OCCUPATIONAL NOISE-INDUCED HEARING LOSS: ORIGIN, CHARACTERISATION AND PREVENTION

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ABSTRACT: Permanent hearing loss due to noise exposure constitutes premature aging of the ear caused by depletion of the outer hair cell population. Describing it is complex because many other factors also contribute to this depletion. Managing it is still more difficult because reduction of sound levels reaching the ear is not an adequate strategy by itself. Adequate prevention of any disability is only afforded by predetermination of individual risk coupled with comprehension of its severity. Otoacoustic emission data show that neither have traditional hearing tests given early warning, nor has the terminology 'mild hearing loss' indicated that extensive cochlear damage has accumulated.

1. ORIGIN

The cause of noise-induced hearing loss is, by definition, over-exposure to loud sound. The condition was first described over a hundred years ago when Dr. Thomas Barr of Glasgow realised that boilermakers suffered premature loss of hearing. In modern times the condition is regarded as a very complex problem. Last year it cost over one hundred million dollars in direct compensation costs (Macrae, 1998) and indirect legal costs as well as all the social consequences of poor communication at a personal level.

The primary factor responsible for Noise-Induced Hearing Loss (NIHL) is premature depletion of the three rows of cells in the cochlea called the Outer Hair Cells (OHC). The motor activity of these cells (dubbed the "cochlear amplifier") is essential to normal hearing. When the OHC are subjected to very loud sounds (120 to 130 dB SPL), the basilar membrane on which they sit can be forced into vibrational amplitudes approaching the size of the cells themselves, causing shear forces rupturing cell membranes or, for still louder sounds, producing complete disruption of the surrounding structure. In the mammalian ear new cells do not re-grow – the damage is permanent. Typically the spatial pattern of permanent loss of cells is related to the frequency and level of the sounds. An exposure to one-third-octave white noise for years will typically result in heavy loss of OHC of up to one tenth the length of the basilar membrane; repetitive impact noise can take out one third the starting population OHC (about 12000 in each ear). This adds to the scattered loss of OHC that occurs with aging beginning from birth, with the cells at the high frequency end being more vulnerable.

Recent research has focussed on the many mutually potentiating influences (McFadden, 1986a; Morata, 1998) which act upon the ear reducing the population of active OHC. These include hereditary factors (several lines of defective genes are being studied) and the protective presence of melanin in the cochlea (originally assessed using eye colour). Then there are the acquired defects such as due to maternal infection during pregnancy, birth trauma leading to hypoxia, infections, particularly during the first decade of life, plus a whole gamut of toxic influences ranging from heavy-metal poisoning, naturally occurring toxins and commercially-produced chemicals including solvents such as benzine and toluene (Johnson, 1994) to antibiotics and loop diuretics. To these we have to add physical injury, due to head impacts and raised barometric pressure. In the past these many effects have been regarded as outside the area of interest. The reason for considering all these "unrelated" effects here is that we now suspect that all these other synergistic factors (let us lump them together as determining "individual susceptibility") are swamping the main noise effect we are trying to measure, confounding attempts to control the rate of accumulation of cochlear damage by setting limits on sound exposure. A second reason the problem is difficult to manage is that we have no way of isolating occupational noise exposure from any other kind of excess sound exposure, eg. music exposure – it all appears to add up to deplete the OHC population.

2. CHARACTERISATION – OLD AND NEW

Typically the first clinical signs of noise-induced hearing loss are indistinct speech perception, particularly in conditions of raised background noise, while pure tone audiometry first reveals a "noise-notch" at 4 to 6 kHz. It is generally accepted that this dip in sensitivity occurs because the ear canal and drum has a resonance at 3 to 4 kHz emphasising this component of any sound to peak levels at the ear drum of up to +20 dB higher than entering the ear canal and producing a loss of sensitivity at a higher frequency (McFadden, 1986b).

By the time a person seeks help for a noise-induced hearing loss the noise-notch may be no more than 25 dB in depth, and the person is accorded typically a 5 percent hearing loss (Macrae, 1998). In traditional compensation parlance the disability is termed "mild" by comparison with possible moderate and severe noise-induced hearing loss. Despite this, it is not the loss of hearing sensitivity that drives sufferers to seek help. Ironically, the most common symptom first presented is the loss of voluntary ability to distinguish between sounds of different source location or frequency, particularly under conditions of multiple sources, reverberation or moderately raised background noise. There exist audiometric tests for cochlear selectivity, which is essential for voluntary selection (both pure tone masking and speech in noise tests). However, until now this initial and significant form of hearing disability has not only been too

time-consuming to test, it has been still harder to describe in lay or even legal terms.

The inherent difficulty in raising awareness of, and preventing the most common form of hearing loss is describing what the average person wishes they had avoided only after the symptoms of loss of selectivity developed. Yet there is a simple experiment that any person can conduct on himself or herself which we suspect better describes hearing loss than simply reducing the volume to mimic loss of sensitivity. Turn on the radio to a talk program and have the volume at normal speaking level. Now try to hold a conversation with someone. Next, turn down the radio and experience the relief. Finally, turn it up again and imagine the frustration of never being able to turn the radio down in situations of such conflict. Hearing loss is so subtle and so poorly appreciated because the nature of the complaint is qualitatively no different from the experience of a normal-hearing listener. We learn from birth to wait for a gap in the conservation before beginning to speak. It is not so much that competing sounds are "masking" what our listener is trying to "hear", it is more the case that once the signal-to-noise ratio drops below about 10 dB (where here "noise" is defined as any signal we are not interested in) even the normal hearing listener doesn't cope too well. However, once the active OHC processing power is degraded the central task of voluntarily selection is disabled. The onset of hearing loss is so subtle because qualitatively things are the same as for the normal listener. Quantitatively, however, the presence of competing sound affects the damaged ear much more. For the person with a problem with selection, if they cannot remove the competing sound, such as trying to "hear" in a crowded room, they cannot cope.

The important question investigated at NAL since 1989 is whether the otoacoustic emission technique can provide not just a fast objective measure of hearing ability (LePage et al, 1993), but yield a parameter which better indicates loss of frequency selection ability than behavioural tests. Otoacoustic emissions being objective, there is a good likelihood that they will indicate loss of OHC function as a general slowing of cochlear activity. Further impetus to test this idea came from an animal study by Altschuler et al (1992), in which it was shown that while the inner hair cells and just one row of OHC remain intact, hearing sensitivity can remain normal, which suggests that the mammalian ear uses redundancy, or excess numbers of OHC to cope with progressive aging of, and damage to the hearing organ. Since audiometry is an untimed test it gives absolutely no indication that the loss of OHC amounts to a significant reduction in the rate of adjustment to sound level. If such redundancy is demonstrable in humans then a possible correlate may be the net level or reduction in the rate of activity of the outer hair cells before symptoms present.

A transient otoacoustic emission is the sound re-emitted into the ear canal due to an incident click. Important to this endeavour is the understanding that this stimulus is just large enough to drive all OHC into saturation. The 40 μs pulse delivered to the earphone generates a click, which is preset to 80±1.5 dB SPL peak. Kemp has shown that this level obtains a saturating response suggesting that the net emission power should reflect the remaining number of active OHC. The resulting emission is typically 0 to 10 dB SPL and so signal averaging (sample period of 40 μs, duration 20.48 ms) is used to improve the signal to noise ratio by 24 or 30 dB, taking about one minute. Also because the click response will be determined by the characteristics of the external ear and middle ear as well, in the standard protocol, a method of differencing is employed such that clicks of two different levels are used and any linear component of the response is subtracted away leaving only the nonlinear response due to the level-dependent change in outer hair cell activity. Also alternate responses are summed into two arrays and the reproducibility between the final averaged waveforms is calculated. If the ear has a fast recovery from the previous click it will respond with high waveform reproducibility (a correlation coefficient of 1.0); if the ear is still recovering it will respond differently and the reproducibility will be lower, towards zero. It turns out that this parameter can be thought of as speed of recovery or more loosely, "reaction time". However, being a bounded parameter [-1, 1] and non-normally distributed, the waveform reproducibility is typically used to weight the sound level of the emission so that the net response is a sound level. In our experiments we have used a parameter Coherent Emission Strength (CES dB SPL, which is the average sound pressure multiplied by the square of the reproducibility) to quantify the average reproducible (or coherent) component of the emission sound level. Test-retest variability for CES is ±4 dB SPL (Murray et al, 1997).

By comparing strength of the emission with hearing thresholds for the same frequency range (1 to 4 kHz) there should be a range of emission strengths over which hearing sensitivity does not change. Figure 1 shows the results of a study of 505 ears (LePage and Murray, 1993) if the strength of the emission is compared to hearing level for the same frequency range (1-4 kHz). It is seen that most cases of hearing loss are on the left side of the figure for which the emission strength is below some critical value (LePage et al, 1994) less than 0 dB SPL. The notable exceptions to the pattern, points on the right side of the figure, were cases subsequently confirmed as belonging to two categories: those with a hearing loss which is more central in origin, or those from individuals who at first did not correctly indicate their true thresholds. Naturally the figure does not include points from newborns for which CES values have been recorded up to 38 dB SPL. The complete picture including neonates suggests that there is a range of CES (about 80% of the total) for which the hearing level does not change, supporting the notion of redundancy in OHC motor capacity. This suggests that there is a period of accumulation of latent or subcritical damage during which a person who has had occupational exposure for some years may not be distinguished audiometrically from one whom has led a noise-free life.

In turn Fig. 1 may explain why in the new standard (AS/NZS1269:1998) emphasis upon monitoring hearing thresholds in occupational workers has been reduced in favour of higher attention to noise-level management. Regular hearing tests not only provide no early warning, they essentially do not measure the parameter which most

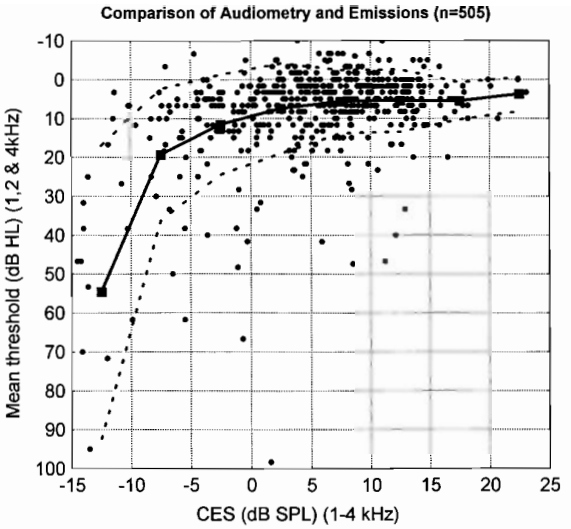

Figure 1. Comparison of behavioural and objective measures of hearing for 505 ears. The ordinate is a 3 frequency average hearing level (at 1, 2 and 4 kHz) as usually plotted in audiograms versus frequency. The abscissa is Coherent Emission Strength (CES dB SPL) - a measurement of the reproducible component of power of the click evoked emission. The heavy line and square symbols represent the mean value of the hearing thresholds for the appropriate 5 dB band of CES values. The dashed lines represent ±1 standard deviation about those mean values.

represents the disability – loss of selection. Our estimates suggest that a 5 percent hearing loss (Macrae, 1988) may constitute in excess of 80% loss of outer hair cells while a 20 percent hearing loss, the most ever typically presented in cases of compensation for noise-induced hearing loss, represents almost total loss of OHC processing power, certainly the case for frequencies above 1 kHz. The advent of the rapid, objective, non-traumatic otoacoustic emission test clearly has highlighted the inadequacies of traditional approaches to occupational noise-induced hearing loss and of compensation issues.

While many hundreds of studies conducted using Transient Evoked Emissions (TEE) have concerned themselves with neonatal screening, Narelle Murray and I have been questioning why the problem of noise-induced hearing loss is inherently difficult to manage and have proceeded to separate the normal aging effect from any accelerated aging effect. We believe the otoacoustic emission results have again shed new light.

Using the more sensitive technique has revealed that population variance in emission strength is huge. Figure 2 shows a scatter plot of CES for teenage and adult subjects between the ages of 10 and 60. These data represent the largest transient emission database (2038 people, pathological cases removed) so far presented in the literature. At any particular age, the range of emission strengths is about 80 percent of the total span of 40 dB. The high level of scatter implies that there are significant additional sources of variability never previously seen in otoacoustic emission data, or alternatively discounted. Of immediate concern is that the scatter represents a problem in the measurement technique (such as variability of transmission through the middle ear) so that the variation is not due to variation in OHC motility (for whatever cause). After nearly a decade of study at NAL we suspect that the scatter in these results irrespective of age is real and not attributable to some form of measurement error or misinterpretation of the origin of the emissions. The variability is more likely to reflect some individual component of the OHC response such as efferent involvement in the determination of susceptibility or maybe systematic variations in conditions of cochlear regulation (LePage, 1993).

Comparing Figs 1 and 2, if subjects with emission strengths below some critical value are more susceptible to acquiring a hearing loss than those with very high values then the scatter indicates that many young people are at imminent risk of hearing loss. Also since the relationship in Fig. 1 is monotonic, we suspect that any lowering of emission strength represents increased risk. Indeed we have studied the apparent dip (Fig. 2) in the values in teenagers and young adults with normal hearing (LePage and Murray, 1998) and conclude that despite the scatter, there are highly significant effects of certain kinds of noise exposure such as personal stereos. The sloping lines show the results of a linear regression for left and right ears separately (left below right) and indicate a significant decline with age. Our current studies also include a cohort in whom we are tracking both TEEs and pure tone audiometry for confirmation.

The interpretation of the scatter (Fig. 2) we are investigating is that it represents high variability in individual susceptibility to hearing loss due to the very many synergistic factors mentioned in Section 1. These must be taken into account in any trend analysis in which the independent variable is aging effect, or noise exposure, or effect of toxic substances or head injury and so on. Although our longitudinal epidemiological study has made several assumptions, our data support the notion of redundancy of OHC function. Since mammalian OHC do not regenerate when permanently damaged it would almost appear that, like many other systems in the human body such as that involved in insulin production, the evolutionary process has arrived at a cochlear structure with considerable excess capacity. We appear to have many more OHC at birth than we need to hear normally (or in terms of the cochlear amplifier hypothesis, than we need to maintain adequate gain) so we can afford to lose the greater portion of them before any disability is evident.

3. PREVENTION

Previous Australian Standards (eg. AS1269-1970) have specified three basic aims: 1) reduce the level of the noise being produced by machinery or enclose it to keep the sound inside the enclosure, 2) if silencing is not possible to an acceptable level then reduce the level of noise reaching the ear drum with obligatory hearing protection devices (ear muffs or ear plugs) and 3) monitor the hearing levels to identify those at risk for noise damage. Until recently most efforts to limit sound exposures have not been supported by convincing evidence of a reduction on numbers affected (Royster, 1993). Why? Is it simply a problem of more effectively enforcing or

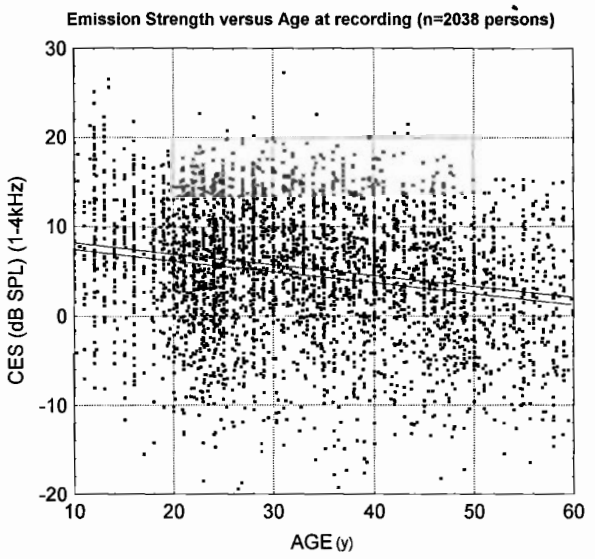

Figure 2. A scatterplot of Coherent Emission Strength as a function of age at the time of recording in a population of 2038 people reporting no current hearing problems, left and right ears. The regression lines indicate a slight but significant decline versus age (left below right). The important features are the normally large scatter in values of emission strength and the fact that having low values can occur at any age, reflecting high risk for hearing loss.

motivating employers and workers to conform to guidelines, or is there a more basic reason?

The key to the success of any prevention program is early warning. In the past behavioural hearing tests such as pure tone audiometry were the only way of monitoring hearing and suffered the inherent problem of trying to use the same parameter both as a measure of disability and also as a predictor for that disability. We now appreciate that behavioural tests have provided no early warning. Accordingly, the title of the latest Australia/New Zealand Standard AS/NZ1269-1998 has been renamed "Occupational Noise Management" to reflect that more emphasis is being given to reducing sound levels at source and less emphasis given to the monitoring of the onset of hearing loss by conventional means, but foreshadows the use of otoacoustic emissions in the future.

The rationale of the new standard continues to be based on the logic that limiting the peak sound levels in the workplace say from $L_{Aeq,8h}$ values of 90 dB to 85 dB SPL must limit worker exposure and therefore should produce a reduction in the incidence of NIHL. It is too soon, however, to tell if these latest measures are effective. The basic principle which has guided the trade-off between acceptable sound levels and time of exposure dates from the so-called Equal Energy Hypothesis – a 3 dB increase in sound level equates to halving the maximum duration of exposure, the point of reference now being an $L_{Aeq,8h}$ of 85 dB. 88 dB equates to a 4 hour limit and so on, to say, 115 dB at which level the rule limits exposure to less than a minute. Set in the context of the discussion in Section 2, we can see this traditional rule is important for protecting the bulk of the population, but it may do very little for the most susceptible people. Without them being identified and targeted for special attention they will likely still be the first in any program to suffer a hearing loss and so their management program will appear to be ineffective, whereas it is only breaking down by failing to detect those most at risk.

Much effort has also been expended on obtaining an adequate method of rating hearing protectors so that the type of device can be matched to the application, not just how its rating must depend upon how they are worn in practice, but taking into account how steeply the rating must be degraded for intermittent use. Because of tremendous variability in real ear attenuation, debate continues as to the best method of rating them so that at least most of the population of users has their hearing protected. The predominant rating method in Australia continues as the so-called "SLC_{80}" – a nominal "real-world" value of attenuation that derived from the pioneering work of Dick Waugh at NAL. This method of rating is designed to stem hearing loss by protecting the bulk of the noise-exposed worker population, but our concern here is for workers who may already be most at risk – in Figure 2, those with critically low emission strengths. The traditional approach may not do much for preserving their hearing because workers whose OHC processing power is reduced may be the very people who feel their immediate need to hear is being compromised further by the wearing of protectors. In addition the notion of redundancy means that any measure designed to reduce the incidence of occupational hearing loss may not be manifest for decades. We are therefore optimistic that the otoacoustic emission approach may be an important adjunct to hearing conservation strategies. Clearly we need to continue to reduce overall rates of accelerated depletion of the OHC population by reducing sound levels, fully realising that irrespective of that measure the most susceptible people will still likely be outside that level of control. Hence we are working towards a new strategy for adoption sometime in the new Millennium. We advocate a two-pronged approach: 1) to reduce sound levels, thus protecting the bulk of the population and 2) to introduce the more sensitive method of assessing the level of redundancy in OHC activity providing the capability of using limited resources to target workers most at risk in plenty of time for all concerned to consider all the career choices still available to them.

4. SUMMARY

We have shown that individual susceptibility may be hampering our efforts to show that industrial hearing conservation programs are worthwhile and we should continue to push for reduction of noise levels. However, it is unrealistic to expect to see an effect except in the long term using behavioural measures such as audiometry. Refinement of the new objective techniques such as otoacoustic emissions may provide a better handle on early warning in terms of the notion of assessing cochlear redundancy. If this new approach can eventually be used with more confidence to quantify the population of OHC in any ear, it is possible to conceive it may be used as a general screening tool for early detection such as has been applied to early warning of glaucoma. Finally

research into noise-induced hearing loss is leading to some exciting developments both in basic hearing science and in practical field strategies which may eventually substantially change the incidence of premature hearing loss.

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