Amplitude Compression in Hearing Aids

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ABSTRACT

In the later part of the 1980's *wide dynamic range compression* (WDRC) amplification was introduced into the hearing aid market. Within a few years it was widely recognized as being a fundamentally important new amplification strategy. Within ten years nearly every hearing aid manufacturer had developed a WDRC product. Compression is useful as a processing stratagy because it compensates for the loss of cochlear *outer hair cells* which compress the dynamic range of sound within the cochlea. Sensory neural 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, which result from and characterize OHC compression and finally outer hair cell physiology. The WDRC processing stratagy is explained, with a short history of the development of WDRC hearing aids, explaining the initial lack of acceptance of the technology, and a summary of areas of future research.

1 Compression and Loudness

Acoustical signal *intensity* is defined as the flow of acoustic energy in *Watts/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 one LU is defined as the loudness *at* threshold. One sone is about 975 LU. Iso–loudness 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 sound is called the *loudness–level*, which has units of *phons*, measured in *Watts/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

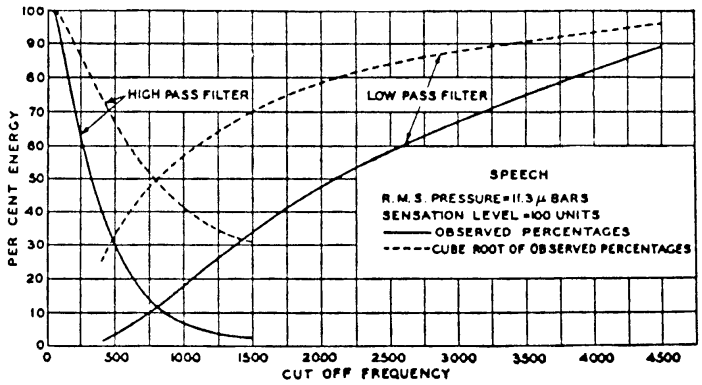

Figure 1: *This figure is from (Fletcher and Steinberg 1924; Fletcher 1929, (p. 236)). It shows the effect of low- and high-pass filtering on the speech loudness-level. The wide band speech is varied in level until it is equal in loudness to low-pass filtered speech. This is done as a function of the filter cutoff frequency. The same experiment is repeated for the high-pass speech. Next the energy of the equally-loud wide band speech is expressed as a percentage of the energy of the low pass speech. An identical calculation is performed on the equally-loud high pass filtered speech. For example, if wide band 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 one since the two solid lines cross at about 11%. After taking the cube root, however, the loudness curves cross at 50%, 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.*

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

This cube–root dependence had first been described by Fletcher the year before (Fletcher 1923a). Fletcher and Steinberg conclude (page 307)

> . . . 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.

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, for tones, was found to be

$$L(I) = I^{1/3},$$

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. For speech the exponent was approximately 1/4. Their method is described in the caption of Fig. 1. *We now know that Fletcher and Steinberg were observing the compression induced by the cochlear* outer hair

cells *(OHCs).*

1.1 Loudness additivity.

In 1933, Fletcher and Munson published their seminal paper on loudness. It details *1)* the relation of iso–loudness 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 (2), rather than thinking directly in terms of loudness growth, they tried to find a formula describing 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 loudness adds. Fletcher's working hypothesis was that each signal is *nonlinearly compressed* by the cochlea, neurally coded, and the resulting neural rates are added. 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* $G(p)$ for tones and speech. *We now know that this function dramatically changed with senory-neural hearing loss.*

Today this model concept is called *loudness additivity*. Their hypothesis was that when two equally loud tones are presented together, separated in frequency so that do not mask each other, the result is "twice as loud." The verification of this assumption lies in the predictive ability of this additivity assumption. For example, they showed that 10 tones that are all equally loud, 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 held, 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 tones, that are equally loud, were matched in loudness by a single tone scaled by α. The resulting definition of α^* is given by the relation

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

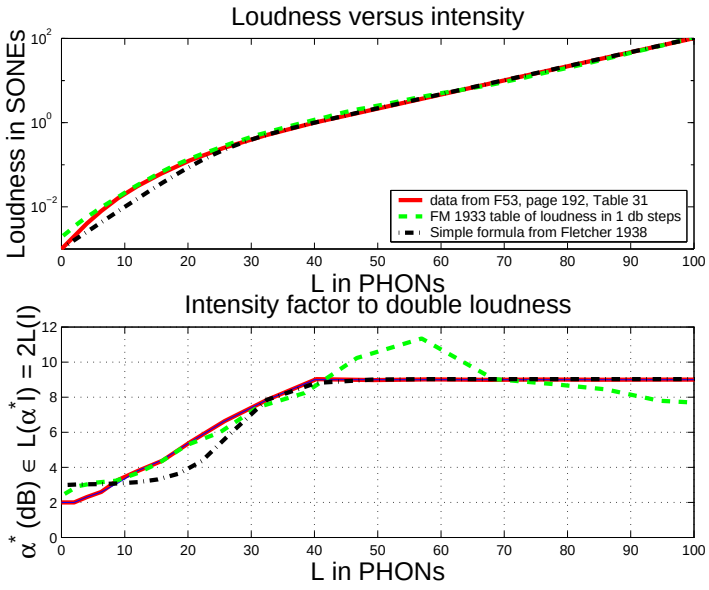

Figure 2: *This figure shows the loudness growth and α^* from p. 192, Table 31 (Fletcher 1953) as a function of the loudness level, in phons. When α^* is 9 dB, loudness grows as the cube root of intensity. When α^* is 3 dB, loudness is proportional to intensity.*

which says that, when the single tone pressure P is scaled by α^*, 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 Fig. 136 (Fletcher 1953, Page 190) and in Allen (1996). The values of $\alpha^*(I)$ found by Fletcher in different papers between 1933 and 1953 are shown in Fig. 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 there is no masking, the relative level α was found to be 9 dB (circa 1953) for P_1 above 40 dB SPL. This value decreased linearly to 2 dB for P_1 at 0 phons, as shown in Fig. 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). 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]. Fig. 2 shows the loudness growth curve and α^* given in (Fletcher 1953, Table 31, page 192), 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 was not 3 dB, but was closer to 2 dB.

Fletcher's statement that loudness is proportional to intensity (α^* of 3 dB) was an idealization that was appealing, but not supported by actual results.

1.2 Recruitment and the rate of loudness growth

Once loudness had been first 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* (personal communication), and first reported by them in 1937 (Steinberg and Gardner 1937; Gardner 1994).

In terms of the *published* record, Fowler (a New York ENT) is credited with the discovery of recruitment in 1936. Fowler was in close touch with the work at Bell Labs and 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."

In a JASA publication on impaired loudness growth, 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 might want to read (Neely and Allen 1996), (Allen 1996a), and (Allen 1999a).

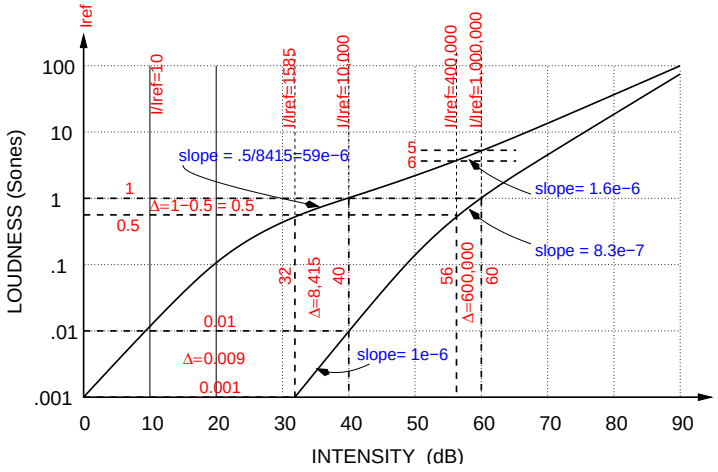

Figure 3: *We show here a recruitment type loss corresponding to a variable loss of gain on the dB scale. The upper curve corresponds to the normal loudness curve whereas the lower curve corresponds to a simulated recruiting hearing loss. For an intensity level change between 60 and 65 dB, the loudness change is smaller for the recruiting ear. The belief that the loudness slope in the damaged ear is greater led to the belief that the JND in the damaged ear should be smaller (e.g., this was the rationale behind the SISI test) (Martin 1986, page 160). Both conclusions are false.*

Loudness growth in the recruiting ear. In Fig. 3 we show 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 dB and log–loudness have resulted in a misinterpretation of recruitment. In the figure we see that for a 5 dB change in intensity at 60 dB SPL, the loudness changes by 2.22 sones in the normal ear and 0.585 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 has lead to some serious conceptual errors about loudness and hearing loss.

Fowler's mistake. After learning about recruitment from Wegel, 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 (your loss could be due to middle ear disease, and you would receive a greater compensation than someone having an identical sensory–neural hearing loss).

In my view, it was Fowler's poor understanding of recruitment that led to such definitions as *complete recruitment* vs. *partial recruitment*, *hyperrecruitment*, and *decruitment*. In fairness, some of these definitions (e.g., decruitment) were introduced in the attempt to diagnose retrocochlear loss from cochlear loss, and are therefore not relevant to the present discussion. *Complete recruitment* means the recruiting ear and the normal ear have the same loudness at high intensities. Steinberg and Gardner described such a loss as a *variable loss* (i.e., sensory–neural loss) and *partial recruitment* as a *mixed loss* (i.e., having a conductive component). 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, page 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 an 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 (Allen 1991), support this view.

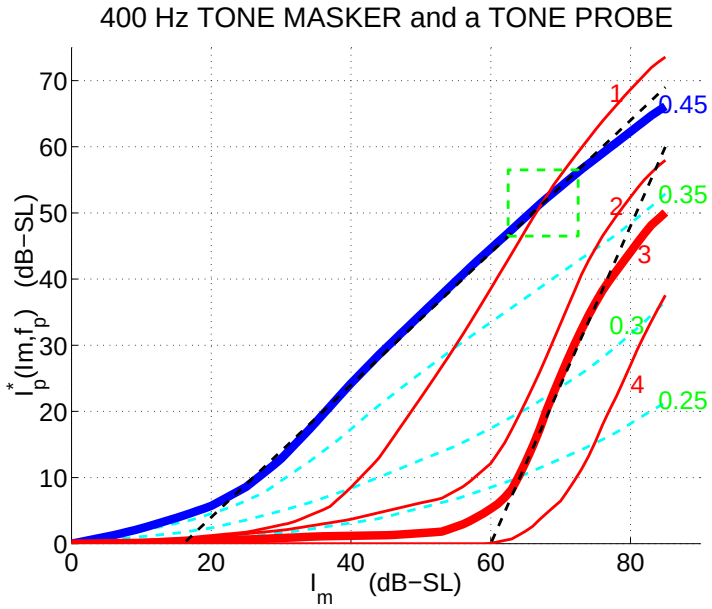

Figure 4: *Masking data from (Wegel and Lane 1924) for a 400 Hz masker. The abscissa describes the intensity of the masker in dB-SL. The ordinate describes the intensity of the threshold probe (maskee) in dB-SL. Each curve corresponds to a probe of a different frequency, labeled in kHz.*

2 Compression and masking

One year after the 1922 threshold measurements in quiet, Fletcher published measurements on the threshold of hearing in the presence of a masking tone (Fletcher 1923a; Fletcher 1923b). Wegel and Lane's classic and widely referenced paper on masking, and the theory of the cochlea soon followed in 1924. In Fig. 4 we reproduce one of the figures from the 1923 Fletcher publication (that 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 Fig. 4, the growth of masking is close to linear. Such

near–linear growth is called *Weber's Law*. The masked-threshold probe intensity[1] I_p^* is equal to the masker intensity I_m plus one 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 appears 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 $\Delta I(I_m)/I_m$.

Upward spread of masking. 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 a *suppression threshold* at about 50 to 60 dB-SL. At this point the masking grows rapidly. The suppression threshold for the dashed-line added to the "solid-fat" f_p = 3 kHz probe curve in Fig. 4 is 60 dB-SL, and its slope is is 2.4 dB/dB. For every 1 dB increase in 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. Namely above 60 dB-SL (i.e., $I_m > 10^6 I_m^*$),

$$\frac{I_p^*(I_m)}{I_m^*} = \left(10^{-6}\frac{I_m}{I_m^*}\right)^{2.4}. \tag{2}$$

From basilar membrane experiments on suppression (Geisler and Nuttall 1997), we know that the probe is being suppressed by 1.4 dB for every 1 dB increase of the masker. To return the probe to its threshold, it must therefore be increased by 1.4 dB more than a linear growth (i.e., 1 dB/dB) (namely 1+1.4 = 2.4 dB) to compensate for the compression effect.

A surprising and interesting crossover occurs near 65-70 dB for the 1 kHz probe. As highlighted by the dashed box in Fig. 4, 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, Figure 8). McFadden (1986) presents an excellent and detailed discussion of this interesting "half-octave shift" effect that is recommenced reading for all serious students of hearing loss.

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

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).

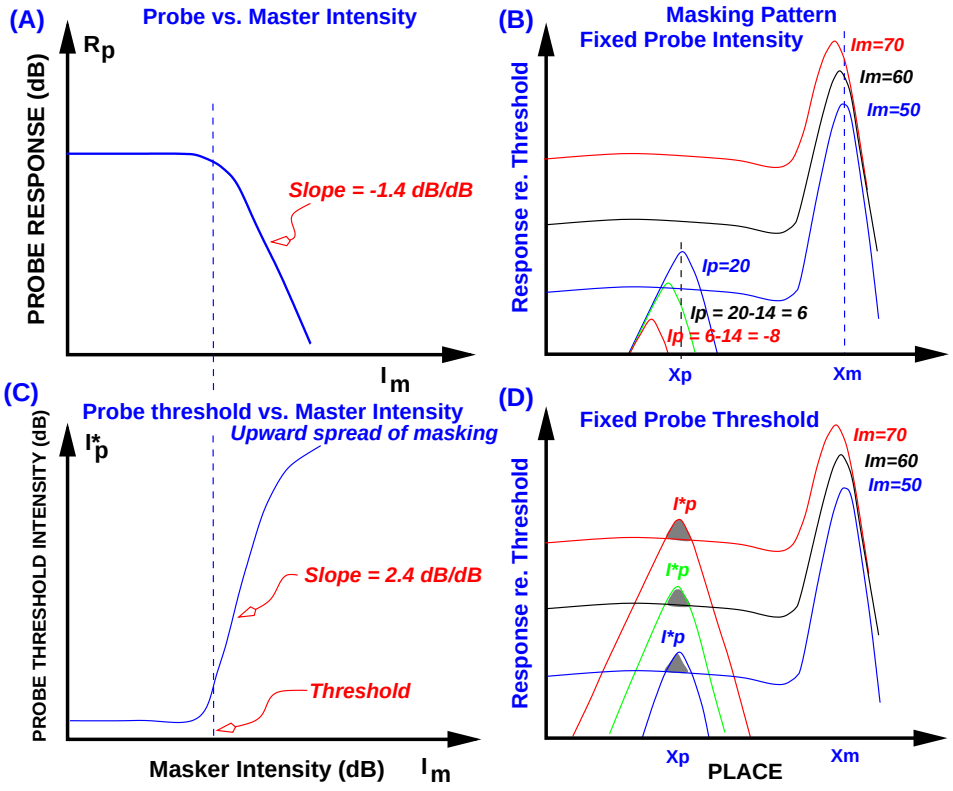

Figure 5: *Cartoon showing the effect of a low frequency masker on a high frequency near-threshold probe. The upper panels show the response of a high frequency low level probe, of fixed intensity, being suppressed by a high intensity I_m low frequency ($f_m << f_p$) masker. Even though the input intensity is fixed, the response intensity R_p is strongly suppressed by the masker. The lower panels show what happens when the high frequency input probe intensity is returned to threshold, indicated by $I_p^*(I_m)$.*

3 The Physiology of compression

What is the source of Fletcher's tonal cube root (and quartic root for speech) 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 outer hair cells (OHC) are the source of the basilar membrane nonlinearity. The history of this insight is both interesting and important.

In 1937, Lorente de No theorized that abnormal loudness growth associated with hearing loss (i.e., recruitment), is due to hair cell damage (Lorente de No 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) (Fahey and Allen 1985; Sachs and Abbas 1976). 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 (Delgutte 1990; Allen 1997b). The two tone suppression (2TS) and upward spread of masking (USM) effect are important to the hearing aid industry because it quantifies the normal cochlear compression that results from OHC processing. In order to fully appreciate the USM and 2TS, we need to describe the role of the outer hair cell in nonlinear cochlear processing.

In Fig. 5 the operation of USM/2TS is summarized in terms of neural excitation patterns.

3.0.1 COCHLEAR NONLINEARITY: HOW?

We still do not know precisely what controls the basilar membrane nonlinearity, although we know that it results from outer-hair-cell (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 due to 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 speculatively theories. The first is a popular but quali-

tative theory, referred to as the *cochlear amplifier*. 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. 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. 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 IHC cilia excitation 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) shifts toward the base, reducing the nonlinear wide dynamic range compression.

3.1 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; Fletcher and Wegel 1922b), thereby establishing the 120 dB dynamic range of the cochlea.

Based on the Johnson (thermal) noise within the inner hair cell (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, namely less than 65 dB (e.g., 55-60 dB) (Allen 1997a). 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.* The IHCs-the cells that process the sound before it is passed to the auditory nerve. 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 noise voltage $|V_c|$ due to cell membrane leakage currents is given by $\langle |V_c|^2 \rangle = 4kTBR$, where B is the cell membrane electrical bandwidth 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 capacitance has been determined to be about 9.6 pF for the IHC (Kros and Crawford 1990) and 20 pf for the OHC. From Eq. 3, $V_c = 21$ μV for inner hair cells 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 1996a).

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

It is known that the OHCs act as nonlinear elements. For example, the OHC 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 inner hair cell (cochlear) signal gain.

4 MULTIBAND COMPRESSION

During the two decades from 1970-80, 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 research results concluded that linear fitting is always superior to compression. When properly adjusted, linear filtering is close to optimum for speech whose level as been adjusted for optimum listen. Papers that fall in this category include Brada *et al*. (1979) and Lippmann *et al*. (1981). However Lippman paper is careful to point out the flaw of pre-adjusting the level.

Another criticism that was quite vurilent was the papers of Plomp (1988,1994) which argued that compression would reduce the modulation depth of the speech. However compression of a wide band 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 wrong (Villchur 1989). The filter bandwidths used in WDRC hearing aids are not narrow enough to reduce the modulations in critical band widths.

Other work that found negative results used compression parameters that were not realistic, and time constants that are too slow. Long time constants with compression produce very different results, and are not in the category of syllabic compression, or automatic gain control (AGC). 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 other 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; Dillon 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; Villchur 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 cant hear it, you can't understand it." This had a certain logical appeal.

Other key papers include (L.M.H. 1994; M.C. 1996; Killion 1996; Killion et al. 1990; Mueller and Killion 1996). A physiology paper that is frequently cited in the compression literature is (Ruggero and Rich 1991).

Fred Waldhauer, a Bell Labs analogue 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 CUNY, we developed a fitting procedure, and ran several field trials (Allen et al. 1990). AT&T licensed its hearing aid technology to ReSound 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 still remains, difficult to show quantitatively the nature of the improvement of WDRC. It is probably fair to say that only with the success of ReSound's WDRC hearing aid in the marketplace, has the clinical community come to accept Villchur's claims.

I think the acceptance issue has to do with the two views of what WDRC is, and why it works. These views strongly influence how people think about compression. They are the *articulation index (AI) view* and the *loudness view*.

The AI–view is based on the observation that speech has a dynamic range of about 30 dB in 1/3 octave frequency bands. The assumption is that the speech 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 the listener 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 the changing of the critical band with hearing loss, or the temporal dynamics of the compression system. The analysis of these important details are interesting only *after* the signals are placed in the audible range.

Summary. In this paper we have 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 senory-neural hearing loss, and this causes the cochlea to have re-

duced dynamic range.

When properly designed and fitted, *wide dynamic range compression* has proven to be *the* most effective speech processing strategy we can presently provide for sensory-neural hearing loss compensation. The reason it works is because it supplements the OHC compressors provided by Mother Nature, which are damaged with sensory-neural hearing loss.

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