The Conductor Model for the Loudness Response in Sensioneural Impaired Human Hearing

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Abstract

We describe a simple model for sensioneural hearing impairment that is consistent with experimental measurements demonstrating threshold elevation and hearing recruitment, decruitment, and hyperrecruitment. Together with our EarSpring model for unimpaired hearing, this model enables us to compute the required degree of hearing correction so that the impaired listener can hear what ought to be heard.

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1 Introduction

Conductor is the name of our model for sensioneural hearing loss in humans. It is the simplest model that produces results consistent with experimental measurements of hearing recruitment, and its variations. Our aim is not to model any specific mechanism of human hearing, but rather, the entire system of cochlea - afferent 8th nerve - brain - efferent 8th nerve, and their complex interactions. Our goal is to derive a method for determining the correct amount of spectral modification needed to enable an impaired listener to hear sounds as they would appear to an unimpaired listener.

Note that hearing sounds as they ought to appear is a different goal than normally pursued by hearing aid providers. Their aim is primarily to assist the impaired listener in verbal communication with others. Accurate sound reproduction is of less interest than that of enhancing the difficult consonant sounds of speech such as sibilance, glottal, plosive, and fricative sounds. Adjuncts such as noise reduction and interference cancellation are also frequently employed. As such, an inaccurate spectral enhancement may provide a better ability to understand speech.

In contrast, our aim is the detailed and correct restoration of musical hearing. Noise reduction and interference rejection techniques normally used by hearing aids would severely corrupt some musical passages, and so we do not pursue those enhancements. Inaccurate spectral band compression would lead to incorrectly reconstructed instrumental sounds, for example, producing an incorrect relationship between harmonics generated by a musical instrument which would change its timbre.

In addition to spectrally selective gain compression, one must pay heed to issues of compression time profiles, loudness masking effects, and maintaining smooth inter-band transitions. These engineering details are dealt with in another paper. This paper confines itself to the basic Conductor model for sensioneural impairment and how that model can be used to derive necessary spectral corrections.

2 Qualitative Description of the Model

Hearing loss is generally described in terms of threshold elevations in each of several spectral bands. It may also include a description of the level at which sounds become uncomfortably loud in each band.

Sensioneural hearing loss may be attributable to the mechanical destruction of some transducer components, e.g., inner hair cells, or it may be attributable to impaired nerve and/or brain function.

We model sensioneural hearing loss as a reduction in the detected sound levels by a subtractive amount. Stated differently, the damaged components produce a noise background, or threshold, that must be exceeded by some amount before sounds can be perceived.

Perceptual loudness is measured in units of sones. In another paper[1] we identified this measure with the average power of vibration of a simple nonlinear harmonic oscillator, which we dub an EarSpring. That paper examined a model for unimpaired hearing. With unimpaired hearing the internal processing threshold is very small and so the identification of sones with that average power was sensible.

But in this paper, we partially retract that identification, and instead identify sones as a measure of how far the incoming signal strength exceeds the elevated processing thresholds caused by damaged subcomponents. We identify signal strength with the mean vibrational power in the EarSpring. Damaged subcomponents may raise the threshold to sizable levels.

A raised processing threshold in any one spectral band simultaneously provides an elevated hearing threshold for the listener, and produces a loudness response characteristic of hearing recruitment. Whatever subcomponents of the system remain, do so with unimpaired effectiveness, in the case of typical hearing recruitment. Hyperrecruitment and decruitment call for additional forms of impairment.

Since measurements of hearing ability are performed in dB intensity measure, taking the logarithm of any quantity from which a constant value is subtracted produces a curve characteristically similar to that of hearing recruitment.

And at high enough sound-field intensities the remaining EarSprings furnish enough signal to overcome the elevated threshold and a significant portion of the recruitment curve, provided the impairment isn't too extreme. Hence at loud levels, even impaired hearing becomes almost normal.

3 Quantitative Description of the Model

As stated above, sound is perceived when the signal strength exceeds internal processing threshold levels. Damaged sensor components elevate that internal processing threshold. The sones loudness is a measure of how far the transducer signals exceed the processing threshold. This processing threshold is different from the elevated threshold measured in audiology examinations. It is innate to the sound processing system.

The sones observed in an impaired system can be described as:

$$\text{Sones}\,(P, P_{thr}) = \max(0, S(P) - S(P_{thr}))$$

[1] *The EarSpring Model for the Loudness Response in Unimpaired Human Hearing*, D.McClain, Dec. 2006

where,

$$\begin{aligned} S(P) &= \text{the signal strength produced by sensors,} \\ &\quad \text{due to sound-field level } P \text{ phon} \\ \text{Sones}(P, P_{thr}) &= \text{the apparent loudness provided by a damaged transducer} \\ &\quad \text{with elevated threshold } P_{thr} \text{ phon} \end{aligned}$$

An unimpaired listener has a zero phon threshold elevation, and so perceives loudness $\text{Sones}(P, 0)$. The threshold signal level $S(0) = 0.002$ sones, which means that for any typical sound levels encountered in daily life, $S(P)$ is so much larger than this threshold level that for all practical purposes, unimpaired hearing has $\text{Sones}(P, 0) \approx S(P)$ as stated in the EarSpring paper.

In that paper, we identified the sound-field level with the phon measure at each particular frequency. At 1 kHz, this is identical to sound-field intensity measured in dBSPL. Signal strength $S(P)$ is identified as the EarSpring mean vibrational power at an excitation level $10^{P/20}$, for a sound field of level P phon.

To demonstrate the difference between the internal processing threshold and an elevated hearing threshold as measured through audiology, consider the case where the listener has a threshold elevation of 60 dB at 1 kHz. In order to produce that much hearing threshold elevation, the internal signal processing threshold must have risen to 4.7 sones. The nominal threshold for unimpaired hearing $S(0)$ is 0.002 sones. Hence the internal threshold has risen by 33.6 dB. As you can see, this appears to have little direct relation to the observed hearing threshold elevation.

We never actually measure sones during audiology testing. Rather, we plot the dB elevation of the listener's threshold levels above agreed upon standard threshold levels for unimpaired hearing. To make sense of such measurements, we must convert the dB elevation to its equivalent sound-field intensity in dBSPL, and then convert this intensity by means of an Equal-Loudness relation to phons in that spectral band.

In an effort to describe one's hearing, we could develop a graph that would show the stimulation level in phons and the listener's hearing in terms of the perceived phon level. Perceived phons is that level of stimulation which, if presented to a person with unimpaired hearing, would provide the same sensation. If one has a quantitative mapping between phons and sones, the perceived phon level P_{sens} can be obtained by solving this equation:

$$\text{Sones}(P_{sens}, 0) = \text{Sones}(P, P_{thr})$$

or, by substitution from the prior equation,

$$S(P_{sens}) = S(P) - S(P_{thr}) + S(0)$$

Of course, when the right hand side of the equation becomes zero or less, there is no solution for P_{sens}. The sound is below the listener's threshold for hearing. That happens when P falls below P_{thr}

So, for example, in Figure 1 we show a straight line representing unimpaired hearing along with the hearing obtained for a threshold elevations of 40 dB and 60 dB at 1 kHz. Typical recruitment is in evidence. Higher threshold elevations imply steeper slopes over the lower regions of the recruitment curves.

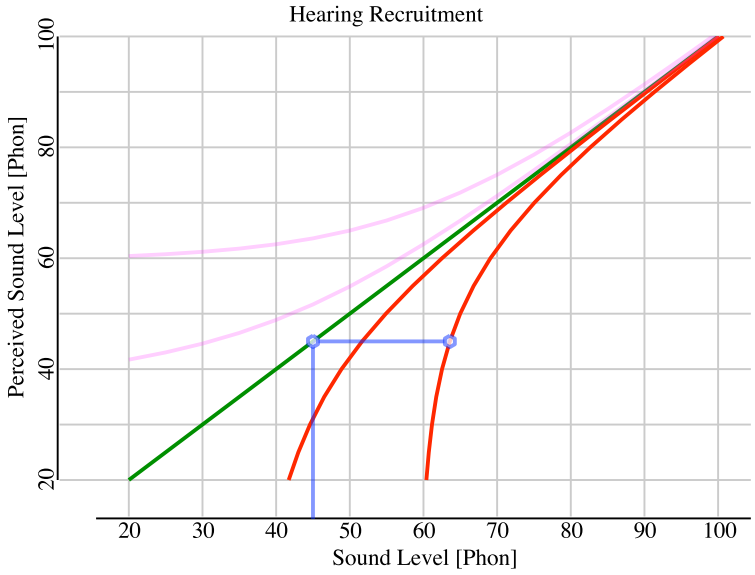

Figure 1: Normal hearing in green, typical hearing recruitment curves in red, for threshold elevations of 40 and 60 dB at 1 kHz. The light-blue lines show the amount of correction needed to enable the listener with 60 dB threshold elevation to hear a tone at 45 dBSPL as actually being that loud. The light-colored curves above the unimpaired hearing line show the mirror image compression curves needed to fully correct the recruitment hearing.

At very loud sound-field levels, the listener's hearing impairment is nearly inconsequential with the recruitment curves asymptotically approaching the unimpaired hearing line. But as the sound level drops, the listener perceives the sound fading ever more rapidly until it disappears below the elevated threshold level.

4 Development of Hearing Corrections

In Figure 1, the amount of hearing correction needed is that number of phons between the presentation level and the phon level required for the listener's hearing to perceive that

same sound level. An example is shown as the length of the horizontal blue line connecting the 45 phon level with the listener's recruited hearing curve.

Clearly, the degree of correction required is dependent on the sound-field level, which implies that dynamic compression in that spectral band will be needed in order to compensate the listener's hearing. As the sound-field intensity increases, less amplification is needed.

We can derive the required correction by solving the following equation for ΔP:

$$\text{Sones}(P, 0) = \text{Sones}(P + \Delta P, P_{thr})$$

or,

$$S(P) = S(P + \Delta P) - S(P_{thr}) + S(0)$$

In other words, the hearing correction ΔP is that amount of signal amplification needed so that the recruited hearing perceives the incoming sound level as an unimpaired listener would.

Incidentally, given the nature of loudness response as a measure of signal strength above an elevated internal processing threshold, it should be the case that persons with unimpaired hearing will also suffer a form of recruitment in the presence of strong background noise. Attempting to hold a telephone conversation in a noisy environment should pose similar difficulties for that person, as would be faced by someone with sensioneural hearing loss in a moderately quiet environment. Hence a hearing correction system would be of considerable use in such situations, even for people with unimpaired hearing.

5 The Phons → Sones Relation

Up to now we have avoided presenting a closed form solution to the hearing correction problem. Various approximations could be used for signal strength $S(P)$ derived from phon levels. The simplest such approximation, with relative error less than 1.5% for sound levels above 36 phon, is:

$$S(P) \approx 10^{(P-40)/30} \quad \text{with } |\epsilon_{rel}| < 1.5\% \text{ for } P > 36 \text{ phon}$$

The relation we use is from our EarSpring paper, valid for all P, where $S(P)$ is the solution of the cubic equation:

$$S(P) = \frac{\left(4\hat{\beta}^2 + \Gamma_{40}^2\right)}{\left(4\hat{\beta}^2 + \left(\Gamma_{40} S(P)\right)^2\right)} 10^{(P-40)/10}$$

and where $\hat{\beta}$ and Γ_{40} are constants obtained from solving the EarSpring system subject to experimental boundary conditions. It is possible, but cumbersome, to write this in closed form so that $S(P)$ is given as an explicit function of P.

When the first approximation is utilized, one can develop closed form solutions to the problems of perceived phon levels and correction gains. Otherwise, one must resort to numerical solution of a transcendental equation.

6 Algebra of Hearing Operators

We can develop a simple algebra of parametric operators that represent impaired hearing and the components needed to correct that hearing. An operator algebra will considerably simplify the description of hyperrecruitment and decruitment conditions and their required corrections.

Let,

$$\begin{aligned} H(P_{thr}) &= \text{the recruited hearing corresponding to elevated threshold } P_{thr} \\ C(P_{thr}) &= \text{the hearing correction needed for elevated threshold } P_{thr} \\ A(\Delta P_{atten}) &= \text{an attenuation of } \Delta P_{atten} \\ G(\Delta P_{gain}) &= \text{a gain of } \Delta P_{gain} \end{aligned}$$

These operators all take a sound level for their argument and produce another sound level as their operational result. The items in parentheses following each operator's name are parameters of the operator, as distinct from the sound level argument upon which an operator acts. In general, operator parameters are fixed values, but sound level arguments may vary with time.

We denote the application of an operator to a sound level by square brackets enclosing the argument and following the operator. So, for example,

$$A(\Delta P_{atten})[P] \equiv P - \Delta P_{atten}$$

which states that an attenuator acting on a sound level diminishes that sound level by the parameter of the attenuator. The square brackets enclosing the sound level argument should be read as *apply*.

The linear additive nature of gain and attenuation results from our operating on phon arguments. Phons are identical to dBSPL at 1 kHz, and so behave similarly under gain and attenuation. However, a phon gain or attenuation operation is interesting for frequencies other than 1 kHz. A ΔP phon gain at one frequency may not be the same change in sound intensity, measured in dBSPL, as the same phon gain at some other frequency. For now we postulate the existence of such gain and attenuation operators.

Composition of operators, denoted by an infix meta-operator $\circ$, implies nested application. For example:

$$(A(\Delta P_{atten}) \circ C(P_{thr}))[P] \equiv A(\Delta P_{atten})[C(P_{thr})[P]]$$

This equation states that the composition of attenuation with hearing correction, applied to a sound level argument, is the attenuation of the corrected sound level. Composition is an operation between operators, producing a composite operator. Application of an operator to a sound level is different from composition. It is meaningless to compose an operator with a sound level. And it is meaningless to apply an operator to another operator.

Operator composition is associative, and so can be performed in any order:

$$A \circ C \circ G = (A \circ C) \circ G = A \circ (C \circ G)$$

Each grouping of composition produces a new composite operator that takes part in the next composition.

A and G are both linear additive operators. Such operators commute under composition with other like operators:

$$A \circ G = G \circ A$$

Operators A and G are inverses of each other, in the sense that

$$A(x) = G(-x)$$

and

$$A(x) \circ G(x) = I \quad \text{where } I \text{ is the identity operator}$$

The cancellation, which produces the identity operator, holds only when the parameters of the attenuator and gain are identical.

The H and C operators are both nonlinear. Nonlinear operators cannot commute under composition with other operators. Their effect varies nonlinearly with presented sound level arguments, unlike the linear additive operators A and G.

The hearing correction derived from a pre-amplified signal is not the same as the post-amplification of the base hearing correction:

$$C \circ G \neq G \circ C \quad \text{unless } C = I \text{ or } G = I$$

This inequality is easy to verify by visual examination of the recruitment curve in Figure 1. Suppose the G gain $\Delta P_{gain} > 0$. Any signal injected into a pre-amplifier will emerge at

a higher sound level, and hence the required hearing correction will be smaller than that needed for the the un-amplified sound, so $C \circ G < C$, which means $C[G[P]] < C[P]$.

Conversely, taking the un-amplified input signal, deriving its required hearing correction, and then post-amplifying that, will result in a larger sound level than the base hearing correction, so $G \circ C > C$. These two paths diverge:

$$C \circ G < C < G \circ C \quad \text{when } \Delta P_{gain} > 0 \text{ in } G$$

This, incidentally, is why any equalization, or other amplitude modifying effects, applied to music must be performed ahead of any hearing corrections. Doing it the opposite way will not produce a correct sound.

If C were correctly matched to H, then all presented sound levels would be corrected by just the right amount so that the listener's hearing H always perceives the presented sound level. In that case,

$$H(x) \circ C(x) = I$$

This identity relation holds only when the parameters of the two operators are the same. Matched C is a compression curve that is the mirror image of the recruitment hearing curve H.

There can be no commuting these two operators, and it isn't even sensible to imagine doing so. Since H is in the head of the listener, H is always the leftmost operator. It cannot be removed from the head of the listener and so we cannot interpose between H and the listener by any external means.

If a listener's headphones or speaker system modifies the intensity of sound in a spectrally varying manner, and is being used to convey the output of the hearing correction system, then a post-equalizer inserted after the hearing correction and ahead of the speaker system would be appropriate. In that case the equalizer should tune out the spectral effects of the speaker system. If such effects as spectral coloration were desirable, then they should be reapplied ahead of the hearing correction.

We can represent the speaker system as an attenuation operator A_{spkr}. Interposing the speaker system between the hearing correction and the listener's hearing ($H \circ A_{spkr} \circ C$) interferes with the ability of the hearing correction to cancel the recruitment of the listeners hearing. But, by interposing a complementary equalization gain G_{spkr} between the hearing correction system and the speaker system, we once again enable the cancellation of hearing recruitment.

$$H \circ (A_{spkr} \circ G_{spkr}) \circ C = H \circ C = I$$

7 Hearing Correction for Hyperrecruitment

As exhibited in Figure 2, the red curve for hyperrecruitment can be obtained by presuming a higher threshold than measured, preceded by a gain equal to the difference between the higher threshold and the measured threshold.

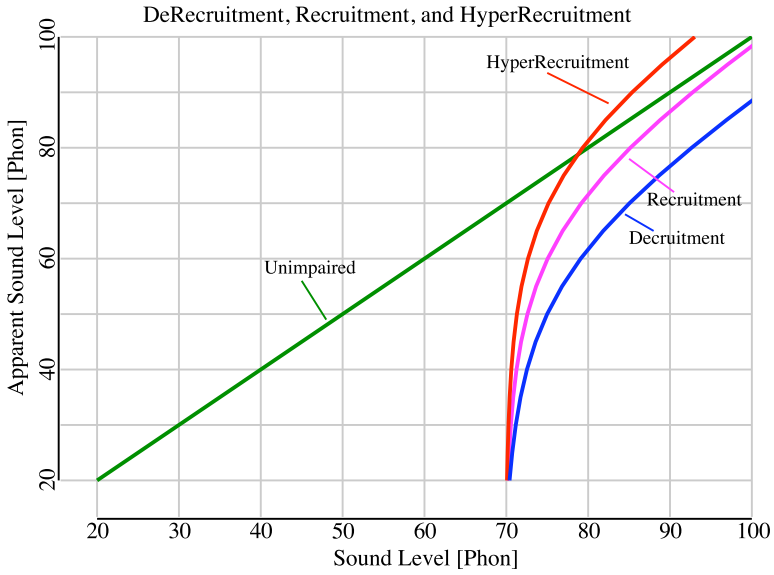

Figure 2: Normal hearing in green plus variations of hearing recruitment, all for an elevated threshold of 70 dBSPL at 1 kHz.

Hperrecruitment hearing:

$$H_{hyp}(P_{thr}, \Delta P) = H(P_{thr} + \Delta P) \circ G(\Delta P)$$

Innate pre-gain G shifts the innate recruitment curve H to the left. Using a higher than measured threshold produces a steeper recruitment curve than would be had for the measured threshold. The composition of these two effects exactly reproduces the measured elevated threshold, and ensures that the recruitment curve will cross above the unimpaired hearing line. Hence, sounds above that crossover level grow too loud – the hallmark of hyperrecruitment.

The amount of ΔP in hyperrecruitment controls the point at which the recruitment curve crosses above the straight-line unimpaired hearing. It also controls the amount by which the recruitment curve rises above the unimpaired hearing at high sound-field levels. This in turn can be estimated by the level at which the listener begins to feel discomfort. The greater the ΔP, the greater the crossover rise, and the lower the measured uncomfortable

level. No other direct measure of the degree of hyperrecruitment is normally obtained with routine audiology examinations.

The physical mechanism responsible for the innate amplification G is unknown. It may be that our Conductor system model is simply lucky in so closely reproducing the hyperrecruitment behavior. It is only a model by analog, and possibly not closely related to any physical mechanisms.

In order to correct hyperrecruitment, we estimate ΔP and perform a correction at the increased threshold elevation $P_{thr} + \Delta P$, followed by attenuation ΔP. The amount of attenuation should equal the amount by which the measured threshold elevation was increased in the correction operation:

$$C_{hyp}(P_{thr}, \Delta P) = A(\Delta P) \circ C(P_{thr} + \Delta P)$$

The resulting operator sequence produces an identity:

$$H_{hyp}(P_{thr}, \Delta P) \circ C_{hyp}(P_{thr}, \Delta P) = H(P_{thr} + \Delta P) \circ (G(\Delta P) \circ A(\Delta P)) \circ C(P_{thr} + \Delta P) = I$$

The post-correction attenuation operator A cancels out the innate gain operator G of hyperrecruitment hearing, provided we have correctly estimated the required ΔP. And then the correction operator C cancels the recruitment in H.

In our EarSpring paper, we surmised that some of the cause of hyperrecruitment may be due to an ineffective suppression mechanism for controlling large scale oscillations of the basilar membrane at high sound-field intensities. But we show here that we also need an elevated threshold that may be partly due to impaired sensory organs.

Impaired control of basilar membrane vibrations may be due to damaged outer hair cells, while some sensioneural hearing loss in that spectral region may be due to damaged inner hair cells. Our experience has shown that in persons with profoundly deep narrowband notches in their hearing, the frequency bands just above that destroyed spectral region may develop hyperrecruitment.

It seems reasonable that this could happen. If one had been subjected to damaging enough sound levels to cause profound loss in one spectral region, then the adjacent higher frequency bands, nearer to the entrance window of the cochlea, would also sustain a high level of mechanical damage – enough so that both inner and outer hair cells could be damaged. The loss of the inner hair cells produces elevated thresholds. The loss of outer hair cells also affects the degree of threshold elevation, and, more importantly, it impairs the ability to control basilar membrane oscillation amplitudes. That, in turn, leads to hypersensitivity toward loud sounds.

As an aside, the same hearing curve as shown for hyperrecruitment occurs when a simple-minded correction of hearing loss is attempted with constant gain equalization instead of

dynamic level-dependent compressive equalization. As mentioned above, pre-amplification moves the entire recruitment curve toward the left. Therefore, a normal recruitment curve, moved laterally toward lower sound levels will necessarily cross above the unimpaired hearing line, mimicking hyperrecruitment.

Sounds at levels below where the resulting hyperrecruitment curve crosses above the normal hearing line fade away too quickly with diminishing sound levels. And sounds above that crossover level become much too loud too quickly, and quite possibly with damaging results. Such simple-minded correction can only be correct at one particular phon level – that level at which the recruitment curve crosses the unimpaired hearing line. Static equalization does nothing to rectify the recruitment condition.

8 Hearing Correction for Decruitment

Decruitment hearing is the converse of Hyperrecruitment. In operator form, it appears as a gentler recruitment than the threshold elevation would indicate, preceded by an attenuation. The amount of attenuation is equal to the difference between the measured threshold and the threshold corresponding to the gentler recruitment curve:

$$H_{decr}(P_{thr}, \Delta P) = H(P_{thr} - \Delta P) \circ A(\Delta P)$$

The key feature of decruitment hearing is the failure for the listener to ever perceive sounds as loud as they are, even at extreme high sound levels.

The way to correct decruitment is to apply the opposite corrections to the innate operators involved – a gentler recruitment correction followed by a post-gain:

$$C_{decr}(P_{thr}, \Delta P) = G(\Delta P) \circ C(P_{thr} - \Delta P)$$

That this is so, can be seen by writing out the full equivalent form for $H_{decr} \circ C_{decr}$. One sees that, as for hyperrecruitment corrections, the innermost G and innate A cancel out, leaving the C to cancel with innate H. But deciding on the correct value for ΔP is a matter of trial and error. And the aforementioned cancellations cannot occur completely unless the correct value is found.

9 A Model for Sensory Damage

We have experimental evidence for both threshold elevation and recruitment. Any model we develop must satisfy those two characteristics. We will suppose that sensioneural hearing

damage occurs by way of destruction of inner hair cells. This is really too simplistic in ignoring 8th nerve and brain function, but it will provide some interesting boundary conditions.

First of all, we can show that simple scaling by the number of remaining live hair cells cannot explain both elevated thresholds and hearing recruitment. If we postulate that N cells function in unimpaired hearing, and that impaired hearing has a fraction f of those cells dead and unresponsive, then the fraction of remaining hair cells can be estimated by the threshold elevation. The number of remaining live cells, operating at the elevated threshold must produce an aggregate signal that exceeds the unimpaired hearing threshold for sounds to be perceived:

$$(1 - f)NA(P_{thr}) = NA(0)$$

where, $A(0)$ is the signal amplitude delivered by each hair cell at 0 dBSPL, and $A(P_{thr})$ is the signal delivered at P_{thr} dBSPL, the elevated threshold. The actual number N of hair cells involved cancels out of the equation, and we find that $f = 1 - A(0)/A(P_{thr})$.

Examining the case for a 60 dB threshold elevation shows that f would be 99.96% dead cells. Even a modest threshold elevation of 20 dB would require that 98.6% of the cells were damaged and inoperable. Furthermore, the perceptual equivalent sound level for any signal at level $P > P_{thr}$ would be so incredibly weak as to defy experimental evidence.

For example, a sound as loud as 120 dBSPL at 1 kHz (about as loud as a nearby jet engine roaring), presented to a person with a threshold elevation of only 20 dB (very mild and would be considered as bordering on normal hearing), would have a perceptual equivalent level of 64 dBSPL (about as loud as adult conversation at 1 meter separation). This flies in the face of experience. An incredibly loud sound applied to a person with a barely noticeable hearing impairment is supposed to sound only as loud to him as an adult conversation sounds to an unimpaired listener?

Beyond this absurd conclusion, there is no way to produce hearing recruitment with simple scaling by the number of remaining live hair cells feeding the brain. What one gets is simply a uniform decrease in loudness at all levels. So, instead, we must seek out an explanation for what might cause the elevated internal thresholds for processing which operate as described in our qualitative and quantitative descriptions of the Conductor model.

Anyone who has ever burnt their finger knows well the raging, intense, persistence of painful nerve signals from damaged nerves at their fingertips. This pain persists long after the damaging influence has been removed. Suppose that damaged hair cells likewise produce a raging loud objection to their status by sending the equivalent of the loudest possible signal. Suppose, for the sake of argument, that loudest signal corresponds to 120 dBSPL at 1 KHz. The actual level chosen won't matter much, as we shall see.

In that case, we have a fraction f of dead cells sending a signal whose amplitude corresponds

to this loud extreme. At the elevated threshold we have a fraction $(1-f)$ of hair cells sending the signal amplitude corresponding to the elevated threshold. The good cells must just overcome the screaming dead signals in order to permit the perception of threshold level sounds, so $(1-f)NA(P_{thr}) = fNA(120)$. Again, the total number of cells N cancels out of the equation, and we find that the fraction of dead cells f, even for an elevated threshold of 60 dBSPL is only slightly less than 1%.

Even profound hearing loss, the onset of which occurs when the threshold elevation is greater than 90 dB at 1 kHz, corresponds to a net damaged fraction of cells $f = 9.1\%$. The screaming of the few drown out the voices of the many.

This model not only reproduces the observed relationships in perceptual loudness, but also provides for recruited hearing, from its thresholding behavior. The same example of a 120 dBSPL sound at 1 kHz, heard by a listener with 20 dBSPL elevated threshold, now hears a sound that seems like 120 dBSPL. A person with 60 dBSPL elevated threshold hears a sound that seems like 119.9 dBSPL. This matches the notion that recruited hearing becomes essentially normal at loud sound levels.

The interesting thing here is that, with this damage model, it doesn't really take profound damage to cause profound hearing loss. Which speaks to the need to guard one's hearing very carefully. It only takes a small amount of physical damage to cause great hearing loss.

Under this model for damaged hearing, the original equation for $\text{Sones}(P, P_{thr})$ should be modified to the pair of equations:

$$\text{Sones}(P, P_{thr}) = \max\left(0, (1-f)\left(S(P) - S(P_{thr})\right)\right)$$

and,

$$(1-f)S(P_{thr}) = fS(P_{dead})$$

where, P_{dead} is the equivalent sound level for the signals emitted by the dead cells. For all but the most profound hearing loss, any reasonable choice for this dead level will do. The higher this level, the smaller the fraction f of dead cells needed to accomplish the threshold elevation. And the closer to unity $(1-f)$. At most, the change amounts to a very small percent modification of the estimated recruitment curves.

The hearing correction equation becomes that of solving for ΔP from:

$$S(P) = (1-f)\left(S(P+\Delta P) - S(P_{thr})\right) + S(0)$$

and the perceptual equivalence equation becomes one of solving for P_{sens} from:

$$S(P_{sens}) = (1-f)\left(S(P) - S(P_{thr})\right) + S(0)$$

When the right hand side of this equation dips below zero, there is no equivalent sound level of perception. The sound level is below the threshold of the listener.

In every case, when the fraction of damaged hair cells f is very small ($f < 1\%$) there is very little change to the outcome that would have been produced by using the equations outlined earlier, which effectively estimated $f = 0$.