8

TALKING HAIR CELLS: WHAT THEY HAVE TO SAY ABOUT HEARING AIDS

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When I submitted the title to this talk, I hoped that I would be able to play a demonstration of the ear talking. I didn't succeed in that, but I do have a demonstration of the ear playing music.

Thus I will begin by describing a method for "playing by ear"—using music by two famous composers to demonstrate this entertaining but meaningful use for otoacoustic emissions—and then discuss the changes in physiology and loudness growth accompanying three types of hearing loss. Different hearing losses require different hearing aid characteristics, which can now be chosen with increasingly solid theoretical justification. This leads to a discussion of loudness growth data and a brief discussion of individual differences in loudness growth and the repeatability of loudness measures.

TARTINI & BACH: PLAYING BY EAR (THE HAIR CELLS PERFORM)

Figure 8–1 illustrates the generation of a Tartini tone. Tartini was a violinist back in the 1700s who is often credited with being the first to describe what violinists call the Tartini tones. If you simultaneously play the notes C and E on the A and E strings of a violin, for example, you will hear the

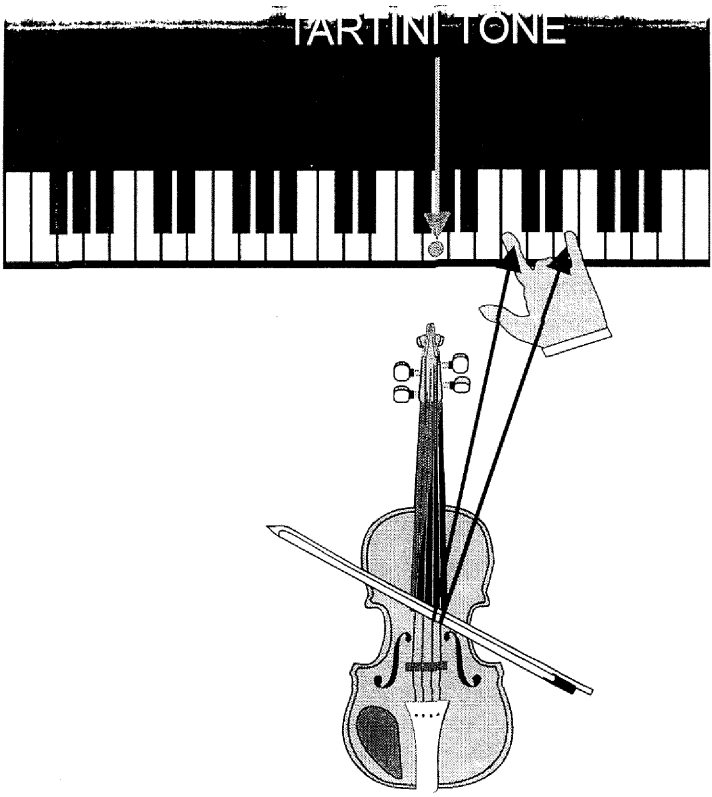

Figure 8–1. Illustration of audible Tartini Tone generated when a violin is played "double stop."

note G as illustrated in the figure.[1] The extra tone you hear—the same one violinists have been noticing for centuries—is the tone we are now measuring objectively in the earcanal. The difference is that now we call it the "cubic distortion product" tone at the frequency 2f1-f2 rather than the Tartini tone.

In the previous example, where f1 = 1047 Hz (C two octaves above middle C) and f2 = 1319 Hz (E), the cubic distortion tone is at the frequen-

[1]Checking this example out on a violin while this manuscript was being prepared, the author realized that the Tartini tone chosen for the sake of illustration was not the easiest one to hear. A better example might have been any of the pairs of notes he regularly plays on a piano to check out intermodulation distortion in hearing aids: The musical fourth C_5 and F_5 produces an audible note F_4 if the aid distorts badly. Similarly, B_4 and F_5 produce D_4; $Bb_4$and F_5 produce Bb_3; A_4 and F_5 produce F3. (C_4 is middle C in this annotation.)

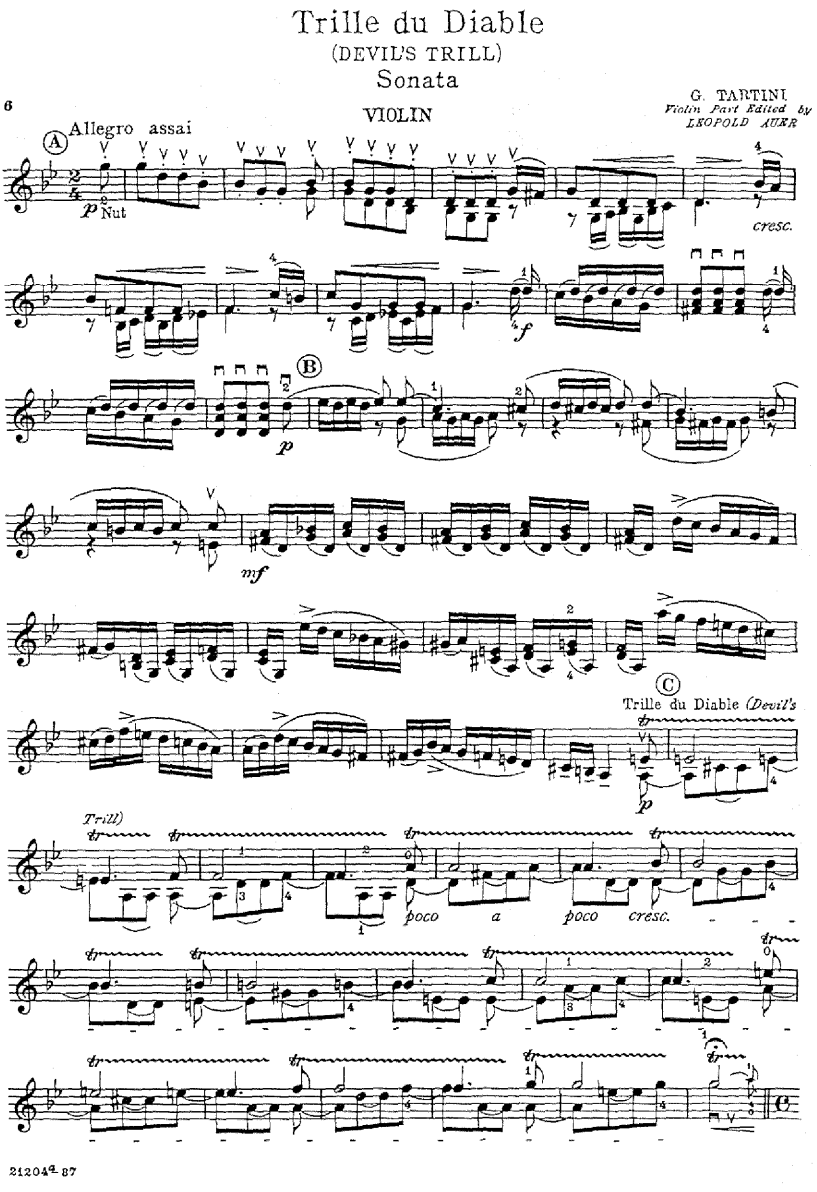

Figure 8–2. Portion of Tartini's Sonata in G.

cy 2f1-f2 = 775 Hz (G). In point of fact, the frequency of 775 Hz is about 20 cents, or 1/5 of a semitone, below the musical note G. Many of the Tartini tones do not fall exactly on a note of the musical scale, which may have been what called them to Tartini's attention in the first place.

Just as Tartini tones are the products of intermodulation distortion in the ear, any nonlinear system exhibiting "cubic law" nonlinearity will generate Tartini tones. (Traditional starved-Class-A hearing aid amplifiers, for example, generate Tartini tones, although in hearing aids such tones are usually called intermodulation-distortion or cubic-distortion products.) In the past, it was difficult to provide a violinist with satisfactory hearing aids: the 105–115 dB SPL input on the violin side of the head created intermodulation distortion products much stronger than normal aural Tartini tones, making the violin nearly unplayable because the distortion from the hearing aid interfered with intonation.

In honor of Tartini, I should like to play a short recording from his Violin Sonata in G, the portion shown in Figure 8–2. The passage of music starting at circle C is the "Devil's Trill," a difficult section requiring two of the violinist's fingers to play the melody while the other two are engaged in a trill. According to Tartini, the Devil himself appeared at the foot of Tartini's bed in a dream one night in Assisi in 1713, playing the now-famous Devil's Trill on Tartini's own violin. (The section of music shown in Figure 8–2 was played for the attendees. It is Band 3 on the enclosed CD.)

Figure 8–3 shows the basic approach to "playing music by ear." One simply chooses successive pairs of frequencies that will generate each of the desired notes as Tartini tones. In the example shown, the desired note is an F, generated as the cubic distortion product of Bb and D. To make these recordings, we held Bb constant as f1 (using an oscillator, not a piano, of course), and then chose the value of f2 to produce the desired resultant tone. The data shown in Figures 8–3 to 8–5 were obtained with the aid of a low-noise ER-10C otoacoustic emissions probe sealed into the earcanal and the CUB^{e}DIS^{TM} analysis system (Allen, 1990). The CUB^{e}DIS system uses a synchronous averaging technique so that 4 seconds of averaging produces an equivalent 0.25 Hz bandwidth. The ER-10C uses a separate earphone driver for f1 and f2 to avoid the possibility of intermodulation distortion generated by the probe itself, and uses two microphones whose averaged output minimizes the noise level of the probe pickup.

As shown in Figure 8–4, as the frequency of f2 is increased, the frequency of the cubic distortion tone will decrease. The data shown in Figure 8–4 were obtained from the ear of Tim Monroe, a 27 year old male engineer with normal hearing and good aural emissions. Although there is a limit to the usable tones that can be produced with f1 = Bb, quite a few of the cubic distortion tones show a level of 5–10 dB SPL. These can

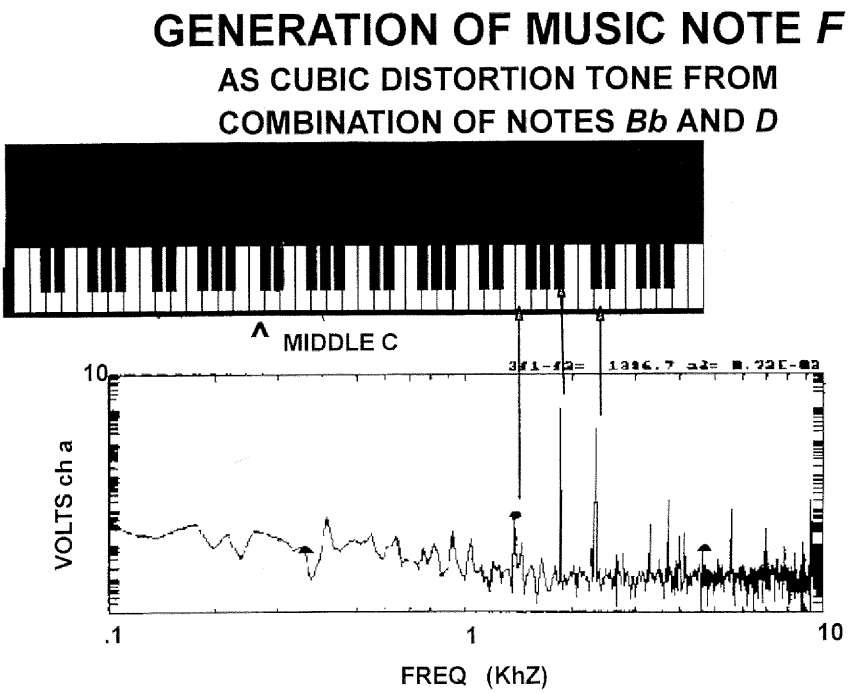

Figure 8–3. Generation of music note F played—literally—by ear. Note: In these figures, the piano keyboard has been scaled and positioned to line up with the frequency axis on the graph. The lowest octave on the piano—extending down to 27.5 Hz—has been omitted.

be heard without signal averaging, for the reasons discussed in the following paragraph.

The minimum pressure audible at the eardrum is about 12 dB SPL, so tones from the ear would be audible *directly* only if they exceeded 12 dB SPL.[2] By using a microphone quieter than the ear, however, tones below 12 dB can be detected and made audible by amplification. The internal noise of the ER-10C microphone, for example, has a spectrum level of about –20 dB SPL. The apparent bandwidth of the ear as a filter in this frequency region is roughly 100 Hz, based on the masking data of French & Steinberg (1947), who reported that a tone 18–22 dB above spectrum level is just audible to the average ear in the 1000–2500 Hz frequen-

[2]A good single-number approximation to the minimum audible pressure at the eardrum is 12 dB SPL; between 500 and 4000 Hz it lies within 3 dB of that value (Killion, 1978).

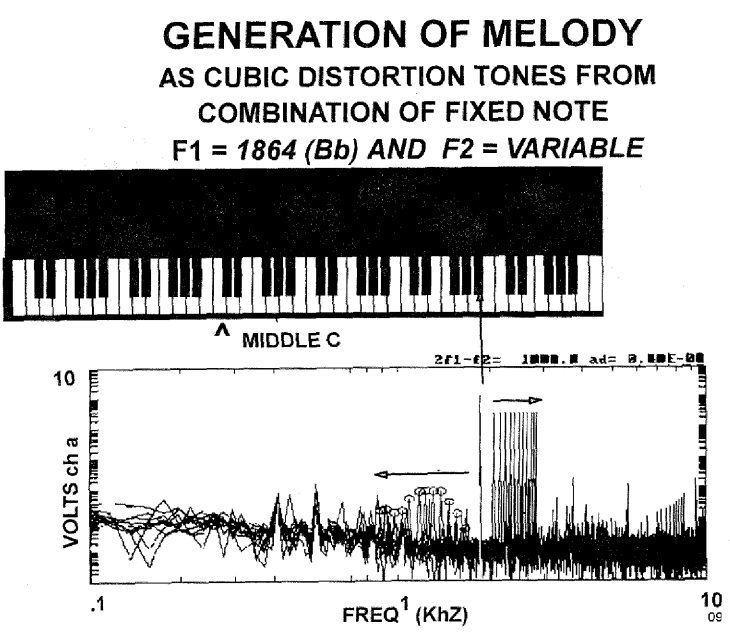

Figure 8–4. Generation of series of tones played by ear (Tim Monroe's).

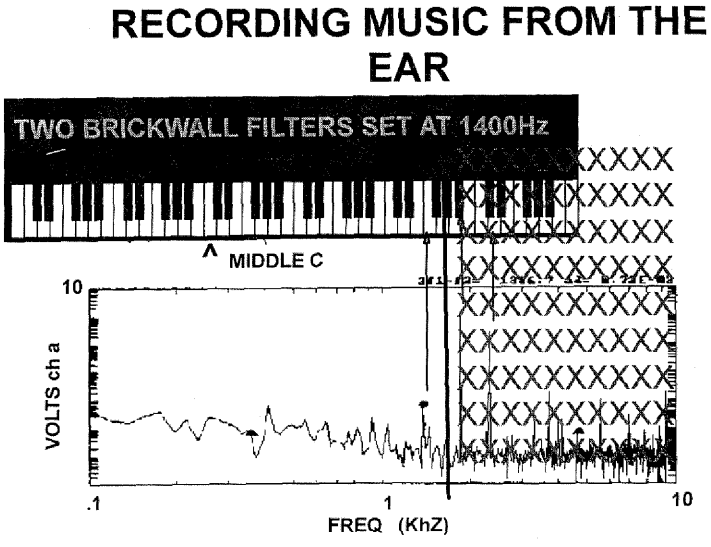

Figure 8–5. Use of two brick wall filters to filter out generating primaries f1 and f2 so cubic distortion tone 2f1-f2 can be heard.

cy region. Listening to the amplified output of an ER-10C microphone placed in the ear would allow detection of tones at roughly 0 dB SPL; such tones would be just at the masked threshold produced by the amplified internal noise of the microphone. Otoacoustic emission tones at 5 to 15 dB SPL should be sufficiently above masked threshold to be both audible and tonal.

The only remaining problem is the presence of the primary tones. To filter out the primaries, we used two "brick wall filters" in series, each set to 1400 Hz as shown in Figure 8–5. Without the filter, the 65 and 55 dB SPL primary tones would mask the weak distortion tone we want to hear.

Figure 8–6 shows the section of Bach Partita #3 used for our demonstration recording. I am grateful to Dr. David Preves for suggesting this passage, whose rapidly alternating notes suggest two violins, and which uses notes that we could evoke in Tim's ear. (His ear doesn't play some notes as well as others, as you will hear when we play the demonstration tape.)

First I will play an old recording of the passage of music shown in Figure 8–6, so you have some idea of what you will be listening for when it is played by ear. (PLAYS: Band 4 on the enclosed CD.)

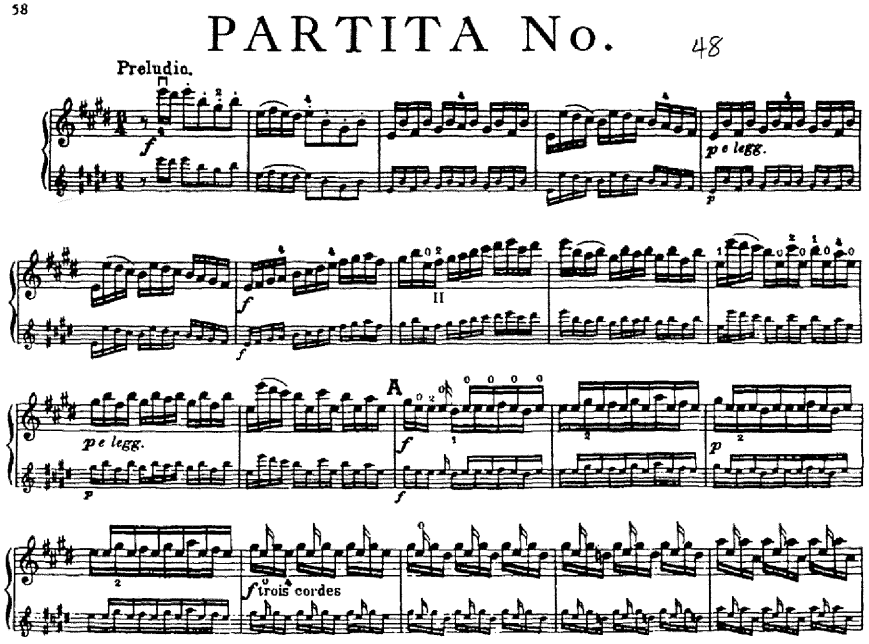

Figure 8–6. A portion of Bach Partita No. 3 played by ear.

The section starting at the bar marked "A" is the section that you will now hear as a recording of the cubic distortion tones produced in Tim's earcanal. We used computer control of the f2 oscillator to produce the frequencies shown in Table 8–1. Table 8–1 also shows the resultant cubic

TABLE 8–1. Frequencies used for generating the first two bars of the Bach Partita #3 demonstration, with primary f1 = Bb = 1864 Hz. All succeeding bars were generated in a like manner to those shown here.

CDT NOTE NUMBER	CDT NOTE NAME	CDT TONE FREQUENCY	f2 TONE FREQUENCY
1	G	1567	2161
2	Eb	1244	2484
3	Eb	1258	2470
4	Eb	1244	2484
5	D	1174	2554
6	Eb	1244	2484
7	Eb	1258	2470
8	Eb	1244	2484
9	F	1396	2332
10	Eb	1244	2484
11	D	1174	2554
12	Eb	1244	2484
13	Eb	1258	2470
14	Eb	1244	2484
15	G	1567	2161
16	Eb	1244	2484
17	F	1396	2332
18	Eb	1244	2484
19	G	1567	2161
20	Eb	1244	2484
21	A	1660	2068
22	Eb	1244	2484
23	F	1396	2332
24	Eb	1244	2484

Notes: 1. In an attempt to add realism, a small difference in frequency (10 cents or 10% of a semitone) was added between "downstroke of the bow" (e.g., note 3) and "upstroke of the bow" (note 4). The effect was hard to hear, probably not worth the bother, and slightly confuses the appearance of Table 8–1. 2. A subsequent calculation generated "vibrato," as described in the text. This *was* audible and improved the musicality of Tim's performance. 3. The Partita was transposed down a half-note for this recording.

distortion tone. Only the first two bars of music are displayed in Table 8–1. For naturalness, a small amount of vibrato was added to each note, changing f2 successively by 0, +20, 0, and –20 musical cents from the value shown (20 cents = 1.2%). (PLAYS: Band 5 on CD.) We later made recordings at half these frequencies (this one was an octave above the written part), which were also successful (Plays: Band 6 on CD).

Any musician in the audience with perfect pitch is perhaps wondering why we had the ear play the passage 1/2 note flat. I confess I can't remember why I transposed the passage down a 1/2 note on the computer before generating the f2 file, although it seemed to make sense at the time.

The experimentalists in the audience are probably asking how we know that the tones we hear came from Tim's ear rather than being generated by some form of intermodulation distortion in the equipment.

The question can be answered by making a recording in an ear which presents the proper acoustic impedance but generates no emissions; that is, a dead ear. An ideal dead ear is the Zwislocki Coupler. The next recording was made identically to the last, except that the probe was removed from Tim's ear and placed in Zwislocki's coupler. Since Tim's ear has a measured acoustic impedance close to that of the Zwislocki coupler, any equipment distortion should show up as tones in the "dead ear." This distortion problem occasionally plagues some commercial otoacoustic emission measurement equipment; the test is the same. (PLAYS: Band 7 on CD. I trust you did not hear Bach playing this time. There is a small bleed-through Bb tone, which is the f1 primary, but no melody.)

A REVIEW OF HEARING, WITH THE GOAL OF DESCRIBING THREE TYPES OF HEARING LOSS

Distortion Products as Indication of Outer Hair Cell Function

We have already had quite a bit of discussion about the operation of the outer hair cells, but the next two figures may be interesting nonetheless. Figure 8–7 shows the different distortion products generated in a normal ear by the nonlinearity of the outer hair cell motion. These were measured using the CUB^{e}DIS™ system. Note that the harmonic-distortion products are evident at 2f1 and 2f2, as is the difference tone at f2-f1. Both third-order distortion tones are visible: the Tartini tone is the lower cubic distortion tone (CDT) at 2f1-f2. The upper CDT is at 2f2-f1. The 2f1-f2 tone is the one we measure clinically to generate the "DP-gram."

Figure 8–8 contains data that I find particularly interesting. It shows the DP-gram and audiogram of a 35 year old female Swedish musician who is also a television producer. When she visited our laboratory, she

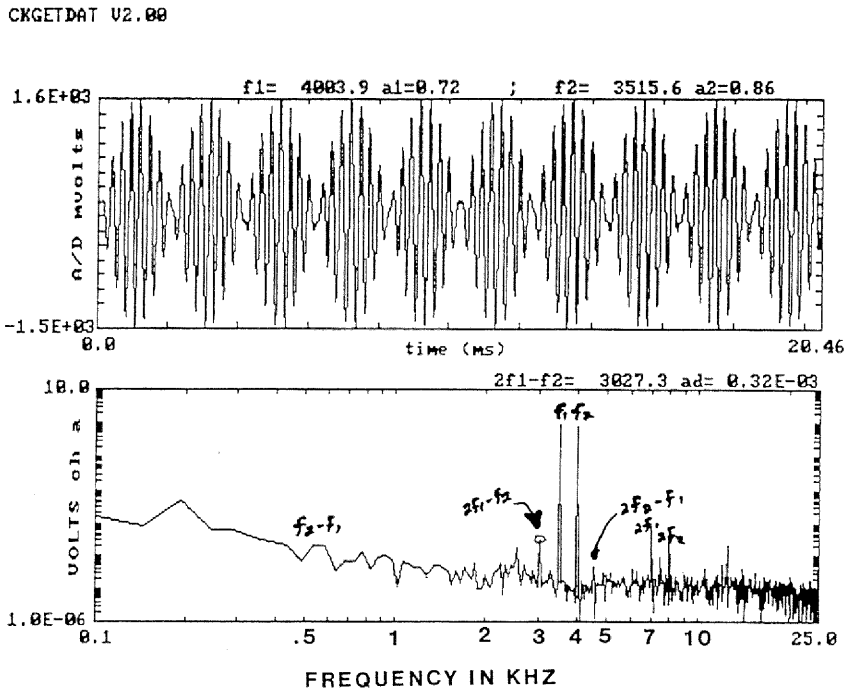

Figure 8–7. Spectrogram of distortion products from the ear: Waveform of combined 65-dB SPL primary tones shown in top graph, spectrum analysis of earcanal microphone output shown in bottom graph.

confessed she produced a lot of rock music and she thought she had a small noise-induced notch in her audiogram. When I measured her otoacoustic emissions, we saw that her emissions were 20–30 dB above the noise floor until we got to 6 kHz, where they dropped not quite to the noise floor and then returned to normal at 8 kHz. I measured her threshold audiogram with the result you see: normal hearing through 4 kHz, a dip at 6 kHz, and normal at 8 kHz. (Before you buy otoacoustic-emission equipment to measure audiograms, I should confess that Figure 8–8 represents the nicest correlation between audiogram and DP-gram that I have seen. By the time most such subjects come in for testing, they have a 50–60 dB hearing loss at the notch frequency, and appreciable loss at adjacent audiometric frequencies.)

For our present purposes, the interesting question is: "What happens to loudness growth as outer hair cell (and inner hair cell) function is lost?"

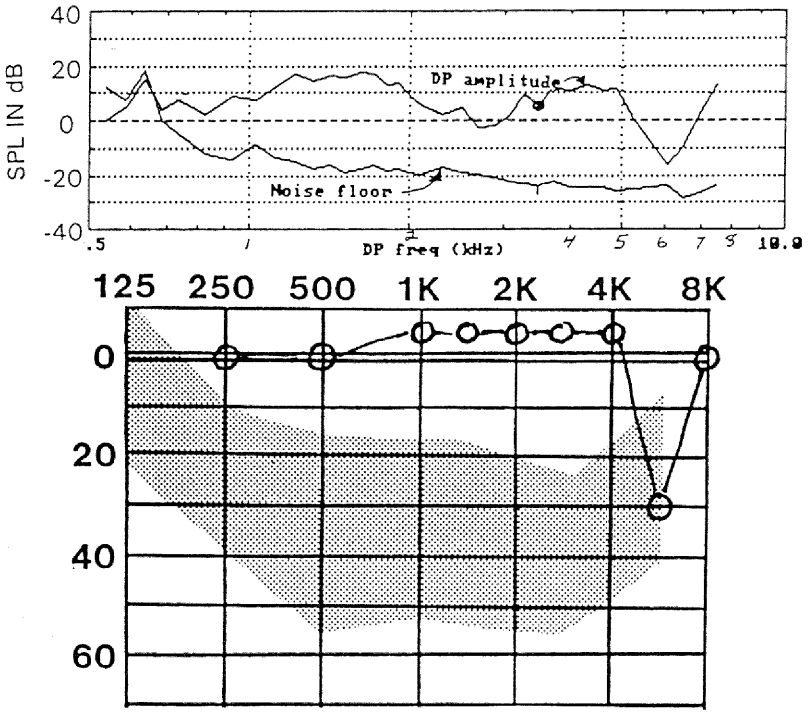

Figure 8–8. DP-gram and audiogram of 35 year old woman with apparent loss of outer hair cell function only near 6 kHz.

The Operation of the Cochlea According to Berlin

First, I'd like to give a demonstration of the operation of the cochlea, a demonstration I learned from Dr. Berlin and have used in nearly every lecture since then.

Figure 8–9 shows a drawing of the cochlea with outer hair cells, inner hair cells, neurons, and the tectorial membrane. The drawing shows the outer hair cells embedded in the tectorial membrane and the inner hair cells as nearly or just touching the tectorial membrane.

From thousands of 8th nerve recordings, we know that all the signals going to the brain come from the inner hair cells. So what do the outer

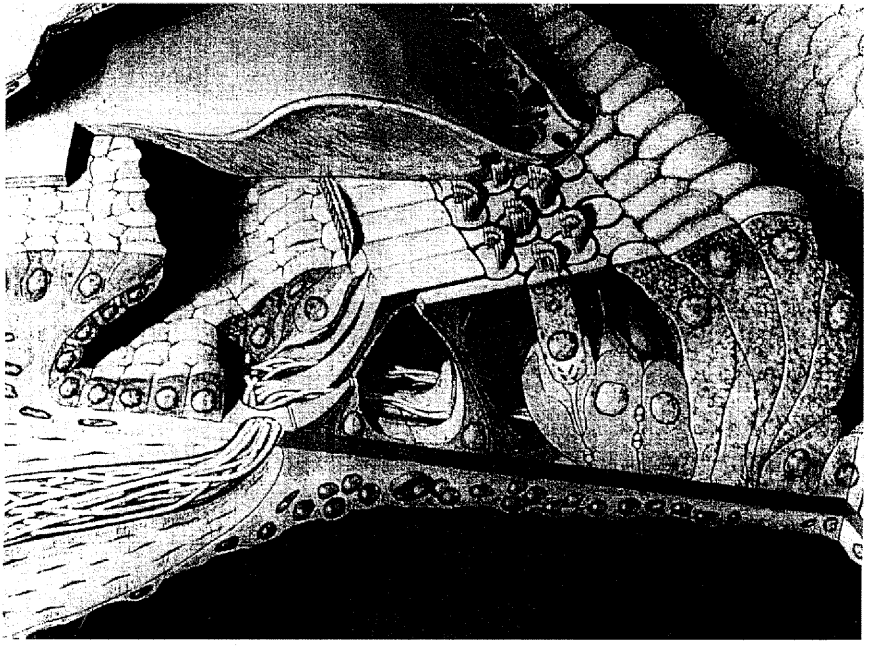

Figure 8–9. Drawing of cochlea showing inner and outer hair cells, neurons, and tectorial membrane.

hair cells do? Let us assume Dr. Berlin's arm is the tectorial membrane, and my fingers intertwined with his fingers represent the embedded outer hair cell fibers. There are various hypotheses on the exact mechanism by which the inner hair cells are induced to fire, and on how the outer hair cells increase the sensitivity of the cochlea by 40 dB or more. My personal favorite is that the motion of the outer hair cells modulates the gap between the tectorial membrane and the stereocilia of the inner hair cells. Because the fluid flow resistance varies as the third power of the gap spacing, only a small motion would produce the required 40 dB change in sensitivity: changing the gap from 10 microns down to 2 microns would do the trick, for example. For our present purposes, however, we are going to assume that the inner hair cells—represented by the fingers of my free hand—fire when they bump into the tectorial membrane, represented by the underside of Dr. Berlin's arm above the elbow. For this simulation, I am holding my fingers in the rest position about 2 inches away. With no help from the outer hair cells, the inner hair cells will fire when a 40 or 50 dB SPL input causes enough motion to bridge that 2-inch gap so the inner hair cell fibers bump against the tectorial

membrane, as the fingers of my free hand in motion bump against my assistant's upper arm.

With the assumed mechanical amplification correlated with normal outer hair cell motion, however, only about 10 dB SPL is required in order to produce the required amount of motion between the inner hair cell stereocilia and the tectorial membrane. With the help of the outer hair cell amplifiers, firing occurs with only 10 dB SPL or so (roughly 0 dB HL) at the eardrum.

If the outer hair cells are damaged or missing or paralyzed, it will take something like 50 dB SPL (40 dB HL) to cause the inner hair cells to fire. In other words, we will see a 40 dB hearing loss.

What happens to loud sounds with this model? Let us consider a 90-dB SPL input. If 50 dB causes 2 inches of motion in our simulation, then 90-dB SPL would cause 200 inches of motion: the required motion of my free hand would extend down 16 feet from where it is to somewhere in the basement below us. With 200 inches of motion available, the 2 inches of motion available from the outer hair cell amplifiers becomes irrelevant. With such a large motion available, the inner hair cells will fire whether the outer hair cells move or not. We conclude that with high-intensity signals the inner hair cells should fire normally.

The remaining question is: "How can one experience more than a 40- or 50-dB cochlear hearing *loss* if the outer hair cell amplifiers only provide 40–50 dB of *gain*?" The answer is that the same aural abuse—from shooting, using a chain saw, or whatever you did that broke off the hairs of your outer hair cells—the same abuse that broke off your outer hair cells will sooner or later be likely to break off the stereocilia of the inner hair cells as well. Loss of inner hair cells results not only in a loss of sensitivity, but a loss of "channel capacity" in the auditory system: Fewer transmitting sites are available to send information to the brain. (It is also possible for the tectorial membrane to float up and away—I think I first heard this suggestion from Dr. Berlin—causing a loss of firing sensitivity for the inner hair cells at high levels even though the inner hair cells themselves may be undamaged.) In most cases, however, histological evidence suggests that the mechanism for hearing loss above 40–50 dB HL is a loss of inner hair cells and the consequent loss of auditory channel capacity.

LOUDNESS GROWTH DATA

Most of the early loudness-growth data were obtained at Bell Telephone Laboratories. Those earlier data look much like the more recent data of Lyregaard (1988) and Lippman et al. (1977), shown idealized in Figure 8–10.

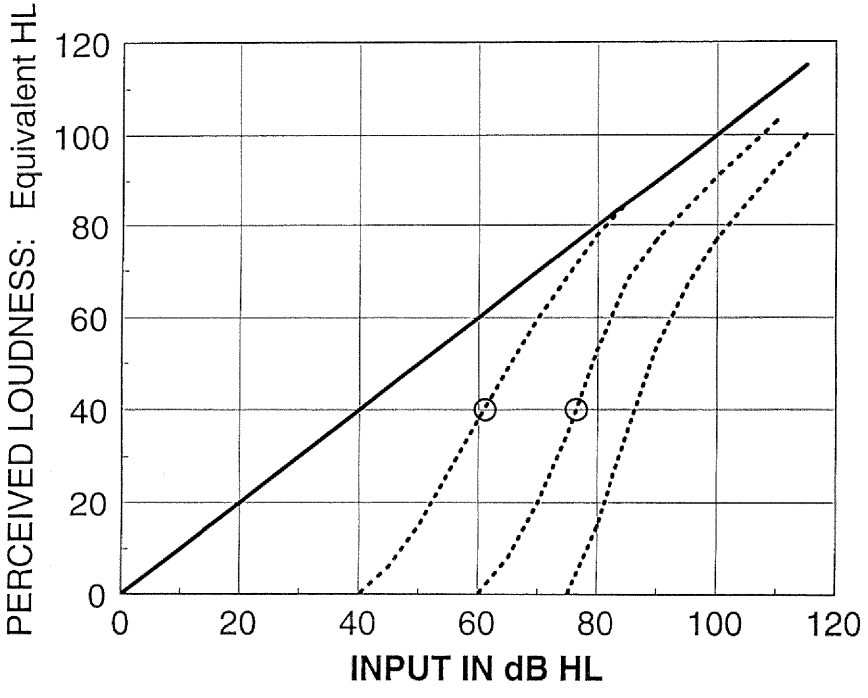

Figure 8–10. Idealized normal and impaired loudness growth curves for 40, 60, and 75 dB hearing loss, after data of Lyregaard (1988) and Lippman (1977).

Note that somewhat like using the early phon scale from Bell Labs, Lyregaard choose to plot loudness not in terms of judged magnitude (as is often done in psychoacoustics), but in terms of equivalent loudness for subjects who hear normally. I like this simplification: If the average subject with normal hearing judges a tone presented at 40 dB hearing level as having a loudness magnitude of 2.3 on the Pascoe/Hawkins scale (2 = soft, 3 = comfortable but soft), the corresponding point is plotted as 40 dB HL on the Y axis, not 2.3. If someone with a hearing impairment requires a 61 dB HL tone in order to have a loudness experience of magnitude 2.3, you plot that point in the same manner, as 40 dB HL on the Y axis above 65 dB on the X-axis. If another individual requires 76 dB HL for the same loudness judgment of 2.3, this is plotted as the x,y point (76,40). (These examples correspond to the circled points on Figure 8–10).

The are two other important sets of recent loudness data to consider. Figure 8–11 shows the data of Pascoe (1988), who reported on 500 ears tested at 0.5, 1, 2, and 4 kHz. Finding nearly the same curves applied at all frequencies, he combined data across frequency. By taking the normal

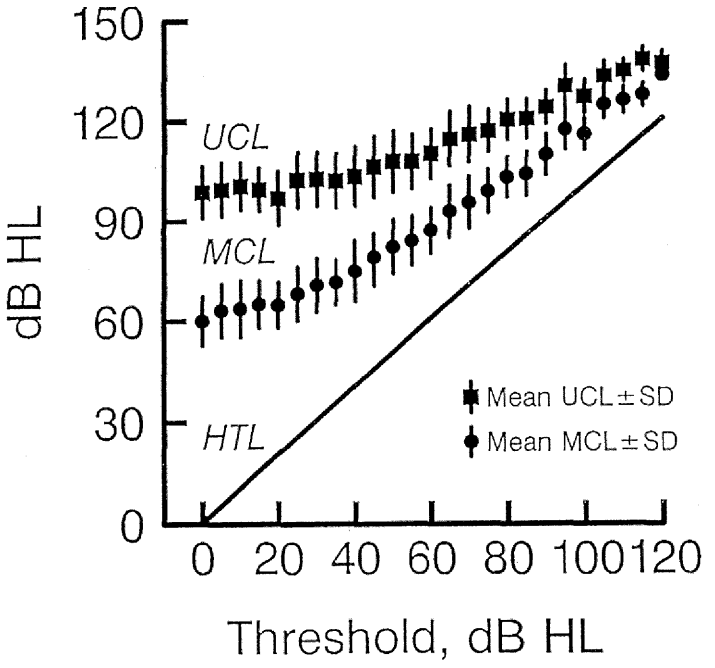

Figure 8–11. MCL and LDL data on 508 ears (Pascoe, 1988).

LDL (Loudness Discomfort Level) as reference, and noting the amount of elevation above normal LDL accompanying a given degree of hearing loss, we can obtain an estimate of whether or not a given amount of gain for loud sounds is likely to cause discomfort. More precisely, it provides an estimate of the *amount* of gain for high-level sounds that can be safely provided if the average individual with impaired hearing is to be no more at risk of loudness discomfort than a neighbor with normal hearing sitting nearby. This is an extraordinarily important piece of information that we will use later.

The other recent data are those of Hellman and Meiselman (1993). Hellman and her colleagues have been doing a variety of loudness studies using cross-modality loudness matching, direct loudness magnitude estimation, and loudness production (where the subject chooses the signal level that matches an assigned loudness magnitude target). Pleasantly enough, they obtained basically the same answer with each approach. Even more importantly, they have studied a large number of subjects with hearing loss: 17 people with 45 dB loss, 24 with 55 dB loss, 20 with 65 dB loss, and 20 with 75 dB loss. (Each loss category included losses within 5 dB of the nominal value.)

Figure 8–12 shows the Hellman and Meiselman data plotted as Lyregaard had done. (A note of thanks: At the time I became aware of the Hellman and Meiselman data, I had already extracted curves from the Lyregaard and Lippman et al. data. Dr. Hellman was kind enough to transform her data to a form ready for the plot of Figure 8–12.) Figure 8–13 shows a comparison between the Hellman and Meiselman data and the previous estimate for a typical subject with 40-dB loss. Her data, taken on a large number of subjects, look quite similar to the earlier estimate based on the Lyregaard and Lippman data. Figure 8–14 shows a similar comparison for 60-dB loss. Again, the estimated 60-dB curve is largely bracketed by the Hellman and Meiselman data for 55- and 65-dB losses.[3]

[3]The recent high-level data are sometimes sparse because of Institutional Review Boards, even though there is sometimes little logical reason for the limitations that are set. For example, 100 dB HL is, depending on the frequency, about 105 dB SPL in the sound field, no big deal for a short exposure. Country and western bands sometimes produce 100–105 dB SPL for hours at audiology meetings! During routine practice, an amateur violinist (the writer, for example) can produce 106 dB SPL at the external ear for a short period of time. A virtuoso can produce over 112 dB, 4 times the power, *and* a beautiful tone! The use of short-duration signals of 110 dB SPL and higher is needed in order to obtain clear-cut LDL measurements in order to facilitate hearing aid design research.

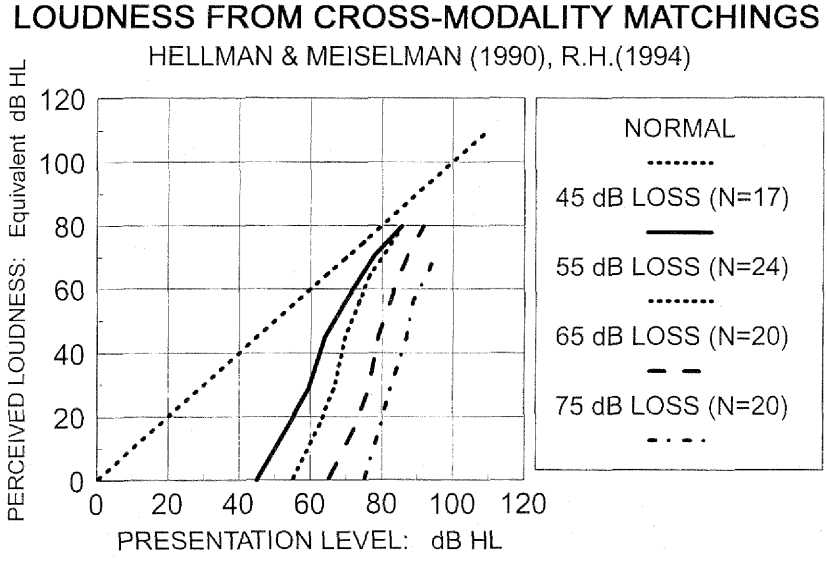

Figure 8–12. Loudness from cross-modality matching.

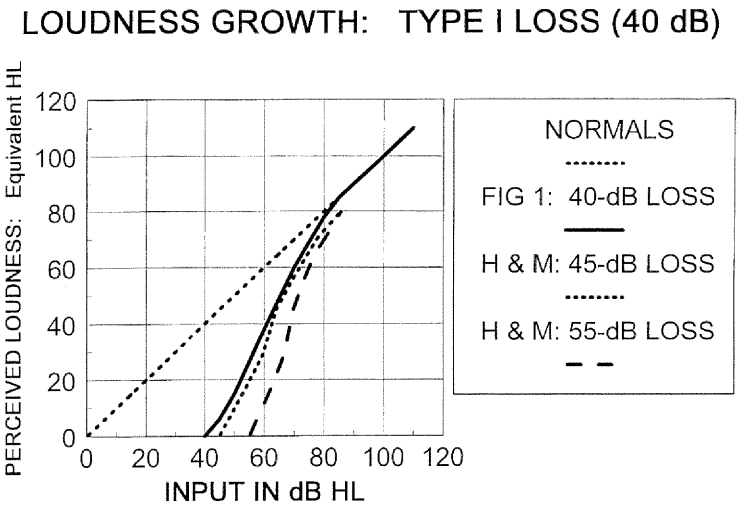

Figure 8–13. Killion & Fikret-Pasa comparison of the earlier loudness-growth estimate for a 40 dB loss with the Hellman & Meiselman data for 45 and 55 dB losses.

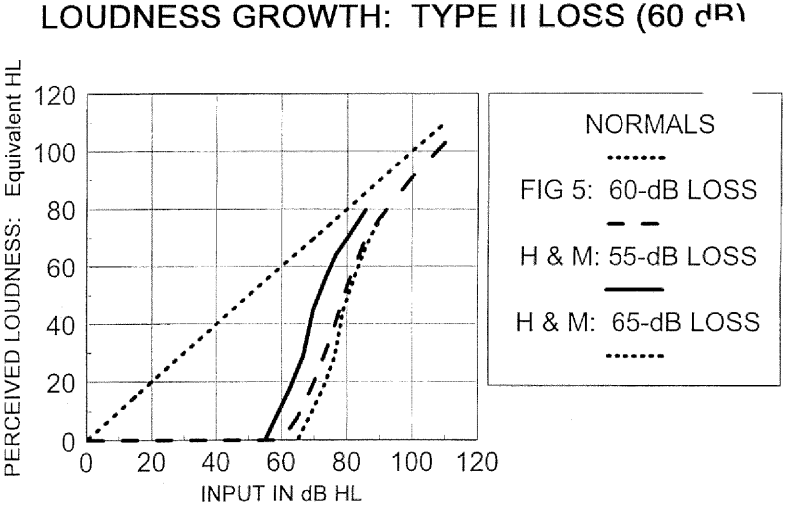

Figure 8–14. Killion & Fikret-Pasa comparison of the earlier loudness-growth estimate for a 60 dB loss with the Hellman & Meiselman data for 55 and 65 dB losses.

We now have everything in place to talk about types of hearing loss, and the types of hearing aid processing they might require.

Types of Hearing Loss

Figure 8–15 illustrates three types of hearing loss (Killion & Fikret-Pasa, 1993). A fourth type—profound loss—is not shown because we know less about it.

Type I hearing loss is depicted in the upper left panel of Figure 8–15, where the loudness growth for a typical cochlear loss of 40 dB is illustrat-

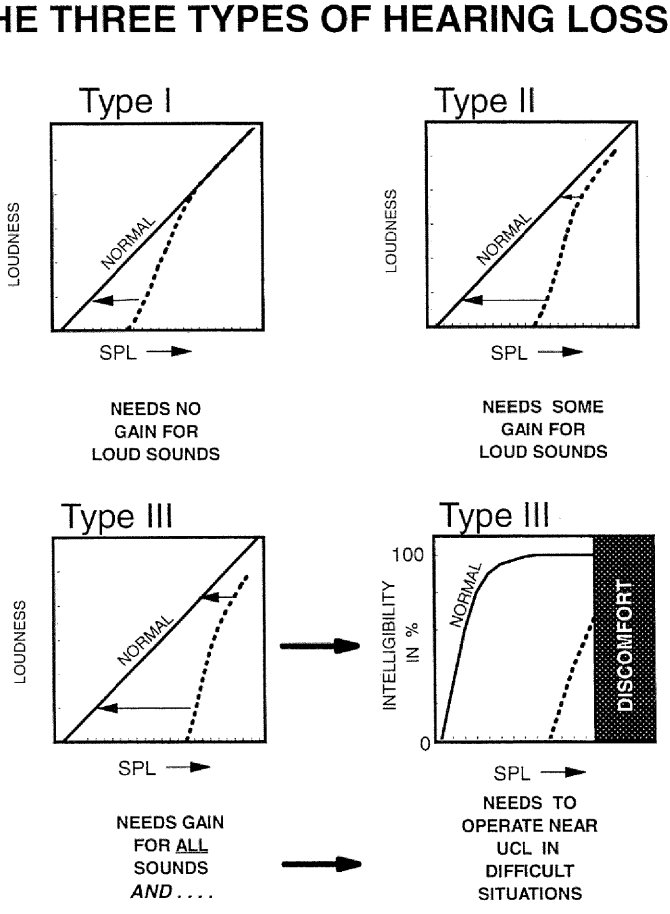

Figure 8–15. The three types of hearing loss.

ed. A Type I loss shows complete recruitment: loudness sensation for intense sounds is the same as normal, but sounds below 40-dB HL are inaudible. This finding is consistent with a loss of outer hair cell function with normal inner hair cell function. With increasing level above 40 dB HL, loudness gradually returns to normal. Not only has *loudness* returned to normal for high-level sounds, but you can find subjects with 40-dB threshold loss who appear to have normal or near-normal high-level hearing by *any* measure you apply. This is true whether you measure high-level difference limens for intensity or frequency, psychoacoustic tuning curves, electrical auditory brainstem response (ABR), or speech discrimination in noise. In animals, where it is possible to place an electrode in the 8th nerve bundle, individual neural recordings can be normal at high levels in the region of loss. Many of these findings were reviewed 15 years ago (Killion, 1979).

To repeat: An individual with Type I hearing loss has a *loss of sensitivity for quiet sounds*, but may have little or no loss of hearing for loud sounds. The hearing loss is restricted to low-level sounds. Although we will discuss hearing aids in greater detail later, it already seems evident that *an individual with a Type I loss doesn't need loud sounds to be made any louder than they already are*; what is needed is gain for low-level sounds in order to make them audible and clear. Not so evident—as witnessed by some hearing aid designs—is the conclusion that the individual with a Type I hearing loss doesn't need loud sounds to be made any *quieter* than they are. In other words, no output *limiting* for loud sounds is needed, at least not unless people with normal hearing also need hearing protection in the same environment. A corollary of this conclusion is that is a mistake to choose a low maximum output "SSPL-90" for a Type I loss; it will only result in premature clipping and distortion of everyday loud sounds. This conclusion is consistent with the experimental findings of Fortune, Preves, and Woodruff (1991), who reported fewer complaints of discomfort with the Class D hearing aids, which had the *highest* undistorted output.

A Type II hearing loss is shown in the upper right panel of Figure 8–15, where the loudness growth for a Type II loss of 60 dB is illustrated. A loss of 60 dB is probably too great to explain solely on the basis of a loss of outer hair cell function, and requires that we assume some inner hair cell loss as well. With a Type II loss, we thus have not only a loss of sensitivity for quiet sounds, but also a loss of some speech cues as well. A loss of inner hair cells means there is less information available to be transmitted to the brain, even for intense sounds. So not only is more gain required for low-level sounds with a Type II loss, but some gain will be required to restore even *loud* sounds to normal loudness.

The loss of inner hair cells means, however, that even with the 8 or 10 dB of gain required to make intense sounds normally loud (see Figures 8–14 & 8–15), there will usually still be a deficit in intelligibility for speech, especially in noise; fewer of the redundant speech cues will be available to the brain's processing centers. It is the availability of redundant speech cues that gives persons with normal hearing their remarkable resistance to interference from noise (Villchur, 1993). Persons with normal hearing can listen simultaneously to four talkers, for example, and selectively choose which conversation to follow. Individuals with a Type II (or worse) loss typically have a reduced ability to do that even with the best of hearing aid fittings (although they can do dramatically better than with the traditional hearing aid fittings of yesterday).

A Type III loss is shown in the lower panels of Figure 8–15, where both the loudness growth and the *intelligibility* function for a Type III loss of perhaps 75 dB are shown. When the hearing loss has progressed into the 70 to 80 dB region, loudness ceases to be a primary concern; the inner hair cell loss (and the resultant loss of normally redundant speech cues) is so great that one concern dominates: intelligibility. Moreover, the range of input SPLs over which speech is intelligible—especially when noise is present—is often very narrow. We no longer have the luxury of a large range of levels over which speech can be understood in noise.

Data for one subject with Type III hearing loss are shown in Figure 8–16. This subject was the first of eight Type-III-loss subjects studied by Fikret-Pasa (1993). This subject obtained a word recognition score of 60% in four-talker babble noise when the test was presented at a level just below discomfort. With a 5 dB reduction in level, the score dropped 10%; with a 10 dB reduction the intelligibility had dropped to half. The useful dynamic range of this subject in difficult listening situations is 5–10 dB on the HL dial.

Hearing-Aid Input-Output Desiderata

It is clear that a Type III hearing loss requires a totally different hearing aid than the first two. In a difficult listening situation, the only chance for someone with a Type III hearing loss to understand speech is to have everything presented at close to discomfort level.[4] At a party, for example, such an individual needs a hearing aid that allows sufficient gain to

[4]I am indebted to Harry Teder, one of Fikret-Pasa's subjects, for first bringing this phenomenon to my understanding. He observed that an experimental Adaptive-Compression® limiting hearing aid, similar to the one he normally used but with a 5 dB reduction in output, was inadequate for his loss. With that reduced output, he found himself handicapped at a party, migrating toward the quieter fringes because of greatly increased difficulty in carrying on a conversation in noise. Type III loss might well be called "Teder loss," since he first clearly understood and described the phenomenon.

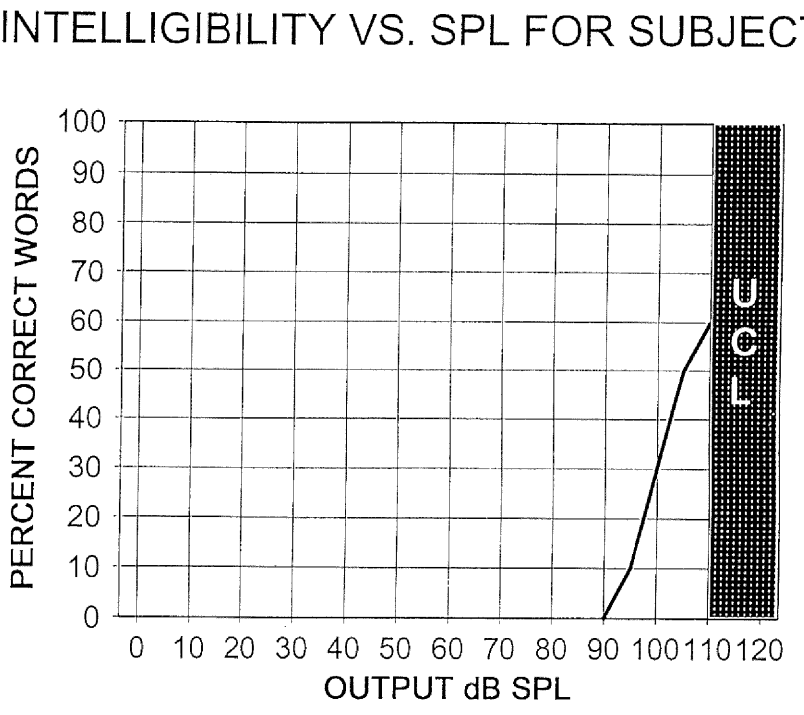

Figure 8–16. Measured word recognition ability of a subject with Type III hearing loss (Subject 1 from 1989 Fikret-Pasa study).

bring all of the ebb and flow of conversation to *beyond* discomfort, accompanied by appropriate compression limiting to keep all sounds just *below* discomfort. With this approach, all sounds will automatically be brought to the level of maximum intelligibility below discomfort. The only alternatives for an individual with a Type III loss are repeated volume control adjustment, regular discomfort, or loss of communication.

With Type I and Type II losses, on the other hand, restoration of normal loudness experience is a reasonable first goal. It is clearly the sensible goal in the case of a Type I loss. The argument goes as follows: Given (a) the need for sufficient gain to make quiet sounds audible and (b) the lack of need for any gain for high-level sounds, what is the most logical choice of gain for levels in-between?

The question can be answered by a reference to music. Assuming that the enjoyment of music is nearly universally desired, the existence of a loudness scale that has been in use in musical scores for more than 200 years would suggest that restoration of normal loudness experience

would be a desirable thing in and of itself. The musical loudness scale ranges from ppp (extremely soft) through pp, p, mp, mf, f, and ff to fff (extremely loud),[5] not unlike recent 7-point loudness scales recommended by Pascoe (1988), Hawkins, Walden, Montgomery, and Prosek (1987), and the others.

What should we do about discomfort? The 102–106 dB SPL fff produced by the Chicago Symphony Orchestra at a 1st balcony seat would be uncomfortably loud for those with normal hearing if it lasted for more than a few seconds at a time. This observation suggests that normal musical enjoyment for individuals with Type I hearing loss would probably *not* be enhanced by arbitrary output limiting circuits to prevent discomfort, especially of the peak-detecting variety. Such circuits would detract from musical enjoyment by limiting the excitement of a great performance. (That has been my personal hearing-aid listening-tes perience.) As argued below, such individuals don't need such protection unless they are forced to endure gain for loud sounds.

The argument for loudness restoration in the case of a Type II loss is not quite so self evident, although it appears to be the most sensible answer to the question "What should the hearing aid do at levels between quiet—where some 40 dB of gain is required—and intense—where only 8–10 dB is required to restore loudness?" Certainly *failing* to attempt some loudness restoration leads back to traditional linear hearing aids and the frequent volume-control adjustments they require. ("I have to adjust my volume controls *all* the time during a church service," complained one wearer of good Class D linear hearing aids.)

If we assume for the moment that restoration of normal loudness is the first order of business, then it readily follows from the curves in Figures 8–13 to 8–16 that the gain of the hearing aid should decrease gradually as the input level increases. This is exactly what wide dynamic range compression accomplishes, and it is the *only* compression or automatic-gain-control (AGC) system that does so.

Again note that the goal suitable for Type I and Type II loss—loudness restoration—is in sharp contrast to the goal for Type III loss. What is needed with Type III loss is a high-gain linear hearing aid with output *limiting*, preferably using variable-release-time compression, with the

[5]I have recently seen a marking of ffff for the final chord of a church-choir anthem—the composer presumably trying to exhort the choristers to some kind of superhuman effort—but a 7- or 8-point loudness scale appears adequate for most musical and clinical purposes. Some forms of rock music provide a possible exception. A VU meter hardly wiggles while monitoring some rock-music broadcasts, indicating a 2-point loudness scale might be adequate.

limiting set just below discomfort.[6] With the volume control turned up, everything is brought *near* discomfort to maximize intelligibility, but *below* discomfort to avoid discomfort.

Figure 8–17 shows that available wide-dynamic-range-compression (WDRC) hearing aid circuits can provide nearly perfect loudness compensation for a typical Type I hearing loss. Note that no gain *or loss* is provided for high-level inputs, consistent with the normal or near-normal high-level hearing with Type I losses.

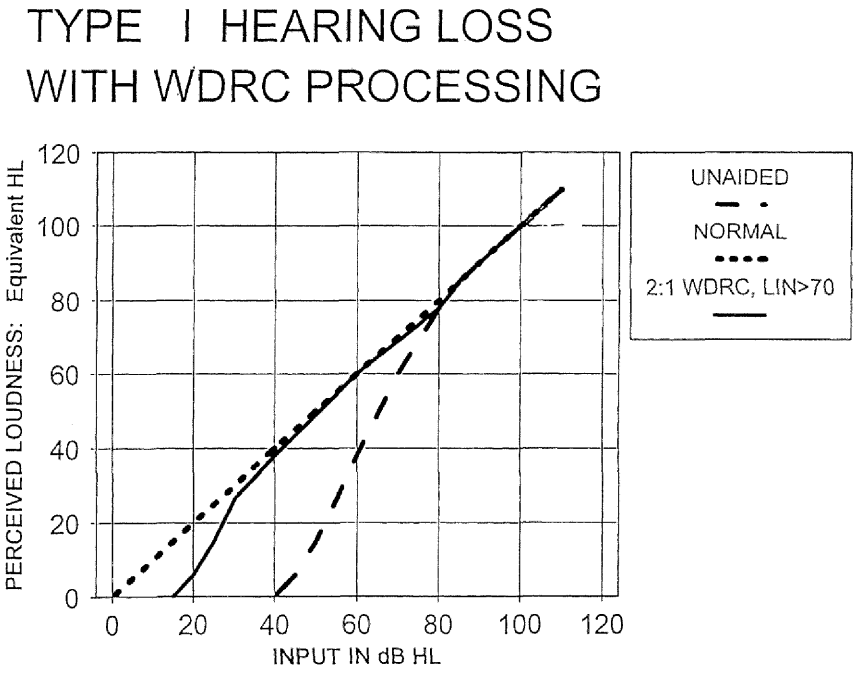

Figure 8–17. Restoration of loudness for a 40 dB Type I hearing loss using a commercial wide-dynamic-range-compression hearing aid amplifier. Note: The full fortississimo (fff) output of the Chicago Symphony Orchestra does not cause clipping.

[6]The data of Fikret-Pasa (1993) indicate variable-release-time compression limiting can provide some individuals with Type III hearing loss an improvement of 5 dB or more in effective signal-to-noise ratio, compared to conventional compression limiting with 50 mS recovery time, when both are driven deep into limiting.

Physiological Data and WDRC Hearing Aids

Dr. Brownell was kind enough to lend me one of the slides he used during his talk, a slide of the Ruggero and Rich (1990) data relating measured basilar-membrane velocity to input SPL. That slide is shown here replotted as curve B.M.#L14 in Figure 8–18. As described earlier, a large gain is seen for low-level inputs, a gain which disappears for high-level inputs. When the outer hair cells are paralyzed with furosemide so they can't move, the gain disappears at all levels, giving evidence that the gain is provided by the action of the outer hair cells themselves.

Since the time of my verbal presentation, I have been unable to resist generating the comparison shown in Table 8–2 between the action of the outer hair cell amplifiers in the cochlea, using the B.M.#13 curve in Figure 8–18, and the action of a popular hearing aid amplifier which uses wide-dynamic-range compression (WDRC). The compression ratio for the ear was derived by first converting velocity to dB. The compression ratio was then calculated as (dB change in input)/(dB change in output). Note: In Figure 3 of the same paper, Ruggero & Rich (1991) reported what they

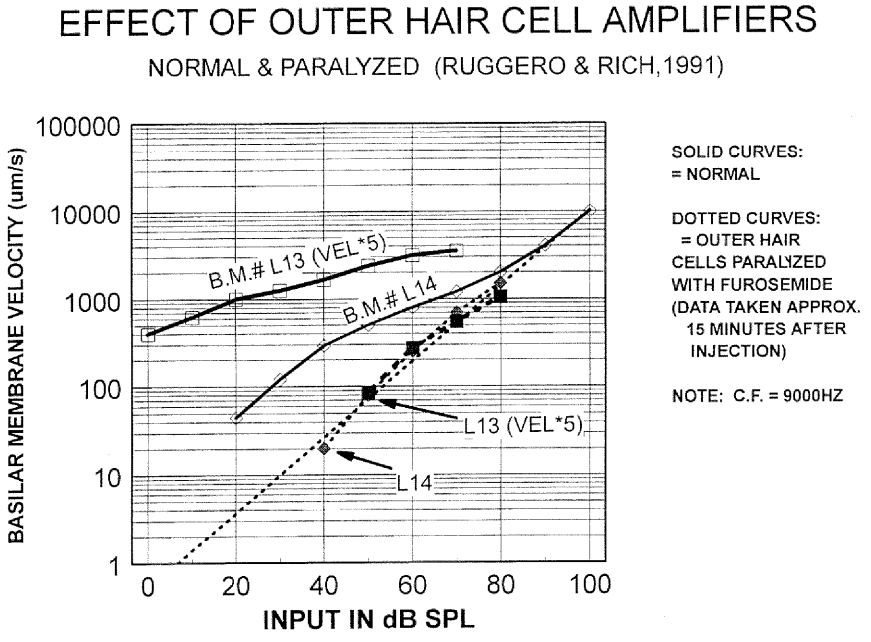

Figure 8–18. Ruggero and Rich (1991) data on the operation of Corti's organ as a wide-dynamic-range-compression amplifier.

TABLE 8–2.

AMPLIFIER TYPE:	Outer Hair Cell Amplifier (Sample #14, 9 kHz C.F.)	Wide-Dynamic-Range Compression Amplifier (4 kHz test frequency)
LOWER THRESHOLD OF COMPRESSION	35 dB	40 dB
UPPER THRESHOLD OF COMPRESSION	85 dB	90 dB
COMPRESSION RATIO	2.3:1	2.1:1
GAIN INCREASE FOR QUIET SOUNDS	20-50 dB	25 dB
POWER CONSUMPTION	50 uWatts	230 uWatts

consider to be a better measurement on another animal, data that can be extrapolated to indicate 50 dB of gain at 10 dB SPL, and little or no gain at 90 dB SPL. Those data (B.M.#L13) have been normalized and added to Figure 8–18.

It is clear from Table 8–2 that nature provides a substantial battery economy: Some 15,000 outer hair cell amplifiers operate on less than 50 uWatts total;[7] a single WDRC hearing aid amplifier requires 230 uWatts.

It is also clear from Figure 8–18 that nature does not normally provide us with linear hearing, but hearing which provides increased sensitivity for quiet sounds and a gradual reduction of sensitivity with increasing level. Thus, nature normally provides wide dynamic range compression to increase gain for low-level sounds! The only time the human ear shows a linear input-output function is when the outer hair cells are missing or temporarily paralyzed. Either causes a 40–50 dB hearing loss. Linear amplification is "pathological."

[7]The power consumption of the cochlea was estimated two ways. First, the vascularization of the cochlea was extracted as a list of diameters by microscopic examination of a temporal bone slide. The sum of the inverse fourth power of those diameters gave a total relative flow, which was compared to the inverse fourth power of the diameter (1 cm) of the aorta. This gave a presumed power ratio of 70:10^9. Considering the short-term 1 horsepower (745 watt) maximum output of a human, this implies approximately a 50 uWatt supply to the cochlea. A similar figure was derived from the measured resistivity/mm of the cochlear fluid and the 80 mV battery supported by the stria vascularis. The two estimates were within a factor of two of each other, indicating that 50 uWatts is probably a good figure.

Wide Dynamic Range Compression (WDRC) Amplifiers

Figure 8–19 shows a presumed ideal input-output characteristic suitable for a hearing aid, inferred 16 years ago from available loudness-growth data for sensorineural hearing loss (Killion, 1979). This graph shows substantial gain for quiet sounds, gradually decreasing as input SPL increases, until no gain (or loss) is provided for loud sounds. A similar ideal characteristic was suggested by Barford (1978). The striking similarity to the physiological input-output curve shown in Figure 8–18, added to this figure as the dotted curve, is evident. The input-output function of the commercial WDRC amplifier looks very much like the normal nonlinear input-output function of the cochlea.

It might seem surprising that an electronic amplifier—designed in the 1970s before the recent physiological data became available—should match the ear's physiological amplifier so well. Recall, however, that psychoacoustic data on the input-output characteristic of normal-hearing and impaired ears have been available since the 1930s. The physiological data have simply supplied a reassuring explanation for what we already believed to be true.

Indeed, Steinberg and Gardner (1937) first concluded from loudness data on impaired ears that what we now call wide-dynamic-range com-

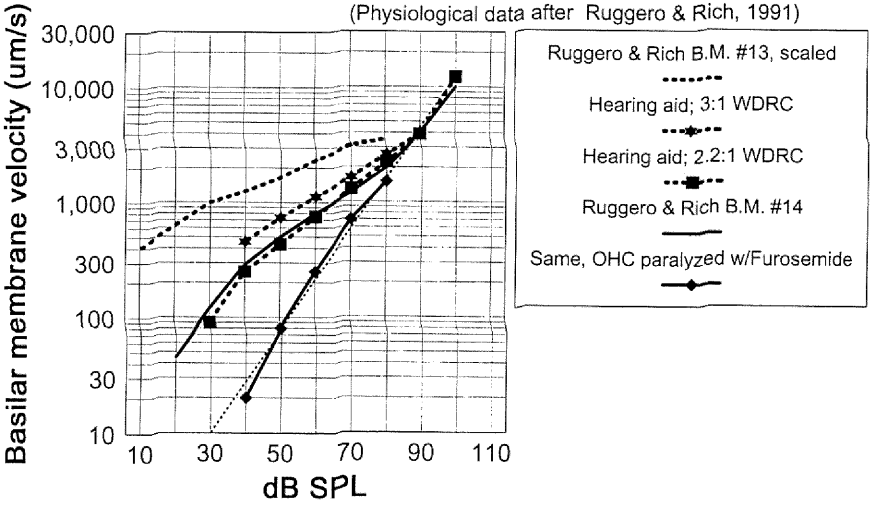

Figure 8–19. Presumed ideal characteristic for hearing aid amplifier (Killion, 1978), compared to physiological input-output function measured by Ruggero and Rich (1991).

pression[8] was required in a hearing aid amplifier: "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" (p. 20). Ironically, it took nearly 30 years before Goldberg (1966) introduced wearable low-distortion WDRC hearing aid amplifiers into the marketplace. I can recall listening to one of his BTE "Computer" hearing aids in 1968, and marveling that his factory-programmable-compression-ratio amplifier was not used in every hearing aid. (Goldberg's amplifiers still sound good.)

Only in this decade, however, have WDRC amplifiers become widely accepted; over 50 years after Steinberg and Gardner's observations. Today several commercial hearing aid amplifiers in addition to Goldberg's can produce excellent compensation for the pathological loudness growth of sensorineural loss. The hearing aid design based on Villchur's research (1973) allows choice of compression ratio (Waldhauer & Villchur, 1988); another design (Killion, 1979) has trimpot control (soon to be computer-programmable control) of compression ratio.

Figure 8–20 shows that not all "compression" hearing aids can be set to compensate for the impaired loudness function. The irony is that the one which fails most visibly has often been described in lectures as having the capability of being programmable to be "just like" a popular WDRC amplifier with treble-increases-at-low-level (TILL) operation. The thin solid curve shown in Figure 8–20 was plotted from the manufacturer's published data. This circuit is a substantial improvement over linear hearing aids, and does a good job of loudness compensation up to about 70-dB SPL input, but the 40 dB range of normally loud and very loud sounds is suppressed. This aid is *not* just like a good WDRC-TILL hearing aid, which preserves normal loudness experience for *all* input levels! For all practical purposes, the aid programmed as shown in Figure 8–20 operates as a compression limiter above 70 dB. This should be a good aid for a Type III hearing loss, where its programmable limiting should be useful, but hardly ideal for Type I or Type II losses.

This argument deserves more discussion. The region between 70 and 110 dB is where most of the excitement in music resides. When the members of the orchestra are performing at physiological limits in order to produce the excitement of a Mahler finale, it will come out of the thin-solid-curve aid sounding a little like a tiny tinkle. The last 40 dB of excitement has been thrown away. It is analogous to saying "I want no more

[8]Wide-dynamic-range compression was what was meant by the word "compression" at Bell Labs in those days; what we now call compression limiting was simply called limiting. Limiting due to peak clipping was called peak clipping.

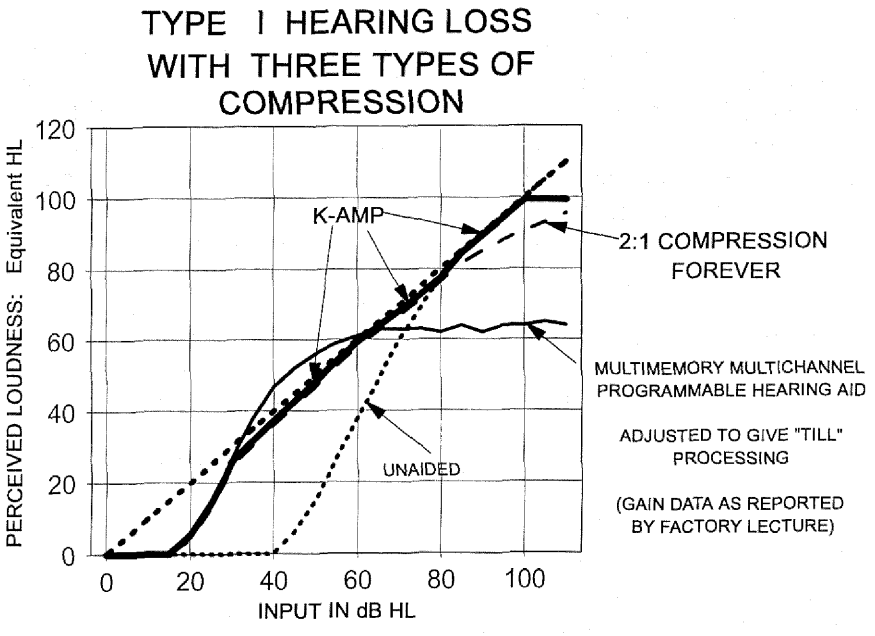

Figure 8–20. Amplified loudness growth for several compression hearing aids.

than a 150 watt light bulb to illuminate my life. I am going to accept the range between moonlight and 150 watts, nothing above that. Never do I wish to see bright sunshine again." Even worse from the standpoint of naturalness would be the use of an AGC system that forced all sounds to the *same* loudness (the most comfortable loudness level has been suggested). That would be equivalent to eliminating the brightness both from 150 watts up to sunlight *and* from 150 watts down to moonlight.

The auditory equivalent of bright sunshine *can* be obtained with an output-limiting aid by turning up the volume control, but then most *everyday* sounds will be too loud, as illustrated in Figure 8–21, and a 40-dB wide range of sounds will be brought to just short of discomfort. Moreover, visual and auditory discomfort is a time-dependent phenomenon. I find that driving down the road on a sunny day is no problem for short periods of time, but after 5–10 minutes my eyes start to ache. Similarly, the 100–105 dB levels at a country and western dance are OK for a few minutes, but after that I get out my Musician's Earplugs. Levels that provide an exciting few seconds of finale to an orchestral performance become uncomfortable when prolonged into minutes. This means that sunglasses sufficiently dark for hours of light exposure, or output-limit-

ing levels sufficiently low for hours of high-level sound exposure, may provide excessive attenuation for transient conditions.

Output limiting for Type I and Type II loss is so obviously wrong that we must ask ourselves how we arrived at the use of limiting (compression or peak clipping) for Type I and Type II losses in the first place. I believe it came about because the only amplification available over the years was linear amplification. With only linear amplification available, some form of limiting is absolutely necessary because unlimited amplification of high-level real-world sounds by the amount required for quiet sounds would cause pain or physiological damage. Such linear amplifiers inherently included limiting by peak clipping, which, chosen appropriately, would prevent discomfort. Low-distortion compression limiting was such an obvious and dramatic improvement over limiting by clipping (see, e.g., Hawkins & Naidoo, 1993, or Fikret-Pasa, 1993) that we

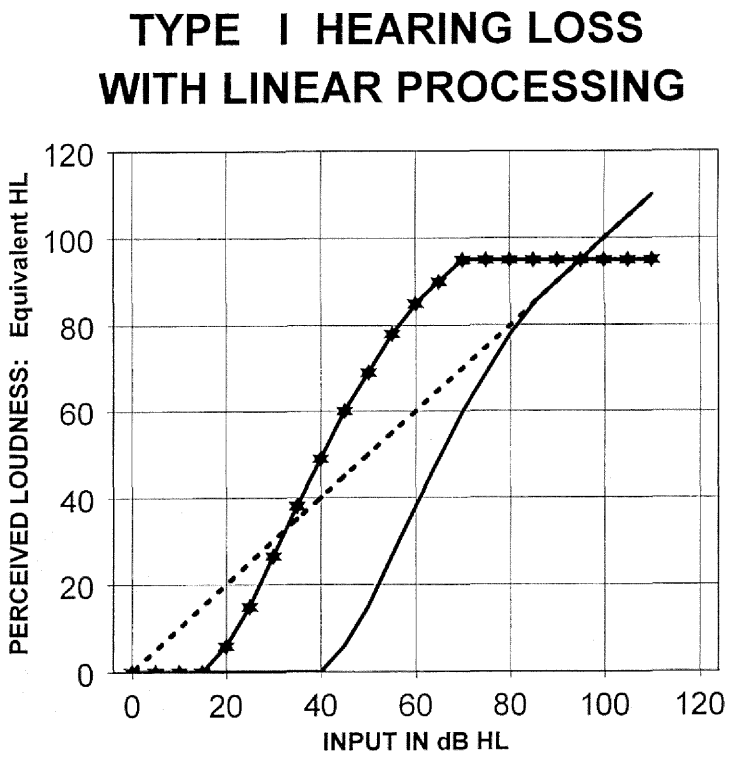

Figure 8–21. Linear amplification with limiting.

often ignored the fact that the typical user of a linear hearing aid would either have to set the gain too low for proper hearing of quiet sounds or else experience excessive loudness over a wide range of sounds in the real world, as illustrated in Figure 8–21.

Figure 8–22 is an attempt to dramatize the fact that limiting is a good solution to a problem that should not be present in the first place. Linear hearing aids unavoidably create a monster, shown here figuratively as the Excessive-Loudness Lion. (The lion was the only animal in the clip art catalog that looked like a monster.) Output limiting is required to tame him, but why did we create him in the first place? The *only* reason for output limiting in hearing aids for Type I loss is because we have *created* this excessive-loudness monster by using *linear* amplifiers that don't have enough intelligence to gradually turn the gain down. Our great prowess in taming him conceals our shame in his creation.

The UCL data of Pascoe argue that even for Type II loss, where some gain is needed for loud sounds, limiting is not required if the proper high-level gain is chosen (Killion & Fikret-Pasa, 1993). Given that most individuals have Type I or Type II hearing loss, we can safely conclude that *output limiting is the wrong approach most of the time.*

Should the Frequency Response Stay Fixed?

Use of fixed-gain linear amplifiers was one error in traditional hearing aid design; use of a fixed frequency response appears to have been anoth-

Figure 8–22. Linear Loudness Lion.

er. Most hearing loss is frequency dependent, that is, is greater at some frequencies than others. A 40 dB loss at 1 kHz will more often than not be accompanied by a much greater loss at 4 kHz. Skinner (1976) studied the effect of hearing aid frequency response in her Ph.D. research, using the set of 5 frequency responses shown in Figure 8–23 with subjects having ski-slope hearing loss.

In Figure 8–24, two of Skinner's five frequency responses are shown. The frequency response having the most high-frequency boost (40 dB)

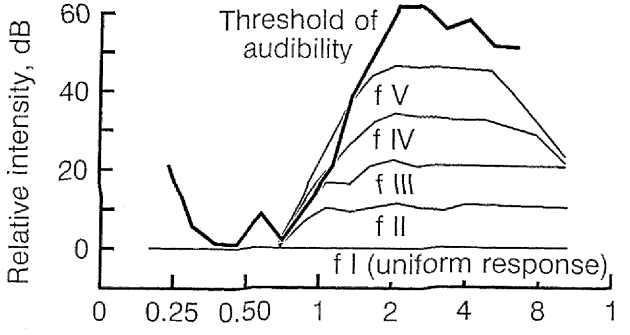

Figure 8–23. Hearing aid frequency responses studied by Skinner using subjects with ski-slope high-frequency losses. (One subject's threshold is shown in dB SPL.)

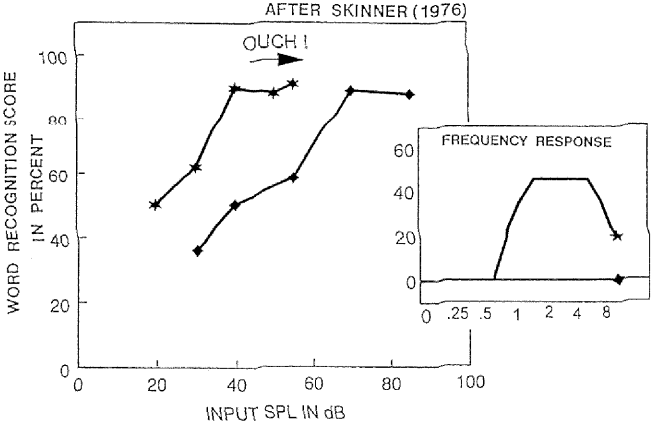

Figure 8–24. Two of Skinner's frequency responses and corresponding intelligibility curves for one of her subjects. (The "Ouch!" is visual shorthand for "exceeds discomfort level").

gave her subjects the best word recognition score for quiet sounds. This should not be surprising, given their high-frequency loss. Although this response gave the best score for low-level inputs, it became intolerable by the time an input of 55 dB SPL was reached (10 dB below conversational speech). At that point, the *output* had reached discomfort. The flat frequency response, on the other hand, gave poor word-recognition scores at low levels, but at high levels it gave scores nearly as good as the best high-frequency-emphasis scores and did not cause discomfort. Intermediate responses (not shown in Figure 8–24) sometimes gave slightly higher maximum scores.

Skinner herself stated the obvious conclusion: It would be a good thing if the frequency response of hearing aids was level dependent. A high-frequency emphasis for quiet sounds and a relatively flat response for loud sounds would represent much better processing than fixed-response linear amplification for most subjects.

Figure 8–25 shows the level-dependent frequency response designed into one single-channel WDRC-TILL amplifier. The acronym TILL was assigned by Killion, Staab, and Preves (1990) to describe hearing aid signal processing which provides "Treble Increases at Low Levels." A commercial two-channel programmable WDRC amplifier can be programmed to provide even greater changes in frequency response with level, changes of the TILL type, suitable for high-frequency hearing loss,

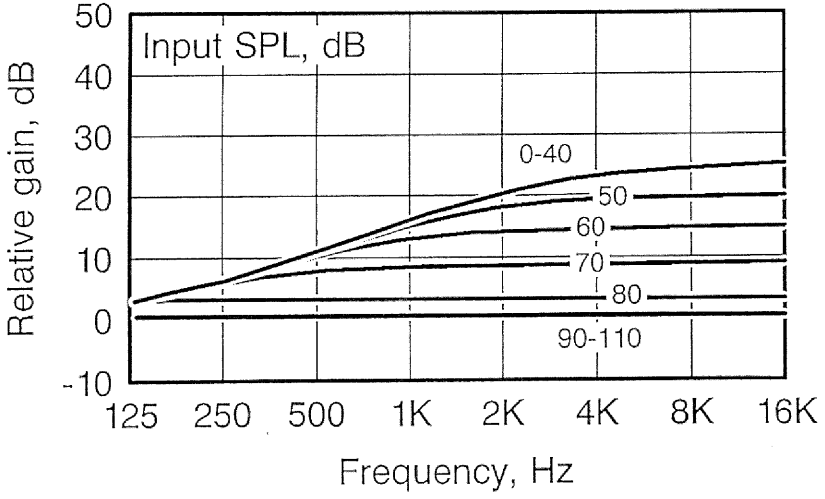

Figure 8–25. Level-dependent frequency response characteristics of commercial WDRC-TILL amplifier.

or of the BILL type (Bass Increases at Low Levels), suitable for reverse-slope loss or for rejecting high-level low-frequency noise.

Leijon (1989) reached a similar conclusion to that of Skinner. Leijon took the Zwicker loudness model, modified it for the widened critical bands typical of moderate-to-severe hearing loss, and calculated the frequency response required of the hearing aid in order to compensate for abnormal loudness perception. Figure 8–26 shows a hearing loss he used as an example, and Figure 8–27 shows the calculated TILL frequency-response set required to compensate for the abnormal loudness corresponding to that loss. Note that we would label the audiogram in Figure 8–26 as having "Type I loss" at low frequencies, but as having "Type II" loss at high frequencies. At high frequencies, therefore, we would expect some loudness loss even for loud sounds. Leijon's calculated required-gain curves shown in Figure 8–27 do indeed show some gain for loud sounds at high frequencies.

Most available WDRC-TILL hearing aid amplifiers can be adjusted to accommodate a Type I loss at low frequencies and a Type II loss at high fre-

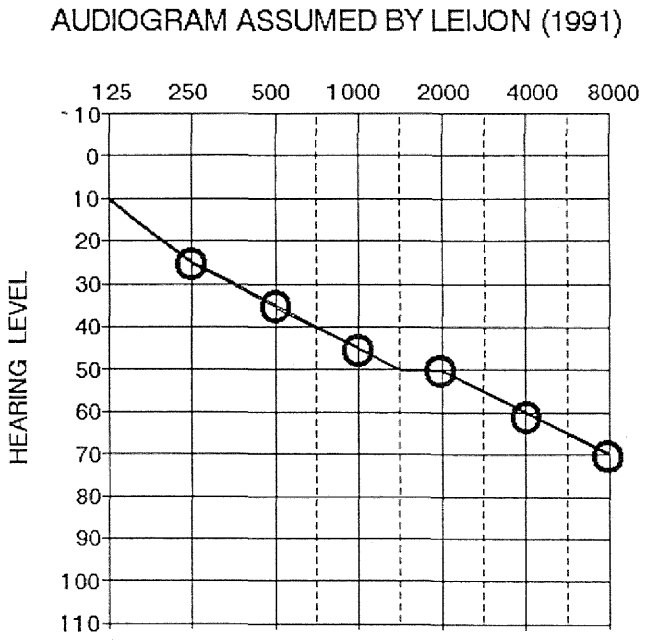

Figure 8–26. Audiogram assumed by Leijon (1991) for Figure 8–27 results.

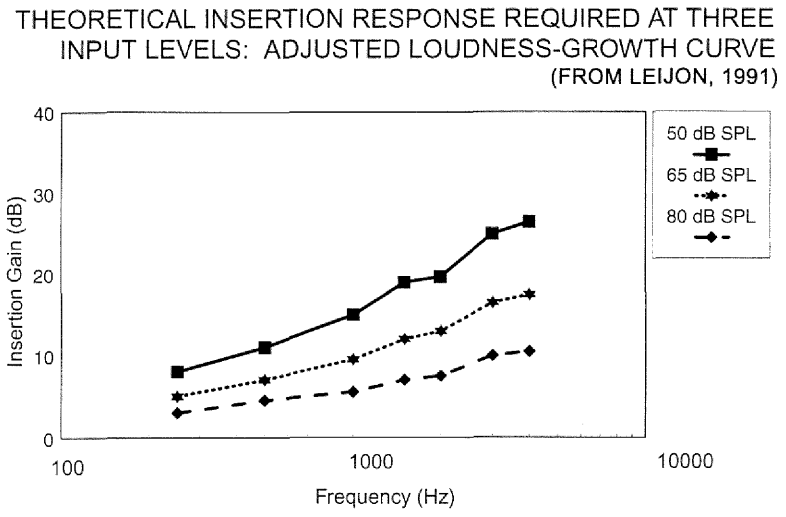

Figure 8–27. Gain-frequency response calculated by Leijon as required to restore normal loudness for hearing loss with Figure 8–26 audiogram.

quencies. Figure 8–28 shows a set of estimated real-ear curves for one such hearing aid, adjusted for someone whose loss progresses from minimal at very low frequencies, through Type I at mid frequencies, to Type II at high frequencies. The curves show 20 dB of gain for loud sounds and 45 dB of gain for quiet sounds at high frequencies, with little or no gain at low frequencies.

Much has been said about the advantages or disadvantages of loudness-based targets. It is certainly true that loudness is not the only important auditory dimension. I believe it is also true, however, that loudness provides the clearest noninvasive picture of the operation and residual capability of the impaired cochlea. Thus, although loudness may not be the final consideration, it appears to many of us to be the best place to start.

Fitting Formulae: FIG6

The need for a systematic approach to fitting the speech sounds into the auditory area has been obvious for years. Fitting formulae of Lybarger, Wallenfels, Burger, Byrne and Dillon (NAL-R), Lyregaard and McCandless (POGO), Cox, Libby, and Seewald (DSL) are currently in use. Several of these are illustrated in Figure 8–29 for a case of high-frequency hearing loss. The pluses represent the NAL-R curve (Byrne & Dillon, 1987), which has received the greatest number of validation studies and is probably the most popular of the formulae suitable for linear hearing aids. A modi-

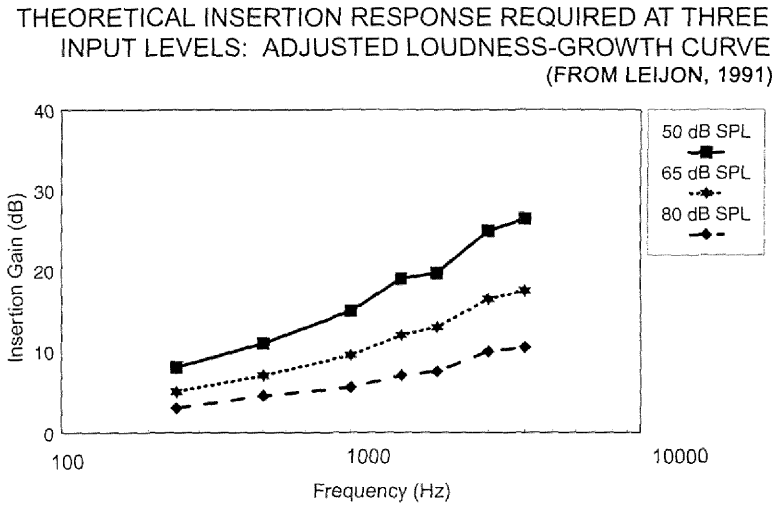

Figure 8–28. Hearing aid showing some gain for high-level sounds at high frequencies, with little gain for high-level sounds at low frequencies, while maintaining basic TILL increase for quiet high-frequency sounds.

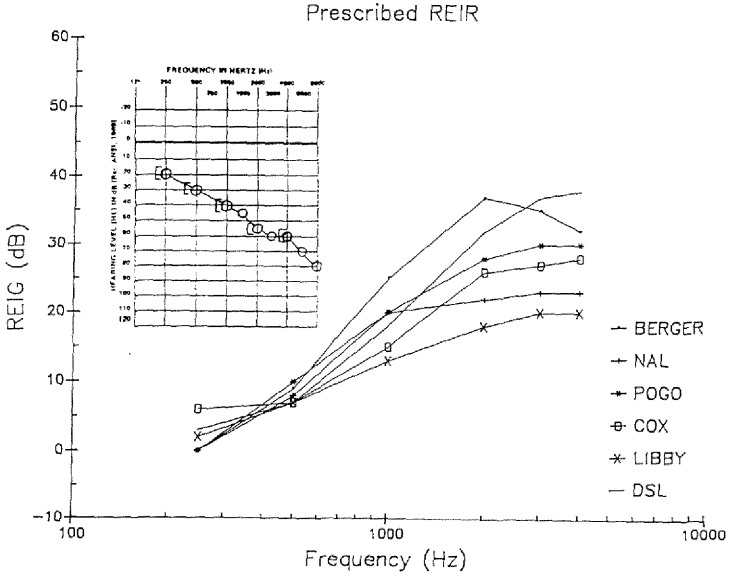

Figure 8–29. Target gain for fixed-gain, fixed-frequency-response hearing aids according to several popular formulae (from Hawkins, Mueller, & Northern, 1992).

fied form of NAL is used by many hearing aid manufacturers when audiologists send in only the impression and an audiogram. I understand that this implicit "you figure it out" request still accompanies the majority of the orders manufacturers receive.

NAL and other targets have served us well, but we need more sophisticated target curves if we wish to move beyond linear, fixed-gain, fixed-response amplification. First and most obvious, one gain and frequency-response target isn't enough if different input levels require different frequency responses.

Various loudness-based fitting methods have been introduced in recent years to provide more sophosticated targets: ELCVIL8, DSL-I/O, P3, and VIOLA. Some use individually-measured loudness data (VIOLA and P3-LGOB), others use audiogram information only, predicting the individual loudness growth from average data (DSL-I/O & P3-Aud+), and some use MCL plus audiometric data to better predict the individual loudness growth from average data (ELCVIL8). The oldest of these appears to be the ELCVIL approach, which was based on Villchur's 1973 and 1987 fitting suggestions.

FIG6, the triple-target method that will be discussed in detail, falls in the audiogram-only class. FIG6 estimates pathologic loudness growth data from published data on loudness growth versus hearing loss. FIG6 gives a set of three fitting targets; for quiet or low-level sounds, conversational sounds, and loud or high-level sounds. The derivation of the loudness-based FIG6 is described in what follows.

The basis for estimating required gain-to-restore-loudness as a function of hearing loss are the data in Figure 8–30, a reproduction of Figure 6 from Killion & Fikret-Pasa (1993). This was an attempt to estimate the hearing aid gain required for different amounts of hearing loss (assuming the loss fell in the Type I and Type II categories):

1. The gain required to restore audibility for low-level (40 dB SPL) sounds;
2. The gain suggested by loudness data for restoring loudness at conversational speech levels (65–70 dB SPL); and
3. The gain required to restore full loudness to intense sounds (85–105 dB SPL). Note: For speech in the sound field, 40, 65, and 95 dB SPL correspond to about 25, 50, and 80 dB HL, respectively.[9]

[9]The relationship between 0^0-incident speech and SPL is 15 dB, based on the references given in ANSI standard S3.6-1989, resulting in the 50 dB HL equivalence for 65 dB SPL face-to-face speech. In a Northwestern University classroom experiment last year, pairs of students (ages ranging from their 20's to 40's) and their instructor carried on more or less normal two-way conversations at a comfortable conversational distance, with a sound level meter held near the ear of the listener in each case. The range of frequent peak readings was 58 dB to 70 dB, with an average of 64.8 dB. This supports 65 dB SPL (50 HL) as a reasonable choice for the level of typical conversational speech.

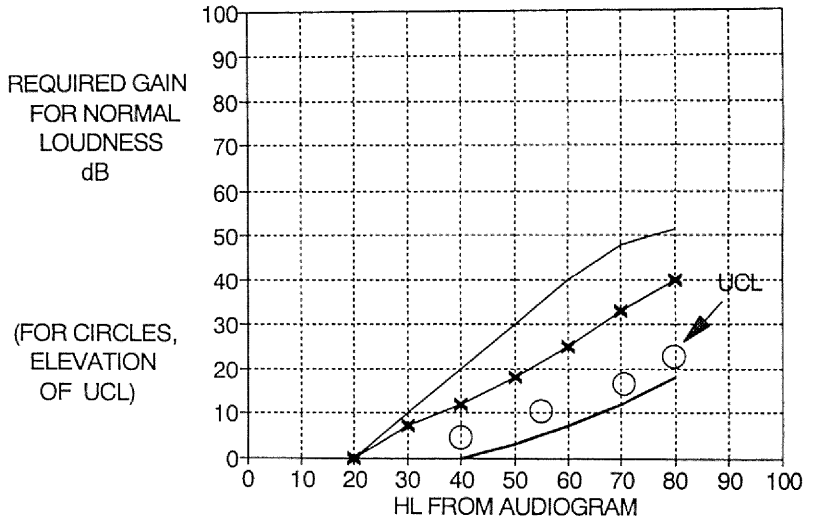

Figure 8–30. Gain required to restore loudness for low-level (——), comfortable-level (X——X), and high-level (——) sounds. UCL data (o) from Pascoe (1988) indicate that the required high-level gain is safe. (Reproduced from Killion & Fikret-Pasa, 1993).

Figure 8–31 shows an example application of the FIG6 procedure. The determination of the three target curves of FIG6 turned out to be fairly straightforward, as explained in the following paragraphs.

1. For quiet sounds, the primary consideration is that of audibility. The hearing aid must provide adequate gain to make quiet sounds aud ble. At first thought, restoring aided sound-field thresholds to 0 dB H seems like a good idea in order to restore "normal sensitivity." (Audiologists once thought that way. I can recall Darrell Teter—who was trained at the same university that I was—saying "In those days, baby, if you had a 60 dB loss, I gave you 60 dB of gain!") As discussed by Killion and Studebaker (1978), however, room noise in most locations limits even those with normal hearing to approximately 20 dB masked thresholds.[10] So giving hearing aid wearers sufficient gain to bring their aided sound-field thresholds in a test booth below 20 dB HL will, in most cases, be giv-

[10]The Killion and Studebaker paper contains a rule of thumb useful in many situations: Subtract 20 dB from the A-weighted sound level meter reading in a noisy environment and you have a good estimate of the masked sound-field thresholds you could obtain in that environment. Driving 70 mph in many cars produces a 70 dB(A) SPL noise level, for example, so the occupants experience a good simulation of a 50 dB hearing loss. With the most common noise spectra, the masked threshold will be approximately flat from 500 to 4000 Hz.

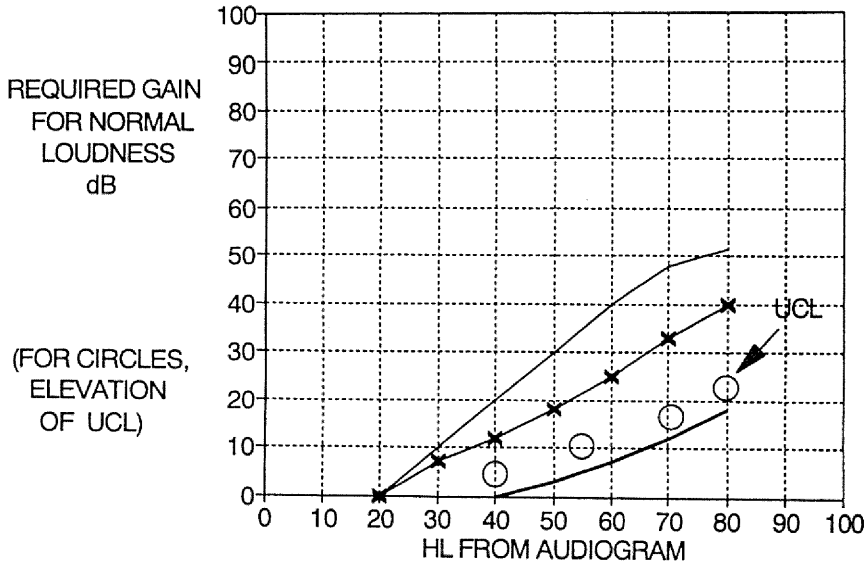

Figure 8–31. FIG6 target gain curves for WDRC hearing aids with level-dependent frequency response. The target curves (and the NAL-R curve) correspond to the audiogram at the top of the figure.

ing them empty (and probably annoying) gain. On the other hand, the quietest elements of conversational speech fall at about 20 dB HL, so *less* gain would leave some conversational speech cues inaudible. The formula for low-level gain thus seemed obvious: G = HL – 20. This is the gain required to produce 20 dB HL sound-field thresholds. No gain is required until the hearing loss exceeds 20 dB. (For losses above 60 dB, some modification is required for practical reasons because of feedback difficulties. A half-gain rule was thus adopted above 60 dB: A gain of 40 dB for a 60 dB loss [60 – 20 = 40], but only 45 dB of gain for a 70 dB loss, 50 dB for 80 dB, etc.). The low-level target shown in Figure 8–31 is labeled "40 dB" meaning 40 dB SPL input.

2. For conversational speech, Pascoe's (1988) data on Most Comfortable Loudness (MCL) as a function of hearing loss were used to estimate required gain. For example, a hearing loss of 60 dB is typically accompanied by an MCL elevation of 24 dB, so 24 dB of gain would be required to restore MCL. The target curve resulting from this application of Pascoe's

data, corresponding to normal 50 dB HL conversational speech,[11] has been labeled "65 dB."

3. For high-level sounds, the required gain was estimated from the loudness-growth data of Lyregaard (1988) and Lippman et al (1977). Their loudness-growth curves for individuals with 40 dB hearing loss became asymptotic to the normal loudness-growth curve at high levels. For individuals with significantly greater hearing loss, the loudness-growth curves also became asymptotic, but not to the normal loudness-growth curve. The curve for a 60-dB loss, for example, appears to approach an imaginary line shifted 8 dB to the right of normal. In their original Figure 6 therefore, Killion & Fikret-Pasa indicate that 8 dB of high-level gain is required for a 60 dB loss, whereas 0 dB of high-level gain is required for a 40 dB loss. Other high-level gain data were estimated in a similar way. The high-level target has been labeled "90 dB" rather than 95 dB because most probe-microphone and test-box equipment will only test up to 90-dB SPL.

The UCL elevation data of Pascoe (1988) were also added to the curves in Killion and Fikret-Pasa's Figure 6 (Figure 8–30 here), with a surprising result: On the average, at least, *the gain required for loud sounds is always less than the elevation in discomfort level.* For example, at 60-dB loss about 8 dB of gain is required for high-level sounds, *but the elevation in UCL is about 12 dB.* Let me say that differently, because it is important. Assume you have a typical 60-dB loss. Your *discomfort* level will be elevated by 12 dB, but you will need only 8 dB of gain in order to experience the same *loudness* for high-level sounds as a person with normal hearing. If you and I sit together at a Chicago hockey game and you wear a hearing aid with 8 dB of gain, by the time the sound level rises to your discomfort level, I will already be 4 dB past my normal-hearing discomfort level. When I first reach discomfort, you will still have a 4 dB margin left before you reach *your* discomfort level.

What the available data and the reasoning of the previous paragraph imply is that we really *have* been doing it wrong all these years. We have been using limiting to cover up our original sin in fitting linear amplifiers. Had we instead used amplifiers that restored loudness sensation to approximately normal, we would not have needed limiting except in

[11]Pascoe's data, which show a normal MCL of 60 dB HL, were obtained with pulsed tones. A reasonable single-number approximation to the minimum audible field data is 5 dB SPL (across the audiometric frequencies), so 65 dB SPL would be a reasonable approximation to 60 dB HL. The choice of a slightly different test level will not affect the results significantly with wide-dynamic-range compression hearing aids. (Choosing a 5 dB different level will affect the measured gain only 2.5 dB, for example, if a hearing aid provides 2:1 compression at that test frequency.)

those circumstances where someone with normal hearing would need hearing protection. For those cases, where hearing protection is required, the user of a WDRC aid can simply turn down the volume control.

Based on the deduced gain requirements of the previous figure—and the fact that these are largely independent of frequency as reported by Pascoe (1988) and Lyregaard (1988)—it became a simple matter to fit each of these curves with mathematical formulae. With formulae in hand, can a spreadsheet be far behind?

The FIG6.EXE Computer Spreadsheet

FIG6.EXE is a computer spreadsheet that automatically produces target curves once the audiogram data have been entered. It provides targets for three input levels: 40, 65, and 90 dB SPL. The target curves are plotted to the standard 50 dB/decade hearing-aid scale. This allows ready comparison to measured probe response curves taken at 40 (or 50), 65, and 90 dB SPL. (Hold the measured and calculated curves up to the light and slide them until the scales match; one or the other curve may need to be magnified on a copy machine so both are the same size.) Obtaining the real-ear 40-dB-input curve requires an unusually quiet room and special equipment as of this writing. It is easily checked, however; aided sound-field thresholds should be close to 20 dB HL. (The 40 dB target formula was chosen to produce exactly 20 dB HL aided thresholds.) The NAL-Revised target curve is also displayed by the FIG6 computer program for comparison. Although the NAL-R target is based on an empirical formula and not directly on loudness data, the revised-NAL curve usually lies close to the target curve for 65 dB SPL input except at low frequencies (where NAL always calls for a rolloff) and at 1 kHz (where NAL calls for more gain).

Two cc coupler targets for BTE, ITE, ITC, and CIC hearing aids are available in FIG6, which incorporates the CORFIG data of Killion and Revit (1993). These target response curves can be printed and sent in to the factory if desired.

FIG6.EXE and associated graphical printing software are contained on Band 8 of the enclosed CD. A PrntScrn program is included in the FIG6 disc, so graphs can be printed directly to a LaserJet or Dot matrix printer. Color graphs can be PrntScrn'd to an HP 320® or 500C or 550C Inkjet color printer. The same software package will be sent (on a 3 1/4" disk) gratis to anyone who sends in a self-addressed envelope to Etymotic Research.

Individual loudness growth

The FIG6 program uses average-loudness-based targets. Will these be valid for everybody, or will individual differences be too large to make

any average-based approach truly useful? The answer surely lies somewhere in between.

The longstanding success of average-based fitting formulae since Lybarger introduced his famous stored-program fitting computer (a circular slide rule) in 1955 would argue against discarding average-based approaches. On the other hand: (1) loudness tolerance can increase with accommodation to loud sounds, (2) acoustic trauma can produce hypersensitive ears, and (3) some people are simply different.

LDL Shift with Prolonged Exposure

Patients who typically produce aberrant loudness growth judgments include those who have learned to live comfortably with sounds most of us would judge to be uncomfortably loud. Individuals who have accommodated to powerful linear hearing aids will sometimes say WDRC aids are not loud enough.

Figure 8–32 shows interesting loudness accommodation data that came out of an experiment originally aimed at another question: do subjects with hearing impairments judge sound quality in the same way as people with normal hearing, assuming the sound is loud enough to be readily heard by all subjects? Palmer (1994, personal communication)

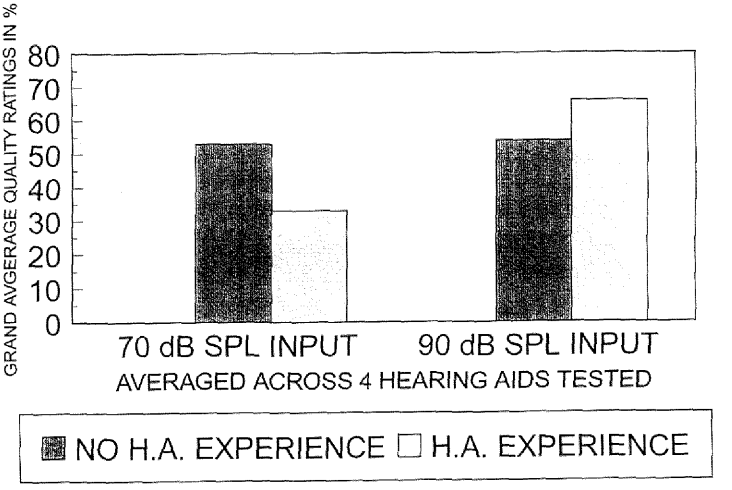

Figure 8–32. Quality judgments for two groups of subjects with hearing impairment. Those experienced in linear hearing aids and those with no hearing aid experience. Note that the experienced users liked it loud!

found an interesting confirmation of the old "Wear it awhile and you'll get used to it" advice. Subjects with hearing impairments who had not been wearing aids judged sound quality in about the same way as people with normal hearing, down-rating the distorted sound of starved Class A hearing aids with 90 dB inputs. Subjects with hearing impairments who had been wearing such hearing aids, on the other hand, didn't mind distortion nearly as much. What they *did* like was *loud*! As shown in Figure 8–32, subjects with no hearing aid experience judged the across-aids average quality for 70 dB and 90 dB inputs about the same. Subjects with hearing aid experience, on the other hand, judged the 70 dB and 90 dB inputs differently: Their across-aid average quality ratings were significantly higher for the louder presentation.

Figure 8–33 shows the shift in loudness function with prolonged exposure to intense industrial noise. These data were obtained roughly 25 years ago in Germany by Niemeyer (1971), who reported that in his measurements, normal-subject loudness discomfort level (LDL) averaged 10 dB above acoustic-reflex threshold (AR), and that the LDL-AR difference did not increase with hearing loss until a very large hearing loss was encountered. At the time, acoustic reflex and loudness discomfort levels

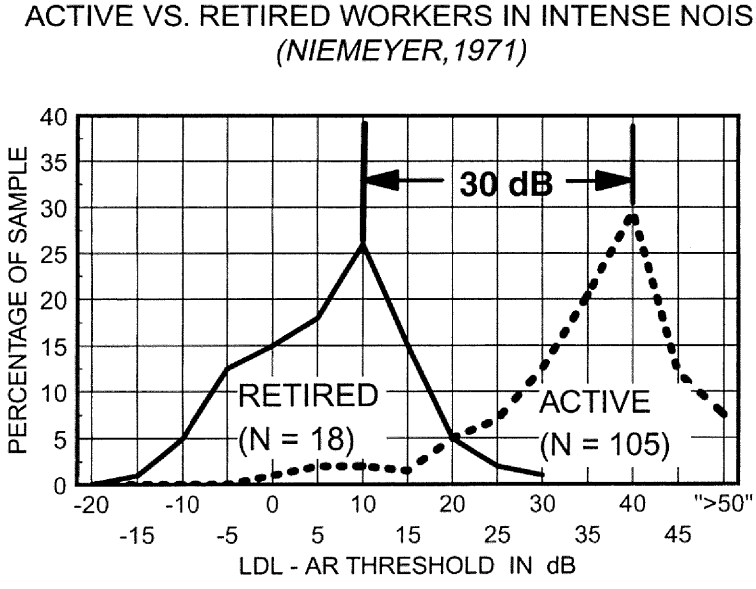

Figure 8–33. Loudness Discomfort Level Shift for workers exposed to intense noise (data of Niemeyer, 1971).

were popular things to compare. More importantly, it was a time when it was possible to find subjects who were still being subjected to intense industrial noise without hearing protection. Niemeyer's surprising finding on some 105 active workers in intense noise was that their LDLs were elevated by 30 dB, exhibiting a typical difference between acoustic reflex and LDL of 40 dB. Loud sounds simply didn't bother these subjects.

The thoughtful thing that Niemeyer did was to double-check his findings not only against individuals with normal hearing but against 18 subjects who had been retired for at least 2 years from working in that same intense-noise environment. When he tested those subjects he found *their LDLs had come back to normal*! So here we see a 30 dB change in LDL from "getting used to it." As he noted, their *hearing* thresholds did not recover, only their LDLs.

When you first fit a hearing aid, it helps to know where the patient's ears have been. I fit my first wife's father with hearing aids when he retired as foreman of a paint plant some 17 years ago, and I couldn't measure his loudness discomfort at the limits of the audiometer. Nothing bothered him. When I fit him with hearing aids, I automatically ordered compression limiting and set the output conservatively, but I did it mostly out of habit; certainly not because I had any LDL problems to worry about. You can perhaps guess the outcome: He retired to Maine where it was quiet, and 2 years later I got a call from him saying, "You know, those hearing aids are a bit uncomfortable [loud] sometimes."

The conclusion appears to be that measurement of individual loudness growth can be valuable in some cases, but any history of high-level noise exposure should be taken into account. Moreover, Knier, and Bentler (1994) reported that approximately 20% of subjects could not reliably perform the loudness task. In cases such as those, the average data provide a good place to start.

PRESENT AND FUTURE PROGRESS

Hearing Aids at High Sound Levels

One of the reasons hearing aids often don't work well is because they distort high-level sounds. Figure 8–34 shows distortion measurements we made recently on three commercial hearing aids: Two Class A hearing aids and one WDRC-TILL hearing aid.

At normal conversational speech levels of 65 dB SPL (frequent peaks on the sound level meter), all three hearing aids operate without distortion. This is true even taking into account the 75 dB SPL instantaneous peaks which accompany 65 dB SPL peak sound level meter (SLM) readings.

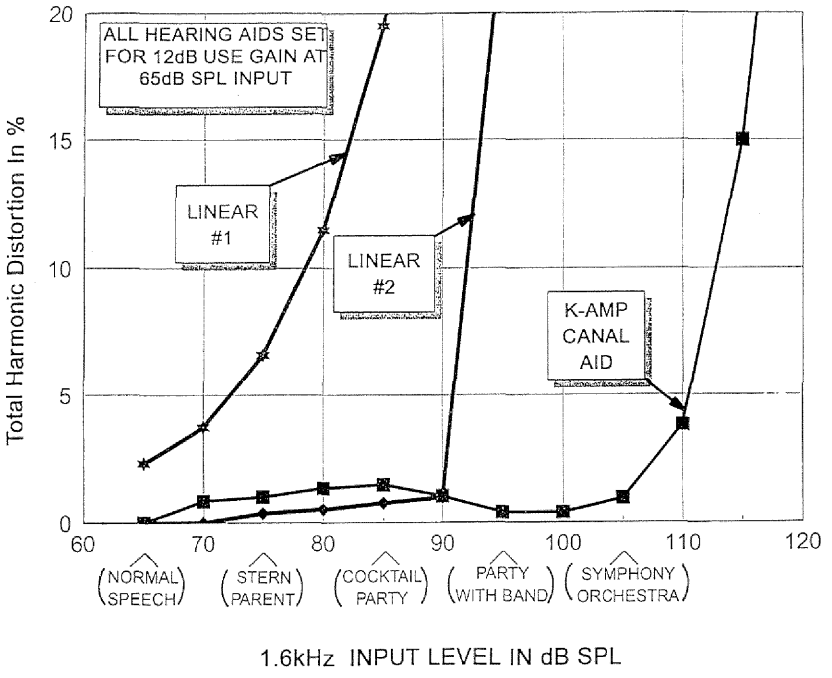

Figure 8–34. Distortion at real-world SPLs for three widely sold hearing aids: two using starved-class-A linear circuits and one using a low-distortion WDRC Class D circuit.

But many circumstances in social situations require low-distortion operation at much higher levels. A raised voice ("stern parent") can easily exceed 75 dB SLM readings. If you take a $32.00 Radio Shack sound level meter with you to parties, you will find that after a few drinks the "cocktail party effect" (Pollack & Pickett, 1957) sets in and the overall SPL rises into the 80s. If a band is playing—whether at a national audiology convention or a Wisconsin farmhouse party—the conversational levels rise into the high 90s. At the two country and western dances where I had a sound level meter, the levels ranged from 100–105 dB SPL.

Although the FDA will not presently permit any benefit claim for reduced hearing-aid distortion (Killion, 1994), it would otherwise seem self-evident that designing hearing aids that can be worn without distortion is a good thing. Distortion erodes intelligibility.

As an amateur violinist, the slide that I most enjoy showing is reproduced in Figure 8–35. As described elsewhere, the violin is a powerful

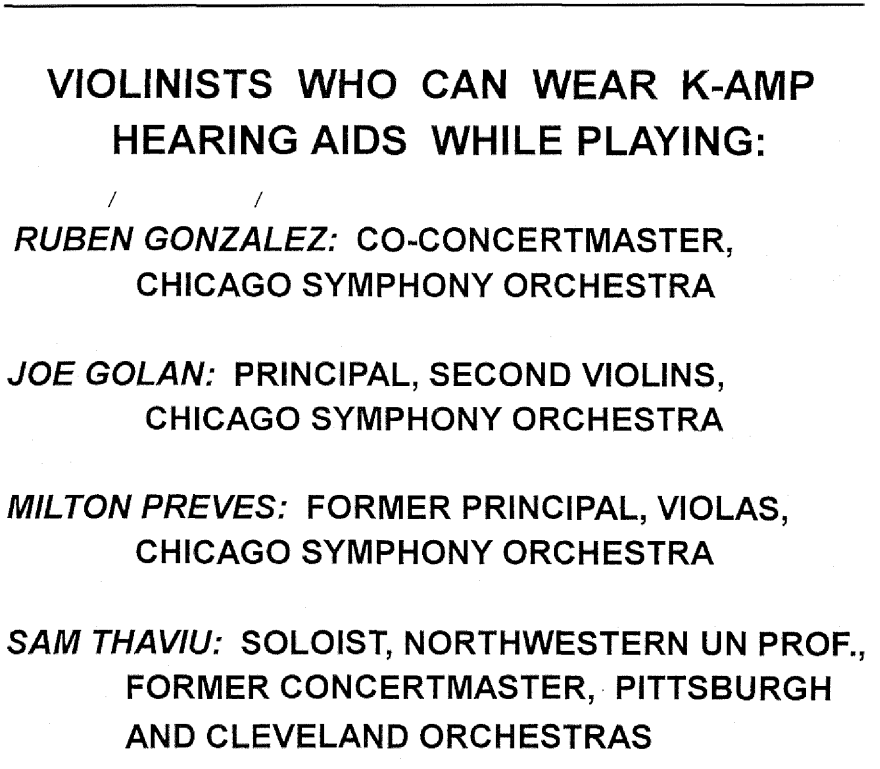

Figure 8–35. Four world-class musicians whose ears attest to the improved performance of modern hearing aids.

instrument, and soloists find their left ears especially at risk (Royster et al., 1991). The sound pressure levels routinely produced by musicians were a major challenge in the design of the hearing aids these musicians can now wear. I am grateful that each of them gave me permission to publish their successful use of these hearing aids.

Acknowledgments: Steve Iseberg, Tim Monroe, Don Wilson, Dennis Ervin, Matt Roberts, and Jonathan Stewart each made a significant contribution to the instrumentation and/or software necessary to provide the "Playing Bach by Ear" recordings of objective Tartini tones. Don Wilson did much of the higher-quality graphics.

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