My remarkable trip from Delft 1983 to Denmark 2022: 50 years of cochlear modeling

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Abstract. The goal of this presentation is multi-fold: First, to reflect back on the role of the MoH meetings, and our present understanding of cochlear function. Second, to briefly review the contributions of three close friends during this intellectual trip: Egbert deBoer, Steve Neely, Paul Fahey and George Zweig. Finally, and perhaps most importantly, I would like to review my personal view of the experimental data on cochlear function, from the tympanic membrane, middle ear, basilar and tectorial membranes, inner and outer hair cells, auditory nerve, and the cochlear amplifier.

INTRODUCTION

The path to new friends: After obtaining my MS (electrical thawing of frozen dog kidney) and PhD (modeling a 1 [m] $\times$ 1 [cm] 18 kC° plasma arc-jet) from Univ. Penn (2003) in Electrical Engineering, I joined the *Acoustics Research Department* at AT&T Bell Labs. I soon was introduced visiting scholar Egbert deBoer, who was widely known for his significant wide-range of hearing-science contributions. Egbert then informed me that I would start modeling the inner ear. I took the "hint" from both him and my brilliant supervisor David Berkley, who had also published on this topic. I was immediately in the able hands of Mohan Sondhi who greatly upgraded my otherwise meager understanding of the necessary mathematics. Several papers followed, most notably Allen[3,9], Allen and Sondhi[14], Sondhi[23,24,25].

Researching the inner ear: Bell Lab's Acoustics Research Department was mostly involved in speech recognition (ASR) and various acoustic applications (speaker phone and electrete microphones), but historically there was always one person doing hearing research, and I quickly morphed into that lucky sole. At Bell Labs, limits were only determined by ones imagination.

The next major event in my life was the arrival of Steven Neely from Cal Tech, who was assigned to work with me, and work we did. This turned into a life-long seminal friendship, with many important joint publications. Soon after (cira 1982) I was setting up a lab at Columbia University in the Black Building (168th St, NYC), to record from the auditory nerve of the Cat. For this work University of Scranton physicist Paul Fahey joined in, resulting in another life-long friendship/collaboration, along with, in our view, several key publications on how the nonlinear cochlea processes sound.

But equally importantly I spent much of my time studying the "window to hearing," the middle-ear. This interest lead to a PhD relationship with Sunil Puria, also from Columbia University. Today Sunil is one of the leading, if not the number-one expert, on middle ear science. Throughout this time up to the 2020 MoH meeting (Canada), I was in a continuous and wonderful and fun conversation with Egbert, in which, it seemed by design, that we almost never agreed. These years have been carefully document due to the extensive work of Chris Shera, who set up the MoH website, with all the relevant MoH publications freely available.

About 1982-3 AT&T started the successful development of the first multi-band wide dynamic range hearing aid, which within five years, was sold to the ReSound Corporation Allen[7]. ReSound is one of the few remaining large hearing aid companies in the highly competitive industry. My role at ReSound molded my future in may ways, largely

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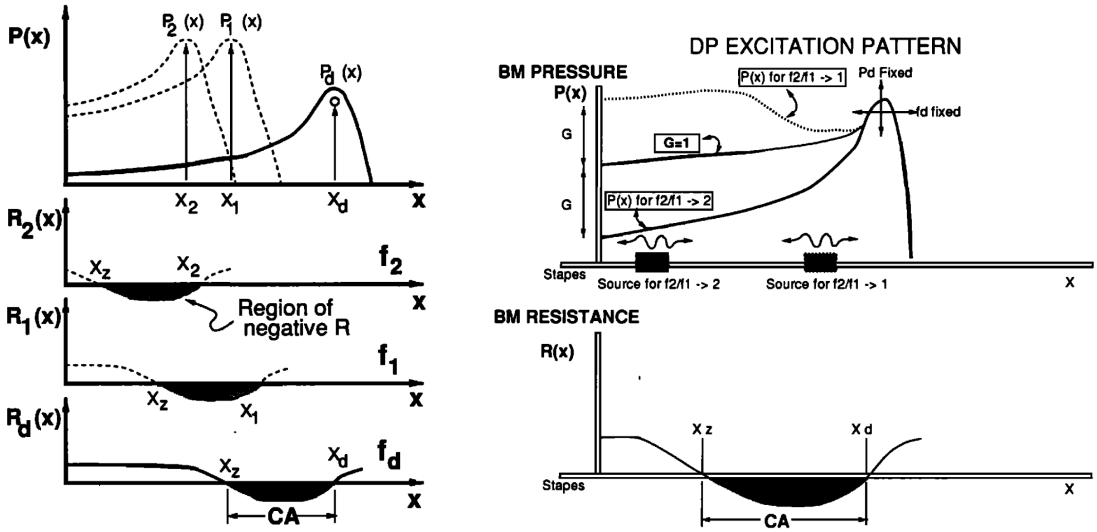

FIGURE 1. *These figures, from Allen and Fahey [10] (1992), explain the protocol for measuring the cochlear amplifier gain on the basilar membrane. In the LEFT panel two tones at frequencies $f1 < f2$ excite the cochlea at locations X_1, X_2. Such a pair of tones generate a nonlinear (NL) distortion product at frequency $f_d = 2f_1 - f_2$, which appears in the cochlea at location X_d. The primary levels of the two tones are $P_1(f_1), P_2(f_2))$ are adjusted so that the distortion product excites a neuron tuned to frequency f_d. In this way the source is on the basilar membrane rather than being in the ear canal. If we assume that there is an active region of negative resistance, corresponding to a cochlear amplifier generation site, show in black, then as the signal from the distortion product propagates back to the ear canal, where it is measured, it would be amplified by the active region. RIGHT: Since we know the DP level given the response from the auditory nerve response at the DP frequency, and we can measure the level in the ear canal, we can determine the gain of the CA as the pressure ratio.*

due to to its founder, and another very close friend, Stanford's ENT Dr. Rodney Perkins, via another equally special friend, Mead Killion.

Following the amazing experience with ReSound, I returned to Bell Labs research to continued my research on modeling the middle ear and cochlea, and quantifying auditory speech decoding features. Spoiler: my present view is not the common one. Two papers that best outline these views are AF-93 [10] and FA-85 [18].

Experiment I (AF-93)

The 1992 experiment (AF-93) was significant for several reasons. First because it rigorously defined the term *cochlear amplifier* (CA), and second because of the introduction of the experimental outcomes of an important and insightful new experiment to measure the magnitude of the CA.

The 1993 Allen-Fahey experiment: As shown in Fig. 1, the experimental paradigm was defined as follows: A near threshold pure tone signal was generated using the nonlinear (NL) outer hair-cells (OHC) coupled basilar membrane (BM) response, by placing two primary pure tones having frequencies $f_2 > f_1$ having intensities $I_2 < I_1$, such that they generated a NL distortion product at *place* $X_d(f_d)$ on the BM. Here $f_d = 2f_1 - f_2 = f_1 - (f_2 - f_1)$, and $X_d(f_d)$ defines the frequency to place relationship defined by the *second cochlear map* [11].

The intensity of the $f_d = 2f_1 - f_2$ generated tone on the BM was determined by measuring the neuron's threshold response to f_d, relative to that of a tone at f_d, presented in the ear canal.

Methods: Thus the protocol was as follows:

1. A neural frequency tuning curve (FTC), having its CF (f_{cf}) and threshold intensity I^*_{cf} was first measured for the frequency range of interest.

2. Primaries $(f_1, L_1), (f_2, L_2)$ were introduced, such that the resulting product $f_d = f_{cf}$ was at a frequency where the response is maximum, as determined by the second-cochlear map function [11].

3. Once this NL neurally-calibrated place-fixed source intensity was introduced on the BM, its ear canal pressure threshold level $P_{ec}(f_d)$ (i.e., $I^*_{ec}(f_d)$) was measured.

The source transducer design for these experiments was exceptionally linear, with an acoustic source impedance close to that of the ear canal. Thus reflections in the cochlear were measurably negligible. No nonlinear artifacts were observable in an acoustic cavity. cite 4 cav cal?

Given the Allen and Fahey [10] measurement paradigm, we compared the ear canal pressure level $P_{ec}(f_d)$ (that is, the sound intensity required to excite the nerve fiber at threshold) to the pressure propagated from the BM source $P_{ec}(f_d)$ on the BM at $X_d(f_d)$. These results are provided in figures from Allen and Fahey [11] (1993).

Results: The findings of these experiments found that the magnitude of the CA amplification was less than 6 [dB].

Conclusions: If an active (negative resistance) positive cochlear amplifier gain were responsible for the low-side suppression, it would show up as a difference between forward and reverse propagation gain. Based on the results of these experiments, *the CA gain in the cat animal model must be less than 6 dB at all measured frequencies (between 0.6-8 [kHz]).*

In the years following Allen and Fahey [11] (1993), similar experiments were repeated in several laboratories. All these experiments confirmed the observations of AF-93.

Experiment II (FA-85)

Several years earlier (1983-1985) Fahey and Allen did a related but quite different experiment, quantifying the NL suppression near the tip of the neural FTC, due to a suppressor tone in the low frequency "tail," always at least 1/4 and 1/3 octave below the best frequency (Fig 2, LEFT) of the suppressed tone Allen and Fahey [11] (1993). This NL response could be viewed as an alternate definition of the *nonlinear cochlear amplifier* (NL-CA). In this case, it represents an attenuation. The two panels of Fig. 2 are modifications of figures taken from Fahey and Allen [18] (1985), and later successfully modeled by Allen and Sen [13] (2003).

Interpretation of the OHC suppression effect: When originally published in 1993, we did not provide a physical interpretation of these suppression curves. Today, 30 years later, we have a plausible physical explanation. Figure 2 now tells this story.

The RIGHT lower panel of Fig. 2 shows the big picture. When the suppressor is below 65 dB-SPL (bold-dashed **red** line in the RIGHT lower panel), there is no low-side neural suppression effect. In these cases the threshold levels of the frequency tuning curve FTC is identical to the unsuppresed FTC. When the low-side suppressor is below 65 [dB], the FTC is identical to the unsuppressed FTC.

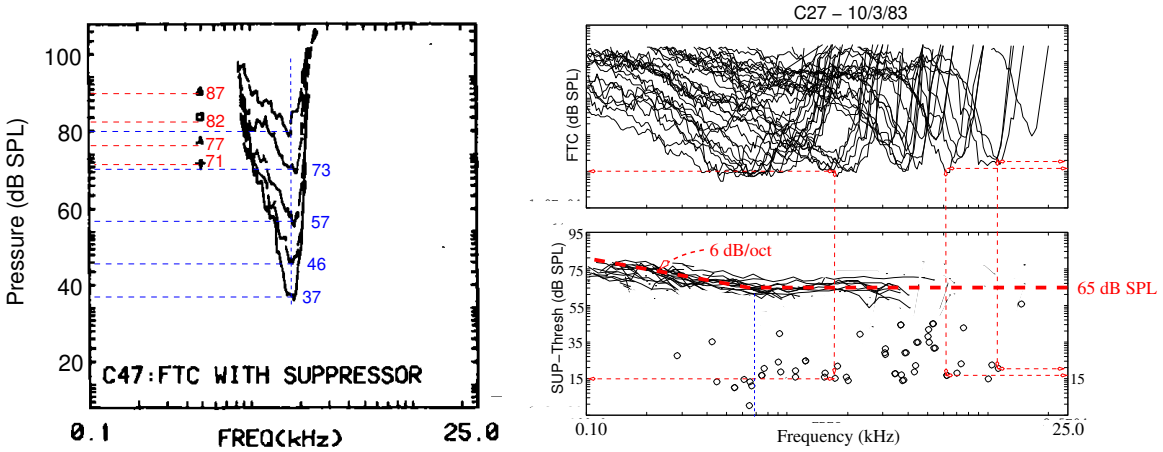

FIGURE 2. *Data from Experiment II, animals #27 and #47 from Fahey and Allen [18].*
LEFT: *Raw data from animal #47 of AF-85, Fig. 11. The neuron has a $f_{cf} = 1.8$ [kHz] and a threshold of 37 [dB-SPL]. The suppressor frequency was 0.5 [kHz] at intensities of {71, 77, 82, 87} [dB-SPL]. The suppressed threshold for these four suppressors was found to be {46, 57, 73, 80} [dB-SPL]. The suppression starts at 65 [dB-SPL], as shown in the LOWER–RIGHT panel.*
RIGHT: *This figure is a bit busy, but simple once explained. The animal was #27 from the experiment run of Oct 3, 1983. On the top panel are all the tuning curves. This animal was chosen because tuning curves were found over a wide range of frequencies, from 0.5 [kHz] to 18 [kHz]. Four examples are show out of the 44 neurons tested. These four have best frequencies of {0.7, 1.9, 6.0, 10.9} [kHz]. The locus of suppression threshold is 65 (±3) [dB-SPL]. Around 0.7 [kHz] the suppression threshold curves slowly rise, with a slope of 6 [dB/Oct]. This effect is due to the middle ear transfer function, which acts as a low-pass filter (causing the threshold to increase). See the text and Fahey and Allen [18] for an further discussion.*

Explanation of the OHC nonlinear suppression: In the LEFT panel of Fig. 2 we show an example FTC with suppressors above 65 [dB]. Note that the threshold for this neuron is elevated relative to the majority, which have 15 [dB] thresholds. As the suppressor level is increased above its threshold of 65 [dB], the threshold is dramatically elevated, with a slope much greater than 1. For example the 6 dB increase from 71 to 77 [dB] results in an 11=57-46 [dB] higher CF threshold, thus a slope of $11/6 = 1.833$.

The now obvious explanation is that above 65 dB the suppressor is exciting the outer hair cells (OHC), triggering the cochlear compressing nonlinearity, thus reducing the quiescent gain expressed the tuning curve. There is substantial evidence that the source of the cochlear nonlinearity is due to cochlear OHC cells. If the OHC are only excited for tones above 65 dB, as characterized by the red-dashed line in the RIGHT-lower panel of Fig. 2, the BM will be linear below 65 dB, Thus the suppression thresholds are defined by the OHC pure tone thresholds of 65 [dB]. The direct evidence for this clam is the locus of suppression threshold shown in the lower right panel of Fig. 2 (red dashed line).

Each of the lines in Fig. 2 corresponds to the suppress threshold for the circles below which label each tuning curve threshold. These relationships are explicitly shown as red dashed lines in the figure. The one black dash line at 0.7 [kHz] indicates where the suppression thresholds switch from constants at 65 dB, to having a 6 [dB/oct] slope, as labeled in the figure.

Note that about half of these suppression thresholds fall below the bold-red dashed line, while the others support the visual regression line. Since we no longer have the 1985 raw data used for this figure, we cannot say more about these few suppression curves which seem out of sync with the majority. Since this switch at 0.7 [kHz] seem to follow the middle ear response, this effect may have nothing to do with the OHCs, which are still NL but with a different frequency slope.

When a 71 [dB-SPL] 0.5 [kHz] suppressor excites the OHCs at the 1.8 [kHz] place, the nonlinear (NL) suppression raises the FTC threshold from 37 to 46 [dB-SPL], causing the neural tuning curve to lose 9 [dB] threshold sensitive at its best frequency. When the suppressor is then raised to 77 [dB-SPL], the CF threshold increases to 57 [dB-SPL], and for a 87 [dB] suppressor the CF threshold is 80 [dB] (not labeled) resulting in a compression slope of $(80-73)/(87-82) = 7/5 = 1.4$.

Based on the 44 examples in the LOWER–RIGHT panel (the number of small circles is 44), the threshold of the OHC nonlinear suppression is at 65 ± 5 [dB-SPL]. We did not see a similar suppression effect for suppressors above the best frequency.

In summary, the ramifications of these observations seem huge. Our neural measures presented here are highly consistent with different measurements from many other laboratories [3, 6]. After thinking about these data for more than 37 years, I have come to a number of additional conclusions, presented below, perhaps for the first time.

CONCLUSION

There are many:

1. The NL compression threshold at 65 [dB-SPL] has major implications for multi-band compression hearing aid signal-processing.

2. To explain the frequency independent 65 [dB-SPL] low-side suppression effect, there must be some sort of *second filter* effect, that transforms the BM mechanical response to the inner hair cell (IHC) response. For example, the mathematical model of a radial resonant mode in the tectorial membrane, introduced in Allen and Sen [12], is an example of such an process.

   Fix footnotes!

   Related articles discussing BM to IHC transduction include Allen [2,4], Allen and Sen [12], Sen and Allen [22]. The concept of a second-filter is quite old, but was discarded after an unscientific discussion regarding an important paper [17]. This concept was much later revisited by both Cooper [15] and Geisler and Nuttall [19], both of whom independently showed a similar elevated suppression threshold, but on the BM rather than in the neural FTC. The suppression threshold (2), as a function of frequency (not constant and described in terms of BM velocity variables) indicate some form of second filter. However they both avoid using the term "second filter," thereby obscuring the significance of their important observations.

   Their second-filter results confirm the elevated suppression threshold shown in Fig. 2, yet their observations are quiet different, because the suppression of the BM response below the CF differs, in both the frequency slope and the suppressor intensity [15, 19]. In summary, BM and neural thresholds are very different in their

properites, but are similar in terms of the magnitude of the suppression effect. Both studies openly state the need for a "second filter."

This threshold response was previously discussed in Allen and Sen [13] (2003). While a detailed summary of the suppression was presented, there was no attempt at a physical explanation of the source of the OHC suppression. The threshold of strong low-side suppression near 67 [dB] has been seen in many published data-sets as reviewed by Allen [6]. This observation may date as early as the venerable loudness model of Fletcher and Munson (1933), or even Steinberg and Gardner (1924) Allen [7].

I an hopeful, given the very strong suppression effect and the constant threshold of 65 [dB], that we can finally agree on the role of the OHC nonlinear response, given that [5, 8]:

> *There is no (zero) nonlinear effect for suppressors below 65 [dB-SPL].*

It seems likely that we may continue to argue about this interpretation for years to come, but given the many observations of low-side suppression in so many labs and under many different conditions, the observed effects are not likely to change. We may (can) continue to argue over our terminology, but not the conclusions. We are all entitled to our personal opinions, but not the collective facts.

3. The observed suppression levels seen in these examples are similar, if not identical to, many other low-side suppression experiments in the literature, measure in all possible ways Abbas and Sachs [1], Cooper [15], Delgutte [16], Geisler and Nuttall [19], Wegel and Lane [26], and possibly Ruggero et al. [20], as discussed in some detail by Allen and Sen [13] (2004) in the following quote:

   > A third important observation of both the Cooper and Geisler studies was that the displacement (of the OHC cilia) rather than the velocity must control the nonlinear response. This is simply proved by looking at how the iso-suppression response depends on the stimulus as a function of frequency. When BM displacement is used as the control, the suppression is independent of frequency. However when velocity is the control, the suppression threshold depends on frequency. One must conclude that the OHC stimulation is proportional to the BM displacement, not its velocity. When Ruggero [1992] uses BM velocity as the relevant variable in his characterization of suppression, he is viewing the response through a 6 dB/octave high-pass filter. This has important implications to the interpretation of his results, since, as we concluded above, that it takes a high-pass filter to bring the neural and BM measurements into alignment.

   As discussed by Allen [3], Fig. 8, p. 255 (1988), when the suppressor tone is presented, at any level, in the absents of excitatory tone at the CF, the neuron has no signal output (there is only spontaneous activity). Of course, this depends on the source of spontaneous activity (SA). If the source of the SA is before the OHC cilia, then it can be suppressed. If it is after the OHC cilia it cannot be suppressed by a low-side suppressors. *Thus the suppressor action is only see if the excitatory tone is present.* This as a significant, but perhaps under appreciated point.

4. If one were to define the cochlear amplifier in terms of the change in threshold sensitivity then it is seen to change from 37 (no masking) to 80 dB (87 dB masker). However the effect of such a CA has nothing to do with the low intensity signals, as expected from a CA that amplifies the signal due, say, to a negative resistance on the BM. It seems clear that we are observing what happens when the suppressor excites the OHC cilia, which causes the gain (not the amplification) to decrease. This follows from the fact that the cochlea shows no sign of a NL response when the suppressor is below 65 [dB-SPL].

   It is unreasonable to call this change in suppression, an amplifier gain. It behaves more as a compressor, much like the wide band compression built into most modern hearing aids, first introduced into the commercial market by ReSound. These commercially successful circuits were introduced to combat loudness recruitment [7].

5. An interesting parallel may be drawn between the disagreement in our field on cochlear function and the famous scientific "war" over positive feedback in the brain [21]. Scott's book is available in paperback, and online as a pdf.

   After more than 50 years (1930-1980) it was finally resolved that the brain has multi-layer positive feedback. It was a painful battle, and a important lesson for scientists, while hopefully but likely not, learned.

   On pages 294-295 Scott also has a chapter on *Scientific Reductionism*, which is a reasonable theory of science, described as a never-ending cut-and-paste sequence of experimental overlays, that mostly converges to some

fundamental answers about important questions, raised mostly by scientists. For example, reductionism is how in 1853 Maxwell composed his famous equations of electro-magnetics, and how in 1905 Einstein came to his theory of relativity.

Science breaks when the research is caught up in some fundamental disagreement. In the case of a theory of the brain [21] (p. 11-12), this was a question of feed-forward vs. feed-back in brain circuits. By rejecting the now widely accepted view of neural feedback, neuroscience was retarded for at least 50 years.

A dramatic well documented example is mentioned on page 10 of Scott[21]:

> Jerry Lettvin, a noted electro-physiologist in the Electrical and Computer Engineering Department at MIT, told me (in the spring of 1978) that when he and his colleagues reported blockages of nerve impulses in the optic nerves of cats and speculated on the possibilities of this phenomenon for visual information processing, his funding (from the National Institutes of Health) was cut off. "When you start doing science again," he was informed, "we are ready to resume your support." ...
> An encouraging feature of science, however, is that the truth does eventually become recognized.

Due to the venerable principal of *scientific Reductionism*, this last sentence does not bode well for the widely accepted "cochlear-amplifier," and rejected "second-filter" concepts.

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