Impact noise and the equal energy hypothesis

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The equal energy hypothesis (EEH) was evaluated over a limited range of conditions by exposing four groups of chinchillas to impact noise (200-ms *B* duration) presented at a fixed rate of four impacts per second. The intensity of the impacts (107–125 dB peak SPL) and the duration (120–1.87 h) of the four exposure conditions were counterbalanced so that the four groups received the same total energy. The traumatic power of the exposures was assessed by measuring the threshold shift of the auditory evoked response and the amount of hair cell loss. Exposures between 107 and 119 dB were consistent with the EEH in that they produced roughly the same amount of permanent threshold shift (less than 20 dB) and hair cell loss (less than 20%). However, the 125-dB exposure produced substantially more threshold shift and hair cell loss than the three lower intensities. Thus, the EEH may be applicable only at lower impact intensities; above a "critical intensity" the amount of damage increases significantly.

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INTRODUCTION

The Equal Energy Hypothesis (EEH) as first proposed by Eldred *et al.* (1955) represents an attempt at building a unified theory to estimate the auditory hazards of any noise exposure. A simple statement of the EEH is that the trauma associated with a particular noise exposure is a monotonic function of the total amount of acoustic energy received by the organism. It is implicitly assumed that the distribution of energy over time is irrelevant in determining the degree of hearing loss. The EEH is an attractive idea primarily because of its inherent simplicity and the ease with which the concept can be incorporated into instrumentation to produce a "single number index" of the hazards of any noise environment.

Burns and Robinson (1970) have formalized the EEH. They adopted an A-weighted measurement standard and applied the theory to carefully selected demographic data of noise-induced permanent threshold shift (PTS). Their analysis represents one of the primary data bases for the EEH as it currently exists. Atherley and Martin (Atherley and Martin, 1971; Atherley, 1973; Martin, 1976) have extended the Burns and Robinson calculations in an effort to apply the EEH to impulse and impact noise exposures. The Atherley and Martin (1971) formulation is based upon a scheme for estimating the equivalent continuous A-weighted sound level (L_{eq}) for an 8-h work day. The application of their analysis to data acquired from the drop-forging industry is often cited in support of the EEH for impact noise (Atherley and Martin, 1971; Atherley, 1973). Other studies of impact noise also appear to be consistent with the EEH (Guberan *et al.*, 1971; Kuzniarz *et al.*, 1976). However, when reviewing these data, one cannot help but be dismayed by the extreme variability. A cursory inspection of the demographic data reveals as much as a 90-dB spread in the age-corrected hearing levels at some exposure levels and test frequencies (Atherley and Martin, 1971; Taylor and Lord, 1976). Variability is so extreme that a variety of analytic formulations would adequately describe the data.

Other demographic studies of impulse noise-induced PTS appear to be in conflict with the predictions derived from the EEH (Sulkowski *et al.*, 1980, 1983). Sulkowski *et al.* (1980) compared the growth of PTS in workers exposed to industrial steady-state noise and impulse noise of the same L_{eq}. Workers exposed to impulse noise consistently showed greater hearing loss than those exposed to continuous noise. Furthermore, more recent analyses of data from the drop-forging industry indicate that the hearing losses resulting from exposures of ten years or more are equal to or greater than those from continuous noise of the same equivalent level. For exposures of less than ten years, the losses for impact noise were less than those predicted for continuous noise (Taylor *et al.*, 1984). In spite of conflicting evidence, the momentum has been gathering to apply the EEH to impulse noise exposures. As a result, damage risk criteria for impulse noise have been formulated using the energy principle (Pfander, 1980; Smoorenburg, 1983).

Laboratory measures of temporary threshold shift (TTS) and PTS in humans and animals as well as the loss of sensory cells measured in animals have been used to evaluate the EEH. A number of laboratory studies have raised doubts about the applicability of the EEH especially as it applies to impact and impulse noise (Henderson and Hamernik, 1978; Hamernik *et al.*, 1980). Perkins *et al.* (1975), for example, exposed animals to 50 impulses (1-ms *A* duration, 155-dB peak SPL), but used two different repetition rates (1/min or 1/10 s) in order to vary the distribution of energy over time. Even though the total energy was the same for both groups of animals, those exposed at a rate of 1/10 s showed greater TTS and PTS as well as greater hair cell loss than those exposed at a rate of 1/min. The exposure at 1/min may have been less damaging than the one at 1/10 s because the auditory system has a longer period of time to recover between impulses. The interstimulus interval is an important variable which, by definition, the EEH fails to take into consideration.

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The application of the EEH to impulse noise is further complicated by the apparent existence of "critical levels." The implications of the "critical level" are illustrated by an experiment carried out by McRobert and Ward (1973) in which they exposed individuals to nonreverberant impulses and adjusted the level and number of impulses in such a way that all exposures contained equal amounts of energy. Even though the energy remained constant across conditions, only the highest exposure level produced a significant temporary threshold shift (TTS). Price (1981) has summarized much of the evidence supporting the concept of the "critical level" and suggested that it is frequency dependent due primarily to the outer and middle ear transfer functions. Impulse duration also appears to influence the "critical level" (Coles and Rice, 1970).

Above the "critical level," the rules for time-intensity trading may become rather complex. Furthermore, at very high intensities the tympanic membrane can rupture, thereby reducing the amount of energy reaching the inner ear; consequently the EEH would not apply (Eames *et al.*, 1973). A similar problem exists at low intensities, i.e., below some "minimum level" essentially no threshold shift develops even after long duration exposures (Henderson and Hamernik, 1978). Both of the preceding cases illustrate that there are natural boundary conditions for the EEH. The point to be made here is that the EEH may only be valid at moderate intensities and over a restricted range of exposure parameters.

In spite of the negative findings related to the EEH at very low and high levels, the concept does appear to have some predictive value for intensities observed in industrial environments (Martin *et al.*, 1970; Atherley and Martin, 1971). From a practical standpoint, it may be fruitful to attempt to find a range of exposure conditions for which the EEH is generally applicable. This is especially true in light of the fact that the EEH has the potential for producing a unified conceptual framework for international noise standards (ISO, 1984). Consequently, the purpose of this experiment was to further evaluate the EEH by studying the trading relationship between impact intensity and exposure duration using exposure intensities similar to those encountered in industry (Martin, 1970; Dym, 1977; Lahti and Starck, 1980; Nabelek, 1980; Passchier-Vermeer *et al.*, 1980). The experimental paradigm used in this study is similar to one employed by Ward *et al.* (1981) using continuous noise.

I. METHODS

A. Subjects

Twenty-three adult chinchillas were used as subjects. The animals were anesthetized (0.1 mg/kg Ketamine) and made monaural by the surgical destruction of the left cochlea. A chronic bipolar electrode was then implanted in the region of the inferior colliculus according to procedures outlined previously (Henderson *et al.*, 1973). The animals were allowed to recover for several days prior to threshold testing.

B. Auditory evoked response testing

The auditory evoked response (AER) was used to estimate hearing thresholds according to procedures described previously (Henderson *et al.*, 1973,1974; Salvi *et al.*, 1982). Three pre-exposure thresholds were measured at 0.5, 1, 2, 4, 8, and 16 kHz; the mean of the three measures was used to establish the pre-exposure thresholds for that animal. After the exposure, the animals were tested at 0.5, 2, and 8 kHz at postexposure times of 0.25, 2, 8, 24, and 240 h in order to estimate the amount of TTS and the threshold recovery function. At 30 days postexposure, thresholds were remeasured at all frequencies in order to estimate the amount of PTS.

C. Exposure

One or two animals at a time were placed in small separate cages (31 × 51 × 38 cm) located below a loudspeaker and were given free access to food and water. The animals were then exposed to electronically synthesized impact noise. The impact consisted of a burst of broadband noise, whose spectrum was shaped with an equalizer so that it roughly approximated that of an impact generated by striking a steel plate with a hammer (Blakeslee *et al.*, 1978; Henderson and Hamernik, 1982). The electrical signal was generated by multiplying (Analog Devices AD534) the spectrally shaped noise with the output from an exponential waveform generator. The waveform generator controlled the repetition rate and envelope of the synthesized impact. The signal was amplified and transduced by a loudspeaker located above the animal's head.

The acoustic signal was measured with a ½-in. condenser microphone (B&K 4134) and measuring amplifier (B&K 2606). The spectrum of the acoustic signal was determined with a real time spectrum analyzer (Federal Scientific UA-15) using a bandwidth setting of 40 Hz. A representation of the spectrum and pressure-time envelope of the acoustic impact are shown in Fig. 1. The spectrum of the impact had two prominent peaks, one near 1.5 kHz and the other near 4.0 kHz. The duration of the impact at a point 20 dB down from the peak was 200 ms and the nominal rise time of the signal was 12 ms. Table I contains the exposure conditions for each

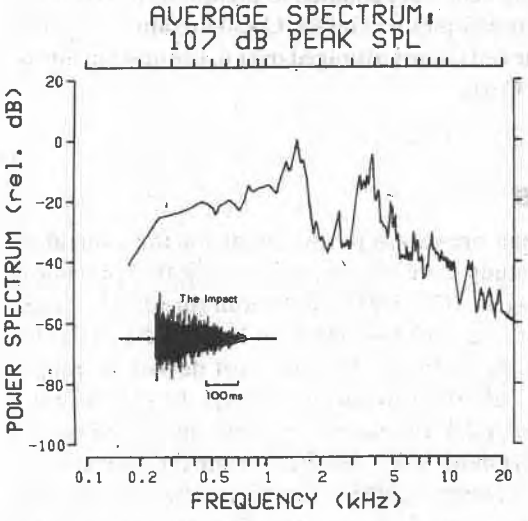


FIG. 1. Relative amplitude of the acoustic spectrum of the impacts using an analysis bandwidth of 40 Hz. Insert shows the pressure-time profile of the impacts.

TABLE I. Exposure conditions.

Peak SPL	Duration	Repetition rate	Subjects
107 dB	120.00 h	4/s	7
113 dB	30.00 h	4/s	5
119 dB	7.50 h	4/s	5
125 dB	1.87 h	4/s	6

of the four experimental groups of chinchillas. The repetition rate of the impact noise was maintained at 4/s throughout the experiment while the exposure intensity and duration were counterbalanced so that the four exposure conditions contained essentially the same amount of energy. The intensity levels in the animal cages were monitored on a daily basis for animals in the 107- and 113-dB groups. Since the 119- and 125-dB exposures were of relatively short duration, the intensity levels were calibrated immediately before the exposure began. There was little variability in the peak level of the exposure within the cages ($\pm$ 2 dB) and the presence of the two animals in their separate cages had essentially no effect on the level. Nevertheless, as a further precaution, animals in the 107-dB exposure group were rotated between cages on a daily basis so that the effect of any slight variation in intensity would be minimized.

D. History

After final thresholds had been measured, the animals were sacrificed and their cochleas were analyzed for sensory cell damage using the surface preparation technique (Engstrom *et al.*, 1966). The animals were decapitated and the right bulla was removed and opened to expose the cochlea. The cochlea was initially perfused through the round window with 2.5% gluteraldehyde in veronal acetate buffer ($pH = 7.3$), fixed overnight, then postfixed in 1% OsO_4 in the same buffer. Following dehydration through to 70% ETOH, the cochleas were microdissected and the organ of Corti mounted in glycerin on glass slides. The sensory epithelium was viewed at approximately 400× using Nomarski optics. Sensory cells were counted as present if the cell-body cuticular plate complex was intact. Cochleograms were plotted using hair cell counts averaged over 0.4-mm segments of the organ of Corti.

II. RESULTS

The mean pre-exposure thresholds for the chinchillas used in this study were similar to those reported previously (Henderson *et al.*, 1973,1982). The mean thresholds ranged from a low of -2.1 dB at 4 kHz up to 21.5 and 20.3 dB at 0.5 and 16 kHz, respectively. The standard deviations ranged from 4.9–7.2 dB at all frequencies except 16 kHz where it increased to 9.4 dB. In order to evaluate the significance of the mean threshold shifts resulting from the exposures, a one-tail, 95% upper confidence limit was computed for the *t*-statistic using a sample size of five. The confidence limits, which ranged from 4.7–9.0 dB, are shown in Fig. 6.

The average TTS was determined for each group at

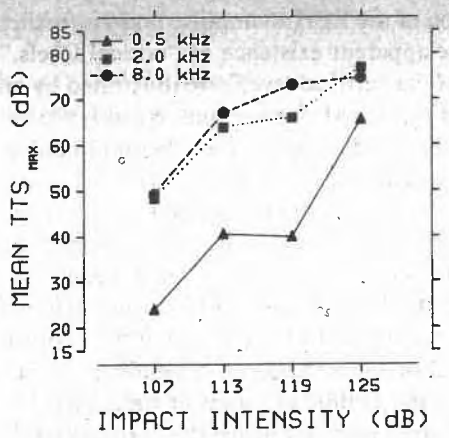


FIG. 2. Mean value of TTS_{max} plotted as function of impact intensity for the three test frequencies.

0.25, 2, 8, 24, and 240 h postexposure. The maximum temporary threshold shift (TTS_{max}) occurred immediately after the exposure. Figure 2 illustrates how the mean value of TTS_{max} varied across the four exposure conditions and three test frequencies. At the lowest frequency, 0.5 kHz, TTS_{max} increased from approximately 24–65 dB as impact intensity increased over an 18-dB range, i.e., roughly a 2.3-dB increase in TTS per dB increase in level. An increase in TTS_{max} with level was also seen at the high frequencies although the effect was not as pronounced as the one at 0.5 kHz. At 2 and 8 kHz, TTS_{max} increased from approximately 49–76 dB as the intensity was raised from 107–125 dB, i.e., about a 1.5-dB increase in TTS per dB increase in SPL.

The TTS recovery curves for the 119-dB exposure condition are plotted as a function of the logarithm of postexposure time in Fig. 3. The trends seen in these data are typical of all the exposure conditions. When the threshold shift exceeded 50–60 dB, there was a plateau in the recovery curve during the first 2–8 h postexposure. This delayed recovery pattern was most often seen at the highest exposure levels and at the test frequencies of 2 and 8 kHz. When the threshold shift was less than 50 dB, the TTS recovered monotoni-

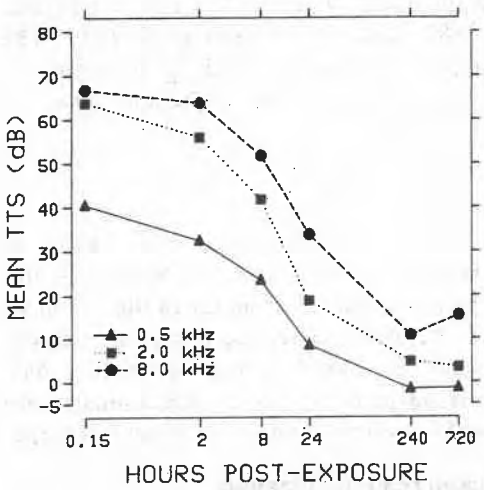


FIG. 3. Mean value of TTS plotted as a function of the logarithm of postexposure time for the three test frequencies. Impact intensity of 113-dB peak SPL.

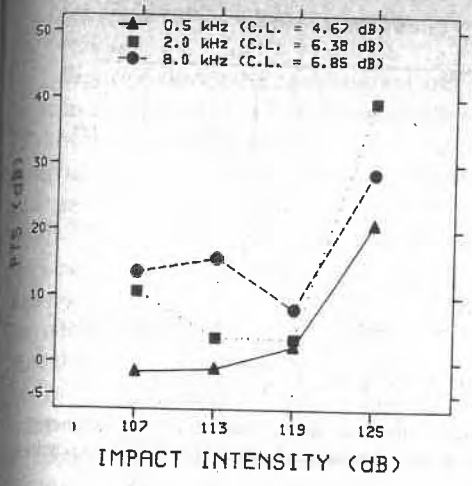


FIG. 4. Mean value of PTS (CI: 95% upper confidence limits) at the three test frequencies plotted as a function of impact intensity.

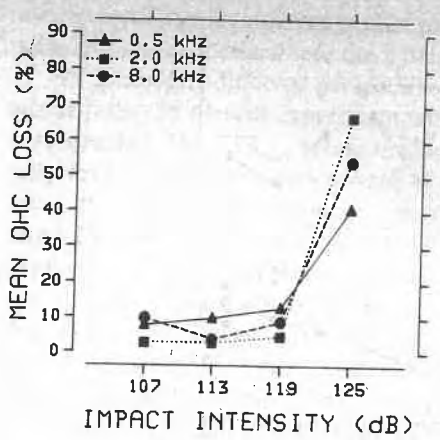


FIG. 5. Mean loss of OHC in the 0.5-, 2-, and 8-kHz regions of the cochlea plotted as a function of impact density. Average hair cell loss computed over a length corresponding to an octave on frequency-place map of Eldredge *et al.* (1981).

cally out to approximately ten days postexposure and there was little or no PTS.

The permanent threshold shifts measured at 0.5, 2, and 8 kHz are shown in Fig. 4 along with the 95% upper confidence limit for the *t*-statistic (Hays, 1963). The mean threshold shifts resulting from exposures between 107 and 119 dB are fairly small, but tend to increase at the higher frequencies. The shifts at 0.5 kHz are not statistically significant whereas the losses at 2.0 and 8.0 kHz fall close to or just outside the upper confidence limits. When the intensity was raised to 125 dB, the PTS values increased to between 20 and 40 dB with the greatest loss occurring at 2.0 kHz. Thus, the 125-dB exposure produced approximately 20–35 dB more PTS than the three lower level exposures even though all the exposures had the same energy. Table II shows the PTS values for each animal for the four exposure conditions. The range of PTS values increased substantially at the 125-dB exposure level due to the fact that one or two animals showed little PTS at one or more of the test frequencies.

Figure 5 shows how the mean outer hair cell loss varied

TABLE II. Individual PTS values.

Subject	Frequency 0.5	1.0	2.0	4.0	8.0	16.0
			107-dB exposure			
367	− 3.3	− 6.6	6.7	− 6.7	− 1.7	0.3
368	− 10.0	− 10.0	− 1.6	10.0	2.8	− 6.7
370	− 6.6	− 1.7	18.3	26.7	16.6	− 6.3
961	− 1.7	5.6	15.0	38.3	5.0	16.7
377	0.0	10.0	13.3	13.4	21.7	6.6
498	− 0.6	1.7	4.0	22.7	13.7	8.0
496	1.0	5.0	2.3	21.0	10.3	11.0
			113-dB exposure			
615	0.0	− 1.3	5.0	6.7	8.3	23.3
688	− 0.1	5.0	3.7	2.6	41.7	13.6
704	− 5.0	− 5.0	3.3	4.3	20.0	20.0
816	1.7	5.0	5.0	6.0	10.0	2.3
817	− 2.3	3.3	1.6	11.0	− 1.7	1.6
			119-dB exposure			
666	3.4	6.7	5.0	11.6	1.6	16.7
667	3.4	5.0	10.4	6.3	10.0	8.3
713	3.3	3.0	0.8	6.7	11.7	10.0
714	1.6	0.0	0.0	1.7	6.7	0.0
813	0.0	6.7	1.7	4.3	10.0	16.6
			125-dB exposure			
717	5.0	13.3	31.7	24.9	13.3	16.3
721	38.3	46.7	61.6	56.6	51.7	46.7
755	28.3	41.7	55.0	46.6	46.3	53.3
799	8.4	13.3	24.0	29.9	8.3	15.0
827	3.3	10.0	8.3	6.6	6.7	10.0
835	41.6	51.7	55.3	53.3	45.0	53.4

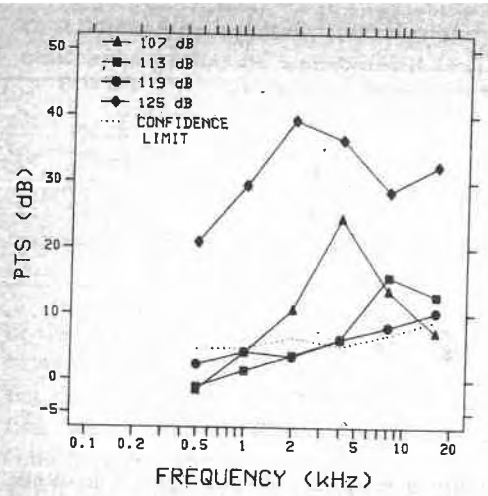


FIG. 6. Mean PTS and 95% upper confidence limits plotted as a function of test frequency for the four exposure groups.

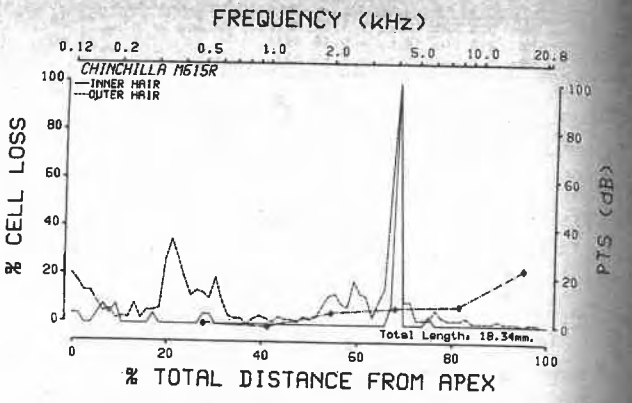


FIG. 8. Typical pattern of hearing loss and hair cell loss for a chinchilla in the 113-dB exposure group. Similar results were obtained for exposures at 107 and 119 dB.

over exposure conditions. Each point represents the average number of outer hair cells that were missing in a length of cochlea corresponding to an octave band of frequencies centered at 0.5, 2, and 8 kHz. These calculations were based upon the frequency-place map developed by Eldredge *et al.* (1981). Qualitatively, the trends in the outer hair cell loss parallel the PTS data, i.e., the averaged OHC loss is reasonably constant at about 5%–10% for the 107- through 119-dB exposures, but when the level is increased to 125 dB the hair cell loss increased dramatically to between 40% and 70% in the 0.5-, 2-, and 8-kHz octave bands. Some minor discrepancies can be seen between hair cell loss and PTS; for example, at the 113-dB exposure there is roughly a 15-dB PTS, but little or no hair cell loss.

Figure 6 provides a more complete description of the pattern of hearing loss from the four exposure groups. Exposures between 107 and 119 dB produced relatively small threshold shifts. Generally, only the threshold shifts between 2 and 16 exceeded the 95% upper confidence limit. The greatest loss occurred at 4 kHz which corresponds to the smaller of the two spectral peaks in the exposure stimulus. By contrast, for all test frequencies, the 125-dB exposure resulted in threshold shifts that were much larger than those seen at lower levels. The threshold shifts, which ranged between 20 and 40 dB, fell outside the confidence limits across the entire range of frequencies. At 125 dB, however, the maximum shift occurred at 2 kHz, which is approximately half an octave above the larger of the two peaks in the amplitude spectrum of the impact (Fig. 1).

The distribution of hair cell loss along the cochlear partition has been transformed into the frequency domain in Fig. 7 using the frequency-place map of Eldredge *et al.* (1981). The points from 0.25–8 kHz in Fig. 7 correspond to the audiometric test frequencies and were obtained by computing the average hair cell loss over an octave interval on the frequency map. Because the endpoints do not contain a full octave, the point at 16 kHz had to be averaged over slightly less than an octave interval while the point at 0.125 kHz was averaged over approximately half an octave. Exposures between 107 and 119 dB produced little or no loss of inner (IHC) or outer hair cells (OHC). By contrast, the 125-dB exposure resulted in average OHC losses of 30%–70% across the entire cochlea with the maximum damage occurring at 2 kHz for OHC. Significant IHC losses of 20%–30% were only seen above 1 kHz with the peak loss occurring in the 4-kHz region. While the overall pattern of hair cell loss mirrored the PTS data, some minor discrepancies can be seen between the two set of data. For example, at the three lowest intensities, threshold shifts of 10–18 dB can be seen at

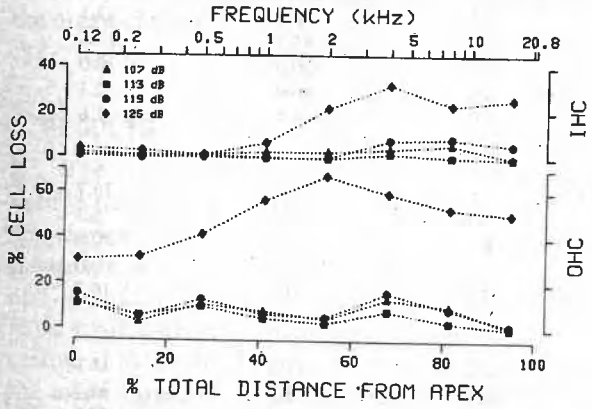


FIG. 7. Mean IHC and OHC loss plotted as function of percent distance from the apex. Cochlear position transformed to frequency according to the frequency-place map of Eldredge *et al.* (1981). Average values computed over lengths of the cochlea corresponding to octave intervals, except for points located near the apex and base (see text for details).

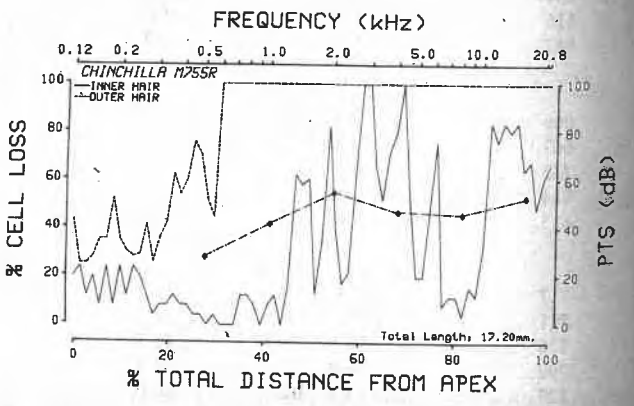


FIG. 9. Typical pattern of hearing loss and hair cell loss for a chinchilla in the 125-dB exposure group.

the high frequencies while there is little or no corresponding hair cell loss.

Figure 8 shows the cochleogram and pattern of PTS of an animal exposed at 113 dB; these results are representative of those animals exposed between 113 and 119 dB. The lesions tended to be rather punctate as illustrated by the narrow patch of missing IHC and OHC near 4 kHz plus the small secondary lesion of OHC near 0.4 kHz. This animal showed little or no hearing loss except at 16 kHz where there was no hair cell loss. Typical results of an animal exposed at 125 dB are shown in Fig. 9. The lesion is much more severe than that seen at lower intensities and consists of a complete loss of OHC extending over the basal three-fourths of the cochlea. Although the IHC loss is also quite substantial in the basal half of the cochlea, the pattern of loss is extremely irregular. The hearing loss followed the general contour of the lesion and was approximately 50 dB from 2–16 kHz and then declined to about 30 dB at 0.5 kHz. Thus, the damage sustained by individual animals in the 125-dB group was consistently more severe than for animals exposed at the lower intensities.

III. DISCUSSION

The EEH has traditionally been evaluated from field studies where PTS is the prime metric and where it is difficult to accurately specify the exposure levels. The present study, on the other hand, provides an opportunity to evaluate the EEH in terms of TTS, PTS, and anatomical criteria as well as under controlled and repeatable exposure conditions. Thus, the reader should keep these factors in mind when comparing results from different studies.

When the present results are evaluated in terms of PTS, three of the four exposure conditions, namely those in the intensity range between 107 and 119 dB, appear to be consistent with the EEH since the amount of PTS and hair cell loss was essentially the same for the three groups. However, since the exposures barely produced any PTS, it is difficult to properly evaluate the EEH using PTS as a metric at these three intensities. When the intensity increased to 125 dB there was a dramatic increase in PTS and hair cell loss, a finding which is inconsistent with the EEH. The intersubject variability in the 125-dB group was also larger than that seen at lower intensities.

Ward *et al.* (1981) carried out an experiment similar to our own with chinchillas, but used continuous noise rather than impact noise. Equal energy exposures in the intensity range between 82 and 111 dB SPL (rms) caused equal amounts of hair cell loss (approximately 10% OHC); however, an exposure of 120 dB SPL (rms) caused a loss roughly 7× as great. The intensities reported in the Ward *et al.* study are expressed in terms of their rms value whereas ours are expressed in terms of peak SPL; thus it is difficult to precisely compare the two sets of results. Furthermore, other aspects of the stimuli were quite different. Nevertheless, the trends in the two studies are quite similar. Both show that the EEH may be applicable at intensities below 115–119 dB SPL; however, above this level, hearing loss and hair cell loss increase dramatically. Thus, below a "critical level," both laboratory studies found a range of intensities for continuous noise and impact noise where the EEH may be applicable.

A somewhat different perspective on the EEH is obtained from the present experiment when the TTS_{max} data are evaluated. The TTS_{max} values tend to increase systematically with exposure intensity at each of the test frequencies. Thus, the TTS_{max} values are not consistent with the EEH. The TTS data have added significance when they are related to previous work on asymptotic threshold shift (ATS) from impact noise. The growth to ATS is intensity dependent such that it can be reached within an hour for high intensities or after several days for low intensities (Blakeslee *et al.*, 1978; Henderson and Hamernik, 1982). Using these earlier studies as a guide, it would appear that our TTS_{max} values are close to ATS. Thus, if ATS is related to PTS, as has been postulated for long-duration exposures (Mills, 1976), then the TTS_{max} values shown in Fig. 2 might approach the level of PTS resulting from long-duration exposures. Assuming that there is a relationship between ATS and PTS, then the TTS_{max} results would imply that the trauma associated with the present exposures is more closely linked to intensity than it is to energy (Mills, 1976). The demographic data obtained by Sulkowski *et al.* (1983) appear to be consistent with this view, i.e., PTS was greatest among the group of workers exposed to the highest peak SPL even though both groups were exposed at the same equivalent level over many years.

The function relating hearing damage to intensity has frequently been shown to rise precipitously once the impulse or continuous noise exceeds a "critical intensity" (McRobert and Ward, 1973; Henderson *et al.*, 1974; Ward *et al.*, 1981; Price, 1981) or "critical energy" level (Nilsson *et al.*, 1982). It seems unlikely that energy *per se* is the critical factor since our results and those of Ward *et al.* (1981) indicate that the degree of hearing loss and hair cell loss can vary dramatically across exposure conditions even when the energy level is held constant. The results of Ward *et al.* (1981) suggest that the "critical intensity" is somewhere around 115 dB SPL for chinchillas exposed for 220 min to a band of continuous noise centered at 1400 Hz. Although the noise spectra are somewhat different, our results obtained with impact noise suggest a "critical intensity" similar to that reported by Ward *et al.* From these limited results, it might be tempting to conclude that the "critical level" for the chinchilla is in the range of 115–119 dB. However, in other experiments, chinchillas that were exposed to 50 reverberant impulses (1 impulse/min, 200-ms *B* duration) showed a dramatic increase in hearing loss and hair cell loss at roughly 150 dB (Henderson and Hamernik, 1978). The high "critical intensity" seen in the Henderson and Hamernik study may be related to either the different type of impulse used (i.e., blast wave versus impact) or to the long period of recovery between impulses, i.e., 1 min vs 50 ms in the present study. The peak in the spectrum of the impulse used by Henderson and Hamernik was also much lower than in the present study and according to Price (1981) this would raise the critical level although it is difficult to predict how much this would be for the chinchilla. Clearly, a broad range of exposure parameters needs to be considered in determining the "critical level," e.g., spectrum, duration, off-time, and repetition rate).

Unfortunately, the relative importance of each of these exposure variables is poorly understood at the present time due to the lack of a systematic body of data relating specific exposure parameters to the degree of damage in a particular species. Experiments are currently underway to further explore the role of several of these variables and the manner in which they interact in determining the degree of noise-induced hearing loss.

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