Amplitude Compression in Hearing Aids

In the latter part of the 1980s, *wide dynamic range compression* (WDRC) amplification was introduced into the hearing aid market. Within a few years it was widely recognized as a fundamentally important new amplification strategy. Within 10 years nearly every hearing aid manufacturer had developed a WDRC product.

Compression is useful as a processing strategy because it compensates for the loss of cochlear *outer hair cells*, which compress the dynamic range of sound within the cochlea. Sensorineural hearing loss is characterized by *loudness recruitment*, which results from damage to the outer hair cells. WDRC compensates for this hair cell disorder, ideally restoring the limited dynamic range of the recruiting ear to that of the normal ear. This article reviews the history of loudness research, loudness recruitment, cochlear compression effects (such as the upward spread of masking) that result from and characterize OHC compression, and finally, outer hair cell physiology. The WDRC processing strategy is explained, and a short history of the development of WDRC hearing aids is provided.

Compression and Loudness

Acoustical signal *intensity* is defined as the flow of acoustic energy in watts per meter squared (w/m^2). *Loudness* is the perceptual intensity, measured in either *sones* or *loudness units* (LU). One sone is defined as the loudness of a 1 kHz tone at 40 dB SPL, while 1 LU is defined as the loudness at threshold. Zero loudness corresponds to zero intensity.[1]

For the case of pure tones, one sone is $\approx$975 LU. Isoloudness intensity contours were first determined in 1927 by Kingsbury (Kingsbury, 1927; Fletcher, 1929, p. 227). Such curves describe the relation between equally loud tones (or narrow bands of noise) at different frequencies. The intensity of an equally loud 1 kHz tone is called the *loudness level*, which has units of *phons*, measured in w/m^2. In 1923 Fletcher, and again in 1924 Fletcher and Steinberg, published the first key papers on the measurement of the loudness for speech signals (Fletcher, 1923a; Fletcher and Steinberg, 1924). In the 1924 paper the authors state

$$10^{-\bar{a}/30} = \int_0^\infty \mathscr{G}(f) 10^{-a(f)/30}\, df$$

... the use of the above formula involved a *summation of the cube root of the energy rather than the energy*.

where a is the relative intensity in dB SL, $\bar{a}$ is the "effective" loudness level, and $\mathscr{G}(f)$ is an empirically determined frequency weighting factor. This cube root dependence had first been described by Fletcher the year before (Fletcher, 1923a). Fletcher and Steinberg concluded that

it became apparent that the non-linear character of the ear['s] transmitting mechanism was playing an important part in determining the loudness of the complex tones (p. 307).

Power law relations between the intensity of the physical stimulus and the psychophysical response are examples of *Stevens' law*. Fletcher's 1923 loudness growth equation, which for tones was found to be $L(I) \propto I^{1/3}$, where L is the loudness and I is the acoustic intensity, established the important special case of Stevens' law for sound intensity and pure-tone loudness. Their method is described in the caption of Figure 1. We now know that Fletcher and Steinberg were observing the compression induced by the cochlear outer hair cells (OHCs).

Loudness Additivity

In 1933 Fletcher and Munson published their seminal paper on loudness. This paper detailed (1) the relation of isoloudness across frequency (loudness level, or phons); (2) their loudness growth argument, described below; (3) a model showing the relation of masking to loudness; and (4) the basic idea behind the critical band (critical ratio).

Regarding the second point, rather than thinking directly in terms of loudness growth, they tried to find a formula to describe how the loudnesses of several stimuli combine. From loudness experiments with low- and high-pass speech and complex tones, and other unpublished experiments over the previous 10 years, they showed that, across critical bands, loudness (not intensity) adds. Fletcher's working hypothesis (Fletcher and Steinberg, 1924) was that each signal is nonlinearly compressed in narrow bands (*critical bands*) by the cochlea, neurally coded, and the resulting band rates are added.[2] The 1933 experiment clearly showed how loudness (i.e., the neural rate, according to Fletcher's model) adds. Fletcher and Munson also determined the cochlear compression function $G(I)$ described below for tones and speech. We now know that this function dramatically changes with sensorineural hearing loss.

Today this model concept is called *loudness additivity*. Their hypothesis was that when two equally loud tones are presented together but separated in frequency so that they do not mask each other, the result is "twice as loud." The verification of this assumption lies in the predictive ability of the additivity assumption. For example, they showed that 10 tones that are all equally

[1] Fletcher and Munson (1933) were able to measure the loudness below the single pure-tone threshold by using 10 equally loud tones. This proves that the loudness at threshold is not zero (Buus, Musch, and Florentine, 1998).

[2] There seems to be some confusion about what is added within critical bands. Clearly, pressure must add within a critical band, or else we would not hear beats. Many books and papers assume that intensity adds within each critical band. This is true in the ensemble sense for random signals, but such a scheme will not work for tones on a single trial basis.

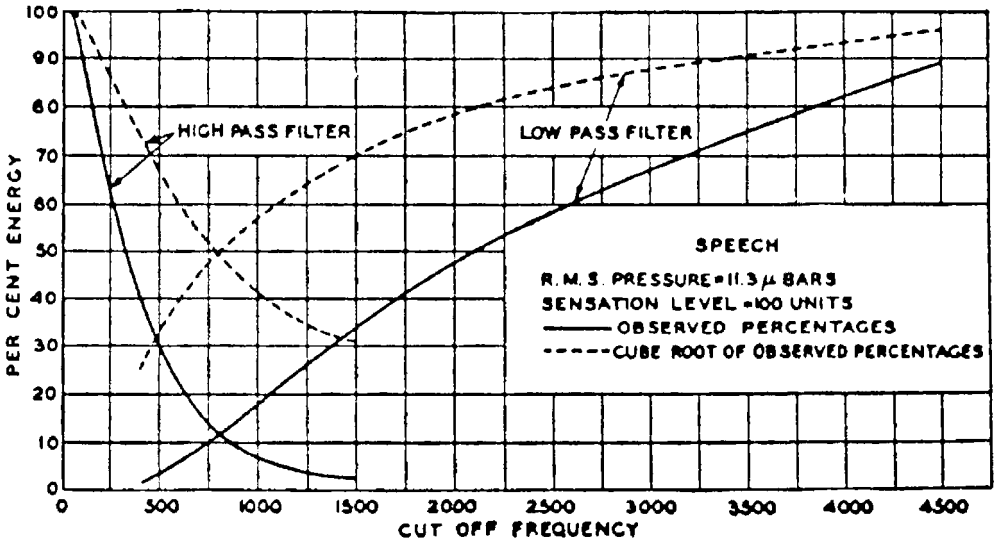

Figure 1. Effect of low- and high-pass filtering on the speech loudness level. The broadband speech is varied in level until it is equal in loudness to the low-pass-filtered speech. This is repeated for each value of the filter cutoff frequency. The experiment was then repeated for the high-pass speech. The percent reduction of the equally loud broadband speech energy is plotted against the filter cutoff frequency. For example, if broadband speech is to be equal in loudness to speech that has been low-pass-filtered to 1 kHz, it must be reduced in level to 17% of its original energy. The corresponding relative level for 1 kHz high-pass-filtered speech is 7%. These functions are shown as the solid lines in the figure. The high- and low-pass loudnesses do not add to 1 since the two solid lines cross at about 11%. After taking the cube root, however, the loudness curves cross at 50% (i.e., at 0.8 kHz, $0.125^{1/3} = 0.5$), and therefore sum to 100%. A level of 11.3 μ BARS (dynes/cm^2) corresponds to 1.13 Pa, which is close to 95 dB SPL. (From Fletcher, 1929, p. 236.)

loud (they will be at different intensities, of course), when played together, are 10 times louder, as long as they do not mask each other. As another example, Fletcher and Munson found that loudness additivity held for signals "between the two ears" as well as for signals "in the same ear." When the tones masked each other (namely, when their masking patterns overlapped), additivity still holds, but over an attenuated set of patterns (Fletcher and Munson, 1933). Their 1933 model is fundamental to our present understanding of auditory sound processing.

The Method. A relative scale factor (gain) α may be defined either in terms of the pressure or in terms of the intensity. Since it is the voltage on the earphone that is scaled, the most convenient definition of α is in terms of the pressure, P. It is typically expressed in dB, given by $20 \log_{10}(\alpha)$.

Two equally loud tones were matched in loudness by a single tone scaled by α^*. The asterisk indicates this special value of α. The resulting definition of α^* is given by

$$L(\alpha^* P) = 2L(P), \tag{1}$$

which says that, when the single tone pressure, P, is scaled by $\alpha = \alpha^*$, the loudness, $L(\alpha^* P)$, is twice as loud as the unscaled signal. Given the relative loudness level (in phons) of "twice as loud," defined by $\alpha^*(I)$, the loudness growth function $G(I)$ may be found by graphical methods or by numerical recursion, as shown in Fletcher (1953, p. 190) and in Allen (1996b). The values of $\alpha^*(I)$ found by Fletcher in different papers published between 1933 and 1953 are shown in Figure 2.

The Result. These two-tone loudness matching experiments showed that for f_1 between 0.8 and 8.0 kHz, and f_2 far enough away from f_1 (above or below) so that

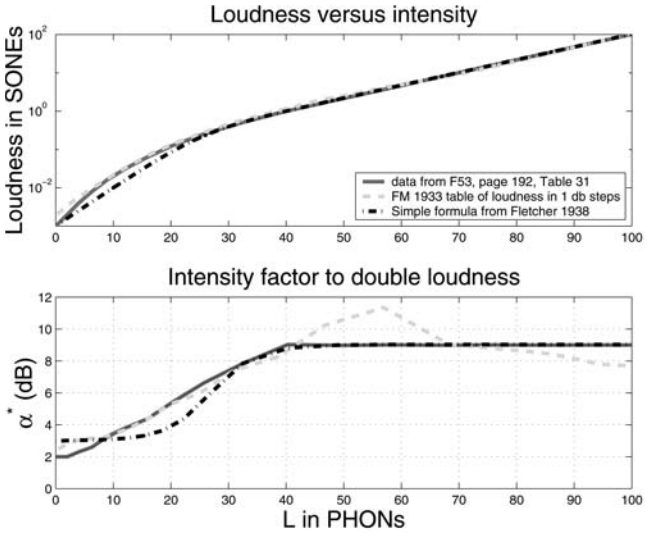

Figure 2. Loudness and α^* as a function of the loudness level, in phons. When α^* is 9 dB, loudness increases as the cube root of intensity. When α^* is 3 dB, loudness is proportional to intensity. (From Fletcher, 1953.)

there is no masking, the relative level α was found to be $\approx$9 dB (ca. 1953) for P_1 above 40 dB SPL. This value decreased linearly to 2 dB for P_1 at 0 phons, as shown in Figure 2.

From this formulation, Fletcher and Munson found that at 1 kHz, and above 40 dB SPL, the pure-tone loudness G is proportional to the cube root of the signal intensity $[G(I) = (P/P_{ref})^{2/3}]$ because $\alpha^* = 2^{3/2}$ (9 dB).[3] Below 40 dB SPL, loudness was frequently assumed to be proportional to the intensity $[G(I) = (P/P_{ref})^2$, $\alpha^* = 2^{1/2}$, or 3 dB]. Figure 2 shows the loudness growth curve and α^* given in Fletcher (1953, p. 192, Table 31) as well as the 1938 and 1933 papers. As may be seen from the figure, in 1933 they found values of α as high as 11 dB near 55 dB SL. Furthermore, the value of α^* at low levels is not 3 dB but is closer to 2 dB. Fletcher's statement that loudness is proportional to intensity (α^* is 3 dB near threshold) was an idealization that was appealing, but not supported by actual results.

Recruitment and the Rate of Loudness Growth

Once loudness had been quantified and modeled in 1933 by Fletcher and Munson, Mark Gardner, a close personal friend and colleague of Harvey Fletcher, began measuring the loudness growth of hearing-impaired subjects. In about 1934 Gardner first discovered the effect that has become known as *loudness recruitment* (Gardner, 1994), first reported by Steinberg and Gardner in 1937.

In terms of the published record, Fowler, a New York ear, nose, and throat physician, is credited with the discovery of recruitment in 1936. Fowler was in close touch with the work being done at Bell Labs and was friendly with Wegel and Fletcher (they published papers together). Fowler made loudness measurements on his many hearing-impaired patients and was the first to publish the abnormal loudness growth results. Fowler coined the term *recruitment* (Fowler, 1936).

Steinberg and Gardner (1937) were the first to correctly identify recruitment as a loss of compression. Since most sensorineural hearing loss is cochlear in origin, it follows that the loss of compression is in the cochlea. Those interested in the details are referred to the following articles (Neely and Allen, 1997; Allen, 1997a; Allen, 1999a).

Loudness Growth in the Recruiting Ear. Figure 3 shows a normal loudness growth function along with a simulated recruiting loudness growth function. It is necessary to plot these functions on a log-log (log loudness versus dB SPL) scale because of the dynamic ranges of loudness and intensity. The use of the dB and log loudness has resulted in a misinterpretation of the rate (slope) of recruitment. In the figure we see that for a 4 dB change in intensity about 58 dB SPL, the loudness changes by

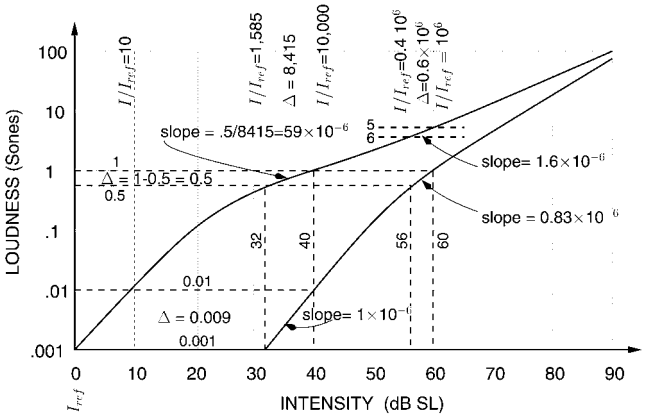

Figure 3. Shown here is a recruitment-type loss corresponding to a variable loss of gain on a log-log scale. The upper curve corresponds to the normal loudness curve; the lower curve corresponds to a simulated recruiting hearing loss. For an intensity level change from 56–60 dB, the loudness change is smaller for the recruiting ear (0.5 sones) than in the normal ear (1 sone). The belief that the loudness slope in the damaged ear is greater led to the concept that the JND in the damaged ear should be smaller (this was the rationale behind the SISI test) (see Martin, 1986, p. 160). Both conclusions are false.

1.0 sone in the normal ear and 0.5 sones in the recruiting ear. While the slope looks steeper on a log plot, the actual rate of loudness growth (in sones) in the recruiting ear is smaller. Its misdefinition as "the abnormally rapid growth of loudness" has lead to some serious conceptual errors about loudness and hearing loss. Correct statements about loudness recruitment include "the abnormal growth of loudness" or "the abnormally rapid growth of relative loudness $\Delta L/L$ (or log loudness)."

Fowler's Mistake. After learning from Wegel about the yet unpublished recruitment measurements of Steinberg and Gardner, E. P. Fowler attempted to use recruitment to diagnose middle ear disease (Fowler, 1936). In cases of hearing loss involving financial compensation, Fowler stated that recruitment was an "ameliorating" factor (Fowler, 1942). In other words, he viewed recruitment as a *recovery* from hearing loss—its presence indicated a reduced hearing loss at high intensities. Thus, given two people with equal threshold losses, the person having the least amount of recruitment was given greater financial compensation (the loss could be due to middle ear disease, and the individual would receive greater compensation than someone having a similar sensorineural loss).

In my view, it was Fowler's poor understanding of recruitment that led to such terms as complete recruitment versus partial recruitment and hyper-recruitment. *Complete recruitment* means that the recruiting ear and the normal ear perceive the same loudness at high intensities. Steinberg and Gardner described such a loss as a variable loss (i.e., sensorineural loss) and partial recruitment as a mixed loss (i.e., having a conductive component that acts as a frequency-dependent fixed

[3]Since $G(I) = (I/I_{ref})^{\beta} = (P/P_{ref})^{2\beta}$, $L(P) = (P/P_{ref})^{2\beta}$. Thus, Equation 1 gives $(\alpha^* P)^{2\beta} = 2P^{2\beta}$, or $(\alpha^*)^{2\beta} = 2$, giving $\alpha^* = 2^{1/2\beta}$. When $\beta = 1/3$, $\alpha^* = 2^{3/2}$.

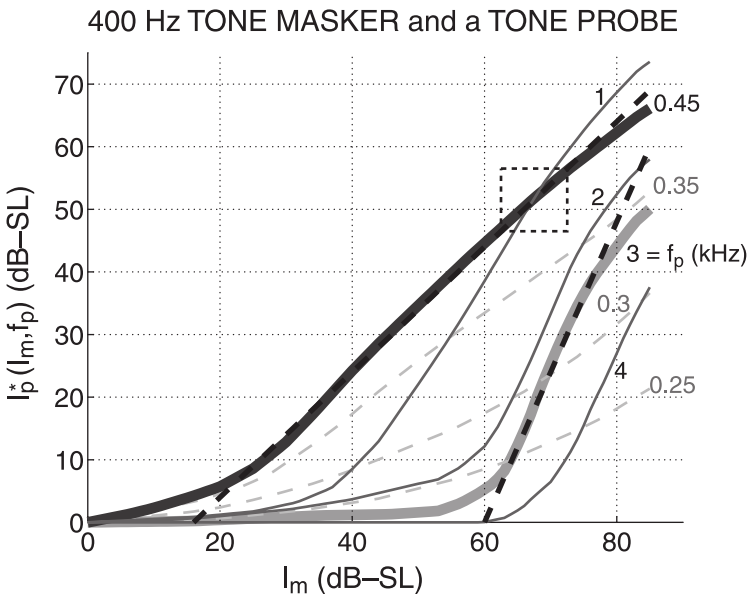

Figure 4. Masking data for a 400 Hz masker. The abscissa is the intensity of the *masker* I_m while the ordinate is the threshold intensity of the probe $I_p^*(I_m, f_p)$ (the *maskee*), each in dB SL. Each curve corresponds to a probe of a different frequency, labeled in kHz. Two dashed lines are superimposed on the heavy curves corresponding to f_p of 0.45 kHz (slope = 1.0 dB/dB) and 3 kHz (slope = 2.4 dB/dB). The curves for f_p of 1, 2, and 4 kHz are shown by light lines. Probe frequencies below 1 kHz are shown as light dashed lines. (Data from Wegel and Lane, 1924.)

attenuation). They, and Fowler, verified the conductive component by estimating the air-bone gap.

Steinberg and Gardner attempted to set the record straight. They clearly understood what they were dealing with, as is indicated in the following quote (Steinberg and Gardner, 1937, p. 20):

> Owing to the expanding action of this type of loss it would be necessary to introduce a corresponding compression in the amplifier in order to produce the same amplification at all levels.

This model of hearing and hearing loss, along with the loudness models of Fletcher and Munson (1933), are basic to the eventual quantitative understanding of cochlear signal processing and the cochlea's role in detection, masking, and loudness in normal and impaired ears. The work by Fletcher (1950) and Steinberg and Gardner (1937), and work on modeling hearing loss and recruitment by Allen (1991) support this view.

Compression and Masking

In 1922, one year after publishing the first threshold measurements with Wegel, Fletcher published measurements on the threshold of hearing in the presence of a masking tone (Fletcher, 1923a, 1923b). Wegel and Lane's classic and widely referenced paper on masking, and their theory of the cochlea, soon followed, in 1924. In Figure 4 we reproduce one of the figures from Fletcher's 1923 publication (which later appeared in the 1924 Wegel paper) showing the upward spread of masking due to a 400 Hz tone. As we shall see, these curves characterize the nonlinear compressive effects of outer hair cell compression.

Critical Band Masking. When the probe is near the masker in frequency, as in the case of the 0.45 kHz probe tone shown in Figure 4, the growth of masking is close to linear. Such near-linear growth is called *Weber's law*. The masked-threshold probe intensity[4] I_p^* is equal to the masker intensity I_m plus 1 JND ΔI, namely

$$I_p^*(I_m) = I_m + \Delta I(I_m).$$

The masking appears to be linear because the relative JND (e.g., $\Delta I/I \approx 0.1$) is small. As the intensity of the masker is increased, the variations in the JND $\Delta I(I_m)$ with respect to the masker intensity I_m appear negligible, making $I_p^*(I_m)$ appear linear. Weber's law is therefore observed when the probe is within a critical bandwidth of the masker. One sees deviations from Weber's law when plotting more sensitive measures, such as $\Delta I(I_m)/I_m$ (Riesz, 1928).

Upward Spread of Masking. The *suppression threshold*, $I_s^*(f_p, f_m)$, is defined as the smallest masker intensity such that the slope of $I_p^*(I_m, f_p)$ with respect to I_m is greater than 1. Since the probe slope is close to 2.4 dB/dB over a range of intensities, this threshold is best estimated from the intercept of the $I_p^*(I_m, f_p)$ regression line with the abscissa. For the 3 kHz probe, the suppression threshold intensity is 60 dB SL. Such suppression is only seen for probes greater than the masker frequency ($f_p > f_m$). For probes that are sufficiently higher in frequency than the masker (e.g., $f_p \geq 2$ kHz in Fig. 4), the masking is close to zero dB SL until the masker intensity reaches the suppression threshold at about 50–60 dB SL. In other words, the *masked threshold*, defined as the intensity where the masking of the probe begins, and the *suppression threshold* are nearly the same. The suppression threshold for the dashed-line, superimposed on the "solid-fat" $f_p = 3$ kHz probe curve in Figure 4, is 60 dB SL; its slope is 2.4 dB/dB. For every 1 dB increase in

[4]An asterisk is used to indicate that the intensity is at threshold.

the masker intensity I_m, the probe threshold intensity $I_p^*(I_m, f_m, f_p)$ must be increased by 2.4 dB to return it to its detection threshold (Delgutte, 1990; Allen, 1997c). Namely, above $I_s^*(f_p = 3\text{ kHz},\ f_m = 0.4\text{ kHz}) = 60$ dB SL (i.e., $I_m > 10^6 I_m^*$),

$$\frac{I_p^*(I_m)}{I_m^*} = \left(\frac{I_m}{I_s^*}\right)^{2.4}. \tag{2}$$

From Figure 4, a surprising and interesting crossover occurs near 65–70 dB for the 1 kHz probe. As highlighted by the dashed box, the 1 kHz probe threshold curve crosses the 0.45 kHz probe threshold curve. At high levels, there is more masking at 1 kHz than at the probe frequency. This means that the masker excitation pattern peak has shifted toward the base of the cochlea (i.e., toward the stapes). Follow-up forward masking studies have confirmed this observation (Munson and Gardner, 1950, Fig. 8). McFadden (1986) presents an excellent and detailed discussion of this interesting "half-octave shift" effect that is recommended reading for all serious students of hearing loss.

Downward Spread of Masking. For probes lower than the masker frequency (Fig. 4, 0.25 kHz), while the threshold is low, the masking is weak, since it has a slope that is less than linear. This may be explained by the migration of the more intense high-frequency (basal) masker excitation pattern away from the weaker probe excitation pattern (Allen, 1999b).

The Physiology of Compression

What is the source of Fletcher's tonal cube root loudness growth (i.e., Stevens' Law)? Today we know that the basilar membrane motion is nonlinear in intensity, as first described by Rhode in 1971, and that cochlear OHCs are the source of the basilar membrane nonlinearity. The history of this insight is both interesting and important.

In 1937, Lorente de Nó theorized that abnormal loudness growth associated with hearing loss (i.e., recruitment) is due to hair cell damage (Lorente de Nó, 1937). From noise trauma experiments on humans one may conclude that recruitment occurs in the cochlea (Carver, 1978). Animal experiments have confirmed this prediction and have emphasized the importance of OHC loss (Liberman and Kiang, 1978; Liberman and Dodds, 1984). This loss of OHCs causes a loss of the basilar membrane compression (Pickles, 1982, p. 287). It follows that the cube root tonal loudness growth starts with the nonlinear compression of basilar membrane motion due to stimulus-dependent voltage changes within the OHC.

Two-Tone Suppression. The neural correlate of the 2.4 dB/dB psychoacoustic suppression effect (the upward spread of masking) is called *two-tone suppression* (2TS) (Sachs and Abbas, 1974; Fahey and Allen, 1985; Delgutte, 1990). Intense low-frequency tones attenuate low-level high-frequency tones to levels well below their threshold. The close relationship between the two effects has only recently been appreciated (Allen, 1997c, 1999b). The 2TS and upward spread of masking (USM) effects are important to the hearing aid industry because they quantify the normal cochlear compression that results from OHC processing. To fully appreciate the USM and 2TS, we need to describe the role of the OHC in nonlinear cochlear processing. In Figure 5 the operation of USM/2TS is summarized in terms of neural excitation patterns.

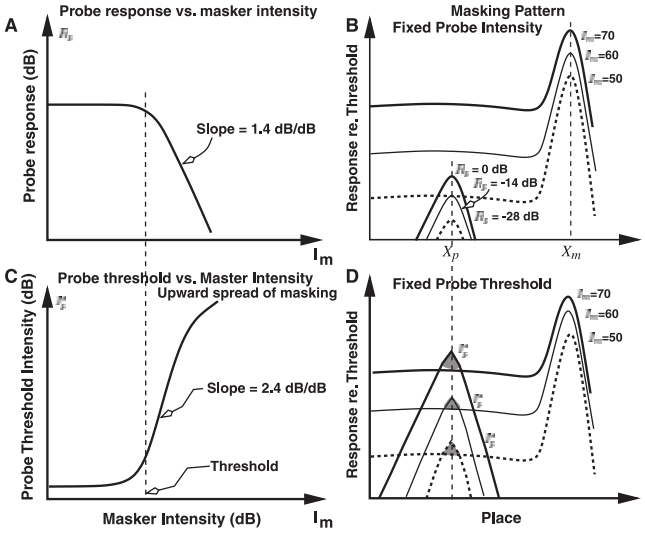

Figure 5. This sketch shows a conceptual view of the effect of a low-frequency suppressive masker on a high-frequency near-threshold probe, as a function of place. The abcissa for A and C is suppressor (masker) intensity in dB, while B and D are a cochlear place axis, where the base (stapes end) is at the origin. Panels **A** and **B** show the IHC cilia response R of a high-frequency, low-level probe, of fixed 20 dB SL intensity, being suppressed by a high-intensity, low-frequency ($f_m \ll f_p$) variable intensity suppressive masker having intensities I_s of 50, 60, and 70 dB SL. Panels A and B correspond to the isoprobe level of 20 dB SL. Even though the probe input intensity is fixed at 20 dB SL, the cilia response to the probe R_p is strongly suppressed by the masker above the suppression threshold, indicated by the vertical dashed line in A and C. The lower panels **C** and **D** show what happens when the high-frequency probe intensity is returned to threshold, indicated by $I_p^*(I_m)$. To restore the probe to threshold requires an increase of 1 dB/dB of suppressor level, due to the linear suppressor growth in the high-frequency tail region of the probe, at X_p.

Cochlear Nonlinearity: How?

We still do not know precisely what controls the basilar membrane nonlinearity, although we know that it results from OHC stiffness and length changes (He and Dallos, 2000), which are a function of the OHC membrane voltage (Santos-Sacchi and Dilger, 1987). This voltage is determined by shearing displacement of the hair cell cilia by the tectorial membrane (TM). The most likely cause of nonlinear basilar membrane mechanics is changes in the micromechanical impedances within the organ of

Corti. This conclusion follows from ear canal impedance measurements, expressed in terms of nonlinear power reflectance, defined as the retrograde to incident power ratio (Allen et al., 1995). In a transmission line, the reflectance of energy is determined by the ratio of the load impedance at a given point divided by the local characteristic impedance of the line. It is this ratio that is level dependent (i.e., nonlinear).

Two Models. It is still not clear how the cochlear gain is reduced, and that is the subject of intense research. There are two basic but speculative theories. The first is a popular but qualitative theory, referred to as the *cochlear amplifier* (Kim et al., 1980). The second is a more physical and quantitative theory that requires two basic assumptions. The first assumption is that the tectorial membrane acts as a bandpass filter on the basilar membrane signal (Allen, 1980). The second assumption is that the OHCs dynamically "tune" the basilar membrane (i.e., the cochlear partition) by changing its net stiffness, causing a dynamic migration in the characteristic place with intensity (Allen, 1997b). Migration is known to occur (McFadden, 1986), so this assumption is founded on experimental dogma.

We cannot yet decide which, if either, of these two theories is correct, but for the present discussion, it is not important. The gain of the inner hair cell (IHC) cilia excitation function is signal dependent, compressing the 120 dB dynamic range of the acoustic stimulus to less than 60 dB. When the OHC voltage becomes depolarized, the OHC compliance increases, and the characteristic frequency (CF) of the basilar membrane shifts toward the base, reducing the nonlinear wide dynamic range compression.

Cochlear Nonlinearity: Why?

The discussion above leaves unanswered *why* the OHCs compress the signal on the basilar membrane. The answer to this question has to do with the large dynamic range of the ear. In 1922 Fletcher and Wegel were the first to use electronic instruments to measure the threshold and upper limit of human hearing (Fletcher and Wegel, 1922a, 1922b), thereby establishing the 120 dB dynamic range of the cochlea.

The IHCs are the cells that process the sound before it is passed to the auditory nerve. Based on the Johnson (thermal) noise within the IHC, it is possible to accurately estimate a lower bound on the RMS voltage within the IHC. From the voltage drop across the cilia, we may estimate the upper dynamic range of the cell. The total dynamic range of the IHC must be less than this ratio, or less than 65 dB (e.g., 55–60 dB) (Allen, 1997b). The dynamic range of hearing is about 120 dB. Thus, the IHC does not have a large enough dynamic range to code the dynamic range of the input signal. Spread-of-excitation models and neuron threshold distribution of neural rate do not address this fundamental problem. Nature's solution to this problem is the OHC-controlled basilar membrane compression.

The formula for the Johnson RMS thermal electrical membrane noise voltage $|V_c|$ due to cell membrane leakage currents is given by[5] $\langle |V_c|^2 \rangle = 4kTBR$, where B is the cell membrane electrical bandwidth, k is Boltzmann's constant, T is the temperature in degrees Kelvin, and R is the cell membrane leakage resistance. The cell bandwidth is limited by the membrane capacitance C. The relation between the cell RC time constant, $\tau = RC$, and the cell bandwidth is given by $B = 1/\tau$, leading to

$$|V_c| = \sqrt{\frac{4kT}{C}}. \tag{3}$$

The cell membrane capacitance C has been determined to be about 9.6 pF for the IHC (Kros and Crawford, 1990) and 20 pF for the OHC. From Equation 3, $V_c = 21$ μV for IHCs at body temperature ($T = 310\,^{\circ}$K).

Although the maximum DC voltage across the cilia is 120 mV, the maximum RMS change in cell voltage that has been observed is about 30 mV (I. J. Russell, personal communication). The ratio of 30 mV to the noise floor voltage (21 μV), expressed in dB, is 63 dB. Thus it is impossible for the IHC to code the 120 dB dynamic range of the acoustic signal. Because it is experimentally observed that, taken as a group, IHCs *do* code a wide dynamic range, the nonlinear motion of the basilar membrane must be providing compression within the mechanics of the cochlea prior to IHC detection (Allen and Neely, 1992; Allen, 1997a).

Summary. Based on a host of data, the physical source of cochlear hearing loss and recruitment is now clear. The dynamic range of IHCs is limited to about 50 dB. The dynamic range of the sound level at the eardrum, however, is closer to 100–120 dB. Thus, there is a difficulty in matching the dynamic range at the drum to that of the IHC. This is the job of the OHCs.

It is known that OHCs act as nonlinear elements. For example, the OHC soma axial stiffness, K_{ohc}, depends directly on the voltage drop across the cell membrane, V_{ohc}. As the OHC cilia excitation is varied from "soft" to "loud," the OHC membrane voltage is depolarized, causing the cell to increase its compliance (and length). The result is compression due to a decrease in the IHC (cochlear) signal gain.

Multiband Compression

During the two decades from 1965 to 1985, the clinical audiological community was attempting to answer the question: Are compression hearing aids better than a well-fitted linear hearing aid? A number of researchers concluded that linear fitting is always superior to compression. When properly adjusted, linear filtering *is* close to optimum for speech whose level has been adjusted for

[5] While the thermal noise is typically dominated by the shot noise, the shot noise is more difficult to estimate. Since we are trying to bound the dynamic range, the thermal noise is a better choice for this purpose. The shot noise reduces the dynamic range further, strengthening the argument.

optimum listening. Papers that fall in this category include Braida et al. (1979) and Lippmann et al. (1981). However, Lippmann et al. are careful to point out the flaw in preadjusting the level (see p. 553).

Further criticisms were made by Plomp (1988, 1994), who argued that compression would reduce the modulation depth of the speech. However, compression of a broadband signal does not reduce the modulations in sub-bands.

All these results placed the advocates of compression in a defensive minority position. Villchur vigorously responded to the challenge of Plomp, saying that Plomp's argument was flawed (Villchur, 1989). The filter bandwidths used in WDRC hearing aids are not narrow enough to reduce the modulations in critical bandwidths. Other important papers arguing for compression include Hickson (1994), Killion (1996a, 1996b), Killion et al. (1990), and Mueller and Killion (1996). A physiology paper that is frequently cited in the compression literature is Ruggero and Rich (1991).

Other work that found negative results used compression parameters that were not reasonable and time constants that were too slow. Long time constants with compression produce very different results and are not in the category of syllabic compression. Such systems typically have artifacts, such as noise "pumping," or they simply do not react quickly enough to follow a lively conversation. Imagine, for example, a listening situation with a quiet and a loud talker having a conversation. In this situation, the compressor gain must operate at syllabic rates to be effective. The use of multiple bands ensures that a signal in one frequency band does not control the gain in another band. Slow-acting compression (AGC) may be fine for watching television, but not for conversational speech. Such systems might be viewed as a replacement for a volume control (Dillon, 1996, 2001; Moore et al., 1985; Moore, 1987).

A key advocate of compression was Ed Villchur, who critically recognized the importance of Steinberg and Gardner's observations on recruitment as a loss of compression. He vigorously promoted the idea of compression amplification hearing aids. Personally supporting the cost of the research with dollars from his very successful loudspeaker business, he contracted David Blackmer of dbx to produce a multiband compression hearing aid for experimental purposes. Using his experimental multiband compression hearing aid, Villchur experimented on hearing-impaired individuals, and found that Steinberg and Gardner's observations and predictions were correct (Villchur, 1973, 1974). Villchur clearly articulated the point that a well-fitted compression hearing aid improved the dynamic range of audibility and that what counted, in the end, was audibility. In other words, "If you can't hear it, you can't understand it." This had a certain logical appeal.

Fred Waldhauer, a Bell Labs analog circuit designer of some considerable ability, heard Villchur speak about his experiments on multiband compression. After the breakup of the Bell System in 1983, Waldhauer proposed to AT&T management that Bell Labs design and build a multiband compression hearing aid as an internally funded venture. Eventually Bell Labs built a digital wearable hearing aid prototype. It quickly became apparent that the best processing strategy compromise was a two-band compression design that was generically similar to the Villchur scheme. With my colleague Vincent Pluvinage, we designed digital hardware wearable hearing aids, and with the help of Joe Hall and David Berkley of AT&T, and Patricia Jeng, Harry Levitt, Arlene Newman, and many others from City University of New York, we developed a fitting procedure and ran several field trials (Allen et al., 1990). AT&T licensed its hearing aid technology to Re*S*ound on February 27, 1987.

Unlike today, in 1990 multiband compression was widely unaccepted, both clinically and academically (Dillon, 2001). Why is this? It was, and remains, difficult to show quantitatively the nature of the improvement of WDRC. It is probably fair to say that only with the success of Re*S*ound's WDRC hearing aid in the marketplace has the clinical community come to accept Villchur's claims.

It may be possible to clarify the acceptance issue by presenting two common views of what WDRC is and why it works. One's adopted view strongly influences how he or she thinks about compression. They are the articulation index (AI) view and the loudness view.

The articulation index view is based on the observation that speech has a dynamic range of about 30 dB in one-third octave frequency bands (French and Steinberg, 1947). The assumption is that the AI will increase in a recruiting ear as the compression is increased, if the speech is held at a fixed loudness. This view has led to unending comparisons between the optimum linear hearing aid and the optimum compression hearing aid.

The loudness view is based on restoring the natural dynamic range of all sounds to provide the impaired listener with all the speech cues in a more natural way. Soft sounds for normals should be soft for the impaired ear, and loud sounds should be loud. According to this view, loudness is used as an *index of audibility*, and complex arguments about JNDs, speech discrimination, and modulation transfer functions just confound the issue. This view is supported by the theory that OHCs compress the IHC signals.

Neither of these arguments deals with important and complex issues such as changing of the critical band with hearing loss, or the temporal dynamics of the compression system. Analysis of these important details is interesting only *after* the signals are placed in the audible range.

Summary

This article has reviewed the early research on loudness, loudness recruitment, and masking, which are relevant to compression hearing aid development. The outer hair cell is damaged in sensorineural hearing loss, and this causes the cochlea to have reduced dynamic range.

When properly designed and fitted, WDRC has proved to be *the* most effective speech processing

strategy we can presently provide for sensorineural hearing loss compensation. It works because it supplements the OHC compressors, which are damaged with sensorineural hearing loss.

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—*Jont B. Allen*

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Assessment of and Intervention with Children Who Are Deaf or Hard of Hearing

The purpose of communication assessment of children with educationally significant hearing loss differs from the purpose of assessing children with language or learning disabilities. Since the diagnosis of a hearing disability has already been made, the primary goal of communication assessment is to determine the impact of the hearing loss on language, speech, auditory skills, or cognitive, social-emotional, educational and vocational development, not to diagnose a disability. It is critical to determine the rate of language and communication development and to identify strategies that will be most beneficial for optimal development.

Plateaus in language development at the 9–10-year age level, in reading development at the middle third grade to fourth grade level (Holt, 1993), and in speech intelligibility at about 10 years (Jensema, Karchmer, and Trybus, 1978) have been reported in the literature. The language plateaus appear to be the result of developmental growth, which ranges from 43%–53% for children with profound hearing loss using hearing aids (Boothroyd, Geers, and Moog, 1991; Geers and Moog, 1988) to 60%–65% of the normal range of development for children with severe loss using hearing aids (Boothroyd, Geers, and Moog, 1991) and for children with profound hearing loss using cochlear implants (Blamey et al., 2001). In contrast, in a study of 150 children, Yoshinaga-Itano et al. (1998) reported that children with hearing loss only who were early-identified (within the first 6 months of life) had mean language levels at 90% of the rate of normal language development through the first 3 years of life. A study of children in Nebraska (Moeller, 2000) reported similar levels of language development (low-average range) for a sample of 5-year-old children receiving early intervention services in the first 11 months of life. Later-identified children were able to achieve language development commensurate with the early-identified/intervened group when their families were rated as "high parent involvement" in the intervention services.

With the advent of universal newborn hearing screening, the population of children who are deaf or hard of hearing will change rapidly during the next decade. By 2001, 35 states had passed legislation to