Six experiments on a 1-D nonlinear wave-digital filter modeling of human click-evoked emission data

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Presented at "Auditory Mechanisms: Processes and Models"
The Ninth "Mechanics of Hearing" Workshop
Oregon Hearing Research Center,
Oregon Health & Science University
Portland, Oregon, USA, July 23 - 28, 2005

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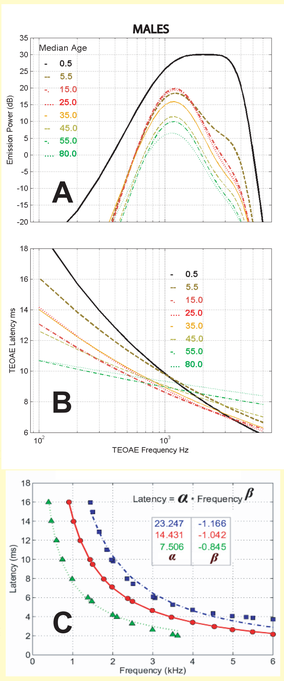
Background

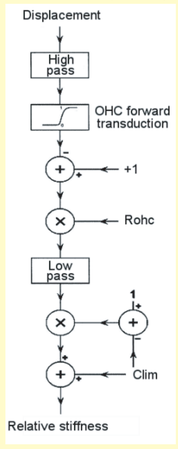
Human Click-Evoked Otoacoustic Emissions (CEOAE) exhibit considerable variation over the long term due to many factors including aging, as seen in a large study [1]. There remains uncertainty as to whether any particular feature in the time-frequency plane has a unique origin place or latency, or whether, as seems likely, there are many solutions to "back-solving" the emission to reveal the pattern of activity responsible for it, e.g. the need to explain the presence of short-latency, low-frequency emissions.

Here, a basic 1-D model is used to gain insights into origins of CEOAE obtained using the ILO™ "nonlinear" test paradigm. The model allows trying various scenarios to mimic known physiological factors, such as the decline in endocochlear potential (EP), long-term change in stiffness, and change in electromechanical time constants. Curves A & B are for pooled data from 2000 males in different age ranges, while C shows the model used to fit both human and model latency data. *Experiments 1-3* describe effect of a global change in activity, while *4 & 5* are for punctate or enlarging "lesions" and *6* describes the effect of varying the warp of the F-P map [8].



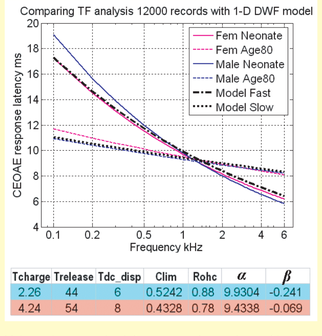
Methods: 1-D WDF model

The discrete time domain model of Nilsson [3] has been reformulated using the wave-digital-filter (WDF) approach [4], by Olofsson [5]. This has been reimplemented as a Mex (.dll) file which is compiled by and called from MATLAB™. The delay-line model is made up of 350 segments like that shown here. Each element employs forward transduction of the sound signal by the outer hair cells (OHC). Stiffness variation in this model achieved at the forward transduction stage (Preyer model [6]). The resistance element is tapered to lower values towards the apex. The mechanical feedback to the basilar membrane is produced by OHC basolateral membrane voltage variations; the level of feedback is set as the fraction Rohc in [0,1]. Lower limit to the decrease in BM stiffness is Clim in [0,1]. The filters are one-pole. Low pass has separate time constants. The OHC reacts rapidly (Tcharge) with sound increase and it holds that state for a while (Trelease). For many healthy OHC the relative stiffness will change a lot depending upon level, and vice versa.



Experiment 1: Finding model parameters to mimic aging effect in human latency data

Tdc_disp	Trelease	Tcharge	Clim	Rohc	b1	b2
1	100	0.1	0.5	0.7	13.649	-1.1
10	10	10	0.25	0.8	13.215	-1.169
10	100	0.1	0.5	0.7	13.171	-0.675
1	100	0.1	0.25	0.9	12.256	-0.83
5	100	0.1	0.5	0.7	11.994	-0.603
5	100	1	0.25	0.8	11.823	-0.426
10	10	1	0.5	0.7	11.563	-0.717
1	10	10	0.25	0.7	11.457	-0.852
1	50	1	0.25	0.8	11.452	-0.577



Tcharge	Trelease	Tdc_disp	Clim	Rohc	α	β
2.26	44	6	0.5242	0.88	9.9304	-0.241
4.24	54	8	0.4328	0.78	9.4338	-0.069

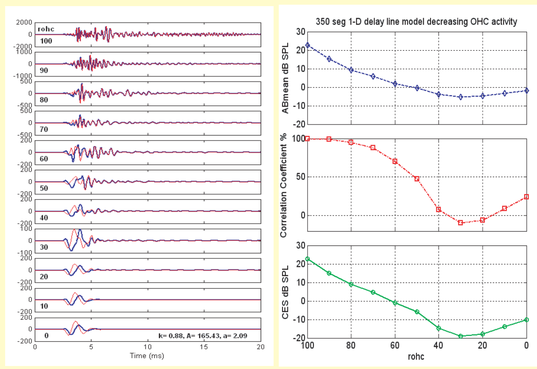
Values chosen to look for appropriate values of latency to mimic human data (see sample from result table above):
Rohc = [0.7 0.8 0.9]; OHC 'kick' measure of f/b resulting
Clim = [0.25 0.5 0.707]; Limit to stiffness decrease
Tcharge = [0.1 1 10]; LP filter charging time constant (ms)
Trelease = [100 50 10]; LP filter release time constant (ms)
Tdc_disp = [10 5 1]; HP filter time constant (ms)

5 parameters of the model are explored, charging and release time constants, baseline time constant (Tdc_disp), lower limit on stiffness decrease (Clim) and fraction of OHC activity (Rohc). 3 values of each are explored (243 permutations) to produce coefficients α and β of the curve used to characterise human data for neonate and 80 year olds. **The curve corresponding to neonates requires higher, faster activity and higher stiffness while aged ears correspond to slower, weaker, slack responses.**

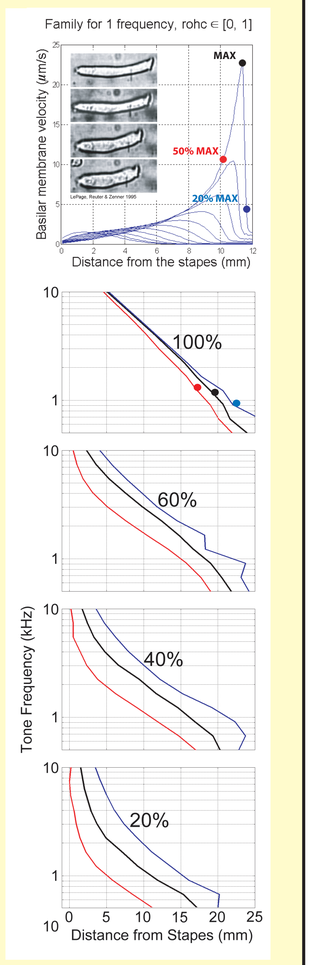
None of the 243 permutations produced a latency curve with a wider range than the neonatal case, suggesting that activity in the pristine cochlea yields maximal spatial span.

Experiment 2: Effect of decline of Rohc upon CEOAE strength

There is evidence from gerbil that the stiffness of the basilar membrane depends on OHC basolateral membrane potential which, in turn, depends upon Endocochlear Poential (EP). The EP declines with age i.e. from >+80mV down to ~ +15mV [7]. So we vary Rohc uniformly over all 350 segments to mimic EP decline, in steps of 10%. **The model displays characteristics very reminiscent of aging human CEOAEs, e.g. decline in the strength and high frequency content [1,2].** Computing scalar measures of emission strength displays qualitatively similar trends for decline of ABmean, Wavepro(%) and Emission Strength (dB SPL) with increase in age; e.g. Wavepro does not change initially, unlike ABmean (the rms value of the emission).

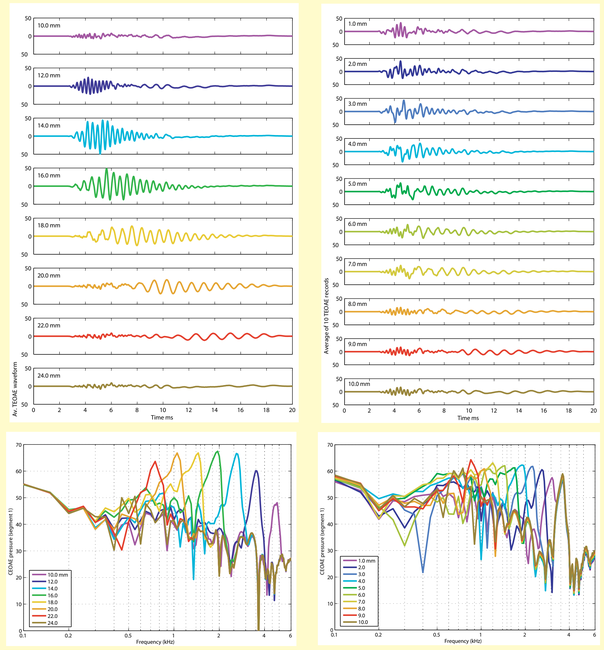


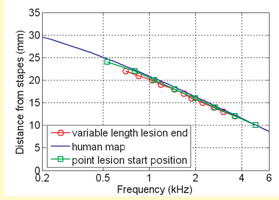
Experiment 3: Freq-place map is affected by Rohc



Although the map is specified in terms of tapered values for mass, stiffness and damping, Rohc affects stiffness and in turn the 'active map'. To see how much the map shifts with Rohc if Clim = 0, (i.e. no limit on stiffness decrease, say due to passive limit), Rohc is reduced here in steps of 20%, and maybe also with OHC degradation (see inset). This is determined by plotting the spatial location and extent of the peak of the traveling wave envelope, as computed from the locations of the 0.5, 1 and 0.2 x MAX values. It seems the active component of stiffness as controled by the EP is significant. **The model suggests that as OHC (drive) is degraded, the map collapses with loss of HF resolution.**

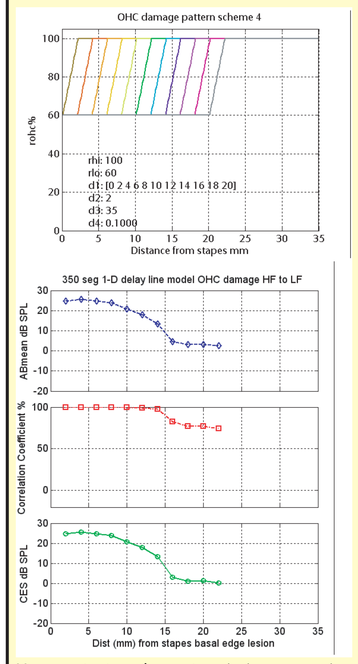
Experiment 4: Punctate lesions





OHC lesions are modeled as ***small*** reductions in Rohc for just a few of the segments of the model. For both sets Rohc = 80% globally, and 75% defining the lesion. The set on the left has a 0.1 mm lesion moved down the cochlea in 2 mm steps. On the right the lesion starts at 12 mm and increases in length by 1 mm steps. Left: maps of moving edges.

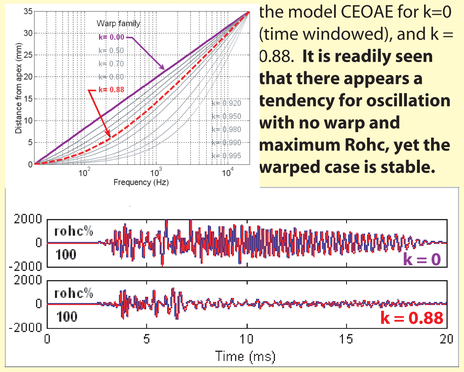
Experiment 5: large spreading lesions



Here we test a scheme to mimic progressive loss of OHC from the base in 2mm steps, such that Rohc drops by 40%. CEOAEs are not strongly affected for the basal 10 mm because of the (2.5, 20) ms time window applied to mimic clinical processing of transient response.

Experiment 6: Map warp

At the last Mechanics Meeting, the notion of warp was introduced to generalise the Greenwood model for the map [8], finding a warp-factor (k=0.88) common to all mammalian species, even if the cochleas were too short for full development. This WDF model allows us to explore how CEOAE would appear for k<> 0.88. Here we compare the model CEOAE for k=0 (time windowed), and k = 0.88. **It is readily seen that there appears a tendency for oscillation with no warp and maximum Rohc, yet the warped case is stable.**



Conclusions

Six experiments are conducted on a 1-D WDF model: **1)** Variation of model time constants to see how CEOAE latency is affected. Younger cochleas correspond to higher stiffness and faster activity. **2)** The influence of age, changing in scalar measure of OHC; activity mimics real data in a large database. **3)** Influence of decline in activity upon the tonotopic map. **4)** The presence of small "lesions" of downgraded OHC activity [9,10] has a profound effect on the size of spectral peaks (10 - 15 dB) in the emission. **5)** Global loss of OHC activity due to noise exposure. The approach may be helpful in understanding clinical emissions. **6)** Effect of warping the map by different degrees. The evolutionary development of map curvature may have occurred to maintain cochlear stability.

Acknowledgements

This work was supported in part as Project HLPR99.1 on location at both the National Acoustic Laboratories, Sydney, and also by the Karolinska Institute, Stockholm.

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