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DIFFERENTIAL SUSCEPTIBILITY TO NOISE-INDUCED HEARING LOSS

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INTRODUCTION

Individual variations of susceptibility to noise-induced hearing loss (NIHL) are a well-established characteristic of field studies of noise damage to hearing. Such variations are manifested as a wide dispersion of hearing threshold levels (HTL) in groups of people exposed for comparable periods of time to the same or equivalent noise environments.

A population of young, otologically normal ears, without undue noise exposure, would be expected to exhibit a distribution of HTL's closely approximating a Gaussian probability distribution. As damaging noise exposure accumulates, it produces a threshold shift (TS), the distribution of which is such that the resulting HTL's no longer follow a Gaussian distribution. Rather, some highly susceptible members of the exposed population suffer relatively large TS's, producing a noticeable positive skewing of the HTL distribution. This paper will put forward a simple empirical model of the statistics of NIHL. The descriptive model has certain novel implications which bear upon the problem of prediction of individual susceptibility to noise damage.

DEVELOPMENT OF NIHL

The change in HTL distribution in response to increasing noise exposure may be derived by adding to the non-exposed HTL distribution for 18 years of age (ISO 7029) the noise-induced threshold shift component as tabulated by Robinson and Shipton (1977) for various values of Noise Immission Level, E_A (a measure which combines noise level and exposure duration). Illustrated in Figure 1 are the distributions for the 4 kHz HTL, which is known to be the audiometric frequency to show the greatest noise damage in the majority of cases. HTL's at other frequencies follow a similar course but of lesser magnitude. In the figure, the horizontal axis is HTL at 4 kHz; the vertical axis is the proportion of the population exhibiting an HTL less than the abscissa value. The distribution shown at the far left, that is with the most acute HTL's, represents the population of otologically normal 18 year old males without noise exposure. As noise is applied to this normal population, its members suffer varying degrees of hearing loss. According to the model described by Burns and Robinson (1970), TS grows as a logistic (S-shaped) function of the Noise Immission Level, to approach a maximum value for severe exposures.

Note from Figure 1 that relatively modest exposures produce HTL distributions which deviate from the Gaussian, most markedly at the upper end. The process of hearing damage is thus not uniform. To account for this, the distribution of noise-exposed HTL's must be the product of a TS distribution acting upon the Gaussian non-exposed HTL's, as observed by Johnson (1975). Such a TS function must be bounded below; in other words, noise exposure cannot be expected to produce negative TS's or improvements in HTL. In addition, the TS function must be heavily skewed in order to produce the non-Gaussian shapes exhibited for the low exposure distributions of Figure 1.

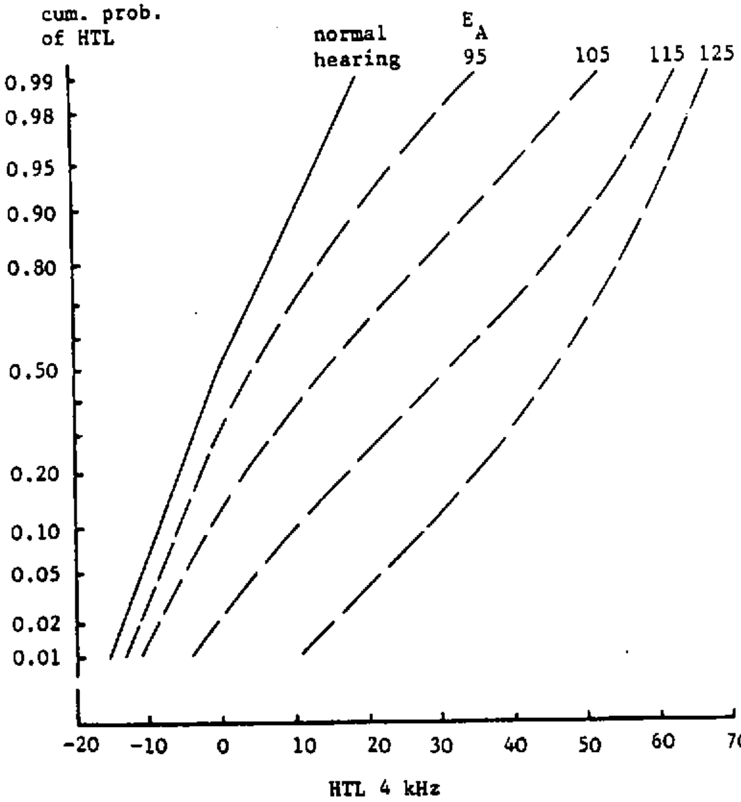

Figure 1. Results of field studies, showing the development of NIHL with increasing noise exposure.

Individuals who are highly susceptible to noise damage may be found anywhere in the initial HTL distribution. For low noise exposures, these individuals would relatively quickly reach large values of TS. As the exposure continued, more and more individuals would suffer high TS's, with the result that the HTL distribution becomes positively skewed, with a long tail of high HTL's. For severe exposures, the HTL skewing is reversed as shown in Figure 1. This long tail of lower HTL's may be thought of as a noise-resistant fraction of the population, who have not yet received a noise dose sufficient to cause very high TS's.

THE MODEL

To build up a simple model of NIHL, the baseline for young, non-exposed ears must be established. The distribution of non-exposed HTL's may be assumed to follow the Gaussian probability density function:

$$P(x) = \{1/(\sigma_1\sqrt{2\pi})\} \exp\{-(x-\mu_1)^2/2\sigma_1^2\} \qquad -\infty < x < \infty, \quad \sigma > o \qquad (1)$$

For the example of interest, the 4 kHz HTL distribution for 18 year old males may be approximated by Gaussian distribution of :

$$\mu_1 = 0 \text{ dB}$$

$$\sigma_1 = 7.5 \text{ dB}$$

The customary bell-shaped curve of this distribution may be seen in Figure 2a. Next, a probability density function must be hypothesised for TS, to be independent of HTL. Several conditions must be met :

(a) the function must be continuous, rather than discrete;

(b) the function must be bounded below; and

(c) the function should have a long positive tail.

These basic conditions are met by the Gamma probability density function, of the general form:

$$P(y) = \alpha^k y^{(k-1)} \{\Gamma(k)\}^{-1} \exp\{-\alpha y\} \qquad \alpha > o, \quad k > o, \quad y > o \qquad (2)$$

$$\text{with mean } \mu_2 = k/\alpha$$

$$\text{variance } \sigma_2^2 = k/\alpha^2$$

$$\text{and } k = (\mu_2/\sigma_2)^2$$

The equation is somewhat simplified if k is an integer; for k=3, the function becomes:

$$P(y) = (1/2)\alpha^3 y^2 \exp\{-\alpha y\}. \qquad (3)$$

In order to characterize this form of the density function, only μ_2 need be specified. For a given μ_2, the function may be represented in Figure 2b; this curve is relatively simple, yet satisfying on conceptual grounds. For

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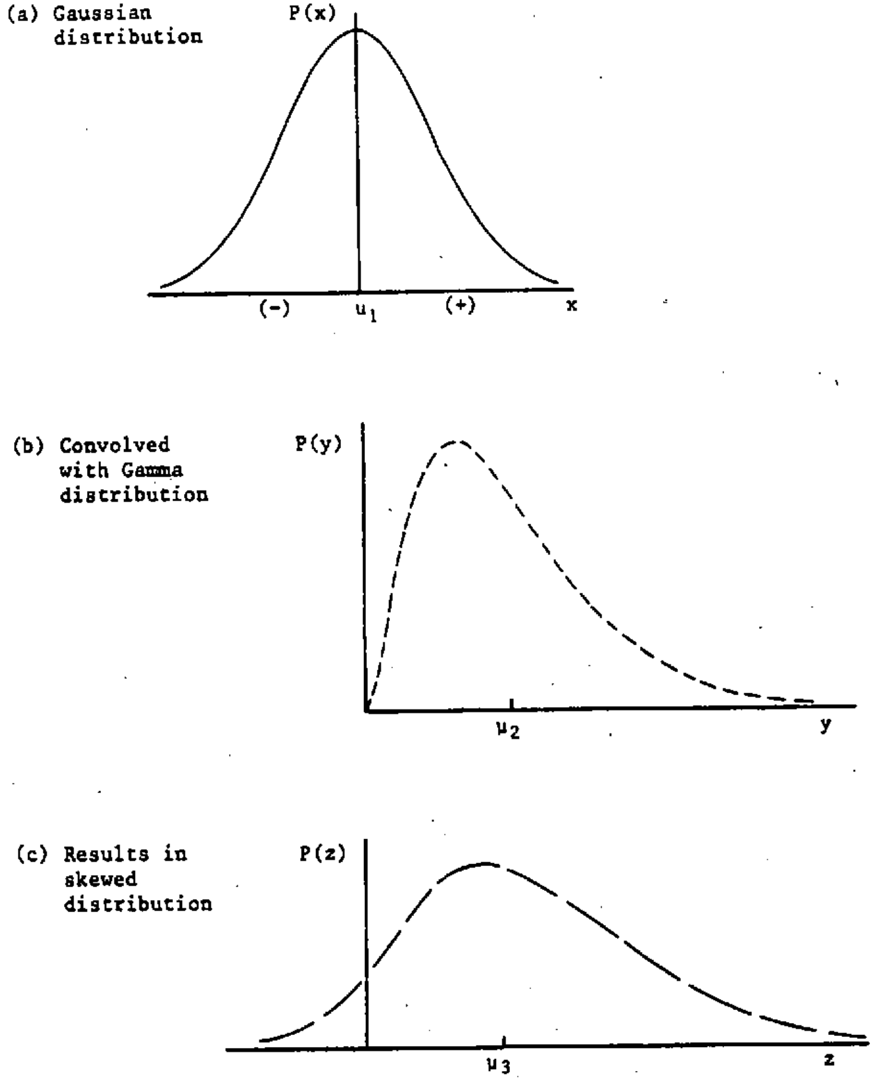

Figure 2. Illustration of the convolution process, achieved by numerical integration of two probability density functions to give the resultant distribution.

practical reasons though, this TS distribution must be bounded above as well as below; a maximum TS of 55 dB will be used for later computations.

Having hypothesised the two probability density functions of x (representing the non-exposed Gaussian or normal HTL's) and y (the skewed TS or acquired hearing loss), the resultant z (exposed HTL's) will have a probability distribution which is the convolution of x and y. The result, illustrated in Figure 2c, is easily obtained by numerical integration once the relevant parameter μ_2 is assigned. This result represents the distribution of HTL's following exposure which produces a mean TS equal to μ_2.

The results of the numerical integration process are presented in Figure 3. The cumulative distribution of non-exposed HTL's (x) is illustrated, along with the skewed HTL's (z) resulting from imposition of various mean values of TS. For small to moderate values of mean TS, note how the HTL distributions become positively skewed, showing that the highly susceptible individuals succumb early to the noxious effects of the noise environment. As the exposure becomes more severe, the HTL distribution changes character with increasingly larger proportions of the distributions parallelling the Gaussian, causing a reversal of the skew. This feature is a consequence of the TS saturation value which is not exceeded in any noise environment. This upper bound on the TS distribution, mentioned above, was set at 55 dB for illustrative purposes. The highly damage-susceptible individuals would relatively quickly approach this maximum loss, at which time the noise would cease to have any damage potential for them. At the other end of the susceptibility continuum, the noise-resistant individuals are still slowly accumulating hearing loss, having not yet received a sufficient noise dose to cause saturation TS. These resistant members of the population make up the negative-skew tail of the exposed HTL's, having identified themselves by sustaining relatively smaller hearing losses.

The statistics of NIHL may be modelled, to the first order at least, by a simple, empirical approach. The intention has been simply to describe, rather than quantify, the development of NIHL in response to increasing noise exposure. The assumptions made are plausible from first principles, and have served well to follow the trends of occupational hearing loss.

IMPLICATIONS

One important observation may be made from the results of field studies, summarized in Figure 1, and from the behaviour of the simple model presented here, as shown in Figure 3. As damaging noise exposure accumulates, a clearly noise-resistant fraction emerges, being those individuals who retain acute HTL's in spite of noise exposure.

As susceptibility to noise damage is distributed throughout the population, damaging exposure will cause the more volatile (susceptible) fractions of the population to "boil away". As exposure accumulates, the distillation process leaves a residue of "hearing survivors". Some of these fortunate individuals retain HTL's even better than the majority of young non-exposed ears. Considering the obvious fact that a person's hearing does not improve with age and noise, this clearly identifiable residue of survivors must necessarily have been the possesors of better-than-average hearing in their youth.

The concept of differential damage susceptibility has provided an obvious basis for defining a sample of non-susceptible ears, and the "parent" population of

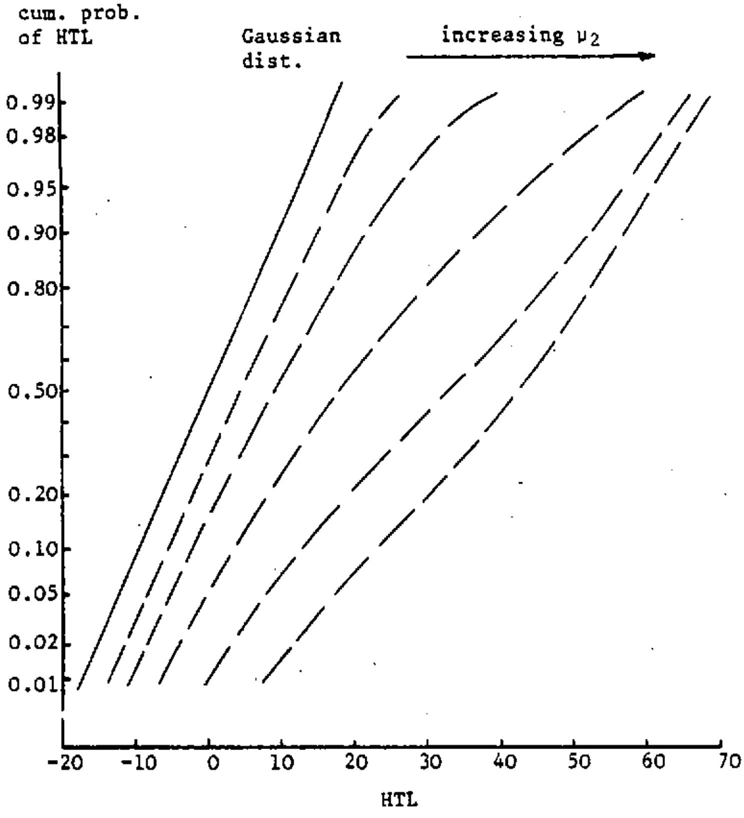

Figure 3. Behaviour of the model, showing the development of NIHL with increasing mean TS.

younger, non-exposed ears from which the survivors have been drawn. Some common characteristic, auditory or otherwise, may prove to be exclusive to these two groups of ears. It would seem to follow that the lack of this characteristic would signal that a given individual was not resistant, but among the more susceptible fraction of the population. Thus we may be able to get a view, arguably through the wrong end of the telescope, of a test for susceptibility in advance of noise exposure.

ACKNOWLEDGEMENT

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