ear filter on signals passing through the oval window both ways.

In our investigations, the following model properties turned out to be the key players for OAE generation:

- Hydrodynamic coupling. The pressure of the fluid filling the spiral canal is a linear combination of the accelerations of all the moving components of the cochlea weighted by positive quantities (effective Green’s functions) that

depend symmetrically on the positions of the acceleration site and the pressure-action site. Consequently, the organ of Corti vibration is heavily conditioned by the inertia of the fluid. In particular, the BM senses a force equal to stapes acceleration times the Green's function  $GS(x)$  that represents the stapes-BM coupling. In turn, the stapes senses a force that is a linear combination of the BM accelerations weighted by the same Green's function. Precisely this is the way by which BM oscillations feed back to the middle ear, being sensed in the external ear as OAEs.

- The TM resonates weakly at the characteristic frequencies (CFs) of the BM. Under these conditions, OHC transduction currents are substantially proportional to TM displacements.
- Mechanical viscosity of the OHC-BM junction formed by the Deiters' cells. This provides a zero-pole type compensation for the frequency roll-off of OHC electromechanical transduction [4].
- Irregularities of the middle-ear transfer functions. In our computations, we used data by Puria and Rosowski [5] as they reproduce the only complete and sufficiently detailed middle-ear data set that we were able to find in the literature (albeit published only as preliminary conference proceedings). Middle-ear forward and reverse effects were computed by signal convolution with impulse responses reconstructed from these data.
- Stapes motion within the oval window is dominated by viscous drag [6]. This implies approximate proportionality between force applied to the stapes and stapes velocity. Stapes acceleration was then computed as a quantity proportional to the time derivative of sound pressure at eardrum convoluted by middle-ear forward impulse response.

As is well known, grading of cochlear partition distributed parameters from base to apex of the cochlea, particularly stiffness and viscosity, together with the instantaneous hydrodynamic coupling between stapes footplate and BM, and amongst the BM oscillating elements themselves, all contribute to generating responses to sounds with characteristic waveforms whose amplitude rarely exceeds tens of nanometers.

Paired to saturation of the cochlear amplifier output, this determines the markedly non-linear properties of the sound processing performed by the cochlea, notably *tone-to-tone* suppression. This property underlies what can be considered the engineering miracle of cochlear filtering: fast responsiveness paired to high-frequency selectivity.

### 3 Simulation method and results

Although bearing necessary simplifications, as all physical models do, and suffering from certain limitations in its performance (maximum amplification up to 55 dB)

and lack of precise estimates for some of its parameters (particularly those regarding viscosity), the model should be expected to agree generally at least qualitatively with experiments.

OAEs were computed as pressure detected in the ear canal and due to the intracochlear hydrodynamic force  $f_{OW}$  produced by BM oscillations at the oval window, filtered through the middle-ear reverse transfer function. Because of fluid incompressibility, which implies no time delay,  $f_{OW}$  was in turn computed as the sum of local BM accelerations  $a_{BM}(x,t)$  [ $x$ =normalized BM position,  $t$ =time] weighted by the hydrodynamic force propagator of the stapes  $G_S(x)$

$$f_{OW}(t) = \int_0^1 G_S(x) a_{BM}(x,t) dx.$$

In general, due to the progressive phase delay of TWS, the effects of BM oscillations tend to cancel out at the oval window [ $f_{OW}(t) \cong 0$ ]. Non-linear modulation of BM oscillation patterns, generally associated to tone-to-tone suppression, may unbalance selected half-wave components in a “spindle”, i.e. the TW wave-train elicited by a click (see Fig. 2), yielding non-negligible  $f_{OW}(t)$ .

### 3.1 Spontaneous OAEs

Unquestionably, increasing the cochlear amplifier gain (CAG) at a given BM site lowers the corresponding hearing threshold possibly priming spontaneous BM oscillations at that site. However, our simulations show that spontaneous OAEs may arise also from localized damage to the cochlear amplifier.

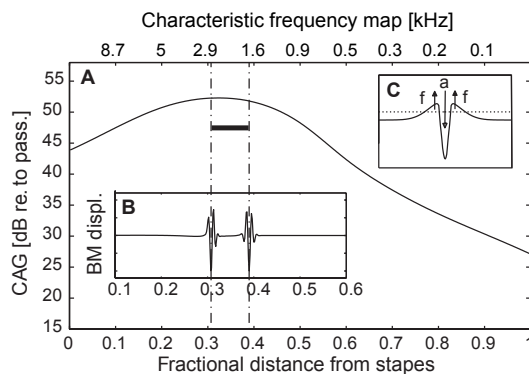


Figure 1. Simulating spontaneous otoacoustic emissions. A: gain vs. frequency profile of the regularised cochlear amplifier gain (CAG). **Thick bar**: location of the interval of lower amplification causing spontaneous basilar membrane (BM) oscillations at the interval ends (B). C: splash effect: the figure indicates how acceleration of a BM portion (*downward arrow*) causes lateral rebound forces (*upward arrows*) responsible for unbalancing undamping in the cochlea.

We discovered that this phenomenon has to be imputed to non-local unbalancing between positional viscosity undamping and shearing viscosity due to the hydrodynamic coupling. The CAG profile shown in Figure 2 was inferred using published psychoacoustic data from healthy and impaired inner ears [7].

### 3.2 Transient evoked OAEs

Our results, densely reported in Figure 2, show what is the role of irregular middle-ear filtering in producing stimulus evoked OAEs. Figure 2 shows the different time course of TW trains (spindles) elicited on the BM by clicks convoluted with the pulse response of an ideal middle ear with regular (smooth) transfer function profile (left panels) and that of a real middle ear with irregular transfer function as reported by Lagostena *et al.* [4] (right panels).

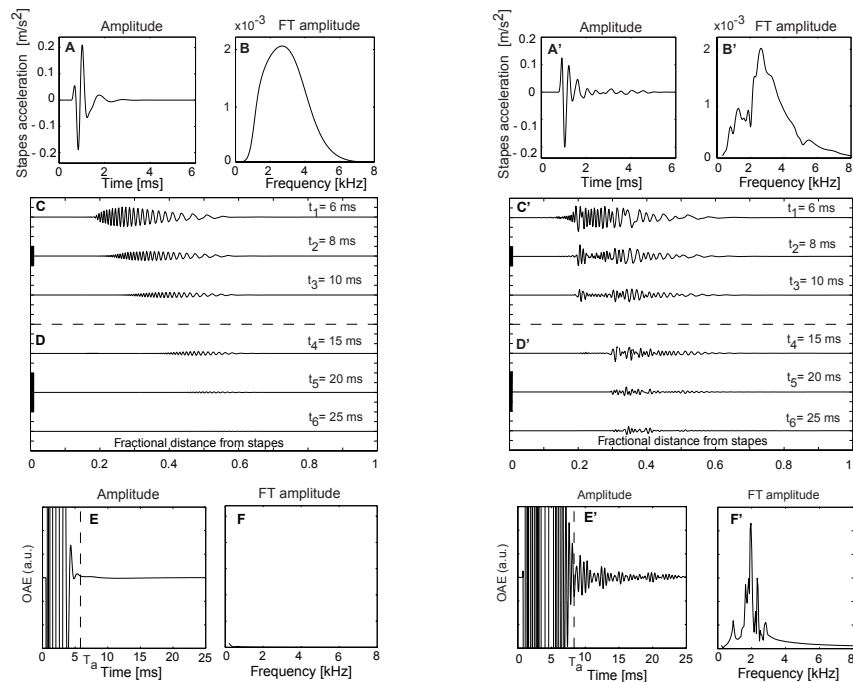


Figure 2. Simulating click-evoked otoacoustic emissions. Left panels show time waveform (A) and Fourier transform amplitude (B) of stapes acceleration following a click filtered through an ideal middle ear with a smooth transfer function. C,D: show the time course of BM oscillation spindles. E,F: show corresponding OAE time-course and the Fourier transform amplitude of its post-transient portion (which is virtually zero). Right panels (A'-F') show similar quantities for a click filtered through a middle ear with transfer functions as in [5]. Spindles as regular as those shown in (C,D) can be obtained only provided that the CAG profile is extremely smooth. After the initial transients due to immediate reverberation at oval window of BM-base oscillation, no transient evoked OAEs are seen in panels (E,F). In contrast, the most remarkable response features in (C',D'), obtained with the same CAG profile, are spindle irregularities, persistence of BM oscillations at characteristic frequency sites close to the sharpest peaks of the middle-ear forward-gain transfer function (see [5]) and transient evoked OAE (E',F') strikingly similar to those well-known to audiologists. These arise as a combination of two main factors, which are both related to tone-to-tone suppression enhancing the irregularity of the middle-ear frequency filtering: *i*) lateral suppression of comparatively smaller BM oscillations at frequencies close to the frequency of dominant oscillations and *ii*) mutual quenching of BM oscillations associated to a continuum of equally expressed responses.

### 3.3 Stimulus frequency OAEs

It is well known that air pressure detected in the ear in concomitance with pure-tone stimulation at the threshold of hearing exhibits frequency dependent modulations that tend to disappear when the input level approaches 30-40 dB sound pressure level (SPL) [8]. We reproduced this phenomenon by simulating the OAEs evoked by low-level stimuli of slowly varying frequency in the presence of a slight localized damage in an otherwise extremely regular cochlear amplifier.

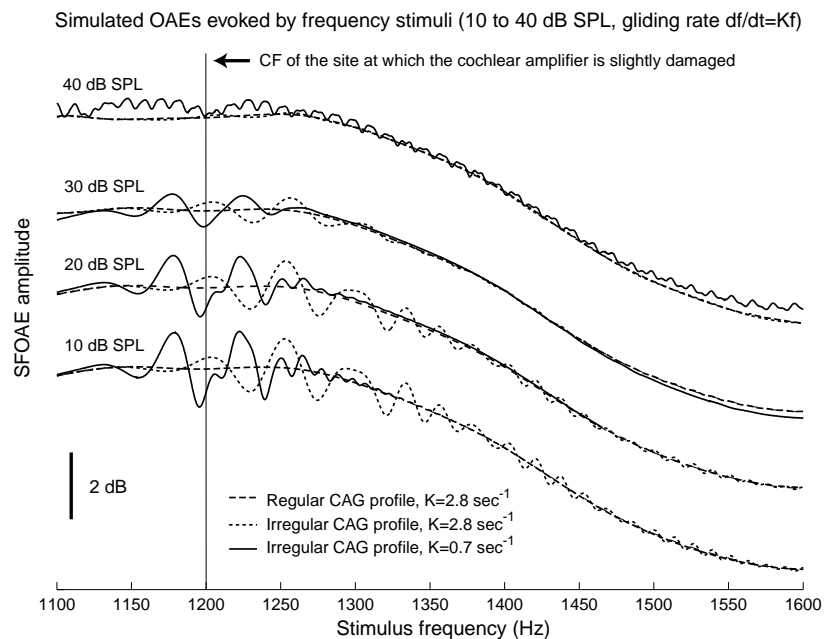


Figure 3. Frequency-stimulated otoacoustic emissions. Emissions as detected at the eardrum and stimulated by stimuli of 10 to 40 dB SPL and frequency  $f$  slowly varying according to the law  $df/dt=Kf$ , were simulated by a time-domain implementation of the model described in the text. **Solid line:** ( $K = 0.7 \text{ sec}^{-1}$ ) the emission generated when the cochlear amplifier is slightly defective at the BM site of CF = 1.2 kHz. **Dotted-line:** the same with ( $K = 2.8 \text{ sec}^{-1}$ ). In both cases, modulations of maximum ~2dB amplitude and ~50 Hz spacing, extending over an interval of ~250 Hz, are noted in the emission profile; their amplitude is larger at smaller input levels and is negligible when the BM response reaches the saturation level of the cochlear amplifier (35-40 dB SPL). **Dashed line:** ( $K = 2.8 \text{ sec}^{-1}$ ) emission of a cochlea with regular (smooth) CGA profile; no modulations are noted.

Our analysis shows that the appearance of stimulus frequency OAE modulations, which are generally associated to the presence a spontaneous emission at the CF of the damage site, is caused by the interference between the TW elicited by the input tone and a perturbation TW component whose phase depends on the distance between the damage site and the CF of the TW.

#### 4 Discussion

The present findings have far reaching implications. Analysis of the model's performance under various conditions indicates that either marked irregularities in the forward gain transfer function of the middle ear, with a regular CAG profile, or slight irregularities of the CAG profile, with regular transfer function, suffice to generate detectable transient evoked OAEs. Very often, in the latter case, spontaneous emissions arise too. Thus, when found in the absence of spontaneous emissions, transient evoked OAEs are likely to be mainly imputable to the characteristics of forward middle-ear filtering. This explanation accords with hypotheses previously advanced on the basis of the similarity between middle-ear transfer function profiles and spectra of transient evoked OAEs.

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