The Allen-Fahey experiment extended


Published in:
The Journal of the Acoustical Society of America

DOI:
10.1121/1.1856229

Citation for published version (APA):
I. INTRODUCTION

Before the 1970s, it was assumed that a “second filter” was necessary to explain the profound difference between mechanical tuning as it had been found on the basilar membrane (BM) and neural tuning as evidenced by auditory-nerve fibers. For a review see Evans (1975). The experiments of Rhode (1971, 1978) and, later, those of Sellick et al. (1982), showed that the sharpness of tuning on the BM is well comparable to neural tuning. These basic findings were corroborated and refined by, amongst others, Robles et al. (1986) and Nuttall et al. (1990, 1991). Accordingly, it has become a problem for theoreticians to explain the sharp tuning of the BM in terms of a mechanical model of the cochlea. An ad hoc assumption was posed by Kim et al. (1980), involving the BM to be locally active. More precisely, the damping of the BM was assumed to be negative over part of the BM, extending basally from the location of largest response, over which segment the BM was assumed to be capable of amplifying the cochlear wave. That ad hoc assumption was supported by an analysis by de Boer (1983), who proved that, in the realm of “short waves,” it is impossible for a standard cochlear model to show the type of peak response that had been measured (amplitude and phase) when that model is passive. The model must have a BM impedance, of which the real part is negative basally from the point of largest response. In the same year Davis (1983) coined the term “cochlear amplifier,” thereby tacitly implying that there is a physiological mechanism that is responsible for local activity and local amplification. This term has come into common usage, although at this point it should be stressed that the concept of cochlear activity is a theoretical one: certain mechanical elements of a cochlear model should be “active” for that model to show a theoretical response resembling a measured one. Zweig (1991) showed with the “inverse solution method” for a one-dimensional model that “local activity” is necessary. We further refer to publications in which the inverse solution method for a three-dimensional model (de Boer, 1995a, b) was used to prove that local activity is absolutely necessary for constructing a three-dimensional cochlear model that simulates measured responses accurately (de Boer and Nuttall, 1999, 2000).

Another very important aspect of all experimental results is that the cochlear mechanical response is fundamentally nonlinear, it is characterized by amplitude compression, shows intercomponent suppression, and includes generation of distortion products for multicomponent stimuli. In addition, the response bandwidth is affected by nonlinearity. Tuning and nonlinearity appear to be intrinsically linked. It follows that cochlear activity and nonlinearity are intrinsically linked, too.

An ingenious physiological experiment, designed to es-

---

The Allen-Fahey experiment extended

Egbert de Boer

Room D2-226, Academic Medical Center, University of Amsterdam, Meibergdreef 9, 1105 AZ Amsterdam, The Netherlands

Alfred L. Nuttall

Oregon Hearing Research Center, NRC04, Oregon Health & Science University, 3181 SW Sam Jackson Park Road, Portland, Oregon 97239-3098 and Kresge Hearing Research Institute, University of Michigan, 1301 E. Ann Street, Ann Arbor, Michigan 48109-0506

Ning Hu, Yuan Zou, and Jiefu Zheng

Oregon Hearing Research Center, NRC04, Oregon Health & Science University, 3181 SW Sam Jackson Park Road, Portland, Oregon 97239-3098

(Received 9 August 2004; revised 15 December 2004; accepted 15 December 2004)

An ingenious experiment has been performed by Allen and Fahey [J. Acoust. Soc. Am. 92, 178–188 (1992)], in which they attempted to estimate the gain of the cochlear amplifier by comparing responses to the $2f_1-f_2$ distortion product (DP) in the outer ear canal (otoacoustic emissions) and from an auditory-nerve fiber. Results were essentially negative: no evidence of cochlear amplification was found in that experiment. A variation of that experiment is reported here, where DP responses in the outer ear canal are compared with mechanical responses of the basilar membrane. This variation does not suffer from the major limitation in the original experiment in the choice of possible frequency ratios. Results confirm and extend those of Allen and Fahey entirely. Apparently, the gain of the cochlear amplifier cannot be measured in this way. It is argued that the retrograde wave going to the stapes is most likely reduced in magnitude by wave interference when the two primary frequencies approach each other. Such a reduction does not take place in the forward-going wave to the location tuned to the DP frequency. This explanation is illustrated on the basis of results of earlier experiments on the movements of the basilar membrane. © 2005 Acoustical Society of America. [DOI: 10.1121/1.1856229]

PACS numbers: 43.64 Kc, 43.64 Bt [BLM]
timate the “gain of the cochlear amplifier,” was performed by Allen and Fahey (1992). It involved two linked measurements of responses of the functioning cochlea, i.e., recording of otoacoustic emissions (OAEs) in the external ear canal along with simultaneous measurement of the response of a primary auditory neuron. The experiments were performed in anesthetized cats. The acoustical stimulus was a pair of pure tones, with frequencies $f_1$ and $f_2$, with $f_2 > f_1$, presented by a transducer in the external ear canal. Where the mechanical responses to these tones interact in the cochlea, distortion products (DPs) are generated. One of these DPs, the one with frequency $f_{DP}$ equal to $f_{DP} = 2f_1 - f_2$, was the object of measurement. In what follows, this component will be designated by “DP.” The frequency $f_{DP}$ was chosen equal to the characteristic frequency (CF) of the auditory neuron under study so that the neuron was maximally sensitive to that frequency. The frequencies $f_1$ and $f_2$ were varied over as wide a range as possible while the DP frequency $f_{DP}$ was kept constant. The stimulus levels of the two tones were equal and chosen such that at every condition a constant DP response at the location of that neuron was obtained by the auditory neuron—presumably reflecting constant BM response at the location of that neuron. Meanwhile, the $f_{DP}$ component of the OAE in the external ear canal was measured.

In its most simplified form, the reasoning employed was as follows. When the frequencies $f_1$ and $f_2$ are varied, the effective locus of the source of the DP varies along the length of the BM. When $f_2/f_1$ is close to 1, $f_{DP}$ is nearly equal to $f_1$ and the DP source is close to the location corresponding to $f_{DP}$, i.e., close to the location of the neuron measured. When $f_2/f_1$ is appreciably larger than 1, the DP source is near the peak generated by the $f_2$ tone. From the source of the DP two waves emerge, one going basally and giving rise to an OAE (pathway #1) and the other apically, towards the location of the neuron (pathway #2). Assume now that there exists a bidirectional cochlear amplifier for the frequency $f_{DP}$. The spatial region of activity would extend basally from the location of the neuron, over a few mm. The total amplification that a wave undergoes as it follows a path through this spatially distributed amplifier is monotonically related to the length of the path. When the source of the DP is near the location of the neuron ($f_2/f_1$ close to 1), the wave of pathway #1 traverses a large amount of the path and thus undergoes maximal amplification, and that of pathway #2 none. When the source is far from the neuron ($f_2/f_1$ appreciably larger than 1) the wave of pathway #1 is not amplified but that of pathway #2 undergoes nearly maximal amplification. Since the response of the neuron for the frequency $f_{DP}$ is kept constant, the amplitude of the OAE in the ear canal should vary as the square of the amplification factor.

The experimental results were, in a sense, unexpected. When the ratio $f_2/f_1$ was varied while constant response of the neuron was monitored, almost no variation was observed in the OAE. Allen and Fahey concluded that the amplification of the cochlear amplifier is less than 10 dB, or, more simply, that “there exists no cochlear amplifier.” There is at least one limitation in parameters in the Allen–Fahey experiment. When the ratio $f_2/f_1$ is close to 1, the three frequencies are close together, and it will not be the $f_{DP}$ component but the (stronger) $f_1$ component that excites the neuron, making it impossible to monitor the DP component’s amplitude at that location. Therefore, the minimum usable value of $f_2/f_1$ is decidedly different from 1. It was reasoned that this limitation would not affect the conclusion. Because it has been proven impossible to generate a cochlear model without amplification which faithfully simulates cochlear responses as they have been measured (see above), the negative result of the Allen–Fahey experiment forms part of a mystery.

With an entirely different technique, the essential elements of the Allen–Fahey experiment were repeated by Shera and Guinan (1997). This very elegant experiment was also conducted in anesthetized cats, and measurement of OAEs was the exclusive technique employed. The basic stimulus consisted, as before, of two sinusoidal signals, with frequencies $f_1$ and $f_2$. The strength of the DP at its best location was monitored via generation of a secondary DP. This was accomplished when a third tone was added to the stimulus, with a frequency not far from the DP frequency $f_{DP} = 2f_1 - f_2$. The forward-traveling wave at the DP frequency $f_{DP}$ and the traveling wave of the third stimulus tone gave rise to a secondary DP which could be detected in the external ear canal. The results were essentially similar to those of Allen and Fahey: when the ratio $f_2/f_1$ approaches 1, there was no sign of cochlear amplification. The mystery remains.

Up to now, several attempts at explaining these—and related—results have been made. These are described further (Sec. V) where we have occasion to comment upon them. In the interest of brevity we forgo here other, secondary, contributing factors such as suppression of the DP by the primary tones. We will come back to these in Sec. IV.

In the present paper we describe an extension to Allen and Fahey’s work, a variation that does not suffer from the principal limitation of that work. Instead of monitoring the response of a neuron “tuned” to the DP frequency, it is the velocity of the basilar membrane (BM) that is directly measured and kept constant. More precisely, the component with the DP frequency $f_{DP}$ of the BM velocity is selected and kept constant. The frequency $f_{DP}$ of the third stimulus tone varies from one experiment to another, approximately $f_{DP} = 2f_1 - f_2$. The forward-traveling wave at the DP frequency $f_{DP}$ and the traveling wave of the third stimulus tone gave rise to a secondary DP which could be detected in the external ear canal. The results were essentially similar to those of Allen and Fahey: when the ratio $f_2/f_1$ approaches 1, there was no sign of cochlear amplification. The mystery remains.

II. EXPERIMENTAL PROCEDURES

We have performed our experiments on guinea pigs. Details about experimental animals and surgical techniques have been described in previous publications (Nuttall et al., 1991, 2004; de Boer and Nuttall, 1997, 2000). The protocols of the experiments described in this paper were approved by the Committee on the Use and Care of Animals, Oregon Health & Science University. Two miniature tweeter loudspeakers were connected with narrow tubes to a plastic cou-
The frequencies given the values 1.01, 1.05, 1.10, 1.15, 1.20, 1.25, and 1.28 m/s remained constant. In the first experiment the ratio of the velocity was kept at the value 8.5 m/s. mary tones the amplitude of the DP component of the BM velocity was measured. By adjusting the levels of the primary tones to the best frequency at the location where BM response in the region upward from 17 kHz, the frequency ratio of the primary tones. The same coupler housed an Etymotic 10 B+ microphone to record the otoacoustic emissions (OAEs). The coupler was not specifically designed to minimize reflection, although the two long and narrow tubes tend to absorb reflected sound.

Basilar-membrane velocity was measured at a location with a best frequency of 16 to 18 kHz. A small opening was made in the first-turn scala tympani bony wall of the cochlea. Glass beads (~20-μm diameter gold-coated) were placed onto the BM. The laser beam of a Doppler velocimeter (Polytec Corp. OFV 1102) was focused on one of the beads. By measuring compound action potentials (CAPs) we verified that in the region from 15 to 20 kHz the hearing loss due to surgery remained below 10 dB. Pure-tone stimuli were generated by computer; each of the two tones was sent to one of the loudspeakers. All OAEs were recorded with a microphone (Etymotic Research ER-10B+, Elk Grove Village, IL) from the ear canal. Two real-time FFT analyzers, Stanford Research Systems type SR770 and Hewlett-Packard type 35665A, were used to observe and monitor the signals from the velocimeter and the Etymotic microphone. We ensured that the system’s intermodulation distortion was well below the OAE level.

III. EXPERIMENTS AND RESULTS

The principal aim of the experiment was to measure the DP component of the OAE in the ear canal as a function of the frequency ratio $f_2/f_1$ whereby the amplitude of the DP component of the BM velocity at the measurement site remained constant. In the first experiment the ratio $f_2/f_1$ was given the values 1.01, 1.05, 1.10, 1.15, 1.20, 1.25, and 1.28. The frequencies $f_1$ and $f_2$ of the primary tones were varied whereby the DP frequency $f_{DP}$ was kept constant at 17 kHz, approximately the best frequency at the location where BM velocity was measured. By adjusting the levels of the primary tones the amplitude of the DP component of the BM velocity was kept at the value 8.5 μm/s; for small frequency ratios the level of both primary tones was around 50 dB SPL.

Figure 1 shows the result of this experiment. The figure shows the level of the DPOAE sound pressure in the ear canal for the series of selected values of the ratio $f_2/f_1$ of the primary tone’s frequencies. According to the simplest possible reasoning (see the Introduction), the level of the DPOAE should rise as the ratio $f_2/f_1$ approaches 1; instead, it decreases. Our experiment does not have a limitation in how near to 1 this ratio can be (within the accuracy of the FFT analyzer, of course), and we observe that the tendency found by Allen and Fahey is confirmed here.

A second experiment was carried out in a different animal. In this case two amplitude criteria were applied to the DP BM velocity component: 21.5 and 62.5 μm/s. For the higher criterion the levels of the primary tones were around 60 dB SPL for small frequency ratios. Results are shown in Fig. 2. The larger ratios could not be used because they would require excessive sound levels. It is clear that the decrease of the DPOAE with decreasing frequency ratio is present in both cases but it is more pronounced for the higher criterion. The tendencies shown in Figs. 1 and 2 were confirmed by two more experiments.

IV. SUPPRESSION

One of the phenomena that could interfere with our simplest interpretation of the procedure is suppression. Two types of suppression should be considered: mutual suppression of the two primary tones and suppression of the DP wave by (one of) the primaries. Kanis and de Boer (1997) indicated that in a very simple nonlinear model of the cochlea mutual suppression of the primaries could give an explanation of the decrease of the DP wave when the two frequencies approach one another. We decided to include mutual suppression in our experiments. Two tones were presented, at the levels used (and varied) in the experiments, and the BM response to each of them was measured with the other tone being switched on and off. This experiment would give an indication of the importance of mutual suppression. Figure 3 shows the result, the percentage of suppression, for both the low and the high criterion, as a function of frequency ratio. Except for the smallest ratio, 1.01, suppression is small to moderate. However, for the ratio 1.01 suppression of the tone with frequency $f_1$ on that with frequency $f_2$ is so large that it can at least partially explain the decrease seen in Figs. 1 and 2 in going from ratio 1.05 to 1.01.

To assess suppression of the DP wave by the primary complex is somewhat more complicated. A first-order estimate is obtained by presenting only one tone and measuring...
FIG. 3. Mutual suppression of primary components. Levels are varied for different values of the frequency ratio as in the actual experiment #2. Results are shown for both the low and the high criterion for the mechanical DP component.

The suppression of another tone at the DP frequency. Again, the levels of the tone were chosen the same as in the actual experiments. As Fig. 4 shows, suppression of a tone at the DP frequency is substantial, and mainly due to the tone with frequency $f_1$, the tone that is closest in frequency to the DP. However, somewhat unexpectedly, this suppression does not depend on frequency ratio. A substantial variation of suppression between ratios 1.01 and 1.05, for instance, is not found here (such a variation only occurs when both tones are strong, as Fig. 3 shows). In summary, we do not find a possible cause for the decline of DP acoustic emissions between ratios 1.05 and 1.01 here.

V. POSSIBLE EXPLANATIONS

The most mysterious property of the results described by Allen and Fahey (1992) is that the nearer the frequency ratio $f_2/f_1$ is to 1, the smaller is the DPOAE amplitude. In this respect we should remember that the DP measured at the level of the BM does not show evidence of a decrease between the ratios 1.1 and 1.01 (Robles et al., 1997). Obviously, there is a fundamental asymmetry in play. Going back to DPOAEs, our results confirm Allen and Fahey’s findings in a more general setting, without a limit to how close to 1 the frequency ratio can be. When we concentrate on the region between frequency ratios of 1.01 and 1.1, we may note a resemblance with the “tuning” of DPOAEs that has been observed (see, e.g., Brown et al., 1992). In those tuning experiments the DP frequency was not constant but it varied little over this ratio interval. Furthermore, the primary-tone levels were kept constant. Nevertheless, the general tendencies are the same. Several studies have attempted to explain this fundamental finding. For instance, Lukashkin and Russell (2001) described how a no-memory nonlinear device would react to patterns of primary tones with varying amplitudes. For certain amplitude and ratio conditions they found a notch in the amplitude of the DP response and reasoned that this notch and its level dependence could be at the root of the decrease of the DP when $f_2/f_1$ approaches 1. In addition, they point to mutual suppression of the primary tones as an important contributing factor, a subject we treated in the preceding section. The analysis of Lukashkin and Russell (2001) does not involve the entire region of overlap, and does not include the phase of individual wavelets. A further impediment to considering it in relation to the Allen–Fahey paradigm is that it would apply to both DP waves, of pathway #1 as well as pathway #2. A similar argument can be applied to the idea launched by Allen and Fahey (1993) about a “secondary cochlear map,” where it is assumed that DPs are undergoing a “filtering;” there seems to be no reason why the waves in the two directions would be affected differently. In summary, none of the earlier attempts at explaining the variation in the frequency-ratio region between 1.01 and 1.1 would explain why only the basally going wave is affected and not the apically going wave.

Up to now, only one comprehensive attempt at explaining the results of the Allen–Fahey experiment has been made. Shera (2003) computed DP waves generated by a twotone complex in a cochlear model according to a number of simplifying assumptions. He stressed that the source of DPs extends over a certain distance and that this region can encompass an appreciable variation of phase. In particular, when $f_2/f_1$ approaches 1, a good deal of destructive interference will occur in the wave of pathway #1, whereas no such interference occurs in the wave of pathway #2. Accordingly, the signal in pathway #1 would be reduced in amplitude considerably and cochlear amplification would not be observable at all in the external ear canal. Incidentally, the same mechanism should be responsible for the tuning of DPs. To wit: for ratios close to 1 the interference should decrease the observed OAE amplitude, whereas for ratios above 1.2 the increasing separation between the response peaks corresponding to the frequencies $f_1$ and $f_2$ diminishes the overlap between the excitation patterns and thus diminishes the strength of the DP.

Shera’s arguments can be extended and applied to realistic responses. Consider the low-level frequency response of the BM as measured in a guinea pig. With the assumption of a cochlea that scales the logarithm of frequency to location along the BM, this response can be transformed to the place domain. The so-derived response is a function of place (let us introduce the variable $x$) for a particular value of frequency. For the latter frequency we take the best frequency at the location where measurements have been made, in our case 17 kHz. It is now possible to estimate also the response
as a function of place at other frequencies. This is done for the frequencies 21.3 and 25.5 kHz, chosen such as to have a ratio of 1.2 and to correspond to a DP of frequency \( f_{DP} = 2f_1 - f_2 \), equal to 17.1 kHz. It is stressed that this only involves the transformation from frequency to location (the “cochlear map”) and not any specific model of cochlear mechanics. The resulting amplitude patterns are shown by the two solid curves in Fig. 5. The thick dashed curve shows the equivalent response of a fictitious tone with the frequency \( f_{DP} = 2f_1 - f_2 \). We now can imagine that a DP with this frequency is generated, and certain parts of the two solid curves are made thicker to indicate the \( x \) region where the overlap between the response patterns is largest, i.e., the region where the (distributed) source of the DP is assumed to be. From every point in this region two waves emanate, one going towards the stapes and one towards the apex of the cochlea. These waves correspond to the pathways #1 and #2 mentioned earlier. We can estimate the phase course of each of these waves as follows. Let \( \varphi_1(x) \) and \( \varphi_2(x) \) be the phases of the cochlear patterns of tones 1 and 2, respectively, measured at location \( x \) referred to the stapes. Similarly, let \( \varphi_{DP}(x) \) be the phase of a fictitious tone with the DP frequency, again measured at location \( x \). Then, the phase \( \varphi_{st}(x;0) \) given by

\[
\varphi_{st}(x;0) = 2\varphi_1(x) - \varphi_2(x) + \varphi_{DP}(x) - \varphi_{DP}(0),
\]

is the phase of the retrograde wave of pathway #1, originating at location \( x \) and measured at location 0 (the stapes). Note that the same phase shift \( \varphi_{DP}(x) \) that the DP wave incurs in going from location 0 (the stapes) to location \( x \) has to be added for the retrograde wave along pathway #1. In Fig. 5 the dash-dot line shows the phase of the wave of pathway #1 as a function of location \( x \). In contrast, the phase \( \varphi_{ap}(x) \) given by

\[
\varphi_{ap}(x) = 2\varphi_1(x) - \varphi_2(x) - \varphi_{DP}(x) + \varphi_{DP}(x_{DP}),
\]

of these waves as follows. Let \( \varphi_1(x) \) and \( \varphi_2(x) \) be the phases of the cochlear patterns of tones 1 and 2, respectively, measured at location \( x \) referred to the stapes. Similarly, let \( \varphi_{DP}(x) \) be the phase of a fictitious tone with the DP frequency, again measured at location \( x \). Then, the phase \( \varphi_{st}(x;0) \) given by

\[
\varphi_{st}(x;0) = 2\varphi_1(x) - \varphi_2(x) + \varphi_{DP}(x) - \varphi_{DP}(0),
\]

is the phase of the retrograde wave of pathway #1, originating at location \( x \) and measured at location 0 (the stapes). Note that the same phase shift \( \varphi_{DP}(x) \) that the DP wave incurs in going from location 0 (the stapes) to location \( x \) has to be added for the retrograde wave along pathway #1. In Fig. 5 the dash-dot line shows the phase of the wave of pathway #1 as a function of location \( x \). In contrast, the phase \( \varphi_{ap}(x) \) given by

\[
\varphi_{ap}(x) = 2\varphi_1(x) - \varphi_2(x) - \varphi_{DP}(x) + \varphi_{DP}(x_{DP}),
\]

is the phase of the retrograde wave of pathway #1, originating at location \( x \) and measured at location 0 (the stapes). Note that the same phase shift \( \varphi_{DP}(x) \) that the DP wave incurs in going from location 0 (the stapes) to location \( x \) has to be added for the retrograde wave along pathway #1. In Fig. 5 the dash-dot line shows the phase of the wave of pathway #1 as a function of location \( x \). In contrast, the phase \( \varphi_{ap}(x) \) given by

\[
\varphi_{ap}(x) = 2\varphi_1(x) - \varphi_2(x) - \varphi_{DP}(x) + \varphi_{DP}(x_{DP}),
\]
is the phase of the apically going wave of pathway #2, which originates at location \( x \) and is measured at location \( x_{\text{top}} \), the location of the peak for the frequency \( f_{\text{DP}} \). The finely dotted line in Fig. 5 shows the phase \( \varphi_{\text{eq}}(x) \) of the wave toward the apex (pathway #2), again as a function of \( x \). For each of the two functions shown the zero point is not important; what should interest us is only the variation of the phase. The wave of pathway #1 does not show large variations in the overlap region, which means that DP wavelets created there will arrive at the stapes with almost the same phase and will mainly reinforce one another. The phase variations in pathway #2 are slightly larger in the overlap region but the total variation does not exceed \( \pi \) radians (see the phase scale on the right of the figure). For the wave traveling in this direction reinforcement is slightly smaller.

In Fig. 6 we show the condition where the frequency ratio is 1.05. The phase variations of pathway #2 are minimal but those of pathway #1 are much larger than in Fig. 5. This means that in this case the DP wavelets in pathway #1 emanating from the overlap region have a pronounced tendency to cancel; as a result the OAE will be much reduced in amplitude. The other wave is hardly diminished in amplitude. The effect shown by Figs. 5 and 6 (derived from realistic responses) lies at the root of the computations carried out by Shera (2003) and substantiate his theoretical analysis but are more general. At this point it is not possible to go into any deeper detail, for instance, computing the integrated contribution of all wavelets to the total waves, because we would need more refined assumptions and a more detailed three-dimensional model of the excitation of the outer hair cells to do that. This extension falls outside the scope of the present paper.

VI. DISCUSSION

From the experiments described we can draw the conclusion that, in contrast to expectation, the DPOAE does not show evidence of cochlear amplification when the frequency ratio \( f_2/f_1 \) approaches 1. On the contrary, the DPOAE level decreases in this case. In this respect we corroborate the experimental results of Allen and Fahey (1992). Our measurement method differed in one respect from that of Allen and Fahey: we did not specifically try to minimize reflection at the stapes. We consider it unlikely that in the frequency ratio range from 1.01 to 1.1 variations of reflectivity could be so large as to explain the course of the results.

The most likely reason for the decrease of the DPOAE when \( f_2/f_1 \) approaches 1 lies in the wave-interference effect emphasized by Shera (2003), which we have illustrated in the preceding section. One important question remains. According to the simplest theory, the ratio of the DPOAE to the BM velocity at the DP frequency should vary as the square of the cochlear amplification factor (see the Introduction and the Allen–Fahey paper) when \( f_2/f_1 \) is varied over a large range. Would the interference effect be large enough to compensate that factor? In this respect we have to consider the nature of cochlear amplification. In many papers “amplification” is taken as the ratio of the response at the peak measured in the live animal to the response at the same frequency in the dead animal. This value may be up to 80 dB. In other work it is taken as the ratio of the peak responses in these two cases; values of up to 50 dB have been quoted. In the physical sense, neither value is correct because it is not the power which is compared. de Boer and Nuttall (2001) published a report in which the actual power amplification was computed in a (three-dimensional) model of the cochlea designed to simulate measured responses. Note that in this work the term “power amplification” refers to the actual amplification of the acoustical power that is flowing in the fluid of the model, from the stapes to the destination. The result was that the power amplification was maximally 17.7 dB. It is this power amplification that has to be “camouflaged” by wave interference, and it is not difficult to see that this would be possible.

ACKNOWLEDGMENTS

The authors are grateful to Christopher A. Shera for his contributions to our understanding, and to Paul F. Fahey for pointing out and discussing omissions in our manuscript. This study has been supported by NIH NIDCD DC 00141.


