

*Intense blue sky*

7 Miles

6 Miles

5 Miles

4 Miles

3 Miles

# Acoustics Today

Refraction,  
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A Rather Dangerous Balloon Adventure



# AUDITORY COMPRESSION AND HEARING LOSS

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A truly remarkable aspect of human hearing is the vast range of levels over which we are able to process sound. Those with normal hearing can hear the rustling of leaves caused by a gentle breeze as well as tolerate (at least for brief periods) the roar caused by a low-flying jet. The range of human hearing is illustrated in Fig. 1. The bottom curve represents the absolute thresholds for a wide range of frequencies; these are the levels where the various pure tones can just be detected. The top curve, on the other hand, represents the levels where the pure tones are considered uncomfortably loud. The area in between these two curves is often referred to as the dynamic range of hearing. For a mid-frequency tone (about 1–4 kHz), this range is at least 120 dB, corresponding to a truly impressive range of  $10^{12}$  in intensity units ( $\text{watts/m}^2$ ). As we will see in this article, the enormous range of hearing is accomplished via a form of amplitude compression that exists in the cochlea of the inner ear. This compression allows the extremely large range of levels in the acoustic environment (inherent, for example, in speech and music) to be “squeezed” into a much smaller and physiologically manageable range of responses.

## Cochlear compression

Sound waves in the atmosphere enter the external ear canal and impinge upon the tympanic membrane (ear drum), causing vibrations that are transmitted via three tiny bones in the middle ear to the cochlea of the inner ear. This vibration causes a displacement of the cochlear fluids which ultimately leads to a pattern of vibration along the basilar membrane of the cochlea. In particular, there is a wave of displacement that travels from the base of the cochlea to the apex. In response to a pure tone, the displacement increases

in magnitude until it reaches a peak at some place on the basilar membrane and then it decreases rather precipitously. This is known as the traveling wave. The response to a high-frequency tone peaks near the base of the cochlea whereas the response to a low-frequency tone peaks near the apex. There is thus a frequency-to-place mapping along the basilar membrane; each place tends to respond best to one frequency (sometimes called the “best frequency” or “characteristic frequency”), although it will respond to other frequencies as well. Much of what is known about the traveling wave comes from the pioneering work of Nobel Laureate Georg von Békésy<sup>1</sup>.

Lying directly above the basilar membrane are two distinct types of sensory cells, the inner hair cells (IHCs) and the outer hair cells (OHCs). Movement of the basilar membrane results in stimulation of these cells, and the greater the movement the greater the stimulation. Most auditory nerve fibers synapse directly with IHCs, and it is thought that neural activity in the auditory nerve *directly* reflects the response of these cells. If so, then what do the OHCs do? As we have learned over the last decade or so, the OHCs play a very important role in hearing. The emphasis in this article is on their role in the compressive response of the cochlea. To gain an understanding of that, let us consider how the magnitude of response at a given point along the basilar membrane changes as a function of the input stimulus level.

The movement of the basilar membrane in response to a stimulus is usually measured in terms of displacement or velocity. That metric is then plotted as a function of the stimulus level, to yield a so-called input-output (I-O) function. The solid line in the main part of Fig. 2 illustrates one such I-O function. In this case, the velocity of basilar membrane response has been converted to a response in dB (a 10-fold increase in velocity corresponds to a 20-dB increase in response). The input stimulus was a 10-kHz tone, and the measurements were taken from a place in the chinchilla cochlea that responds best to a frequency of 10 kHz<sup>2</sup>. The inset shows a cartoon of the traveling wave envelope to a 10-kHz tone at a given level. The arrow indicates the measurement site. Notice that the basilar membrane motion increases and reaches a peak at the measurement site.

As can be seen in Fig. 2, the magnitude of basilar membrane response increases with increasing stimulus level, but the growth is generally quite compressive. This is clear by comparing the I-O function with the linear function shown by the dashed line. Throughout its most compressive region (at moderate to high stimulus levels) the I-O function has a slope of about 0.2 dB/dB, corresponding to a compression ratio of about 5:1. In other words, over that range, a 50-dB increase in stimulus level (input) results in only a 10-dB increase in basilar membrane response (output). As a result of this type of compression, a given point along the basilar membrane is able to respond to an extremely large range of

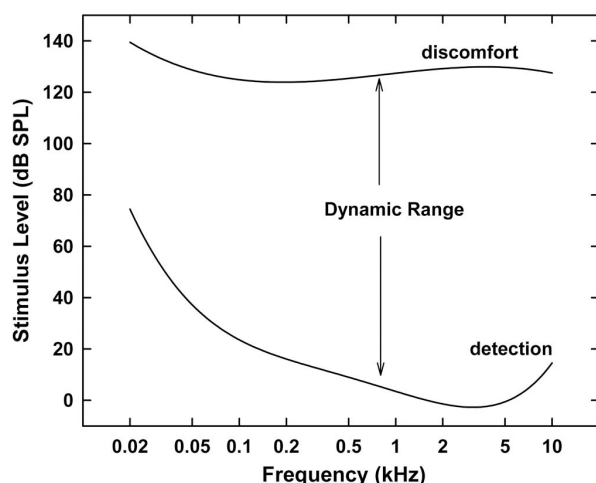


Fig. 1. The bottom curve represents the sound pressure levels where pure tones of various frequencies can be just detected. The upper curve represents the levels at which those tones become uncomfortably loud. The area in between the two curves represents the dynamic range of human hearing.

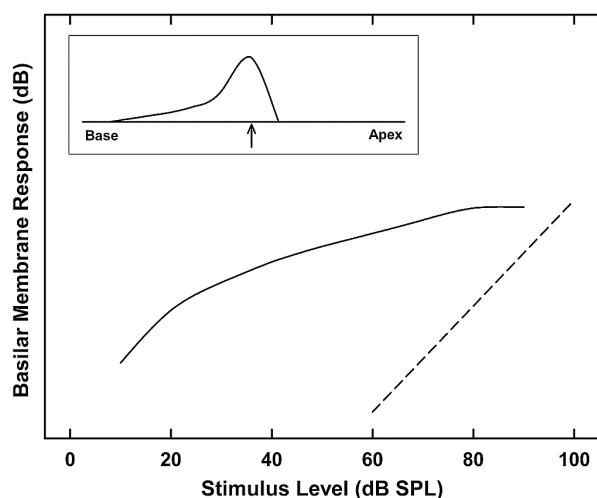


Fig. 2. The solid line in the main part of the figure represents the basilar membrane I-O function for a pure tone whose frequency (10 kHz) matched the best frequency of the recording site. The function is highly compressive. The dashed line represents linear growth (1 dB/dB). The inset is a cartoon of the traveling wave envelope in response to the 10-kHz tone at a given level. The arrow indicates the measurement site. The data are taken from Ruggero et al. (1997), with permission from the American Institute of Physics.

stimulus levels. Because basilar membrane motion serves as the proximal stimulus for IHCs, and subsequently the auditory nerve fibers, the compression that is observed at the basilar membrane greatly extends the dynamic range of the peripheral auditory system.

Although compression can be measured at the basilar membrane, it is not due to the mechanics of the membrane *per se*. In other words, the basilar membrane by itself does not function compressively. Instead, basilar membrane compression is actually the consequence of normally functioning OHCs. Temporary or permanent damage to those cells results in a basilar membrane I-O function that is less compressive, and in fact significant amounts of damage result in a linear I-O function. This is shown schematically in Fig. 3. The solid line illustrates the compressive growth of response that is observed under normal conditions. The dashed line illustrates the linear growth of response observed when the OHCs are severely damaged or functioning abnormally. Although the precise way in which the OHCs affect the motion of the basilar membrane is unclear, it is likely the result of OHC electromotility. Indeed, an interesting finding from recent research is that OHCs have motor capability resulting in their being motile and, in isolation, being capable of changing shape at rates in the audio frequency range. The motor protein (prestin) responsible for this electromotility has recently been identified<sup>3</sup>. These shape changes are thought to alter the micromechanical properties of the cochlea so as to increase the response of the basilar membrane. This, in turn, will increase the response of the IHCs and the auditory nerve fibers that synapse with those hair cells. In other words, the OHCs provide local mechanical amplification in the form of feedback. For this reason they are often referred to as the “cochlear amplifier.”<sup>4</sup> Damage to the OHCs results in a loss of that amplification, as shown in Fig. 3. The amount of gain that normally exists has been esti-

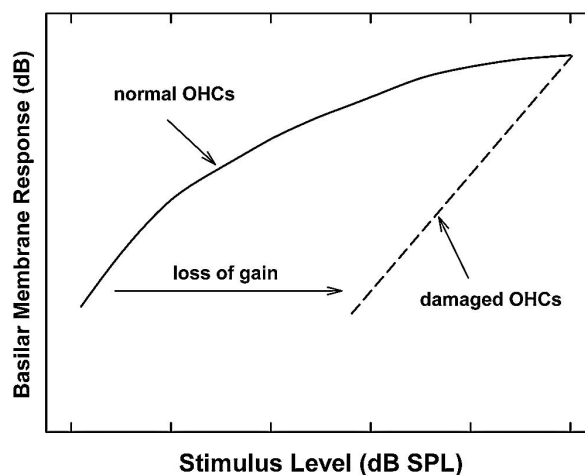


Fig. 3. This is a summary of the effects of OHC damage on the basilar membrane I-O function. The solid line shows an I-O function that might be expected from a cochlea with normally functioning OHCs. The dashed line shows a function that might be obtained from a cochlea with severely damaged OHCs. The horizontal distance between the two curves represents the amount of gain that was lost due to the hair cell damage.

mated to be as large as 50-80 dB for lower stimulus levels<sup>2</sup>, but the gain decreases with increasing level and is negligible at high levels (as evidenced by the horizontal difference between the solid and dashed lines in Fig. 3). This level-dependent gain results in a compressive growth of response under normal conditions.

An interesting and important aspect of cochlear compression is that it is frequency-selective. In other words, the amplification or gain provided by the OHCs at a given place along the basilar membrane depends upon the frequency of stimulation. As we have described thus far, the basilar mem-

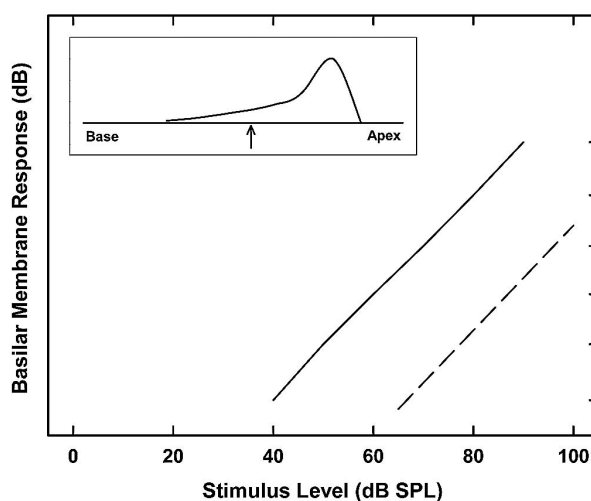


Fig. 4. The solid line in the main part of the figure represents the basilar membrane I-O function for a pure tone whose frequency (5 kHz) is one octave lower than the best frequency of the recording site (10 kHz). The function is linear, as can be seen by comparing it with the dashed line, which shows linear growth (1 dB/dB). The inset is a cartoon of the traveling wave envelope in response to the 5-kHz tone at a given level. The arrow indicates the measurement site. The data are taken from Ruggero et al. (1997), with permission from the American Institute of Physics.

brane response growth is normally highly compressive in response to a tone whose frequency is equal to the best frequency of the recording site (in Fig. 2, this was 10 kHz). However, in response to other input frequencies, the response growth *at that same site* may be less compressive or even linear. This is illustrated in Fig. 4, which shows the I-O function (solid line) at the 10-kHz place in response to a 5-kHz tone. The inset shows that the measurement site (indicated by the arrow) is basal to the peak of the traveling wave envelope. In this situation, the growth of response is linear (compare the I-O function with the linear reference shown by the dashed line). Had the measurements been taken at the 5-kHz place, where the traveling wave envelope peaks, the growth would have been compressive in response to the 5-kHz tone. Thus each place along the basilar membrane will exhibit a compressive growth of response, but only for tones with a frequency near the frequency of the measurement site.

### Some perceptual consequences of normal and reduced cochlear compression

We began this article by describing the incredibly large dynamic range of human hearing, and indicated that it was due to a form of amplitude compression in the inner ear. It should now be clear that the cochlear compression described in the previous section is largely responsible for this dynamic range. We will now explore this further and consider some of the other perceptual consequences of cochlear compression. In addition, we will highlight the consequences of reduced (or absent) compression in individuals with a hearing loss involving damage to the OHCs. Although not all hearing losses result from such damage, most sensorineural losses involve at least some damage to those sensory cells.

When the OHCs are damaged in individuals with a sensorineural hearing loss, the dynamic range of hearing is reduced, sometimes severely. This is manifest as an elevated absolute threshold (this is typically what defines a hearing loss) and usually an *unchanged* uncomfortable loudness level. (For someone with a hearing loss, the bottom curve in Fig. 1 would shift up, whereas the top curve would not shift.) In individuals with a reduced dynamic range, the loudness of a sound goes from relatively soft to uncomfortably loud over a smaller range of sound pressure levels than it does in individuals with normal hearing. This is generally referred to as loudness recruitment, a relatively common phenomenon in individuals with sensorineural hearing loss. An example of this is shown schematically in Fig. 5. The solid line represents normal loudness growth, whereas the dashed lines represent loudness growth for different amounts of hearing loss. As the loss increases, the initial portion of the loudness growth function shifts to the right (to higher levels), but all functions meet at a high sound pressure level. Thus, the dynamic range decreases as hearing loss increases. The reduced dynamic range can be understood in terms of a loss of the level-dependent gain that normally exists in the cochlea (see Fig. 3). At low stimulus levels, the OHCs provide large amounts of gain, enabling sounds such as the rustling leaves to be heard by individuals

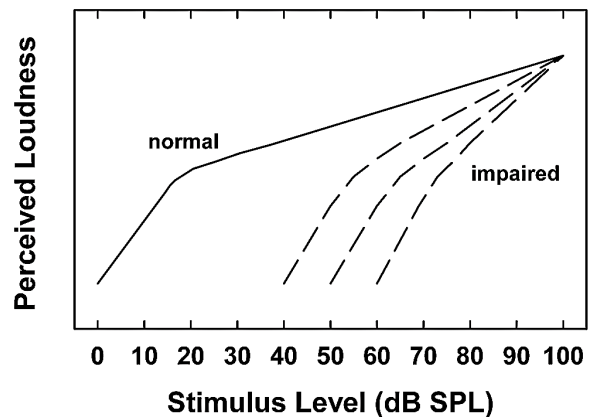


Fig. 5. The solid line shows how perceived loudness grows as a function of stimulus level in individuals with normal hearing. The dashed lines show loudness growth for individuals with different amounts of hearing loss.

with normal hearing. When the OHCs are significantly damaged, the subsequent loss of that gain will result in those sounds being inaudible. At high stimulus levels, on the other hand, the OHCs provide little or no gain anyway, and thus the response to those sounds will be largely unaffected by sensorineural hearing loss.

As discussed in the preceding section, the gain provided by the OHCs is frequency-selective. An important consequence of this is a rather exquisite tuning that can be observed in both physiological and psychophysical measures. One such measure is a so-called tuning curve. These curves can be envisioned as filter functions: they are a plot of the stimulus level needed to achieve a given response as a function of the stimulus frequency. The solid line in Fig. 6 shows a tuning curve that might be expected from an auditory system with normal OHCs. The tuning is sharp. At the tip of the curve, only a small sound pressure level is needed to elicit the criterion response. As the stimulus frequency diverges from there, however, a greater and greater level is needed to elicit that same response. An unfortunate consequence of hearing loss is a degradation of tuning. This is illustrated by the dashed line in Fig. 6, which shows what might be expected from a system with damaged OHCs. The tuning curve from the impaired system is shifted upward, but only in the frequency region around the tip, resulting in broader tuning. This can be understood in terms of a loss of the frequency-selective gain provided by the OHCs. Without the cochlear amplification for frequencies near the best frequency, the sound pressure must be increased to elicit the criterion response. A perceptual consequence of this broader tuning is a greater difficulty processing sound in the presence of competing sounds—it is just more difficult to “filter out” the unwanted sounds with broad tuning. Thus, it may be especially difficult for someone with hearing loss to understand speech in a noisy environment. Indeed, and most unfortunately, this is typically the case for individuals with hearing loss.

Our exquisite sensitivity to sound, enormous dynamic range of hearing, and fine frequency resolution are all closely

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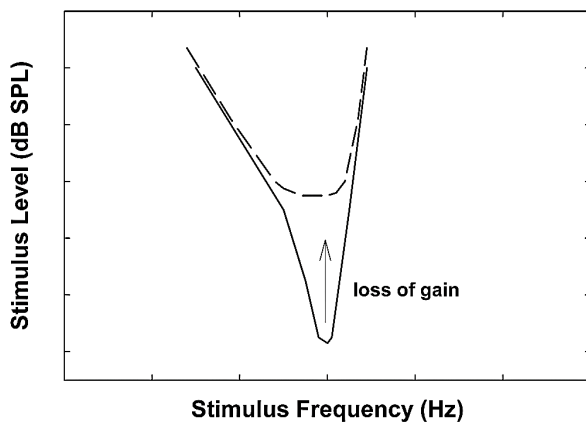


Fig. 6. These are tuning curves for an auditory system with normal OHCs (solid line) and damaged OHCs (dashed line). The upward displacement of the tip of the tuning curve in the impaired system represents a loss of the frequency-selective gain normally provided by OHCs.

associated with the level-dependent gain resulting from normally functioning OHCs in the cochlea. Damage to those cells results in elevated detection thresholds, a reduced dynamic range, and broader tuning. As discussed in several recent reviews<sup>5-8</sup>, it is likely that cochlear compression plays an even more pervasive role in hearing than outlined here. It

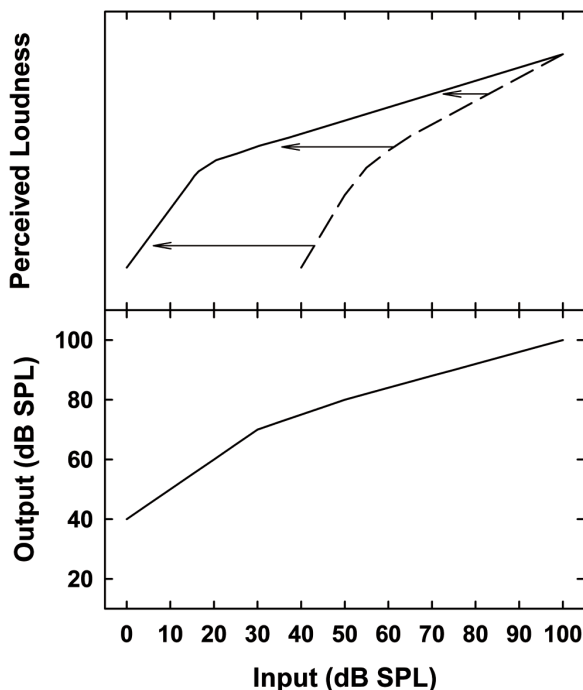


Fig. 7. The top panel shows the growth of loudness for a normal (solid line) and an impaired (dashed line) ear. The length of the arrows indicates the amount by which the sound would need to be amplified to shift the impaired ear to be equal to the normal ear. The bottom panel shows the I-O function for a hearing aid that employs compression. The amount of gain provided by the hearing aid is simply the difference between the output and the input.

may, for example, play an important role in auditory temporal processing, thereby improving our ability to process the time-varying or dynamic aspects of sound.

### Compression in hearing aids

In closing, we consider one form of aural rehabilitation for individuals with damaged OHCs, namely amplification via a hearing aid. The intent of hearing aids is to amplify sounds so that the individual with the hearing loss can hear them. The reduced dynamic range, however, provides a considerable challenge. This is illustrated in Fig. 7. In the top panel the growth of loudness is shown schematically for a normal (solid line) and an impaired (dashed line) ear. The goal of a hearing aid might be to amplify low-level sounds a great deal, but high-level sounds only a little, if at all. This would shift the response of the impaired ear to be more in line with the response of the normal ear (as indicated by the arrows). As noted previously, OHCs normally provide this type of level-dependent amplification: the amount by which they amplify the vibration of the basilar membrane decreases with increasing input level. The goal of level-dependent amplification will not be accomplished by simple linear amplification where all sounds are amplified by the same amount. Instead, compression amplification has become an increasingly more popular type of amplification for individuals with sensorineural hearing loss in order to deal successfully with their reduced dynamic range. An illustration of one type of compression amplification is shown as an I-O function in the bottom panel of Fig. 7. The output increases with increasing input, but the gain of the hearing aid (the difference between the output and the input) decreases with increasing input level. This type of amplification has accomplished the goal of providing considerable gain at low levels and increasingly smaller amounts of gain at higher levels. In a broad sense, compression hearing aids are attempting to restore the compression that is normally observed, and consequently extend the dynamic range of people with hearing loss. Although such aids may provide a more comfortable listening environment, they unfortunately do not restore hearing to normal. Indeed, a review<sup>9</sup> has shown that thus far they have had rather mixed success in terms of improving speech recognition in noisy environments. This may be at least partly related to the fact that the compression in hearing aids does not mimic the frequency selectivity that is observed in the compressive response of a normal cochlea. An important challenge, then, is to determine the best way to map the large range of acoustic levels in the environment to an auditory system that does not benefit from the compression that normally exists at the basilar membrane in the cochlea.<sup>AT</sup>

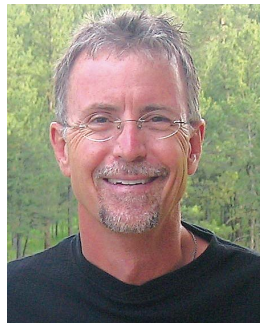
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### References for further reading:

1. G. von Békésy, *Experiments In Hearing*. (McGraw-Hill, New York, 1960).
2. M.A. Ruggero, N.C. Rich, A. Recio, S.S. Narayan, and L. Robles, "Basilar-membrane responses to tones at the base of the chinchilla cochlea," *J. Acoust. Soc. Am.* **101**, 2151–2163 (1997).
3. J. Zheng, W. Shen, D.Z. He, K.B. Long, L.D. Madison, and P. Dallos, "Prestin is the motor protein of cochlear outer hair cells," *Nature* **405**, 149–155 (2000).
4. H. Davis, "An active process in cochlear mechanics," *Hear. Res.* **9**, 79–90 (1983).
5. B.C.J. Moore and A.J. Oxenham, "Psychoacoustic consequences of compression in the peripheral auditory system" *Psychol. Rev.* **105**, 108–124 (1998).
6. A.J. Oxenham and S.P. Bacon, "Cochlear compression: Perceptual measures and implications for normal and impaired hearing," *Ear and Hearing* **24**, 352–366 (2003).
7. A.J. Oxenham and S.P. Bacon, "Psychophysical manifestations of cochlear compression: Normal-hearing listeners," in *Compression: From Cochlea to Cochlear Implants* edited by S. P. Bacon, R. Fay, and A. N. Popper, pp. 62–106. (Springer Verlag, New York 2004).
8. S.P. Bacon and A.J. Oxenham, "Psychophysical manifestations of cochlear compression: Hearing-impaired listeners," in *Compression: From Cochlea to Cochlear Implants* edited by S. P. Bacon, R. Fay, and A. N. Popper, pp. 107–152. (Springer Verlag, New York 2004).
9. H. Levitt, "Compression amplification," in *Compression: From Cochlea to Cochlear Implants* edited by S. P. Bacon, R. Fay, and A. N. Popper, pp. 153–183. (Springer Verlag, New York 2003).



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