A RETROSPECTIVE ON STUDIES ON COCHLEAR MECHANICS, OTOACOUSTIC EMISSIONS AND HEARING LOSS DUE TO OVEREXPOSURE - A MODEL FOR DYNAMIC MAPPING

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Music-Induced Hearing Loss (MIHL) is a special case of noise-induced hearing loss (NIHL) insofar as the methodology of quantification is concerned, while it presents a particularly acute challenge in respect of music's conflicting demands. These include the desire, particularly in the young, to gain euphoria from music which may be loud enough to traumatize the cochlea. If going to a loud music event were to produce actual physical pain or giddiness we would not do it. Enigmatically, the cochlea does not possess pain receptors. Maybe this is because the evolution of our very wide dynamic range (120+dB) seems to have obviated the need, at least up until modern times. But now the music industry appears to be going through a phase of believing that amplification with compression is necessary to bring music alive and that modern music capabilities constitute an important new cultural modality. To hearing-industry professionals, although the contra-indications of exposure to loud sound are clear, the message is ignored. By now we have incredible macroscopic and microscopic detail about cochlear mechanisms, but perhaps we do not have a sufficiently good holistic model of how they all work together to make a convincing case? There is also the issue that our best measures of measuring audibility lack precision due to huge data variability, the source of which has never been explained. Our presentation draws together the key issues and reviews two of our early results to restate the problem. The first result concerns what we in 1998 termed "latent hearing loss" due to the rise in preclinical damage to the outer hair cell population measured using click-evoked otoacoustic emissions (OAE). We showed that this can be determined with a better overview than pure tone thresholds and thus can serve as the basis of personal dosimetry e.g. for those who frequently dose-up on loud music. The second result questions the traditional assumption that the frequency-place map is fixed in the manner depicted by the grid of a standard audiogram. We re-introduce our 1987 model which expresses the leading edge of the excitation pattern in spatial co-ordinates and show that many kinds of hearing data are actually consistent with the notion that mapping from frequency to place is dynamic. The potential mechanism responsible follows the suggestion by Henson that the type IV fibrocytes of the spiral ligament actively tension the radial fibres of the basilar membrane. There is substantial recent literature to support this view and the relevance to this presentation is these cells appear to be the direct targets of noise-fatigue. Taken together we have the key ingredients to explain the so-called "half-octave shift", a key indicator of fatigue. We will present new evidence that we can quantify this behaviour readily in humans during the usual click-evoked OAE test. Thus the model accounts for variability in pure tone thresholds, and may provide a direct measure of susceptibility to fatigue given directly by the OAE test. We outline several other consequences from this model such as a new distinction possible between place pitch and periodicity pitch. Since the music is strongly carried by the timing of neural responses, music-goers can be oblivious to the onslaught of cochlear damage which will eventually affect speech perception and ruin their thriving social lives. A clear message of this presentation is that it is not true that there is no human cost for unjustified or extreme amplification. The OAE technology gives us the capability, not just to track the growth of hidden cochlear damage, but also the mechanical operation of the fibrocytes through a process which is outlined. We should be using, in moderation, the OAE technology for personal dosimetry in respect of MIHL.

INTRODUCTION

This work began in 1973 with impulse-response studies on the displacement of the basilar-membrane in guinea pig and provided the first documented confirmation[1] of the nonlinear compression in the vibration shown in 1971[2]. It continued in next decade with more studies looking at low-frequency components in the motion of the membrane[3][4]. Such assertions then had no place in the common conception of basilar membrane motion and were disputed[5][6]. Such views back

then were not compatible with the standard model. However, today such 'dc-shifts' are highly relevant because of the growing evidence that endolymphatic hydrops produces displacement of the basilar membrane towards scala tympani[7][8]. A decade later at the National Acoustic Laboratories in Sydney we looked at ways to prevent noise-induced hearing loss in industrial and military work situations. This government research organisation was founded to address noise-induced hearing loss in World War II veterans, industrial workers and also children following a rubella outbreak in the 1940s. The mainstream assessment technique was pure-tone audiometry and the primary means of rehabilitation was design and application of hearing aids (HA) based upon providing external gain, very often in excess of 40dB. Since the primary disability produced by noise-induced hearing loss (NIHL) is loss of speech communication, tailoring hearing aids to optimise speech was a strong aim.

Hearing conservation had been served by a team studying the ways of attenuating sounds reaching the ear drum in connection with the so-called "equal-energy" hypothesis (EEH) modelling the effects of temporary threshold shift[9] (TTS) due to loud sound exposure. The fundamental reason humans lose hearing as a function of age and noise-exposure was *not* because they chose not to protect it in noisy situations. Hearing loss is secondary to the loss of OHC function since they clearly perform a vital role. We needed an objective test for OHC function.

1 PERMANENT CHANGES IN OHC PERFORMANCE -- OUTER HAIR CELL REDUNDANCY

The world is grateful to David Kemp for discovering such a test[10]. His discovery not only cemented the vital role of OHC, it provided an approach to try to objectively obtain cyto-cochleograms[11][12] in humans, paving the way to a knowledgeable method of dosimetry of OHC loss. It demonstrated that a chinchilla could lose two out of the three rows of outer hair cells before their behavioural sensitivity showed significant change[13]. At the same time we were aware that one can have OHC present yet not operating[14]. They could be essentially turned off or saturated by virtue that the operating point is removed from the active region due to a steady displacement of the basilar membrane.

In 1990 most investigations were assessing their applicability to neonatal screening on the basis that emissions were present or absent. From the outset of acquiring our first ILO88 system, using the standard "nonlinear" TEOAE protocol, we viewed an enormous variability of emission levels across all subjects, including the four extremes, neonates with high emission levels, and those whose emissions were absent, as well as elderly with emission strengths which varied across the range. In this national centre for hearing assessment we were in the fortunate position of having access to many groups with differing lifestyles and noise-exposure profiles. We carried out cross-sectional studies across all strata of society. These included defence-force personnel, deep coal miners, workers in light industry, office workers, normal and premature neonates, pre-school, elementary school and high-school students, orchestra and freelance musicians including visiting concert artists, even a group of piano-tuners meeting at a convention. We participated in a large state-government run general health survey of inmates in 37 prisons and were able to correlate our results with some 290 general health variables[15].

Since our job was the study of NIHL we wanted to extract a scalar metric which we could reasonably compare in the same subjects over many years. The Otodynamics protocol provided what seemed an ideal pair of seemingly orthogonal parameters. The first was the emission level (dB SPL) and the second the Whole Waveform Reproducibility (a correlation coefficient % comparing the pair of waveforms A and B, time-windowed to remove the stimulus artifact). Using a standard definition of Coherent Output Power, we constructed from these two a third variable Coherent Emission Strength (CES, dB SPL defined as the emission sound power scaled by the square of the correlation coefficient appropriately zero-adjusted).

We did pure-tone audiometry only on selected groups because of the additional time needed. These included the annual survey of musicians in the Australian Opera and Ballet Orchestra routinely playing in the Sydney Opera House and the coal miners, plus the office workers readily at our disposal. In the end we had good numbers to obtain satisfactory statistics comparing pure tone thresholds with emission strengths (see Fig. 1).

The cross-sectional studies were not obtained on the basis of equal numbers in each age range, but we nevertheless had hundreds in each range. In general, emission strengths declined with age. However, within three years we became preoccupied with an anomaly in the data. It persisted no matter how we assessed the strength or how we split the groups (into 5, 8 and 10 years ranges). The decline in emission strengths was non-monotonic with age. There was a pronounced dip in TEOAE emission strengths between the ages of 18 and 24 and it was more pronounced in males i.e. the strengths in the 30 and 40 year groups were higher than for the late teenagers entering

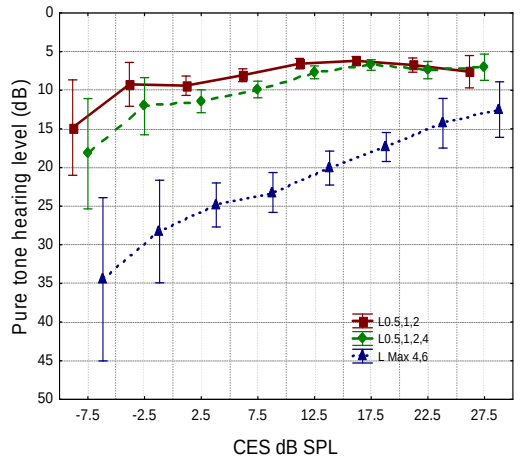

Figure 1 Capacity of TEOAE to characterise OHC redundancy (excess capacity) and serve as early warning for the onset of hearing loss (see text). Here we plot coherent emission strength (CES dB SPL i.e. means ± 95% confidence limits) for the large population (1700 records) (abscissa). The different curves represents different lumped measures of mean hearing level: the 3 frequency average 0.5,1,2 kHz; then mean 0.5,1,2,4 kHz, finally the most variable pure tone measure to show an early effect, Max(4,6) kHz. The CES declines over half of its range (the highest neonate record was +38dB SPL) before a critically low level is reached between -1dB SPL matching the "Worse Ear" condition and -4dB SPL for the "Better Ear".

adulthood. The pure tone audiometry did not show this dip. From the audiological perspective this dip in the aging curve was not believable. Yet to us it suggested that if the trend continued these young people with low emissions, might be suffering premature hearing loss. So we did a simulation based upon the fact that they presumably had normal emissions during infancy so we estimated their current rate of decline of CES and continued that rate onward for each 2 year age range until their emissions reached the critically low levels shown in Fig.1. We took the Australian Bureau of Statistics projections for the population for the next 20 years and calculated the numbers affected for males and females separately (see Fig.2). This was presented at the Better Hearing Australia workshop in Adelaide in 1994. Being "hidden" there was then little sign of a trend in rehabilitation needs and a generally passive response. However a national report which appeared a decade later[16] expressed concern at the rise in number of cases.

The "early-warning" period, down to CES values of -2dB did allow as to distinguish significant differences between different groups such as comparing personal stereo earphone use with non-users and industrial hearing loss[17]. Our most recent comparison around 2003 between different groups is shown in Figure 3.

The notion of redundancy of OHC numbers also is consistent with the notion of varying susceptibility to NIHL[9][18][19]. Our own work showed a distinction between individuals with "tough ears" and those with "tender ears". We encountered a mine worker aged 58 with essentially normal hearing, but on the same shift, there was a young man of 25 with already a severe occupational NIHL. We will show a major difference between the ears of the two workers using time-frequency distributions click-evoked otoacoustic emissions.

With our growing evidence for pre-clinical damage we introduced the notion of "hidden (latent) hearing loss"[17][20][21] which at the time was quite controversial because it challenged the intuitive notion that pure tone thresholds are the most sensitive indicator of ear damage. So it was not intuitively obvious that such a sensitive mechanoreceptor would not be sensitive to its own state of damage. We reasoned that this may be characteristic of highly redundant systems which "fail gracefully".

In hindsight, we prefer to jettison such nebulous terms as "hearing damage" and "hidden hearing loss", in favour of a term with more specificity like *pre-clinical damage.* Better still we should represent the phenomenon more positively like so many other systems in the body as having *reserve*

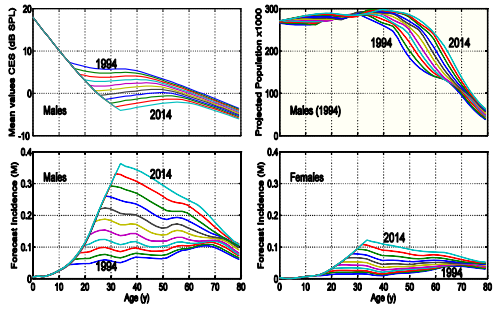

Figure 2 Early simulation of projected rise in actual hearing loss in the Australian community (LePage, 1994), using a 2-year iteration span. The exercise incorporates an anomaly in the age dependence of the TEOAE strength. For many late teenagers and young adults the CES was markedly lower than predicted from high neonatal values. Depicted is a steady decline in CES with age (top left panel) occurs at an initial estimated rate until a critically low value is reached (range [-4, -1] dB SPL). The definition of CES affords a noise floor (emissions absent) of about -10dB SPL. The numbers estimated to be affected (bottom panels, ordinate values are for each 2 year age range) are based upon the (then) latest Australian Bureau of Statistics 20-year projection shown in the top right panel (males; similar for females).

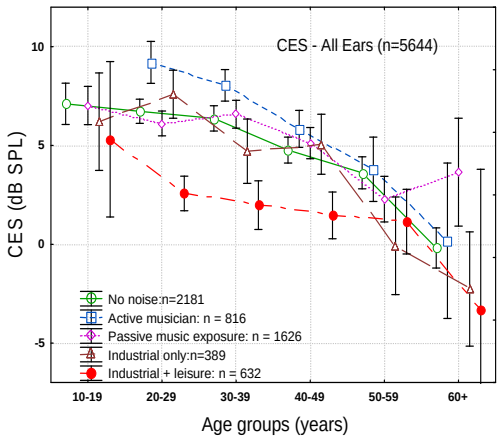

Figure 3 Effects of Music Exposure compared with industrial exposure. Shown are the results of TEOAE analyses from some 2839 subjects (here left and right ears are lumped so the numbers appear doubled) for means and standard deviations in each case comparing five mutually exclusive groups according to their degree of noise exposure extracted from written questionnaires completed by many of the subjects or through personal communication. Group 1 consisted of subjects not exposed to any industrial, music or leisure noise except for activities of modern daily living ('No Noise', n=2181); Group 2 consisted of 408 professional musicians or senior school music students none of whom had any other noise or amplified music exposure ("Active Music", n=816); Group 3 consisted of subjects exposed any type of leisure music, including personal stereos ("Passive Music", n=1626); Group 4 consisted of industrially exposed subjects such as those working in coal mines or in the rural sector ('Industrial noise only', n=389) and Group 5 those industrially noise-exposed subjects who also answered to leisure noise exposure, such as trail bike riding, power tools ('Industrial + Leisure', n=632).

performance capacity for which a better simple analogy is a burning candle stick. A candle generally performs well shedding its light for its whole shortening life — until it doesn't.

2. TEMPORARY EFFECTS: FATIGUE DUE TO HIGH LEVEL EXPOSURE

Our studies also attempted to look at dynamic effects in TEOAE and DPOAE, either simply watching how the emissions behaved during a recording with no external perturbation, but also we have both in the field measurements of TEOAEs from workers who were being subjected to quite extreme on-the-job noise exposures. We will show examples of the data before and after working shifts. Acute noise exposure data were also collected on several groups: attendees at a high-school discotheque, coal miners before and after shifts over an 8 day period, air-force personnel tested in response to repetitive impact testing over a week. We will show examples from a high-school science project which we assisted testing the hearing of young males with up to 1kW amplifiers in their cars, before and after a ten minute exposure.

In general, growing experience is that acute noise exposure has a dramatic effect upon TEOAEs[19]. It is obvious to want to quantify the effect of any exposure upon the cochlea[19][22] but in our experience the measurement noise-level in the recordings increases markedly post-exposure while the record stability decreases. This means that it becomes quite difficult to compare before and after records on the same basis. In our view, the "noise-level" of recordings is mostly likely *not* due to non-specific noise in the conventional sense but an increased electromechanical response to the trauma, which may or may not be a medial efferent response[23]. Addressing this need has been top priority for the last decade[24].

One of our primary pieces of published evidence occurred because we too were musicians and we identified with music-induced hearing loss (MIHL). The largest cohort studied was the Australian Opera and Ballet Orchestra performing routinely at the Sydney Opera House. That study over 9 years of the same orchestra players totalling 150, of which 39 were repeated every year. We collected detailed histories from questionnaires. In general while individual groups showed big changes (e.g. the horn players and those sitting in front of the brass) the musicians seemed highly aware of prevention issues. Charts of measurement statistics versus position in the orchestra pit are revealing. Violinists tend to show asymmetries between ears. Possibly the most consistent anecdotal reports of discomfort are issued by players sitting next to the piccolo player. By contrast, we also encountered a large number of popular musicians, mostly freelance who were less aware. Most notable exception were the cars with high wattage amplifiers. We will show examples of those data. They are a minority. What is evidently a major social issue is exposure to personal stereo devices[25] and an aspect which has not yet been addressed is how much more efficiently button earphones can couple high frequency content in comparison with free field delivery of the same music. That said, the very low frequencies from bass-boosted units are also of concern[8].

We conducted lab-controlled experiments on temporary emission shifts (TES) using our own hardware and software for continuous recording, employing no signal averaging. We recorded TEOAE and plotted dynamic time-frequency displays of the responses, introduced a 100dB tone for 10 seconds and watched how the response reflected the challenge. Examples will be shown. The object was to produce a clear indication that we could recognise some subtle change in response

such as some measure of "half-octave shift"[26][27] or a bounce phenomenon[19], [28], [29] to a lower noise "dose" than typically needed to show a temporary threshold shift.

2.1 The TEOAE "slipping-clutch" effect

Using click-evoked emissions there is one dynamic phenomenon which is very apparent within just a handful of subjects. The ILO88 standard TEOAE paradigm is an impulse response test. It has two modes ("linear" or "nonlinear" stimulus modes) for collecting emissions and involves signal averaging of the click response synchronous with the stimulus depending upon the size of the response and the noise-floor. The averaging can be terminated when the summed response is clearly visible, but we normally allowed the recording to proceed for the full number of 260 repetitions of the "nonlinear" stimulus train lasting about one minute. On this basis records across subjects and across time could be better compared not just for emissions present or absent, but for their detailed content. The signal-to-noise improvement thus afforded is around 24dB which is useful when the noise level is typically between 20 to 30 dB SPL. During this averaging process, the ILO88 screen displays the correlation coefficient ("Waveform Reproducibility%") between the two collection arrays A and B. As the two responses "grow out of the noise" so does the correlation coefficient history rise which is shown from 50% towards 100%. In individuals with strong emissions (most typically young women) the rising curve rises rapidly and reaches near 100% within a few seconds. In individuals with lower emissions this rise occurs more slowly, to the point that with absent emissions, it does not rise at all. Most interesting is the in-between case where the curve rises steadily until it drops back to a lower level and begins its rise again so that the final net value reached is lower than it would have been. It is highly suggestive of a mechanism which is sitting in a "State X" initially allowing a high rate of rise, but then it abruptly slips into "State Y" then State "Z" etc., where the instantaneous correlation coefficient and the rate of rise is lower. Such apparent "losses of traction" (hence the analogy with manual car transmission) are common, indeed possibly the majority of recordings. The effect suggests that the repetitive click stimulus train is itself responsible for some level of fatigue[30].

This phenomenon raises issues as to whether DPOAE display the same kind of phenomenon? Yes indeed they do. Typically emission recording procedures throw away segments of the record which contain artifacts, again non-specific artifacts, mostly epochs of the sampling which contain large transients. Also DPOAEs show phase shifts of the 2f1-f2 component for conditions associated with changes in pressure within the cochlea[31]. TEOAE have the advantage that the slipping clutch effect is obvious in time. By comparison DPOAEs and SFOAEs such an effect is not so obvious; it needs to be interpreted with the framework of models, which typically assume recording conditions are Gaussian and ergodic e.g. "place-fixed" and "wave-fixed" distortion products[32]? In general only the final average of any recording is kept; monitoring slippage of phase between subsegments is not generally carried out, indeed there are serious technical issues[22].

The so-called "half-octave shift" occurs when the ear is subjected to a high level tone[26]. The loss in hearing sensitivity does not occur at the frequency of exposure, it occurs at typically a half-octave above. The suggested explanation of this fatigue effect is that the cochlear partition responds to loud sound by changing its stiffness so that the place of highest trauma is shifted basally from the place mapped at low intensities[27]. After the exposure it takes a higher frequency tone to show hearing deficit. This item actually raises the issue of what information the TEOAE is actually providing in respect of the place of origin of any emission component. Are emissions capable of representing such a half-octave shift? The last part of this presentation considers *whether the phenomenon responsible for the half-octave shift and the slipping-clutch phenomenon are one and the same*. If this is the case we have clear evidence of dynamic mapping in the cochlea which affects not just high level responses. The lower level TEOAE stimuli could also constitute a fatiguing effect[33]. We will consider evidence to suggest that TEOAEs are directly telling us something very important about how noise and music fatigue affects cochlear mechanics. In particular it could also be telling us why auditory data are so variable.

2.2 Dynamic mapping of frequency to place? A potentially important third form of mechanical activity.

We hypothesise that this map plasticity with high level exposure represents a general capacity of the mammalian ear to use map shifting (or perhaps more generally, dithering) to average over local small lesions of the sensory epithelium thereby protecting its function by spreading the focus of exposure[34]. Morphological studies have shown that pure tone fatiguing exposures are spread over a longer length of the cochlear partition than might be suggested from the spatial extent of a tuning curve[35].

A mechanism for this map shift is suggested by many studies which examine the tensioning effects of the type IV fibrocytes in the spiral ligament[36];

they are very active and possess multiple roles[37]–[39], not the least of which is coping with tissue inflammation at the anchor point of the basilar membrane which occurs in response to traumatic sound exposure[40]–[42], [42]. If the noise-fatigue is affecting the fibrocytes, chances are that it is affecting the radial tension on the basilar membrane. It is consistent that the greatest inflammation which they display is at the base of the cochlea, where such tension is regarded as highest. The hypothesis is that the many processes which are working to reduce the inflammation of the fibrocytes will be working more slowly than the processes causing fatigue in the first place. Such a recovery process is of clear relevance to the recovery of tension. What are the potential implications of tension control in respect of mapping and the half-octave shift?

3 A SPATIAL VIEW OF COCHLEAR TUNING

The *cochlear amplifier* hypothesis has become a fixture of the literature of cochlear physiology[43] because it nicely fits into two niches: it is an explanation consistent with the nonlinear compression and sharp tuning peak, e.g. [41], [44] and when it fails it can, with caveats, be replaced by an external amplifier. However the really remarkable thing about the *cochlear amplifie*r hypothesis is that, outside of its original context [45], it has led to no rapid improvement in any explanation of any of the target issues being discussed at this meeting. It does not address auditory psychophysics such as two-tone phenomena, pitch shifts with tone level, nor explain the reason for tails of the tuning curves. It does not explain the "half-octave" shift, nor the EEH pertaining to auditory fatigue[9][19][19], [46]. On the clinical aspects of noise-induced hearing loss it appears to have little to contribute. This seems to be because of two constraining assumptions at the outset, that 1) what is important is how the OHC activity accounts for perception of the lowest level stimuli, not how it handles sound overexposure, and 2) the assumption that the frequency-place map is fixed in the manner described for only the lowest sound levels for many species[47], [48]. This is of course, not to say that viewed in retrospect, the cochlear amplifier theory and all of the resulting experimental results such as negative capacitance will not fit neatly within a more holistic explanation of all these phenomena.

The modelling of the cochlear amplifier has traditionally been conceived as a resonant process using negative damping to achieve the level dependent peaking[45][49]. One question never answered is why the tuning curves, either neural nor mechanical, are *not symmetric about the best frequency (BF)*. The second-order models primarily consider the sharp tip of the tuning curve, but no model yet has addressed the nature of the sharp cutoff on the high frequency side. The feature of interest to me, ever since I started studying the physiology of cell excitation, is this obvious asymmetry of the tuning curve — the low frequency side of the tuning curve is concave-down whereas on the high frequency side the curvature is inverted. This is a stable characteristic of mammals. The mammalian tuning curve is remarkable because of its *asymmetry* not its symmetry. Why should this be important?

When we look at cochlear mechanical tuning we see a frequency tuning curve with a sharp peak with an associated phase lag which is higher at high frequencies than at low frequencies[44]. This means that the high frequency response is following the low frequency response up to 3 cycles later. If hair cells fundamentally work as exponential charging and discharging devices, this implies the initial trigger of OHC activity is coming from the stapes causing a response at the basal flank of the excitation region, viz the *low-frequency side* of the tuning curve. i.e. The low frequency side represents some kind of trigger, whereas the high side some kind of OHC recoil to that response.

The slopes are low on the low-frequency side, but may exhibit 200dB/octave on the high frequency side. To say there is a negatively damped resonant process which fits the tip with the appropriate Q_{10dB} does not capture the essence of the whole tuning curve which to me looks like a hair cell charging then discharging in time, so there must be a limited time aperture during which charging is possible. On the high-frequency side of the neural tuning curve there is no stop-band. It instead displays a cutoff, i.e. there is a frequency about a third octave above the BF, at which any tone, no matter how high its level, will cause our neuron to fire. No second-order model displays such a cutoff. For such a precipitous cutoff to exist instead suggests that the excitation pattern is sliding longitudinally beyond the detectability of the neuron being tested and by implication the hair cells as well. When one looks at the growth curves in either the tuning or the mechanics, the slopes (dB/dB) strongly suggest that they too are the result of some form of longitudinal slippage. Indeed looking at these growth curves one can even see these reaching 0dB/dB and even negative slopes in the region of this high frequency cutoff again suggesting that tuning occurs as a direct consequence of dynamic mapping[34]. Therefore we need to be thinking of the tuning process in terms of the space-time dependence of the flanks of the excitation pattern.

3.1 Modeling the excitation region

Let us begin the model introduction by considering just the low-frequency side of the neural tuning curve, the part of the tuning curve associated with minimum phase shift — established by the first encounter of the neuron with the traveling wavefront. It is after this encounter that OHC activity develops[50]. When the curve is transformed to spatial co-ordinates the shape is found to be invariant[51]. The nonlinear curve-fitting exercise across 4 species (5 extrapolated to bats) was found to have very low errors. Considering the variability of most auditory data, finding these low errors seemed extraordinary. This says that the nonlinear curve fit is very tight, suggesting that the curve strongly characterises shape representing the onset of the OHC response in space-time. Taking into account map scaling for each species, the shape pertaining to the initial response of the activity, remarkably, is fixed by only one variable – the sound pressure of the frequency of the scanning tone (Eqn. 1)

$$\mathbf{\Delta x} \approx \mathbf{p}^{\beta} \qquad (1)$$

where **Δx** is the distance along the cochlear partition from the asymptote (the place for this neuron's characteristic frequency, CF – see Fig. 4). **p** is the minimum sound pressure (dB re 20μPa) required to reach threshold (and by implication evoke a minimally detectable motile response) and **β** is a scalar, which, considered across all species and their respective map gradients[44] takes a value of approximately 0.5. Our equation reduces the complexity of the active segment of the tuning process, to the position of the basal flank of the excitation region as a function of level. Across all mammals this position is simply connected to the threshold sound pressure at any pure tone test frequency (see Fig. 4).

The relationship makes more intuitive sense if we recall that such tuning curves are plotted not for the condition of constant input (constant stimulus voltage or SPL) but for a condition of constant response. The constant quantity is the triggering of activity of the primary afferent nerve, and by inference triggering of OHC motile response[52]. As sensitivity decreases, it takes more signal to trigger such activity.

The field response to this derivation has been non-existent, and indeed it has taken the author more than a quarter century to figure out what **p** might signify.

The clue has come as a result of all the recent work on the spiral ligament and why there should be radial tension in the basilar membrane. Traditional cochlear models assume this tension is passive. The tension has been measured as zero which puzzling considering the need for the basilar membrane to be maintained at some transverse position. The compliance of a trampoline will be strongly dependent upon the tension with its plane. So if there is any tension at all, it is necessary to consider how that is established and regulated.

As observed above the Hensons described the evident capacity of the fibrocytes to generate tension in the basilar membrane. This constitutes a third form of mechanical action in the mammalian cochlea in addition to the mechanisms in the OHC [43]. Force generation is likely the result of just one of their many roles which includes water transport. In turn this tension will establish basilar membrane stiffness [53]. We know that the sound level has a profound effect upon energy consumption by the stria vascularis[24] and that this process is not only strongly age-dependent but is affected by hypoxia but also may be ameliorated by L-NAC[52].

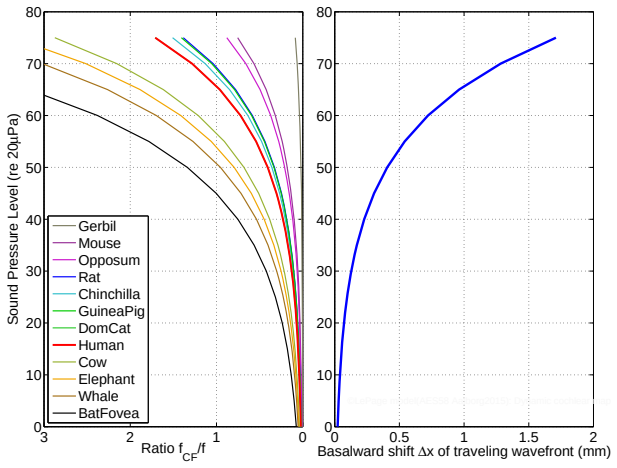

Figure 4. Set of curves representing the point at which the OHC motile response is triggered as a function of frequency and level (dB SPL) taken from representative tuning curves of several species (model from LePage, 1987c, values from LePage, 2003). In sharp contradistinction to the Gold resonant model which does not address NIHL, the tuning is replotted as a frequency ratio, the abscissa thus represents distance along the cochlear partition from the peak place designated by very lowest neural thresholds. The model hypothesises that the mammalian frequency-place map is *normally a multi-valued function* i.e. X = X(f, L) where f is the stimulus tone frequency and L is the tone SPL. In the right panel Δx says *where* the traveling wavefront (relative to its designated place for lowest level threshold) will first trigger an OHC response (minimum phase condition). (The precise scaling of the abscissa is under renewed investigation). The so-called "half-octave shift" represents the maximum extent towards the base that the map may be shifted for the highest tone level. Are we looking at the function of a lens?

If Eqn 1 represents sound level is there any reason why this cannot be generalised to ambient noise level? It would make much sense of having tension if the cochlea could protect itself by changing the position of the excitation region to defocus the acoustic energy. Then we realise that carrying out a threshold test is a special case of ambient noise. In a sound booth where the background noise level is low, the threshold for a pure tone *is* at ambient noise level by definition. For the limiting condition of lowest-level ambient noise (sound chamber) the place where the threshold occurs will be the *most-apical place* corresponding to the *highest radial tension* in the fibres of the basilar membrane. *For every other condition on the spatial curve, the ambient noise level is (so far as the cochlea is concerned) defined by the level of the test tone!*

This scheme allows that varying the tension in the basilar membrane high acoustic levels can be defocussed, while low levels require the energy to be focussed on just a few hair cells. Our curve in Eqn 1 describes the function of a lens. The quiescent state of tension, we hypothesise is indeed set by the *level of ambient noise.* By induction we assert we can generalise our curve to represent ambient noise level. Equation 1 takes on significance when we register that the frequency-place map is likely multivalued:

$$\mathbf{X = X(f , L)} \qquad \mathbf{(2)}$$

where **X** is the place of OHC trigger, **f** is the tone frequency and **L** is the background noise level. As the background noise level rises, the basilar membrane tension *decreases* and the *place for any test frequency moves towards the base.* Thus we can look at Eqn 2 and the curve and it describes the function of a lens. By changing basilar membrane tension we are effectively changing the refractive properties of medium and a compressive wave traveling through that medium is going to display dispersion. We can not only introduce the notion of the **"cochlear lens"** we can attach some timing to its focus or its dispersion of high energy.

In the left panel of Fig. 4 the gradient of the curves has the dimensions $\mathbf{T^{-1}}$. In other words, changes incurring high sound levels will happen quickly, whereas changes approaching very quiet conditions will be slow with rise in slope. The nature of this asymptote suggests that obtaining audiometric threshold is inherently a slow process. In respect of recent noise exposure, this is consistent with expectation that a protective response, tension may be dropped quickly with onset of loud sound, but it will be restored only very slowly after removing the noise source, e.g. leaving a coal mine, an aircraft test facility, a night club or a rock concert. The recovery process is thus characterised as a restoration of tension to the radial fibres of the basilar membrane as anti-inflammatory processes take effect[54][55].
This hypothesised scheme still requires the appropriate tapering of the basilar membrane mass, stiffness and damping, but it is proposed that the presence of a tone is locally perturbing these values. This is a revolutionary concept in that it is suggesting that when a pure tone audiogram is being conducted, the *place* of the neuron most stimulated varies not just with selection of the tone frequency, but also with the tone sound level. The place-frequency map is not fixed in the conventional sense defined by the map obtained from HRP-tracer studies[56][57][58][59]. These maps validly represent the state of the common variable (tension) under the lowest noise level conditions. Such conditions give well-behaved map functions[48].

However, such studies do not address the problem of what the tonotopic map looks like at higher sound levels. The lowest levels are used because these give the least variable maps. *It is implicitly assumed that the spatial location of the peak of the travelling wave envelope is unique* i.e. the same for high tone levels as for the lowest levels. This equation (1) allows us to suggest a very powerful new insight about cochlear processing in general, and why noise-response data are so variable in particular. We have simplistically regarded a single neuron as having a characteristic frequency. It is deeply engrained in our thinking, e.g. from cytocochleograms[35] which show that the percentage distance along the cochlear partition is uniquely associated with a particular frequency. *Distilled, the curve (Eqn. 1) describes how any point on a cochlear map shifts longitudinally as a function of sound level.* Such shifts are most important at high sound levels, but will still be affecting measurements at the levels at which otoacoustic emissions are obtained.

Thus, the half-octave shift is not something that is just a response to loud sound exposure. The mechanism responsible for it appears to be an integral feature of the coding operation of the mammalian cochlea and is responsible for the tuning. It describes for a tone at any given level at what point along the partition a particular tone will evoke a mechanical response from the OHC. If the sound level is reduced, the place of that active response moves towards the apex. Thus the maps for each species describe not the unique place for any frequency but the most apical place, the lowest threshold place.

What can be said about the active region? The part of the tuning curve which represents the activity of the OHC corresponds to the half-millimeter or so

region during which the "amplification" process is occurring[60]. As discussed in the article[51], the gradient of this curve tells how quickly the activity develops. At high sound levels, the OHC activity develops quickly, but at very low sound levels it is slow to take effect.

4 COROLLARIES

The dependence of place also upon ambient noise level provides a conceptual expression of the manner by which tuning might be achieved in a necessarily-redundant system — one which works normally despite loss of patches of hair cells. If the concept has merit perhaps there are new insights to explain age-old problems understanding auditory morphology and data.

4.1 Variability in behavioural sensitivity

For humans almost any frequency used for a pure tone audiogram (e.g. 0.5, 1.0, 2.0, 4.0, 8.0 kHz) will display a test-retest variability, even a few minutes later, of ±5 dB (HL) and this figure degrades rapidly such that at 6kHz, the variability is typically ±15 dB. Such variability is accepted as integral to the nature of the test[61]. The behavioural outcome is indicated by the subject response. The tester places a mark on the vertical line representing that frequency and it is implicitly assumed that the same neuron (or finite patch of neurons) delivers the same frequency information all the way to the cortex. The fact that one can generate similar maps in the cortex, does not mean there is a 1:1 correspondence between places along the basilar membrane and any of the ascending fibres. The simplistic manner of representing an audiogram ignores the fact that the actual ascending neuron for a given test frequency may not always be located at the same place. Some part of the variability in audiometric thresholds may be due to a longitudinal place variation and which inner hair cell is being stimulated on which occasion. The variability is possibly sourced to efferent motor programs which are integrating many more sources of information than pure the sound parameters[62].

4.2 Role for neural convergence

Remarkably large numbers of studies show that the exact place of entry of any tone stimulus to the auditory pathway is not very critical. This is because the auditory brainstem displays neural convergence. The key relay centres from the cochlear nucleus, the superior olivary complex, the inferior colliculus and the medial geniculate body (those in the cochlear nucleus and inferior colliculus most studied) display colateral connections[63]. Each neuron actually gathers firing information from adjacent neurons, often with complex timing relations. This manner of neural convergence means that to a large extent it does not matter which place is currently the site of highest stimulus at the periphery. So in summary the cochlear lens concept goes a long way to explaining variability in the audiogram and fine-structure effects. It also means that small lesions may not be detected by the audiogram. It integrates over a longer length of the cochlear partition.

4.3 Two tone effects and the critical band

While beyond complete treatment here it is necessary to say that the "cochlear lens" appears to function by establishing a form of potential well -- in the form of a local bipolar dc-bias on the cochlear partition -- so that there is superimposed another form of control of OHC operating point established by a pure tone[3], [64]. In the case of two tones with no spatial overlap there is no conflict. However, we will show distortion product data that if two tones spatially overlap, the local map distortion by each will be different; the dominant stimulus appears to "capture" a given place by biasing the OHC operating points affected by the non-dominant tone into a hyperpolarisation condition.

4.4 Pure tone audiometry versus evoked otoacoustic emissions

We have suggested the notion that because of the hypothesised history-dependent dynamic mapping pure tone audiograms will not show the fine structure of OHC damage. It is possible that TEOAEs (and DPOAEs) can show early warning effects by virtue that for constant stimulus conditions (and therefore constant effective background noise) there will be less variation in the generation site. Dynamic mapping does seem to be a natural candidate to explain fine structure in OAE[65] We will show TEOAE examples where the "slipping-clutch" data not only seem consistent, but extreme slippage can occur to the point that low frequencies are being emitted at short-latencies. These appear as large negative-going half-cycles during the stimulus period. In this case we show time-frequency plots with peaks at the same latency apparently shifting from high frequency to low. This is consistent with evidence from cochlear nucleus (AVCN) of cat showing a clear migration of the excitation pattern in response to noise trauma[66].

4.5 Place pitch versus periodicity pitch

A primary and important assertion of this talk is *a relaxation of the notion that the place pitch is resolutely fixed to a particular place*. It is well

known that the mapping of frequency to place varies across species, it varies with gender, it varies with noise exposure. Within limits, while the actually location of a tonal excitation pattern may not be critical, the *maintenance of a smooth (analytic) gradient of the map, is certainly critical for high frequencies*[48]. This function may not lie so very much in the structure of the organ of Corti so much as the tethering of the basilar membrane by the spiral ligament affecting its local resonance frequency[67]. This tethering is particularly critical in the case of echo-locating mammals, the bats and cetaceans (whales and dolphins) where the spiral ligament exhibits specialisations to set the gradient of the map to serve high frequency resolution at ultrasonic frequencies[68]. Periodicity pitch on the other hand is the temporal coding associated with firing of the primary afferent neurons in phase-locked manner to the acoustic vibration. It is independent of place.

4.6 Music-induced hearing loss (MIHL) versus Industrial NIHL

NIHL is mostly the result of a byproduct of another process, whereas MIHL is the product itself. Cutting back on exposure to preserve hearing is a strategy musicians can use, but at the risk of their careers and based upon what criteria? It requires the employer of the musicians to institute organised rostering of musician performance so as to abide by traditional models (such as conformance with the EEH), and indeed the Australian Opera and Ballet Company has instituted such a program in 2000. In general, freelance musicians do not have such a choice. Musicians are 'noble slaves' to their performance situation. Specially designed music-processing aids potentially offer an important new option for the musicians themselves, but not their audiences.

4.7 Ageing of the auditory system

The independence of place and periodicity pitch largely accounts for manner by which human hearing fails gracefully. Our OHC population can be decimated due to a lifetime of natural processes plus noise-exposure. The evident ravages of inflammation within the spiral ligament[38], [40] can mean that the stiffness of the cochlear partition cannot be maintained. The high tension in the fibres at the base of the cochlea is progressively released due to atrophy of the fibrocytes[69] and the map progressively shifts towards the base. The highest frequencies 10-20kHz will then simply drop off the end of the map. *Progressively, lower high frequencies are no longer mapped* resulting in a loss of high frequency reception as a function of age. Adding external gain to such a system will not help in the intended manner assuming all frequency channels are cascaded and independent. This is because an essential nonlinearity in cochlear mechanics is thus our modelled physical shift of the place with intensity. It skews *the auditory pathway* right within the cochlea. So this scheme involving the fibrocytes tends to account for why hearing aids continue to cause discomfort after all the OHC are gone and why lessening the gain is a better tradeoff to retaining speech intelligibility[70] through prolonging high frequency responses, otherwise lost using higher gain.

In the aged cochlea, the remaining basal neurons (now coding low frequencies only) can nonetheless phase lock, so periodicity pitch is unaffected. People with very advanced OHC losses, and hearing aids, may still enjoy their music.

5 CONCLUSIONS

Viewing tuning from a spatial standpoint appears to open many new possibilities to explain auditory data (LePage & Olofsson, in preparation). The realities of the decades of modern research into molecular biology have produced very detailed accounts of the manner in which cochlear damage occurs and it's not just the hair cells. There are scores of mechanisms at play. Perhaps the new main story for NIHL is about the cells in the spiral ligament which actively tension the basilar membrane -- the type IV fibrocytes. These appear to be controlling the mapping between frequency and place. They have a hard time coping with loud sound exposure. Their failure is the main reason for loss of high frequencies. This insight places some imperatives to confront the traditional notions upon which field of audiometry has been built, viz the assumption that high frequency coding is preserved even if the cochlear structure is not. In this respect hearing aid technology (and to a lesser extent cochlear implants) is currently based upon the notion that high frequency channels continue to exist and just need higher stimulus level. Certainly the "vanishing map" idea with increased acoustic trauma tends to explain the steep law of diminishing returns with managing progressive hearing loss and hopefully provides a better basis for understanding acoustic trauma and the nature of risk in respect of MIHL. It seems that not only the OHC may be used to carry out an early warning assessment of imminent risk of hearing loss, TEOAE themselves may directly give a measure of map shift without having to deliver enough trauma to produce a full half-octave shift. Such a measure may be a direct measure of individual susceptibility to hearing loss.

We appear to have a more holistic model of cochlear function, in terms of energy focus or diffusion which changes cochlear response

according to sound level. This makes particular sense in respect of high levels of music exposure, particularly amplified music. This evidently carries a high cost in terms of cellular processes even if there is no acute pain involved. The OHC may be lost, allowing the justification of using external amplification. However, the questions which really need investigation are 1) what levels of mechanical bias may exist in the cochlea to make OHC behave initially like they are dead and 2) how to remove such a bias in the first instance. A new issue is whether the fibrocytes of the spiral ligament are the source of discomfort due to amplification and whether hearing aid prescriptions can be better based upon map sliding measured using TEOAE.

Until now amplified music production has largely been dictated by artistic issues and affective response [71], [72] without having to answer questions as to user health risk and producer responsibility. We suggest this is because *there has been no adequate basis to say how much is too much*. The take-home message is that whereas acoustic music may produce high levels of sound which may affect hearing its damaging effects, as a modality it appears milder by comparison with amplified music, particularly music enjoyed via headsets. We have described a new approach to untangling the issues between cochlea aging and noise trauma, and suggested that judicious of otoacoustic emissions can be used to track progressive latent damage.

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