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COCHLEAR MECHANICS, THE ACOUSTIC REFLEX AND NOISE INDUCED HEARING LOSS

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Abstract

In a noise environment devoid of impulsive sounds the ear responds as a linear system to levels sufficient to destroy it in response to energy input. In such environments a long time average A-weighted measurement will provide a suitable measure of exposure. In an environment characterised by impulsive sounds, however, it is proposed that the response of the ear is determined by the response time as well as the on-time of the acoustic reflex and the durability of the hair cells. Pickles[1] has stated, "the reflex is too slow to protect the ear against impulsive noise," which is true of a single impulse, however, in an environment characterised by many impulses the effectiveness of the reflex may also depend upon the durability of the hair cells, as well as the reflex on-time. It is proposed that the acoustic reflex can respond quickly enough to reduce sound transmission enabling the ear to respond as a linear system in the low frequency range to 2 kHz however, it cannot respond quickly enough to enable linear response in the high frequency range above 2 kHz. Consequently, in the high frequency range of 4 kHz, where the ear is most sensitive, the ear responds to impulsive sounds as a non-linear system where destruction of the ear is proportional to peak pressure. On this basis the well known"4 kHz dip" may be explained.

1. INTRODUCTION

In this article the ear will be characterized as an anatomical implementation of a mechanical sound analyser, which converts information that it receives into a dense code known as a Fourier transform for transmission to the brain at the relatively slow rate of at most 170 cycles per second which upon receipt, the brain converts into the sensation of sound and within physical limits location of the source. The pre-processing of the ear is crucial for a sense of hearing.

1.1 Noise induced hearing loss

Noise induced hearing loss due to noise exposure is recognized as a serious hazard and standardized procedures for quantifying the hazard have been formulated as *the Equal Energy*

Hypothesis (EEH) based upon the assumption that noise induced hearing loss is proportional to energy input to the ear**.** However, it has been known for more than sixty years that workers in industrial environments characterized by a generally flat A-weighted spectrum, such as weaving mills, suffer hearing loss at 4 kHz and higher frequencies inconsistent with the energy input concept, casting doubt upon its general applicability. Some musicians also suffer hearing loss at 4 kHz similar to that of industrial workers[2]. It also has been known for more than twenty years that the experience of industrial workers exposed to industrial noise does not describe the experience of musicians exposed to similarly assessed loud music. These paradoxes are readily explained when the role of ear's cochlear amplifier, based upon the use of variable damping for level control and its anatomical compromise based upon the response of the acoustic reflex, is understood.

1.2 Noise induced hearing loss and the biophysics of the cochlear amplifier

The auditory hazard posed by impulsive noise, particularly at frequencies higher than 2 kHz, has been experimentally confirmed over the past two decades, as has the failure of risk models based upon the Equal Energy Hypothesis (EEH) to predict the hazard. The mechanism responsible for the hazard, however, has remained obscure. Here an explanation is proposed based upon an analytic investigation of the biophysics of the cochlear amplifier. An anatomical compromise is identified which allows the ear to explore the external acoustic field of enormous dynamic range using variable damping but with the crucial restriction that damping must vary slowly to allow quasi-linear response to ensure that the ear may simulate linear response.

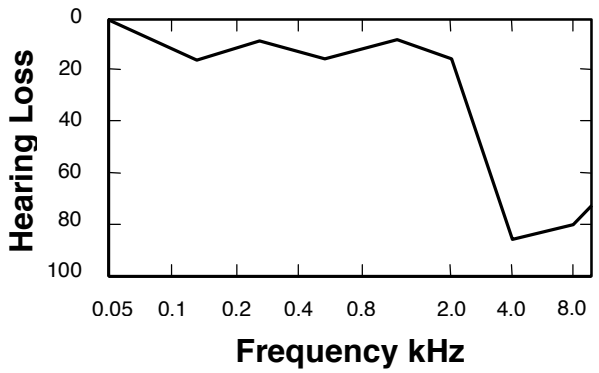

Figure 1. Audiogram of a workman after 10 years work in a weaving mill

The use of variable damping is made possible using an internal energy replacement process however, as will be shown, to use variable damping without deleterious effects on hearing damping must vary slowly to enable the ear to simulate linear response by responding as a quasi-linear system. To simulate linear response the compromise leaves the ear vulnerable to impulsive sounds of high level. The requirement that the ear must respond as a quasi-linear system, combined with limitations of the acoustic reflex, explains the apparent rule change in noise induced hearing loss between 2kHz and 4kHz, as evidenced by the notorious "4kHz dip" (see **Figure 1**).

It is proposed that in humans the acoustic reflex response is fast enough to reduce sound transmission long enough to protect the ear and ensure that the compromise is acceptable in the low frequency range to about 2 kHz. In the low frequency range the ear is able to respond as a linear system where damage is proportional to energy input. In the high frequency range above 2kHz, however, the acoustic reflex is too slow to reduce sound transmission to protect the ear and in this frequency range the ear remains unprotected and vulnerable to loud impulsive sounds, which may over drive and eventually destroy it. In this range where the acoustic reflex cannot provide protection a "Hearing Deterioration Index" may be defined which describes the observed non-linear response, noise induced hearing loss.

2. THE ANATOMICAL COMPROMISE

The apparent dynamic range of the brain (25 to 30 dB) is quite limited and its processing rate of at most 170 Hz is slow consequently in order to investigate the enormous dynamic range of the surrounding audible field (perhaps as much 120 dB) over a frequency range from about 20 Hz to 20 kHz, the ear's pre-processing is crucial for a sense of hearing.

Level control within the ear is necessary to accommodate the analysis of the external audio field within the relatively narrow dynamic range of the brain. As explained the ear uses variable damping for internal level control but to avoid deleterious effects associated with variable damping it is necessary that damping must vary slowly so that the ear may simulate linear response. The effect of the compromise and the limited protection provided by the acoustic reflex means that the ear has no protection in the frequency range above 2 kHz where impulsive sounds of high level are common in many industrial environments. In music, with exceptions, and in the natural environment such impulsive sounds are seldom or perhaps never present.

2.1 The role of the compromise

The discussion begins with the equation of motion of a section of the central partition described by Mammano and Nobili[3], repeated here as amended by Bies[4] for convenience as Equation (1). The aim will be to show that to avoid generation of sounds not part of the original stimulus damping must be slowly varying.

$$m\frac{\partial^2\xi}{\partial t^2} + h\frac{\partial\xi}{\partial t} + \kappa\xi = F^{\,j\omega t} \qquad \textbf{(1)}$$

In the equation the symbols are as follows: mass, m, damping term, h, stiffness, κ, force per unit length, F, time, t, displacement, ξ, and stimulus radian frequency, ω.

For the convenience of simplified presentation complex notation is used where the real part may be taken as representing the observed motion. The imaginary part is in quadrature and may be ignored. The fluid forcing field, $Fe^{j\omega t}$, is due to motion of the stapes and motion of all other parts of the basilar membrane. The basilar membrane displacement will be assumed cyclic, $\xi = \xi_0 e^{j\omega t}$. For the purpose here the damping constant will be assumed to consist of two parts, $h = h_a + h_b$ where h_b will be slowly cyclically variable. It will be assumed that h may range between its lower and upper bounds as follows: $h_a - h_b = 0.01$ and $h_a + h_b = 0.5$ then $h_a = 0.255$ and $-0.245 \le h_b \le 0.245$

Assuming the damping term is cyclically variable, $h = h_a + h_b e^{j\omega_b t}$ then Equation (1) becomes:

$$m\frac{\partial^2\xi}{\partial t^2} + h_a\frac{\partial\xi}{\partial t} + \kappa\xi = Fe^{\,j\omega t} - h_b\frac{\partial\xi}{\partial t}e^{\,j\omega_b t} \qquad \textbf{(2a)}$$

In turn Equation (2a) may be rewritten as Equation (2b), which follows.

$$m\frac{\partial^2\xi}{\partial t^2} + h_a\frac{\partial\xi}{\partial t} + \kappa\xi = F(\cos(2\pi t/T) + j\sin(2\pi t/T)) - h_b\frac{\partial\xi}{\partial t}(\cos(2\pi t/T_b) + j\sin(2\pi t/T_b)) \qquad \textbf{(2b)}$$

The presence of the second term on the right hand side in Equations (2a) and (2b) clearly shows that cyclic variation of the damping generates a second sound, which was not present in *the original sound and would have a deleterious effect upon the process of hearing.*

However, if $T<t<< T_b$ then the coefficient on the (RHS) of the second term in Equation (2b) is approximately 1 and in this case the equation takes the form of a *quasi-linear system*.

$$m\frac{\partial^2\xi}{\partial t^2} + (h_a + h_b)\frac{\partial\xi}{\partial t} + \kappa\xi = Fe^{j\omega t} \qquad \textbf{(3)}$$

The requirement that damping must be slowly varying, which requires that t/T_b must be approximately zero, is the basis for the anatomical compromise, which allows the ear to use variable damping for level control within the ear. Any proposed variation in damping may be represented in terms of a Fourier series of which the lowest order term of greatest period may be as considered here thus the conclusion based upon the simple case is quite general.

2.2 The effect of the compromise

It is proposed that the efferent system controls the level of damping based upon cerebral interpretation of information conducted to the brain by the afferent system. A time lag of the order of that typical of observed psycho-acoustic integration times[5], which range between 0.3 and 0.5 seconds, is implied during which period the ear must be free-running based upon previous instructions. However, during a period of slowly varying damping the ear is vulnerable to sudden loud sounds, which it may amplify rather than attenuate with catastrophic effect. Clearly the use of the anatomical compromise for internal level control requires protection, which as proposed is provided by the acoustic reflex in the low frequency range up to and including 2 kHz but cannot be provided in the high frequency range above 2 kHz.

3. AN ANATOMICAL FREQUENCY ANALYSER

In this article concerned with the deleterious effect of loud noise on hearing, the ear will be described as an anatomical implementation of a mechanical frequency analyser governed by the physics of the central partition. In turn, the central partition will be treated as a linear array of independent mechanical oscillators laterally uncoupled but strongly coupled through the incompressible fluid, which surrounds them. Consideration of the ear as a mechanical system shows that motility of the outer hair cells allows energy to be put back into a resonant oscillator during each cycle so that at the end of each cycle the resonant oscillator behaves as lightly damped. Alternatively, replacement of energy dissipated during each cycle, here called “undamping”, allows variable damping control of an oscillator.

Recent determination of the input impedance at the oval window has shown that it is resistive over the entire audio frequency range[6] thus the acoustic pressure at the window is converted into velocity response within the cochlear duct. Information storage in the “velocity response” of the basilar membrane and associate fluid allows the use of variable damping control for sound level control within the ear. Level control within the ear in turn enables the investigation of the external acoustic field of enormous dynamic range within the relatively narrow dynamic range of the brain. Variable damping control is used by the ear for the detection at threshold of resonance at the place of characteristic frequency in the cochlea.

Level control within the ear allows the ear to explore the external audio field surrounding it over an enormous dynamic range[7] but with the restriction, as explained that damping must be slowly varying to avoid deleterious effects associated with non-linear response. This is the anatomical compromise, which is crucial for a sense of hearing over a wide dynamic range called engineering compromise in the reference[4].

The use of undamping for internal level control is possible because (1) information is stored in the velocity response of the central partition (2) the natural frequency of maximum velocity response, the characteristic frequency, ω_N, is independent of damping while (3) the amplitude of maximum velocity response is inversely proportional to the damping expressed as the "damping factor"[8] and (4) the inner hair cells are velocity sensors as proposed by Billone[9], erroneously rejected by Russel and Sellick[10], Patuzzi and Yates[11, 12], Freeman and Weiss[13], and subsequently vindicated by Bies[14].

The properties listed and especially storage of information in the velocity response and not, as too commonly assumed, in the displacement response causing much confusion in the published literature, enables the use of the anatomical compromise, which is crucial for internal level control and our ability to explore the external audio field of enormous dynamic range.

4. NOISE INDUCED HEARING LOSS

Three examples of noise induced hearing loss will be considered. In the first example, hearing loss will be attributed to energy input in linear response and in the second and third examples, hearing loss will be attributed to high-acoustic pressure amplitudes in non-linear response.

4.1 The role of energy input in noise induced hearing loss

In this section it will be shown that the velocity response of the cochlea is quite linear to sound pressure levels sufficient to cause physical destruction of selected portions of the cochlear partition. The demonstration will be based upon the observation that a linear model can predict the observed distribution of hair cell destruction causing a hearing deficit in two cats brought about by prolonged exposure to an intense stimulus[15, 16].

When a narrow band of noise is introduced at the oval window at the basal end of the cochlear partition a wave will be initiated which travels to the place where the narrow band of noise is resonant. The resonant segment of the cochlear partition will be treated as a one degree of freedom mechanical oscillator. At the place of resonance the system damping will control the oscillator response and mass control above resonance will ensure that wave motion apical will cease. Following the reference[8] an expression for the normalized amplitude of the velocity, u, of the oscillator is obtained as follows.

$$u = \frac{\omega_N F}{\kappa}\left[(1/X - X)^2 + 4\zeta^2\right]^{-1/2} \qquad \textbf{(4)}$$

In the equation the symbols not previously defined above are as follows: characteristic frequency, $\omega_N = \kappa / m$, and the damping factor, $\zeta = h/2m\omega$.

The power introduced into a linear oscillator may be calculated as the integral over time of the product of the acoustic pressure in the fluid of the inner ear and the velocity of amplitude, u, of the cochlear partition. In the case of a narrow band of noise considered here, with the relatively high damping expected of the cochlear partition in passive response[4], it may be assumed that the acoustic pressure expressed as force per unit length, F, is constant. As the

fluid in the cochlea is essentially incompressible in the audio frequency range, it may be assumed that the acoustic pressure amplitude is constant. Consequently, the power distribution, *P*, is controlled by the velocity response given above by Equation (4) and is as follows.

$$P = \frac{F^2}{2\sqrt{\kappa m}}\left[(1/X - X\ )^2 + 4\varsigma^2\right]^{-1/2} \qquad \textbf{(5)}$$

Equation (5) provides a description of the power injection when the stimulus frequency is held constant and the cochlear partition is modeled as a series of oscillators each resonant at a slightly lower frequency as one progresses from the basal end to the apical end of the cochlea.

Two investigations concerned with selective destruction of portions of the cochleae of cats, by two hours exposure to narrow bands of noise centered at 5.5 kHz at sound pressure levels of 109 dB and 110 dB re 20 μPa and subsequently determining the extent of hair cell destruction on their cochleae and their associated hearing deficits, have been reported by Liberman and Dodds[15, 16]. Attention will be given to Figure 6 of the first and Figure 8D of the second reference.

It will be assumed that the observed loss of hair cells is proportional to the work done and thus to the acoustic power input to the damaged areas of the cochlea. Equation (5) allows calculation of the predicted power input distribution, which may be compared with the observed distribution of destruction of hair cells as reported by Liberman and Dodds. It will be assumed that the upper and lower frequency bounds expressed in Hertz, ωu/2π and ωl/2π, of a region of destruction will be determined by corresponding power levels. Equation (5) provides the following prediction.

$$\frac{\omega_u}{\omega_N} = \frac{\omega_N}{\omega_l} \qquad \textbf{(6)}$$

Referring to Figure 6 of Liberman and Dodd[15] an upper bound in terms of frequency in Hertz of 6.4 kHz and a lower bound of 4.8 kHz may be estimated leading to 1.15 and 1.16 for estimates of the upper and lower bounds of Equation (6). Referring to Figure 8D of Liberman and Dodd[16], where the extent of damage is not as clearly defined, a lower bound of 4.0 kHz and an upper bound of about 9.0 kHz leads to estimates of 1.6 and 1.4 respectively for the upper and lower bounds. The estimates of the bounds are in reasonable agreement with Equation (6). Note that *these calculations, based upon a linear description of the ear, show that the ear responds as a linear system to levels of acoustic power exposure sufficient to destroy it.*

4.2 Why hearing loss of industrial workers and musicians similarly assessed diverges.

It has been known for more than twenty years (since1983) that industrial workers suffer serious hearing loss while similarly assessed musicians suffer little or no hearing loss where noise exposure has been determined as a long time average of A-weighted level. It has been shown on the basis of such A-weighted level measurements that exposure of musicians often exceeds recommended maximum levels[17] suggesting, according to accepted industrial criteria, that musicians should show evidence of noise induced hearing loss.

The hearing of musicians has been investigated and no evidence of noise-induced hearing loss has been observed[18]. Waugh[19] has conducted similar investigations including reviews of works of others, and has concluded that for comparable exposures based upon time

averaged A-weighted measurements industrial workers suffer extensively but for musicians, "the auditory dangers of loud music appear almost trivial."

The ear is protected, to a limited extent, from sudden rises in sound level by reflexive tensioning of the stapedius and tensor tympanic muscles of the inner ear. These muscles stiffen the ossicular linkage of the inner ear and reduce sound transmission. However, they are only able to respond within 0.03 to 0.05 seconds. When this response time is compared with the half period of a 2 kHz impulse of the order of 0.00025 seconds it may be concluded that the acoustic reflex is too slow to protect the ear[1]. A more general purpose for the reflex is suggested of reducing noise admitted to the ear in an environment, which suddenly rises in level.

In an environment characterised by many impulses the effectiveness of the reflex will depend upon its rise time and also upon its on time as well as its decay time and the innate durability of the hair cells. It is proposed that the precipitous decline in hearing sensitivity observed at 2 kHz is compatible with the interpretation proposed here that the acoustic reflex can respond quickly enough to reduce sound transmission enabling the ear to respond as a linear system in the low frequency range to about 2 kHz.

It will be shown that energy input may play no significant part in noise induced hearing loss in the frequency range above 2 kHz in noisy industries. It will be shown that typically noisy industrial activity may be characterised by impulsive sounds of high level and frequency, which apparently are responsible for noise induced hearing loss in the frequency range above 2 kHz.

In 1946 Rüedi and Furrer[20] published a paper providing a dramatic example of the effect of failure to satisfy the restrictions imposed upon the ear's use of the anatomical compromise. They published an audiogram of a 28 year old employee who had worked in a mechanical weaving mill for ten years (Figure 1) and a spectral plot of the noise to which he was exposed. The latter plot showed levels of exposure steadily rising from 85 dB at 50Hz to 100 dB at 1.6 kHz flat to 6.4 kHz before dropping off 5 dB at 10 kHz.

In stark contrast to a generally uniform flat level of exposure, the audiogram of the employee shows a generally flat hearing loss ranging between10 and 15 dB from 50 Hz to 2 kHz, which abruptly plunges to a loss of 85 dB at 4 Hz in the region of the ear's greatest sensitivity before climbing up to a loss of about 70 dB at 10 kHz. This observation inconsistent with the equal energy in put concept clearly shows that the equal energy concept does not explain the "4 kHz dip."

4.3 Industrial noise induced-hearing loss

Attention now is directed to Figure 2, which originally appears as Figure 4.3 in the first and second editions, 1988 and 1996 and Figure 4.4 in the third edition, 2003 prepared by Bies, in Engineering Noise Control Theory and Practice[4]. In the figure mean hearing loss at 4 kHz, first published by Burns Robinson in the U.K. in 1970[21] is plotted on the ordinate as a function of percentage risk of developing a hearing handicap plotted on the abscissa, based upon the work of Glorig in the US published by Beranek in 1971[22].

The reader's attention is drawn to the dates. They show that impulsive sounds could have made no contribution to the assessment. The data show exposures at 10, 20, 30, and 40 years at mean levels of 80, 90, 100 and 110 dBA based upon time average A-weighted measurements.

Subsequent inspection of the plotted data allowed the crucial observation to be made that all of the thirty-year exposures lay close to the ten-year exposures at the 10 dBA higher exposure level. The solid line given the name Hearing Deterioration Index (HDI) and given

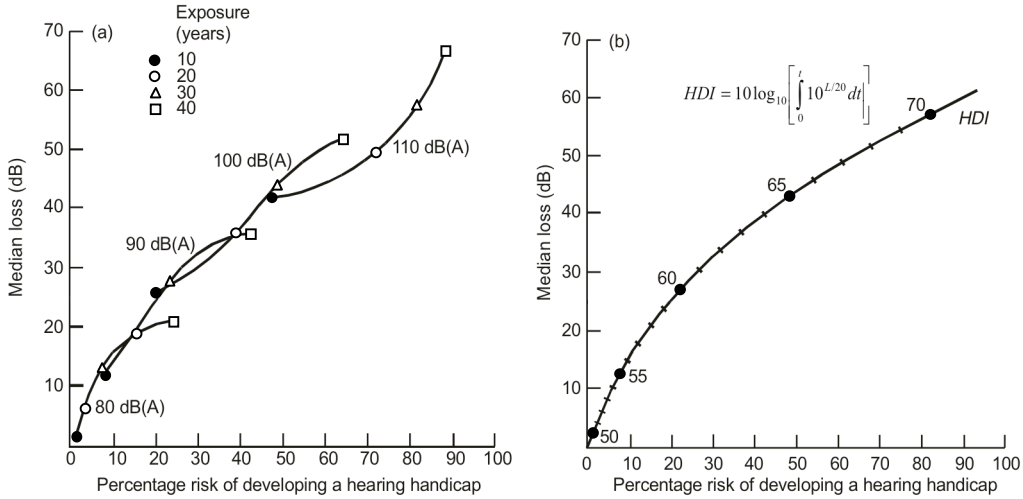

Figure 2. Hearing damage as a function of exposure. The percentage risk of developing a hearing handicap (averaged over 500 Hz, 1000 Hz and 2000 Hz) and the median loss (at 4000 Hz) incurred with exposure are shown (a) as function of mean sound pressure level in the workplace (dB(A)) and exposure time (years); and (b) as a function of hearing deterioration index HDI. The quantity L is the mean exposure level (dB(A)), and t is the exposure time (years).

below by Equation (7) is proposed to summarize the data shown in Figure (2). For comparison the equations (7) and (8) have both been written as daily exposure multiplied by the number of working days, *N*, in a year. Equation (8), which gives the *standard eight-hour noise exposure* based upon the accepted daily energy input multiplied by the number of working days in a year disagrees with Figure 2.

$$HDI = N10\log_{10}[\frac{1}{8}\int_0^8 10^{L/20}dt] \quad \textbf{(7)}$$

$$L_{AeqT} = N10\log_{10}[\frac{1}{8}\int_0^8 10^{L_A/10}dt] \quad \textbf{(8)}$$

6. CONCLUSION

At levels of noise exposure, without impulsive sounds, sufficient to cause hearing loss the ear responds as a linear system and determination of noise exposure as a long time averaged A-weighted sound pressure level measurement is appropriate. At levels of noise exposure experienced by some industrial workers and by some musicians, impulsive sounds in the high frequency range above 2 kHz cause the ear to respond as a non-linear system and a Hearing Deterioration Index (HDI) proportional to pressure may be defined which correlates well with existing data. Consequently, it may be concluded that hearing loss due to noise exposure in the high frequency range above 2 kHz may take place in response to energy input as a linear system, or in response to peak pressures as a non-linear system, or as a combination of both [23]. Thus assessment as a long time average A-weighted sound pressure level measurement alone is inappropriate. This is the basis for the comment that Figure 2 provides the only scientific evidence for a relationship of any kind between noise exposure and noise induced hearing loss.

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