My 50 years of cochlear modeling

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Abstract. The goal of this presentation is two-fold: The primary goal is to discuss my present understanding of cochlear function. A secondary goal is to review my earlier (1970-2021) cochlear modeling work, along with the roles of four close friends: Egbert De Boer, Steve Neely, Paul Fahey and George Zweig.
To understanding of how the cochlea works, one needs an understanding of the experimental data on: 1) cochlear function (both basilar (BM) and tectorial membranes (TM), 2) tympanic membrane, 3) middle ear (ME), 4) inner and outer hair cells (IHC, OHC), 5) auditory nerve (AN), and 6) cochlear amplifier (CA). My views on these topics have been greatly sharpened by looking back and unifying this complex puzzle. A great deal of progress has been made in the last 50 years.
Conclusions: My recent review of neural tuning curve data from 1985, using nonlinear (NL) distortion product generation, has revealed a deeper understanding of cochlear function. The most important, and surprising result, is that the cochlea is much more linear in its filtering properties than I previously assumed. When the suppressor frequency f_s is at least 1/2 octave lower than the characteristic ("best") frequency (f_{cf}), it is best known as "low-side" suppression. There is no "low-side" suppression for suppressors below 65 [dB-SPL]Fahey and Allen [40]. For suppressors above 65 [dB-SPL], suppression is engaged, with a slope between 1-2 [dB/dB]. Since the excitation threshold is also 65 [dB-SPL], we conclude that the neural threshold of excitation to both the inner and outer hair cells have nearly the same threshold. *That is the suppression threshold of the OHC are nearly equal to, the IHC threshold.* This raises the interesting question: If the IHC and OHC 65 [dB] thresholds are the same in the tails of the tuning curves, how can the CA function at threshold levels? Furthermore this is a highly unexpected result because low-side suppression, as measured on the basilar membrane, has a 20-30 [dB] higher threshold[31,43]. *Is the OHC action restricted to the neighborhood of the neuron's best frequency?* This would require that the neural low-side suppression and loudness recruitment (the reduced loudness of low-intensity sounds in the hearing-impaired ear) are closely related (i.e., are the same phenomena). The ramifications of this observation seem significant as they must impact our fundamental understanding of hearing and thus hearing loss[30], (p. 332, Allen90)[9].
In summary: Two-tone suppression acts like an automatic gain control, elevating the loudness threshold, with little audible distortion. We then discuss the properties of the CA, functionally measuring the CA gain. The URL for cited manuscripts:
https://auditorymodels.org/index.php?n=Main.Publications; https://www.mechanicsofhearing.org/

INTRODUCTION

The path to new friends: After obtaining my MS in Electrical Engineering from the Univ. Penn on modeling the electrical thawing of frozen dog kidney (1968), and PhD on modeling a 1 [m] $\times$ 1 [cm] 18 kC° plasma arc-jet (1970), I joined AT&T Bell Labs (Holmdel). Within two years I moved to the *Acoustics Research Department*, Murray Hill NJ.

Once there I was introduced to visiting scholar Egbert De Boer, who was widely known for his significant wide-ranging multi-disciplinary hearing-science contributions. Egbert soon informed me that I would start modeling the cochlea. I took the "hint" from my new friend, as well as my highly knowledgeable supervisor David Berkley, who had also published on the topic. I was immediately in the able hands of Mohan Sondhi, who upgraded my otherwise meager understanding of the necessary mathematics. Many papers followed [7,14,16,29,62,63].

Researching the inner ear: AT&T's Bell Labs *Acoustics Research Department* was mostly involved in automatic speech recognition (ASR), along with various acoustic applications (speaker phone and electret microphones), but historically there was always one person doing hearing research. I quickly morphed into that lucky soul. At Bell Labs, limits were only determined by one's imagination.

The next major event in my life was the arrival of Steven Neely from Washington University, St Louis, who was assigned by BTL management to work with me, and work we did. This turned into a life-long seminal friendship, with many important joint publications[25,26,50–52,55]. Within a few years (circa 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[5]. For this work University of Scranton physicist Paul Fahey joined in, resulting in another life-long friendship/collaboration, and several key publications on how the nonlinear cochlea processes sound [10,18–21,39–42].

Throughout this time, up to and including the 2017 MoH meeting, Egbert and I were continuously engaged in numerous technical conversations. We almost never agreed (but always had great playful fun, e.g., about the quality of Delft's Monk's beer. Mohan Sondhi supported Egbert's view). These years have been carefully documented due to the detailed work of Chris Shera on his MoH website, making all the MoH publications open.

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Cochlear modeling was soon followed with improved models of the middle ear[56,57,64], followed by greatly improved IHC & OHC models Allen and Fahey [18]. This ME work led to a 1990 PhD relationship with Sunil Puria, also from Columbia University. In my opinion, Sunil is one of the leading, if not the number-one expert on middle ear science.

Around 1982-83, AT&T began the development the first a multi-band wide dynamic range hearing aid, which within five years was sold by AT&T to the ReSound Corporation[12,15], resulting in the first ubiquitous commercial new technology.

ReSound remains one of the few remaining major hearing aid companies in this highly competitive industry (it's a large industry because high frequency hearing deteriorates with age). My role in the creation of ReSound molded my future in multiple ways, largely due to ReSound's founder, another close intimate friend, Stanford ENT Professor Dr. Rodney Perkins, and also via another equally very special friend, Mead Killion, founder of Etymotic Research. Such intellectual friendships can lead to large corporations. Microsoft's Gates/Allen relationship is an interesting example.

Following this formidable experience with ReSound, I returned to Bell Labs research to continued my work on modeling the the ear, to quantify auditory speech decoding features[15,16,23,48,49,54].

Six publications that best represent the joint innovative concepts of Allen and Fahey over these 50 years are:

1. Allen-80[3]: Analysis of radial eigen-modes in the TM. Gummer et al. [44] (2006) expands on Allen-80 with new experimental results.
2. Allen-83b[19]: Cat neural tuning magnitude and phase resulting from IHC & OHC cilium excitation.
3. *FA-85[40]: Analyzes why the BM-TM requires a level-dependent second-filter having an attenuation of 20-40 [dB].
4. *AF-92[20]: Proposes a method for estimating the CA gain (a.k.a. the *Allen-Fahey experiment).*
5. AF-90[22]: Defines the *2d cochlear map function*, a key to understanding cochlear transduction.
6. FA-97[42]: The analysis of neural phase and delay for various DPs $f_n = f_1 - n(f_2 - f_1)$ for $n = \pm 1, 2, 3$. Much of the data in this paper is relevant to the second-filter analysis due to radial eigen-modes in the TM, which are correlated with the DP amplitude and phase data, as described in[21]. FA-97 also contains five citations to key works of Ann Brown which provides further results supporting the conclusions of[3,22].

Two of six studies (*AF-92 & *FA-85) are discussed in detail in the following pages. While (AF-90) is not discussed, its results follow from the results of Allen-80, Allen83b, FA-85a and FA-97 and Gummer-06[44].

CA Experiment (AF-92)

The 1992 experiment[20] (AF-92) is significant for two reasons: First it rigorously defines the term *cochlear amplifier* (CA). It does this by introducing an experimental protocol to measure the magnitude of the CA's cycle by cycle power gain, thus quantifying the active CA gain.

The experimental paradigm: The procedure begins by searched for a neuron, and once found, measuring its tuning curve, to determine its BF (f_{cf}). A fixed tone at the BF with an ear canal pressure of $P_{ec}(f_{cf})$ is then presented 6 [dB] above the neuron's threshold. This threshold pressure is denoted P^*_{ec}.

As described in Fig. 1, we next moved the source at frequency f_d, from the ear canal $X_{ec} = 0$, to a variable location at $\approx X_2(f_2)$ on the BM. This DP source at $f_d = f_1 - (f_2 - f_1)$ is generated on the BM by the introduction of two additional tones at $f_1 < f_2$. These two tones created the desired DP third tone at f_d, due to the NL action of the BM. Note that while f_2 is a single frequency, its excitation pattern on the BM is spread over a region, denoted the *characteristic place* (CP) $X_2(f_d)$.

As shown earlier by Fahey and Allen [40], Fig. 7 (1985), the corresponding ear canal pressure, remains approximately the same, once the level of the two primaries P_1, P_2 are adjusted to maximally excite the neuron at its threshold pressure at location $X_d(f_1, f_2)$[66]. This has also been predicted from models of the traveling wave, as long as there are no large ear canal standing waves[6,9,24,42].

In summary: Thus in this scheme we have swapped the ear canal pressure source with a *distributed DP source* on the basilar membrane, near $\approx X_2$, having the threshold intensity P^*_d at X_d.

The distortion product signal at frequency $f_d \approx f_{cf} \ll f_1 < f_2$ is generated on the basilar membrane (BM) at $X_2(f_2) < X_1(f_1) \ll X_d(f_d)$. This DP source was then systematically moved along the BM by changing the frequencies f_2 with $f_1 = \frac{(f_d+f_2)}{2}$. For example, if $f_d = 1, f_2 = 13$ then $f_1 = 7$.

As the two primary frequencies change, their levels were adjusted to keep the ear canal pressure $P_{ec}(f_d)$ at $X_d(f_d)$ fixed at the neural tuning curve's threshold [66]. The magnitude of this adjustment depends precisely on the CA gain. Note that $P^*_{ec}(f_d)$ must be up as much as twice the gain of the CA, assuming the additive round-trip gain.

By varying the DP source location $X_2(f_d)$ along the BM (left-most dashed line), while simultaneously keeping the DP frequency f_d and pressure $P_d(f_d)$ fixed (RIGHT panel of Fig. 1), we may determine the acoustic CA gain coming from $X_2(f_2) \ll X_d(f_d)$. The DP pressure P_d propagates from X_2, where it is generated, to $X_d(f_d)$, where it is detected by the target neuron, having its $f_{cf} = f_d$.

As the source at X_2 moves through the CA region (dark shaded region), it would be amplified, causing the ear canal pressure P_{ec} to vary by more than the CA gain, as a function of $X_2(f_2)$. However the canal pressure $P^*_{ec}(f_d)$ was held constant at the BF as $X_2(f_2)$ was varied, because the two primary levels P_1, P_2 were optimally controlled to keep $P^*_{ec}(f_d)$ constant.

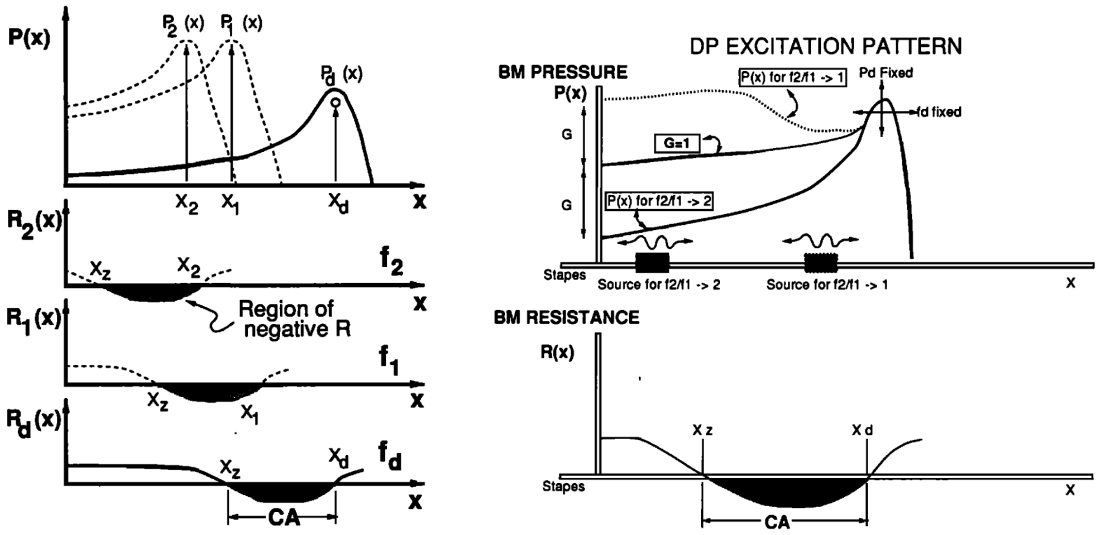

FIGURE 1. *These cartoon figures from*[20] *(1992) describe the experimental protocol for measuring the cochlear amplifier gain on the basilar membrane. In the LEFT panel two tones at frequencies $f1 < f2$ excite the cochlear OHC cilia in the overlapping region $X_1(f_1) > X_2(f_2)$. In this way the experiment excites neurons tuned to f_d at $X_d(f_d)$ from the small region between $X_1 > X_2$. The experimental goal is to record the threshold neural response at $X_d(f_1, f_2)$.*
As discussed in[4] *(1983), the tuning curve slope below f_{cf} is ≈ -50 [dB/oct]. Above the best frequency, the slope is between +100 and +500 [dB/oct]. Thus the region of the DP source must be quite narrow so that no standing waves can be generated.*
As is well known, this narrow overlapping region generates a nonlinear (NL) distortion product at frequency $f_d = f_1 - (f_2 - f_1)$ where $f_2 > f_1$. It was also shown by Ann Brown that these f_d regions appear to be tuned[22]*. This DP propagates to $X_d \gg X_1 > X_2$, where it is isolated from the two primary tones. The primary pressure levels of the two tones $P_1(f_1), P_2(f_2)$ are adjusted so that the distortion product pressure $P_d(f_d)$ at threshold maximally excites a neuron at threshold*[66].
In this way the source at $X_d(f_d)$ is generated on the basilar membrane, *basal to the neuron's best frequency, as show in the RIGHT panel.*
If we assume Rayleigh-reciprocal[17] *for the active region (negative resistance), corresponding to a cochlear amplifier generation site shown in black, then as the signal from the distortion product propagates back to the ear canal, it will again be amplified by the same negative resistance (active region). Thus the change in $P_{ec}(f_d)$ as a function of f_2/f_1 would be amplified by twice the gain of the CA (RIGHT-TOP dotted line), because it would pass through the shaded region twice, once on the way in, and again on the way out. RIGHT: Since we know the DP level at the auditory nerve, and its ear canal pressure, we can determine the gain of the CA as the ear canal pressure ratio which would be twice the gain of the CA since the BF pressure is fixed at P_d^*.*

Results: Thus, as reported by AF-92, P_{ec}^* remained constant. Thus the CA gain was reported to be less than 6 dB. As part of our experimental protocol we then verified that the ear canal pressure is approximately the same value as when it was presented from the ear canal, as we had previously demonstrated[24,40]. As shown on the LEFT panel of Fig. 1, an internally generated distortion product tone at f_d (the neurons BF f_{cf}), generated near $X_2(f_2)$ by the NL action of the outer hair cells, is assumed to modulated the BM stiffness[9], as was demonstrated by Dallos and He[32,33] with their discovery of voltage dependent IHC Prestin, which was experimentally shown to control the mechanical stiffness of the OHC[2,37,46]. If the OHC membrane voltage reduces the OHC stiffness, the BF would shift would migrate toward the base. If the TM acts as a high-pass filter, then the neural response will change due to the slope of the second filter.

As we shall discuss in the next section on FA-85, there was a large amount of nonlinear compression around the best frequency of the tuning curve. We argue that this NL compression is not dependent on the CA gain, since it is only a few [dB]. Thus we concluded that there is no significant CA cycle by cycle active gain. It is exactly this difference we wish to emphasize.

In the years following, similar experiments were repeated in several laboratories (Google "Allen-Fahey experiment."). At least two of these experiments confirmed the observations of AF-92, the most detailed being[34].

Discussion and Conclusions

The source transducer design for these experiments was exceptionally linear, with an acoustic source impedance close to that of the ear canal[6]. Thus reflections (standing waves) in the cochlear were measurably negligible. No measurable DP artifacts were observable when using this transducer in an acoustic cavity[5,6].

Given the AF-92 measurement paradigm, we compared the ear canal pressure level $P_{ec}(f_d)$ to the pressure propagated from the BM source $P_{ec}(f_d)$ on the BM at $X_d(f_d)$, where both are set to the neuron's threshold. If there were a CA, this difference would have been large. However it was shown to be less than 6 dB. Thus we concluded there is no significant cycle by cycle CA gain.

The experiment assumes that the BM is reciprocal, namely that the cycle by cycle gain is independent of the direction of travel. This assumption, known as *Rayleigh reciprocity*, holds for all existing published cochlear models. For reciprocity to be violated (non-reciprocal), it must have internal magnetic fields. The cochlea is Rayleigh-reciprocity because there are no cochlear magnetic forces[17] (Allen 2022, Postulate P6, pages 163 and 343)). The most critical is the causal postulate, P1 Nørgaard et al.[53].

Since the CA power gain is independent of direction, the gain adds in both the forward and backward propagated waves. As a result the round-trip gain must be greater than the one way-gain.

The two primary frequencies are chosen as determined by the *second cochlear map* function (CM_2) $X_d(f_d)$, defined as the frequency $f_d \approx f_{cf}$ where the DP pressure P_d^* is maximum[20,42],[21] (Fig 3). As noted above, FA-97 contains five citations to the research of Ann Brown, which provides further evidence supporting the conclusions about the second cochlear map function[3,22]. Further noted in[5], f_d is also the frequency where the *slope* of the FTC jumps from ≈ -50 [dB/oct], to ≈ 0 [dB/oct] (Allen[5], Figs. 3, 9-15), and where the neural phase jumps by 180° [9,47]. Thus the second cochlear map is the frequency-place map where TM eigen-modes play an important role.

FTC suppression (2d filter) Experiment (FA-85)

Between 1983 and 1985 a quite different but related experiment was performed[40], which quantified the neural FTC threshold pressure $P^*(f_{cf})$ as a function of a suppressor pressure $P_s(f_s)$, where the suppressor frequency $f_s \ll f_{cf}$ (Fig 2, LEFT). In these experiments we studied neurons tuned to all frequencies, which properly sampled the frequencies of speech sounds, which are between 0.1-8 [kHz][11]. Thus these results seem relevant to speech perception, especially for the hearing-impaired[15].

For low-side suppression the suppressor propagates *over* the BF of the neuron we are recording from. This is the opposite of the AF-92 experiment (Fig. 1), where the DP propagates down the BM, to a location far beyond the high-frequency basal generator site. Thus the analysis of FA-85 experiment requires a major revision of the AF-92 setup. There is no measurable DP in this experiment. If we took 1.8 [kHz] as f_2 and 0.5 [kHz] as f_1 then the DP frequency would be $2f_1 - f_2 = 2*0.5 - 1.8 = 1 - 1.8 = -.8$ [kHz].

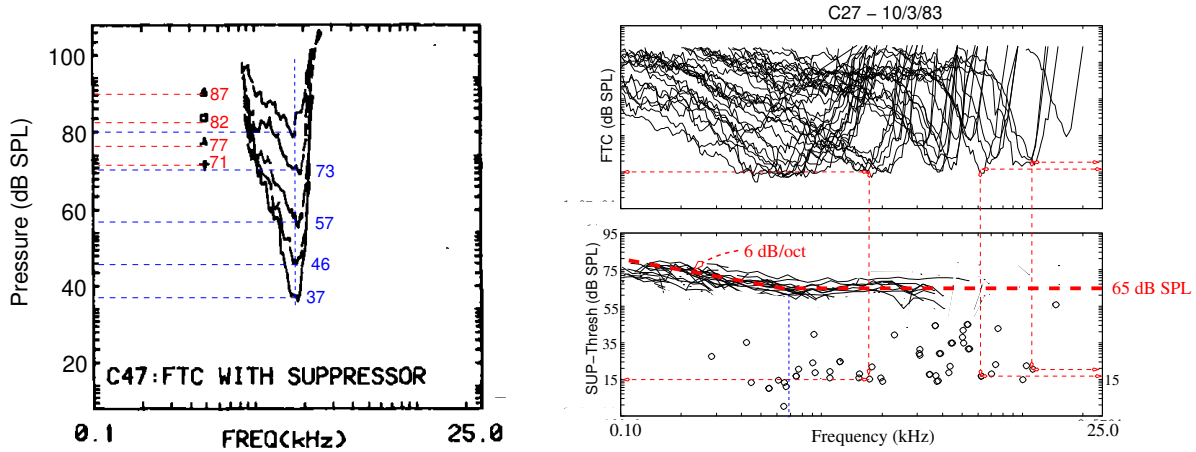

FIGURE 2. *Data from FA-85[40], animals #47 and #27. Compare this with Fig. 6 of [9].*
LEFT: *Suppressed FTC from animal #47 of FA-85, Fig. 11. This neuron has a $f_{cf} = 1.8$ [kHz] and an unmasked threshold of 37 [dB-SPL]. The suppressor frequency was 0.5 [kHz], with pressures {71, 77, 82, 87} [dB-SPL]. The suppressed threshold for these four suppressors was found to be {46, 57, 73, 80} [dB-SPL]. The suppression threshold for C27 starts at 65 [dB-SPL], as shown in the LOWER–RIGHT panel.*
RIGHT: *This figure is busy but easily explained. The tuning curves are from cat #27 on Oct 3, 1983. The top panel is a montage of all the tuning curves. This animal was chosen because tuning curves were observed over a wide range of frequencies, from 0.3 [kHz] to 14 [kHz]. Four examples are highlighted out of the >50 neurons tested. These four have best frequencies of {0.7, 1.9, 6.0, 10.9} [kHz]. the locus of suppression threshold, labeled with a red-dashed curve, hover around 65 (±3) [dB-SPL]. Below 0.7 [kHz] the suppression threshold curves slowly rise, with a slope close to -6 [dB/Oct]. This effect is due to the middle ear transfer function, which acts as a high-pass filter (causing the threshold to increase). If it weren't for the middle ear frequency response, the suppression thresholds would be independent of frequency.]. This Fig. is discussed in[13,27,28,40].*

The LEFT panel of Fig. 2 (Cat #47) provides an example FTC for the case of a suppressor above 65 [dB]. The best frequency is $f_{cf} \approx 1.8$ [kHz] and the suppressor is $f_s = 0.5$ [kHz], as taken from[28,40]. Related data are common in the literature[1,40,65].

The BF threshold for the suppressors 71, 77, 82, 87 correspond to changes in the BF threshold of $\Delta = 9, 20, 36, 43$ [dB]. Plotting the change in thresholds Δ as a function of the suppressor level, gives a least-square nearly linear regression line having a slope of ≈ 2.2 [dB/dB][36]. Below 65 [dB] the slope abruptly goes to zero, since there is no suppression effect. The NL compression effect is much smaller slightly below BF, even at the highest suppression levels, and is goes to zero above the BF, due to the very steep high frequency slopes of the BM tuning curves, of up to 500 [dB/oct][5,35].

The UPPER-RIGHT panel shows the tuning curves for ≈50 neurons, having a wide spread of BFs. In the LOWER–RIGHT panel, each of the lines clustered about the bold dashed red line of Fig. 2 labels the suppression threshold for each tuning curves as a function of frequency, correspond to one of the BF thresholds (circles, connected to the corresponding FTC by a vertical dashed line).

The horizontal bold red dashed line below 0.7 [kHz] indicates where the suppression threshold slopes switch from 0 [dB/oct] to -6 [dB/oct]. The left vertical dashed line labels the middle ear corner-frequency of 700 [Hz]. This change in slope follows the middle ear response commonly found in ear canal eardrum impedance measurements. Note that below

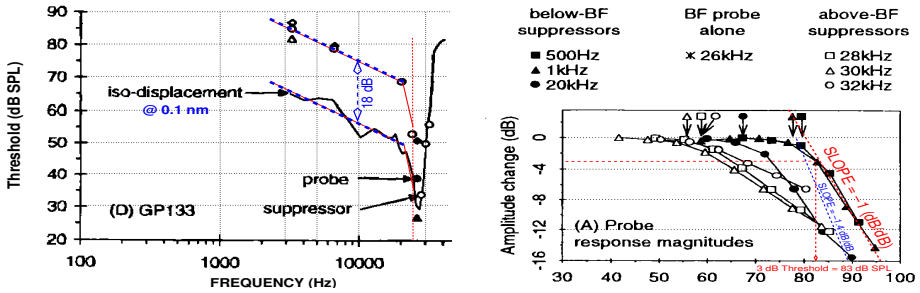

FIGURE 3. *Modified from Cooper figure showing low-side suppression on the BM*[31] *(1996).*

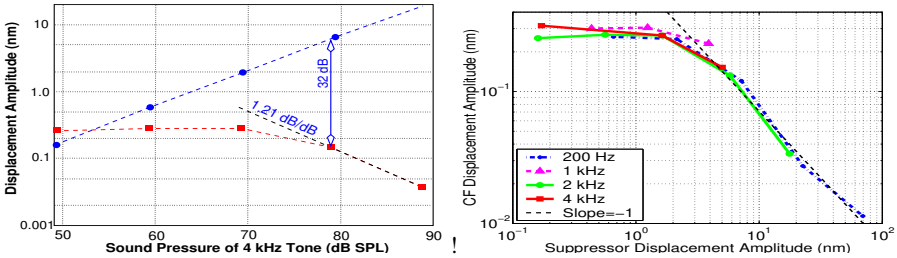

FIGURE 4. *Modified version of Geisler and Nuttal*[43] *(Fig. 3, 1997)*

400 [Hz], at least half of these suppression threshold curves fall below the -6 [dB/oct] dashed bold-red line, while others lie close to it. These two groups differ in the neural spontaneous rates.

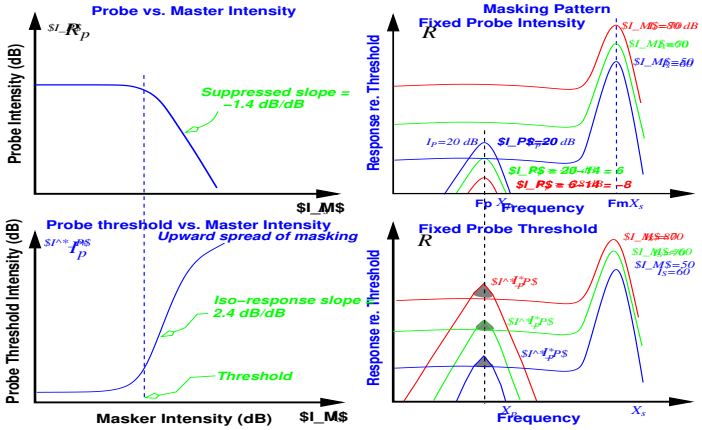

FIGURE 5. *Cartoon shown the effect of low-side suppression which explains why the FTC and suppression thresholds are similar*[14].

Interpretation of the OHC nonlinear suppression: FA-85 didn't provide a physical interpretation of these suppression curves. Today, 37 years later, we have a plausible physical explanation Allen[9], Sen and Allen[59,60,61]. We repeat and summarize this story in the following brief discussion. A well documented Allen-Sen Matlab code of the 3D-NL Cochlea is available upon request.

Physical source of the NL-CA effect: There is substantial evidence in the literature that the source of the cochlear non-linearity is due to the acoustic stimulation of cochlear OHC cells Dallos[32], Dong et al.[37], Iwasa[46]. The obvious explanation is that above 65 [dB] the suppressor is exciting the outer hair cells (OHC), triggering the cochlear non-linearity, thus reducing the quiescent sensitivity expressed by the tuning curve thresholds.

It seems highly significant that for the tails of the tuning curves having CFs greater than 2 [kHz], the IHC excitation thresholds are very similar to the OHC suppression thresholds, since both are close to 65 [dB-SPL] Allen[5], Delgutte[36]. Namely the excitation and suppression thresholds are nearly the same for the majority of neurons having their ($f_{cf} > 2$ [kHz]). The results of FA-97 (Fig. 2) are functionally identical to the low-side suppression results of Abbas and Sachs[1] as discussed in Allen[7,14].

Based on the many examples in the LOWER–RIGHT panel, the threshold of the OHC nonlinear suppression is at 65 ± 5 [dB-SPL]. We did not find a similar suppression effect for suppressors above the best frequency (there was no suppression).

In summary, the ramifications of these observations seem significant. After thinking about these data for more than 37 years, I have come to many conclusions, several for the first time.

CONCLUSIONS

1. Rather than simply citing articles, I have tried to integrate each contribution into the big-picture of cochlear physics vs. function.
2. To proved there is a "second filter" between the BM and cilia motions, FA-93 studied the frequency response of the $2f_1 - f_2$ between the BM (input) and the hair cell cilia (output).
3. The TM is the mechanical structure that sits between the BM and hair cell cilia. Thus the relevant questions is Does the TM have resonances (eigen-modes)? Today due to OCT measurements, the answer to this is clearly yes. By comparing the transfer function between the cilia and BM, we may address this question.

   What Allen and Fahey [21] found is that the TM acts like a high-pass filter, with its high-pass characteristic cut-off frequency about 1/2 octave basial to the BM's BF. Given the evidence that the OHC soma acts like an impedance (voltage dependent stiffness) Iwasa [45], a simple model explains the large 40 [dB] suppression reaponse see at the level of the synapic input to the auditory nerve.
4. The NL compression threshold at 65 [dB-SPL] has major implications for multi-band compression hearing aid signal-processing[15]. Quantifying how the suppression effect switches on at 65 [dB-SPL] is important when designing circuits that compensate for loudness recruitment.
5. The finding that the thresholds for detection and suppression are similar is an important discovery which implies that the thresholds of the IHC and OHC cilia dynamics are similar.
6. Understanding the relations between neural tuning and low-side suppression above 65 [dB-SPL] is important to quantify. I believe this relationship has been significantly quantified with the demonstration of the very similar levels of detection and suppression thresholds. That the slope for suppression is independent of frequency seems perceptually important, and is likely related to loudness recruitment in noise-damaged hearing–impaired ears.
7. The demonstration that there is little or no CA is critical to quantify. What is the definition of the CA? Is it the strong NL effect near the CF, or is it the cycle by cycle amplification?
8. To explain the frequency independent 65 [dB-SPL] neural low-side suppression effect, there must be some sort of *second filter* that transforms the BM mechanical response to the inner hair cell (IHC) response[27]. This leads to the irrefutable (unassailable) requirement for TM eigen-modes. Related articles discussing BM to IHC transduction include[3,8,14,27,61]. The concept of a second-filter was first verified by[38], and much later and independently by[31,43].

   Both showed a large elevation in the BM low-side suppression threshold relative to the iso-displacement threshold. They[31,43] conclude that the suppression threshold is function of frequency, *not* a constant 65 [dB-SPL].

   The following is a quote regarding Cooper (1996) and Geisler-Nuttal (1997)[27]:

   > Cooper's BM results placed the threshold of BM suppression about 1 order of magnitude higher in level than the Fahey and Allen 2TS thresholds, both in absolute terms, and relative to the 0.1 [nm] threshold. The Geisler and Nuttall (1997) study confirms these findings (see their Fig. 2). A second unequivocal finding of the [two] studies is that nonlinear suppression is dependent on BM displacement rather than velocity.

   Ruggero et al. (1992) agreed (page 1096) Allen and Sen [28] (2003):

   > $\cdots$ if neural rate threshold actually corresponds to a constant displacement ($\approx$2 [nm]), $\cdots$, then mechanical suppression thresholds would substantially exceed neural excitation thresholds and would stand in disagreement with findings on neural rate suppression.

   The observed suppression levels seen in these examples are similar, if not identical to many low-side suppression experiments in the literature, measured in various ways by Abbas and Sachs [1], Cooper [31], Delgutte [36], Geisler and Nuttall [43], Wegel and Lane [65], and possibly Ruggero et al. [58].
9. Our view was discussed in detail by Allen and Sen [28] (2004):

   > [An] 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 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.
10. This suppression effect is also clearly seen in the cochlear microphonic (CM) round-window voltage as shown in Allen [4], Fig. 16, which proves beyond question that the suppression is in the cochlea.

ACKNOWLEDGMENTS

I acknowledge the crucial role of my friend and dear colleague Joseph Zwislocki (1922-2018) (see AIP Oral interview).

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