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The Effects of Blast Trauma (Impulse Noise) on Hearing: A Parametric Study Source III

Report No. ARL 90-3

Annual Report

Roger P. Hamernik
William A. Ahroon
Robert I. Davis
Keng D. Hsueh
George A. Turrentine

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State University of New York at Plattsburgh
Plattsburgh, New York 12901

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There are three broad goals to this contract. The first and primary goal is to study the effects of high level blast wave exposure on the conductive and sensory structures of the mammalian ear. This includes the use of the auditory evoked potential to measure hearing thresholds and tuning curves prior to and after exposure to various blast wave exposures. Parameters of the blast waves studied include intensity, spectral composition, number of impulses and repetition rate. Correlations among hearing measures, exposure variables and histology have been developed. To achieve the above, the following two secondary objectives had to be completed: (1) develop a series of blast wave generation devices which are suitable for the laboratory simulation of a wide spectrum of blast waves; and (2) develop a suitable set of software and a PC-based computer system which can interact with crystal and capacitive microphones to capture and analyze blast waves. Four blast wave sources have been developed along with an analysis system. The

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results from Source I (a conventional shock tube) were summarized in reports ADA 206-180 and ADA 203-854. Source II results were summarized in report ADA 221-731. This report summaries the results from Source III, a 3-inch Lamont valve-driven shock tube which generates a blast wave in anechoic (free-field) surroundings whose peak of the A-weighted spectrum is in the 2 kHz octave band.

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SUMMARY

There are three broad goals to this project. The primary goal is to begin the systematic development of a data base from which one could estimate the hazards to hearing resulting from exposure to blast waves or other high level impulse noise transients. To achieve this objective the following two additional objectives must first be achieved: (1) develop a methodology to efficiently acquire data on a large number of experimental animals that have been exposed to a variety of blast wave configurations. This includes audiometric, histological and acoustic variables; (2) develop a set of blast wave simulation devices which can reliably generate blast waves with a variable distribution of spectral energy in a laboratory environment.

Three previous progress reports (ADA 206-180, ADA 203-854 and ADA 221-731) from this contract have documented the results and methodology associated with items (1) and (2) above. In addition, the audiometric and histological results from 109 chinchillas exposed to non-reverberant blast waves, whose A-weighted spectral peak was in the 0.250 kHz octave band, produced by Source I were described in the first two reports. The third report summarized similar results from 105 chinchillas exposed to non-reverberant blast waves with an A-weighted spectral peak in the 1.0 kHz octave band produced by Source II. This report documents the results of parametric experiments performed on 105 chinchillas exposed to non-reverberant blast waves produced by a 3-inch Lamont source (Source III) whose peak of the A-weighted spectrum is in the 2.0 kHz octave band. A preliminary attempt at a synthesis of the data from these three sources is presented in the supplement which is attached to this report. This supplement is a preprint of a recently presented paper which integrates the audiometric data from all three sources with a weighting function derived from narrow band impulses generated by a conventional high frequency audio system.

The general conclusions that can be drawn from these data are very similar to those that were made for the impulses produced by Sources I and II (reports ADA 206-180 and ADA 221-731). However, the three sources do differ in the magnitude of the absolute energy levels of the exposure at which trauma begins to develop and in the frequency (place) of maximum effect. In summary, (1) There was no statistical difference in the amount of hearing loss or the amount of sensory cell loss for exposure to a single impulse at 150, 155, or 160 dB peak SPL. Individual animals showed no permanent hearing loss and no significant sensory cell loss. (2) The variability in hearing and cell losses across animals increases as the severity of the exposure increases. The variability in the results makes it difficult to describe the data with conventional statistics. (3) A general, though not surprising, trend in the data is that as the peak levels and the N increase, permanent effects increase; these permanent effects seem to be dependent upon peak levels more than upon the total energy in the exposure stimulus. Also, for a constant peak and energy level, the more rapid presentation rate (10/min) generally seemed to produce the greater effect although the effect is not consistent.

Since the experimental data reported here were derived from an experimental protocol that was identical to that reported in our previous annual reports, the format of the data presentation has been kept the same. An appendix of the complete individual animal data will be submitted at the conclusion of the contract.

FOREWORD

Disclaimer:

Citations of commercial organizations and trade names in this report do not constitute an official Department of the Army endorsement or approval of the products or services of these organizations.

Animal Use:

In conducting the research described in this report, the investigators adhered to the "Guide for the Care and Use of Laboratory Animals," prepared by the Committee on Care and Use of Laboratory Animals of the Institute of Laboratory Animal Resources, National Research Council (DHHS Publication No. (NIH) 86-23, revised 1985).

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I. INTRODUCTION

There are a number of different suggested standards for exposure to impulse/impact noise [e.g. Coles, et al. (1968), Smoorenburg (1982), and Pfander (1980)]. Although each of these criteria has its proponents, there is a consensus that there is, in fact, an extremely limited empirical data base upon which a standard can be built. The difficulties associated with generating a data base are compounded by the extremely broad range of high intensity noise transients that exist in various industrial and military environments. For example, in industry, impacts often occur as a pseudorandom sequence, having variable peak intensities, and are superimposed on a continuous noise background. This combination produces a highly non-Gaussian noise of variable character often with a very high kurtosis. While rms SPLs' might be within the limits of hearing conservation standards, peaks in excess of 130 dB or more can be very common but irregular in their temporal characteristics. At the other extreme, the diversity of military weapon systems produce impulses which originate as the result of a process of shock wave formation and propagation following high energy discharges. These waves, which can have peak levels in excess of 180 dB, can be either reverberant or non-reverberant in nature depending upon the environment in which they are encountered and they also may be superimposed on a background noise. Trying to develop a single standard to cover this broad range of "acoustic" signals is a formidable task.

The primary goal of this research project is to produce a data base from which one could estimate the hazards to hearing associated with a wide variety of non-reverberant blast wave exposures. To achieve this objective four different blast wave generation devices were designed. Three of these sources are based upon shock tube methods and one uses a high energy electrical discharge to produce a shock wave. The four sources produce pressure-time waveforms whose A-weighted amplitude spectra peak at four different regions of the audible spectrum. The conventional shock tube (Source I) has maximum A-weighted energy in the .250 kHz octave band; the 5-inch "Lamont" rapid acting valve driven shock tube (Source II) has its energy maxima in the 1 kHz octave band; the 3-inch 'Lamont" tube (Source III) has its energy maxima in the 2 kHz octave band; while the spark discharge energy (Source IV) is concentrated at the 4 kHz octave band. These sources in anechoic surroundings produce non-reverberant waves that approximate the ideal Friedlander wave. By varying the exposure variables such as peak sound pressure level (SPL), number of impulses and the presentation rate, the relation between these variables and auditory system trauma can be established. A brief background and literature review which summarizes the current state of knowledge on the contribution of these parameters to hearing loss is presented in the first progress report ADA 206-180 which documents the results of exposure to the very low frequency blast waves that are produced by Source I. The results produced by Source II are documented in report ADA 221-731. This present report, which documents the audiometric and histological results of exposure to the impulses produced by Source III, follows a pattern of data presentation that parallels the presentation format of reports ADA 206-180 and ADA 221-731. In addition to the importance of the parameters mentioned above, when the data collection from all four sources is complete, the relation between the spectral characteristics of the impulse and the subsequent hearing loss can begin to be explored. This spectral question is an important one for which very little experimental data are available. The attached supplement to this report focuses upon the question of spectral sensitivity by performing a preliminary analysis of the data base formed by Sources I, II and III by integrating these data into a similar data base obtained from animals exposed to impulses that were generated using a conventional electro-acoustic source. This integration

of two diverse data sets was made possible through the use of an empirical weighting function developed at the USAARL laboratories. The addendum (Section IV) of this report is the text of a paper which was a product of a cooperative effort between our laboratories and the USAARL through contract DAMD17-86-C-6139. An appendix of all the audiometric and histological data from Source III will be made available at the termination of this contract. [Note: The format of this report follows essentially the same format used for the presentation of the results from Sources I and III.]

II. PARAMETRIC EXPERIMENTS

A. EXPERIMENTAL METHODS

The methodology used to acquire the data presented in this report has been reported in detail in our earlier reports ADA 206-180 and ADA 221-731. Briefly, the basic experimental protocol that is common to all of the experiments consists of the following steps: (1) Preexposure audiograms and tuning curves (TC's) are measured on each animal. (2) The animals are exposed to noise under well controlled conditions. The temporal and spectral characteristics of the noise are recorded. (3) The animal's evoked response thresholds are again measured immediately after exposure and at regular intervals after exposure. At 30 days postexposure, the audiogram is again measured to establish the animal's permanent threshold shift, (PTS), and postexposure TC's are once again collected at all audiometric test frequencies. (4) The animals are euthanatized and their cochleas are then prepared for microscopic analysis. Cochleograms, which provide a quantitative description of the extent and location of the hair cell lesions, are prepared for each cochlea.

Subjects: The chinchilla was used as the experimental animal. Over the years, the chinchilla has been used in a wide variety of auditory experiments and consequently, much is known about its threshold (Miller, 1970; Salvi et al., 1978), psychophysical tuning curves (McGee et al., 1976; Salvi et al., 1982a), threshold for gap detection (Giraudi et al., 1980) and amplitude modulated noise (Salvi et al., 1982b). These psychophysical results indicate that the chinchilla's hearing capabilities are quite similar to those of man. The chinchilla is perhaps the most common animal used in noise trauma research even though there is a general consensus that the species is more susceptible to noise trauma than is man. However, phenomenologically the chinchilla is considered to be a suitable model for man. Thus, the chinchilla was chosen as a reasonable animal model for the blast wave studies described in this report.

One hundred and five (105) chinchillas were used in this study. Each animal was anesthetized [IM injection of Telazol© (Tiletamine-Zolazepan, 30.0 mg/kg)] and made monaural by the surgical destruction of the left cochlea. A chronic electrode was implanted near the inferior colliculus for single-ended near-field recording of the evoked potential (Henderson et al., 1973; Salvi et al., 1982a). Each animal was given Amoxicillin© (100.0 mg/kg, subcutaneous) to reduce the possibility of postoperative infection. The animals were allowed to recover for at least two weeks before evoked potential testing began.

Preexposure testing: Hearing thresholds were estimated on each animal using the auditory evoked potential (AEP). The AEP has been shown to be a valid index of hearing threshold in the chinchilla. The correlation between the behavioral and evoked response measures has been strengthened by directly comparing, in the same animal, estimates of noise-induced behavioral and evoked potential threshold shifts (Henderson et al., 1983; Davis and Ferraro,

1984). There is a close correlation between the behavioral and evoked response thresholds before, during, and after acoustic overstimulation. In other words, the evoked potential threshold estimation procedure provides a good estimate of the magnitude of noise-induced hearing loss. The animals were awake during testing and restrained in a yoke-like apparatus to maintain the animal's head in a constant position within the calibrated sound field. AEP's were collected to 20 msec tone bursts (5 msec rise/fall time) presented at a rate of 10 per second. A general-purpose computer (Digital Equipment Corporation MicroPDP-11/73) with 12-bit A/D converter(Data Translation 3362), timer (ADAC 1601) and digital interface (ADAC 1632) was used to acquire the evoked potential data and control the frequency, intensity and timing of the stimulus via a programmable oscillator (Wavetek 5100), programmable attenuator (Spectrum Scientific MAT) and electronic switch (Coulbourn Instruments S84-04). The electrical signal from the implanted electrode was amplified (50,000x) and filtered (30 Hz to 3000 Hz) by a Grass P511J biological amplifier and led to the input of the A/D converter where it was sampled at 20 kHz (50 msec period) over 500 points to obtain a 25 msec sampling window. Each sampled waveform was analyzed for large amplitude artifacts; and if present, the sample was rejected from the average and another sample taken. Averaged AEP's were obtained from 250 presentations of the 20 msec signal. Each waveform was stored on disk for later analysis.

Thresholds were measured using an intensity series with 5 dB steps at octave intervals from 0.5 to 16.0 kHz and at the half-octave frequency of 11.2 kHz. Threshold was determined to be one half step size (2.5 dB) below the lowest intensity that showed a "response" consistent with the responses seen at higher intensities. The intensity resolution of our method is 5 dB. The average of at least three separate threshold determinations at each frequency obtained on different days was used to obtain the preexposure audiogram.

Tone-on-tone masking functions (i.e., AEP tuning curves. see e.g., Salvi et al., 1982a) were measured on three animals in each group at six probe frequencies between 0.5 and 11.2 kHz presented at 15 dB above the preexposure threshold. A simultaneous masking paradigm was used (McGee et al., 1976). The probe tone had a duration of 20 ms and the intensity was set at 15 dB sensation level at the given test frequency. A simultaneous pure tone masker was presented at increasing levels until the masker just abolished the evoked potential elicited by the probe tone. The procedure was repeated over a range of masker frequencies around the probe tone to yield a "V" shaped masking function. The AEP has been shown to provide as good an estimate of the frequency selectivity as that obtained by behavioral techniques (Salvi et al., 1982a). It also shows that a small population of neurons within a restricted frequency band are contributing to the AEP at near threshold intensities. The advantage of the AEP tuning curves is that they provide an independent method of assessing frequency selectivity and a method that is much easier to apply than behavioral techniques. Ten masker frequencies (from a Wavetek Model 23 programmable frequency synthesizer) distributed in frequency above and below the probe tone frequency were presented in an intensity series with 5 dB steps. The masked threshold was taken as one half a step size (2.5 dB) above the last masker intensity that resulted in a "response". TC's were run on 63 chinchillas (i.e., 3 from each group) from which 378 preexposure TC's and 378 postexposure TC's were obtained. All the individual animal data is tabulated in a three volume data appendix which will be available upon request at the termination of this contract. The results of the analysis of TC data is published in Davis et al. (1989) and will not be repeated in this document. A final analysis of the TC data will be performed after the data have been collected from the fourth and final blast wave source.

Blast Wave Generation, Measurement and Analysis: A principal requirement for this study was the precise measurement and recording of the blast wave. The computer system used for this purpose was a Compaq 286 Deskpro personal computer using the ASYST™ application package (ASYST™ Software Technologies, Inc., Rochester, NY). The blast wave was first digitized and then recorded in storage devices (e.g., hard disk or magnetic tape). By using the customized software developed in our laboratory, each digitized blast wave was analyzed to extract characteristics such as the total acoustic energy, energy spectrum, peak and root-mean-square (RMS) sound pressure level (SPL) etc.

A schematic representation of the blast wave exposure test facility using the 3-inch "Lamont" source is illustrated in Figure 1. A cross-sectional view of the "Lamont" driver is shown in Figure 2. The Lamont source uses a relatively simple rapid acting valve to quickly establish a high pressure discontinuity in the expansion section in order to "drive" the shock front. A force differential generated over the area of the low pressure chamber relative to the high pressure chamber, on the rear plate, maintains the seal of the high pressure chamber. As the low pressure is gradually reduced a point is reached where the net force acting on the value reverses direction and the valve rapidly thrusts forward releasing the "slug" of high pressure gas into the expansion section. N_2 is used as the operating gas and the pressure in the high pressure chamber varies from approximately 100 psig to 1000 psig to achieve peak sound pressure levels of the blast wave of from 150 dB to 160 dB at the exposure location. The SPL of the blast wave can be controlled by systematically adjusting the pressure in the compression section. The pressure-time history of the blast wave was recorded using a transducer located on the center line at a variable distance from the outlet of the shock tube. The experimental animal was mounted next to the transducer.

Two different types of transducers were used to convert the dynamic acoustic pressure into an analog signal. The B&K 1/8 inch microphone (Type 4138) and the PCB crystal microphone (Model 112A22) were selected because of their ability to record high peak levels and their relatively fast rise times. A B&K microphone preamplifier (Type 2639), a B&K measuring amplifier (Type 2606), and a PCB six-channel amplifying power unit (Model 483A08) were used to amplify the analog signals from the B&K and PCB microphones respectively. Both transducers yielded identical results. The amplified analog signals were monitored on an oscilloscope. The output signal from the transducers was amplified and, in order to avoid aliasing problems that can occur in analog-to-digital (A/D) conversion, the amplified signals were filtered using an anti-aliasing filter prior to digitizing. The sampling rate of the A/D convertor (12-bit) was set at 500 kHz and the cut off frequency of the anti-aliasing filter was set at 150 kHz (approximately 1/3 of the sampling rate). For each blast wave, 16,384 samples were recorded for later analysis. Software was written using this PC-based system to perform the following computations: total sound exposure and exposure level calculations (Young, 1970); energy flux calculations; and spectral analysis using a 4096-point FFT; A-weighted analysis, etc.

Thus, for each impact the total sound exposure or exposure level could be calculated (i.e., the time integrated, squared sound pressure). For the impulse data presented here, the total sound exposure was divided by the standard characteristic impedance of air, ρc = 406 mks rayls, to produce a quantity with units of energy flux (i.e., J/m^2). Similarly, all spectral quantities $|P(\omega)|^2$ were converted to units of energy flux spectral density,

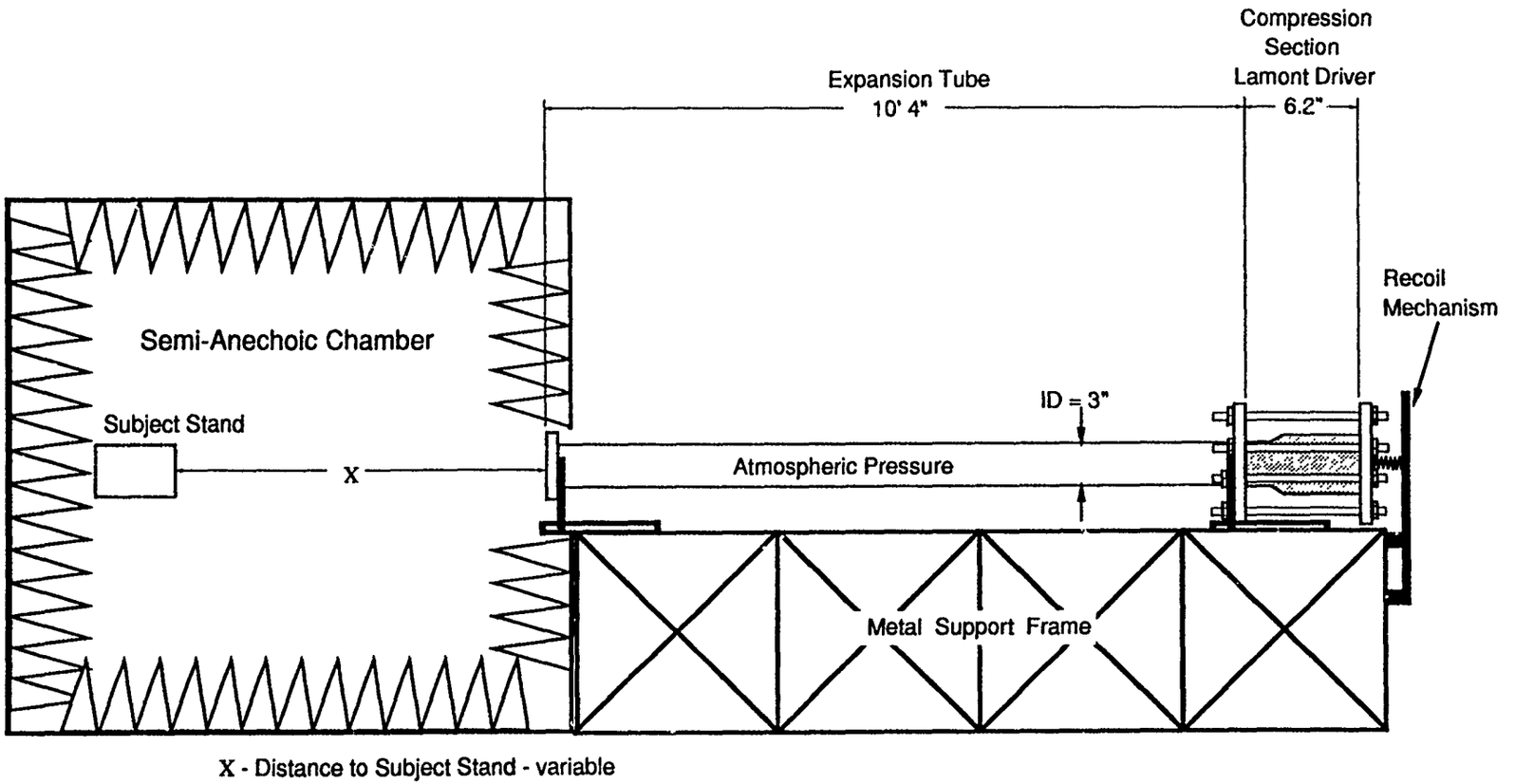

Figure 1. Schematic Side View of the 3" Lamont Shock Tube

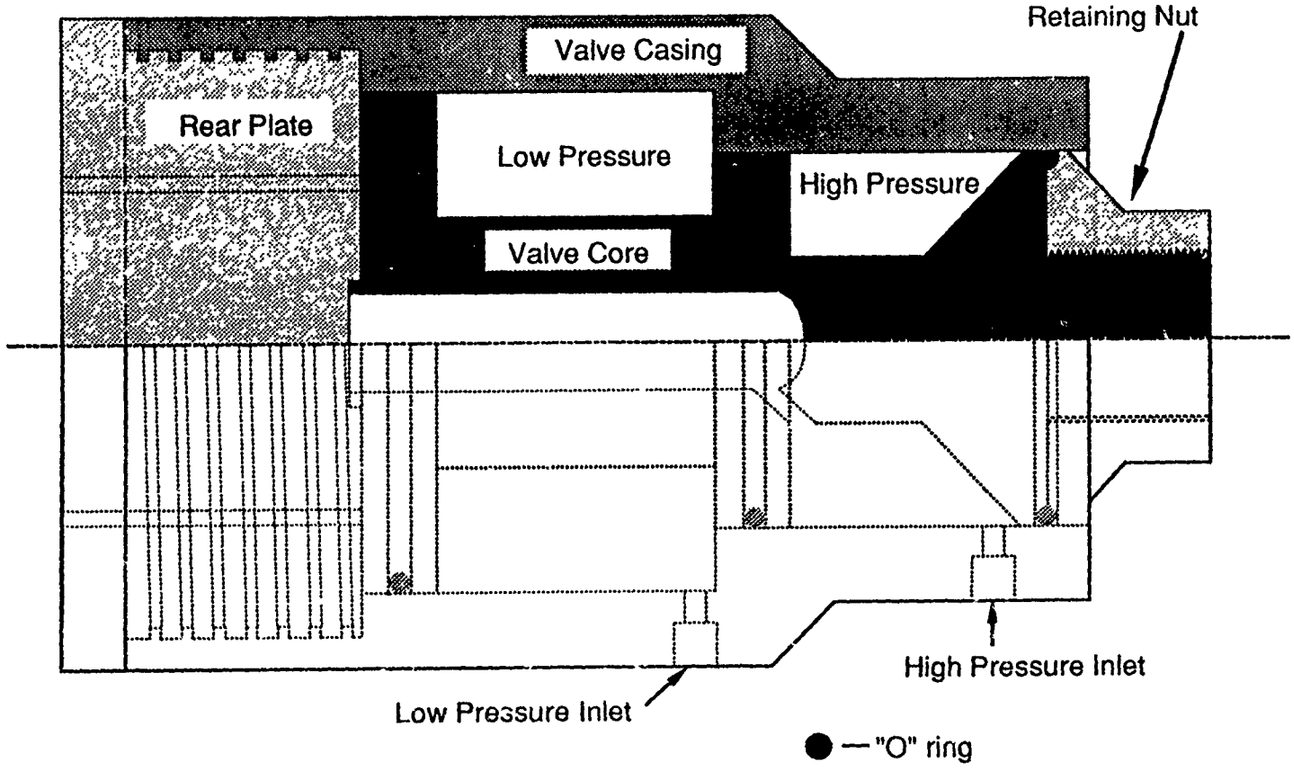

Figure 2. Schematic of the Lamont Quick-acting Valve

and for each impulse exposure, the total "energy flux" in the octave bands having center frequencies at the audiometric test frequencies was calculated. [Since only p(t) was measured, the true energy flux cannot be obtained except in the special case of a plane wave.]

Exposure of Animals: For a given exposure condition, each chinchilla was exposed at the same fixed location relative to the shock tube expansion section outlet. During exposure the animal was unanesthetized but immobilized in a leather harness (Patterson et al., 1986). The right pinna was folded back and fixed in place to insure that the entrance of the external meatus was not obstructed and the position of the entire animal was adjusted so that the cross sectional plane of the meatus was oriented parallel to the advancing shock front (i.e., a normal incidence).

Each experimental group of animals consisted of five animals. Each animal was individually exposed to one of the exposure conditions shown in Table I. A total of 105 animals were used to complete this experimental paradigm.

Postexposure Testing: After the exposure was complete, threshold recovery functions were measured at 0.5, 2.0 and 8.0 kHz at 0, 2, 8, 24 and 240 hours after removal from the noise (using the same method as described for preexposure testing). After at least 30 days, final audiograms were constructed using the average of three separate threshold determinations at each of the seven preexposure frequencies. Permanent threshold shift (PTS) was defined as the difference between the postexposure and preexposure thresholds at each individual test frequency. Postexposure AEP tuning curves were collected at the six preexposure probe tones presented at 15 dB above the postexposure threshold.

Cochlear Histology: Following postexposure audiometric testing, animals were euthanatized by decapitation and the cochleas were immediately removed and fixed. The cochleas were dissected and the status of the sensory cell population was evaluated using conventional surface preparation histology (Engstrom et al., 1966). Briefly, the stapes was removed and the round window membrane opened to allow transcochlear perfusion, via the scala tympani/scala vestibuli with cold 2.5% glutaraldehyde in veronal acetate buffer at 7.3 pH (605 mOsm). Postfixation was performed on the following day with one percent osmium tetroxide in veronal acetate buffer (pH 7.3) for 30 minutes. The cochleas were dissected and the entire sensory epithelium along with the lateral wall structures was mounted in glycerin on glass slides. [See Hamernik et al., (1987) for a more complete description]. The status of sensory and supporting cells were evaluated with Nomarski Differential Interference Contrast microscopy and entered into a data-base on a laboratory computer (Digital Equipment Corporation MicroPDP-11/73 and Macintosh II). Standard cochleograms were then constructed by computing the percent sensory cell loss across the length of the cochlea in 0.24 mm steps. These cell loss figures were then converted into percent loss over octave bands centered at the audiometric test frequencies along the length of the cochlea and correlated with the frequency-place map constructed by Eldredge et al. (1981).

B. RESULTS

The results of the present experiments are grouped into sections devoted to preexposure threshold data, analysis of the exposure stimuli and postexposure threshold and histological data. The audiometric and histological dependent variables were analyzed using mixed design analyses of variance with repeated measures on one factor (frequency). The SPSS^X

TABLE I

A Definition of the Experimental Groups

Group	N	Intensity	Number	Rate
1	5	150 dB Peak SPL	1	
2	5	150 dB Peak SPL	10	10 per minute
3	5	150 dB Peak SPL	10	1 per minute
4	5	150 dB Peak SPL	10	1 per 10 minutes
5	5	150 dB Peak SPL	100	10 per minute
6	5	150 dB Peak SPL	100	1 per minute
7	5	150 dB Peak SPL	100	1 per 10 minutes
8	5	155 dB Peak SPL	1	
9	5	155 dB Peak SPL	10	10 per minute
10	5	155 dB Peak SPL	10	1 per minute
11	5	155 dB Peak SPL	10	1 per 10 minutes
12	5	155 dB Peak SPL	100	10 per minute
13	5	155 dB Peak SPL	100	1 per minute
14	5	155 dB Peak SPL	100	1 per 10 minutes
15	5	160 dB Peak SPL	1	
16	5	160 dB Peak SPL	10	10 per minute
17	5	160 dB Peak SPL	10	1 per minute
18	5	160 dB Peak SPL	10	1 per 10 minutes
19	5	160 dB Peak SPL	100	10 per minute
20	5	160 dB Peak SPL	100	1 per minute
21	5	160 dB Peak SPL	100	1 per 10 minutes
Total	105			

statistical package was used and the probability of a type I error was set at 0.05.

Preexposure Thresholds: The mean preexposure thresholds for all 105 animals are reported in Table II and plotted in Figure 3 along with the behavioral audibility curve published by Miller (1970). The Miller curve was corrected for the effects of temporal integration using the data of Henderson, (1969). The error bars in this figure represent one standard deviation above and below the mean.

Table II

Summary of Mean Preexposure Thresholds (dB) for All Animals (N = 105) Compared to Published Norms

	Test Frequency (kHz)							
Present study	0.5	1.0	2.0	4.0	8.0	11.2	16.0	
	17.3	2.2	6.0	-2.0	13.5	12.4	21.5	$\bar{X}$
	5.8	6.4	6.1	6.4	6.8	8.0	8.4	s
Miller (1970) (750 ms signals)	5.1	3.0	2.7	1.9	5.8	9.9	12.1	$\bar{X}$
	6.1	4.1	4.7	7.1	5.4	6.7	6.9	s
	36	36	36	36	36	34	36	N
Miller (1970) corrected for temporal integration (Henderson, 1969)	16.2	14.1	13.8	13.0	16.9	21.0	23.2	$\bar{X}$

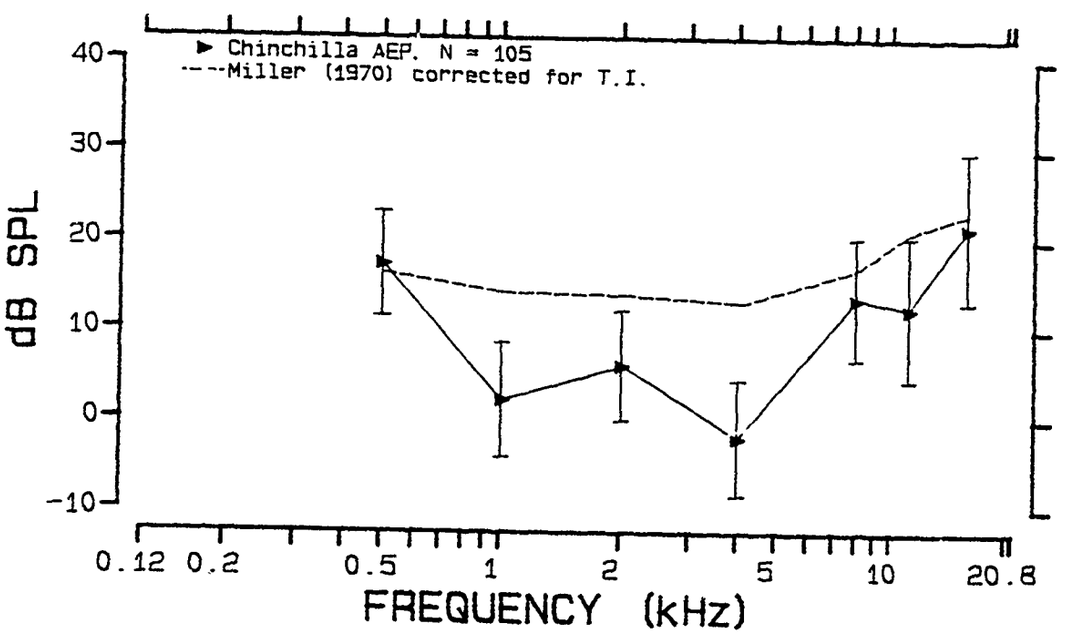

Figure 3. Mean preexposure thresholds for 105 chinchillas.

The mean preexposure thresholds are generally better than Miller's (1970) behavioral thresholds at the mid-frequencies when the (approximate 11.1 dB) effects of temporal integration are taken into consideration. Lower thresholds, which are also found in other published data, probably reflect improvements in the techniques of AEP recording. The mean preexposure thresholds for the 21 individual groups of animals and the mean preexposure thresholds for all 105 subjects are summarized in Table III.

The audiological dependent variables of the study reported are maximum threshold shift (TS_{max}) and permanent threshold shift (PTS). Each of these variables is computed by subtracting the preexposure from the postexposure thresholds. Thus, each animal serves as its own control subject. There were no statistically significant differences in mean preexposure thresholds among groups ($F = 0.48$, $df = 20/84$). There was a statistically significant frequency main effect ($F = 195.30$, $df = 6/504$) that was anticipated on the basis of our previous knowledge of the chinchilla audiogram (Fay, 1988). The interaction between group and frequency was not statistically significant ($F = 1.06$, $df = 120/504$). The analysis of variance for the preexposure thresholds is summarized in Table IV.

Noise Exposures: Pressure-time histories for each of the three intensity waves produced by Source III are shown in Figures 4 (a-c). A time record over a period of 10 ms is shown. The Fourier energy spectrum for these same three waveforms over the entire 33 ms window is also shown in Figure 4 (a-c). The total energy flux for each exposure condition is presented in Table V in relative dB levels as well as in Joules/meter^2. A tabulation of the octave band A-weighted and unweighted energy flux values for a single impulse at 150, 155 and 160 dB peak SPL is presented in Table VI. The data presented in Table V is also shown plotted as a bar graph in Figure 5 to facilitate the comparison of the three waves used for the exposures. For all three peak intensities, each wave had a similar p-t profile and similar A-weighted and unweighted spectral distribution of energy. The A-weighted analysis shows that the peak of the energy spectrum lies in the 2 kHz octave band for each of the three intensity waves.

Postexposure Audiometric Results: There are four independent variables in the present experiments: number of impulses (1X, 10X or 100X), impulse peak level (150, 155 or 160 dB SPL), impulse presentation rate (10/min, 1/min or 1/10m), and frequency (i.e., audiometric test frequency or basilar membrane location). The dependent variables are TS_{max}, PTS, percent outer hair cell loss and percent inner hair cell loss. The independent variable of frequency is the only within-subjects variable. The experimental design did not lend itself to a typical four factor mixed design analysis of variance since there was no rate variable for the three groups exposed to a single impulse. Therefore, several different analyses were performed on each of the four dependent variables. Since the rate variable could not be applied to an analysis which included all groups, the rate variable was analyzed as one variable in a three-factor mixed-design analysis of variance with impulse peak and frequency as the other two factors. In other words, two separate three-factor analyses were performed, the first on the groups exposed to 10 impulses, and the second on the groups exposed to 100 impulses (see Tables VIII and IX). The remaining three groups (i.e., the 1x groups) were analyzed using a two-factor mixed-design analysis of variance with only impulse peak and frequency as independent variables (see Table VII). Thus, each of the above analysis were performed only upon those groups that were exposed to an equal number of impulses. The main effect of the number of impulses was determined using a separate three-factor mixed design analysis of variance

TABLE III

Preexposure Threshold Means (dB) and Standard Deviations for all Groups

dB Peak	#	Rate	N	Test Frequency (kHz) 0.5	1.0	2.0	4.0	8.0	11.2	16.0	
150 dB	1		5	15.8	-2.8	6.8	2.8	15.2	16.5	16.5	$\bar{X}$
				5.1	5.2	8.0	5.2	6.2	5.8	6.7	s
150 dB	10	10/m	5	18.2	4.2	9.5	0.5	18.8	6.5	24.8	$\bar{X}$
				4.8	8.1	4.6	5.3	6.7	5.7	13.8	s
150 dB	10	1/m	5	17.2	2.8	6.5	-0.8	15.5	10.2	17.2	$\bar{X}$
				3.8	5.9	5.8	4.9	5.2	7.8	4.9	s
150 dB	10	1/10m	5	16.2	1.2	4.5	-4.5	13.5	17.8	18.5	$\bar{X}$
				6.8	8.7	6.6	6.2	3.8	5.1	5.7	s
150 dB	100	10/m	5	23.8	9.5	6.2	-0.8	15.2	11.2	22.8	$\bar{X}$
				11.3	12.5	12.8	7.7	10.0	5.9	11.6	s
150 dB	100	1/m	5	20.8	5.5	2.8	-2.8	15.5	7.8	21.5	$\bar{X}$
				5.3	12.2	5.1	7.9	11.0	13.4	8.6	s
150 dB	100	1/10m	5	13.5	-0.5	6.5	-0.2	18.5	13.2	26.2	$\bar{X}$
				2.2	5.5	9.5	5.3	6.0	10.3	15.3	s
155 dB	1		5	17.8	1.5	5.8	-2.2	12.5	13.5	21.2	$\bar{X}$
				4.9	7.8	6.3	7.4	4.9	9.3	6.9	s
155 dB	10	10/m	5	21.2	2.2	2.8	-3.2	12.5	17.5	20.8	$\bar{X}$
				7.2	7.6	2.7	5.8	4.6	8.3	4.7	s
155 dB	10	1/m	5	21.2	0.2	7.5	-2.5	16.8	17.2	22.5	$\bar{X}$
				4.9	4.2	5.7	5.1	7.1	6.9	10.7	s
155 dB	10	1/10m	5	17.5	1.2	6.2	4.8	15.5	15.8	21.2	$\bar{X}$
				3.5	3.0	2.2	4.9	3.6	6.7	8.6	s
155 dB	100	10/m	5	14.2	1.5	4.5	-5.2	12.8	14.5	20.8	$\bar{X}$
				3.9	5.1	4.8	1.9	6.2	3.6	7.5	s
155 dB	100	1/m	5	15.8	2.2	7.8	0.8	11.8	7.8	21.2	$\bar{X}$
				6.1	6.1	7.3	8.5	4.5	6.3	4.0	s
155 dB	100	1/10m	5	18.5	4.2	5.5	-2.8	15.2	7.2	19.5	$\bar{X}$
				5.3	3.3	4.5	4.6	6.7	5.9	4.6	s
160 dB	1		5	16.2	3.8	6.5	-7.2	11.2	9.5	18.8	$\bar{X}$
				7.2	4.6	6.9	4.3	6.4	5.1	6.1	s
160 dB	10	10/m	5	13.5	-1.8	3.8	-5.2	8.8	12.2	25.8	$\bar{X}$
				5.6	3.5	5.1	8.0	9.6	9.5	4.2	s
160 dB	10	1/m	5	17.8	-1.2	3.8	-6.2	9.5	9.8	23.2	$\bar{X}$
				6.3	5.8	4.9	5.2	3.8	5.5	8.5	s
160 dB	10	1/10m	5	17.2	4.2	4.5	-2.8	8.2	7.8	21.5	$\bar{X}$
				3.0	3.7	5.6	4.5	3.7	10.9	5.5	s
160 dB	100	10/m	5	17.5	1.8	4.8	-1.8	8.8	16.8	26.2	$\bar{X}$
				5.9	4.8	7.3	7.5	7.6	10.9	6.1	s
160 dB	100	1/m	5	17.5	3.2	7.2	-2.2	14.5	14.5	21.2	$\bar{X}$
				2.4	1.9	4.6	8.8	7.7	7.7	6.8	s
160 dB	100	1/10m	5	12.2	4.5	11.8	-0.5	14.2	12.2	20.5	$\bar{X}$
				6.6	4.9	5.6	8.7	9.8	7.1	18.0	s

Table IV

Analysis of Variance Summary Table
of Preexposure Thresholds

Source of Variation	SS	df	MS	F	p
Groups	1096.27	20	54.81	.48	.968
Between Subjects	9623.33	84	114.56		
Frequency	44538.81	6	7423.13	195.30	.000
Groups x Frequency	4831.51	120	40.26	1.06	.333
Within Subjects	19156.67	504	38.01		

Table V

Total Weighted and Unweighted Energy Flux (J/m^2)
Values for Each Exposure Condition

Peak	Weight	Absolute Energy (J/m^2)			Relative Energy (dB) re: $1J/m^2$		
SPL (dB)		1X	10X	100X	1X	10X	100X
150	None	0.08	0.79	7.94	-11.00	-1.00	9.00
	A	0.07	0.69	6.93	-11.59	-1.59	8.41
155	None	0.31	3.14	31.36	-5.03	4.97	14.97
	A	0.25	2.47	24.72	-6.07	3.93	13.93
160	None	0.82	8.20	82.04	-0.86	9.14	19.14
	A	0.61	6.07	60.66	-2.17	7.83	17.83

Table VI

Octave Band Unweighted and A-Weighted Energy Flux (J/m^2)
for a Single Impulse Generated by the 3" Lamont Shock Tube.

	150 dB Peak SPL		155 dB Peak SPL		160 dB Peak SPL	
Octave Band CF (kHz)	Unwtg. Energy	A-Wtg. Energy	Unwtg. Energy	A-Wtg. Energy	Unwtg. Energy	A-Wtg. Energy
< 0.125	0.0022	0.0002	0.0115	0.0075	0.0371	0.0221
0.125	0.0012	0.0004	0.0095	0.0004	0.0662	0.0023
0.25	0.0106	0.0012	0.0631	0.0088	0.1262	0.0200
0.5	0.0057	0.0029	0.0421	0.0191	0.1385	0.0649
1.0	0.0112	0.0120	0.0366	0.0397	0.0797	0.0820
2.0	0.0199	0.0259	0.0700	0.0914	0.1522	0.1980
4.0	0.0110	0.0139	0.0430	0.0546	0.1058	0.1332
8.0	0.0087	0.0067	0.0181	0.0136	0.0605	0.0472
16.0	0.0039	0.0017	0.0113	0.0037	0.0278	0.0107
> 16.0	0.0051	0.0051	0.0084	0.0084	0.0264	0.0264
Total	0.0794	0.0693	0.3136	0.2472	0.8204	0.6066

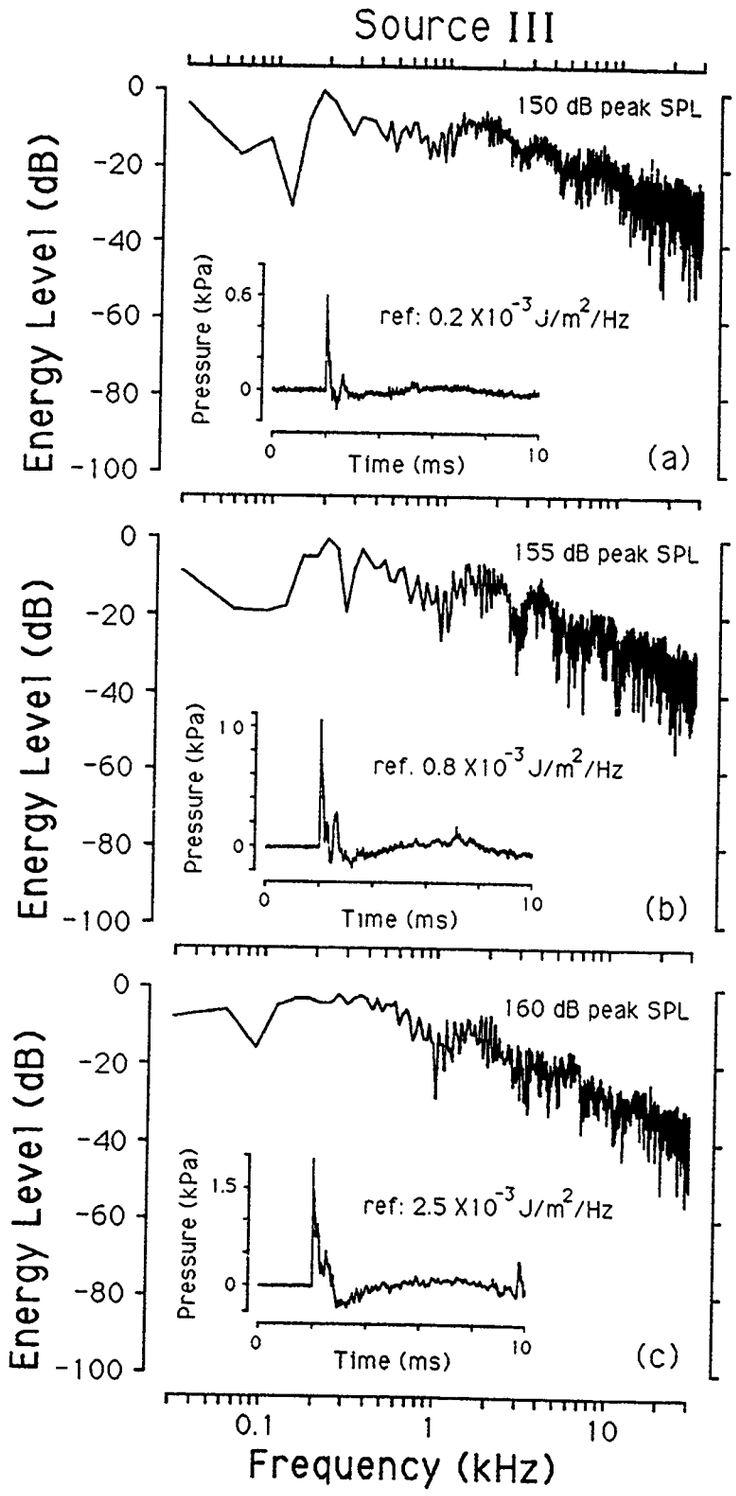

Figure 4. Amplitude spectra and pressure-time waveforms for the blast waves from the 3" Lamont shock tube.

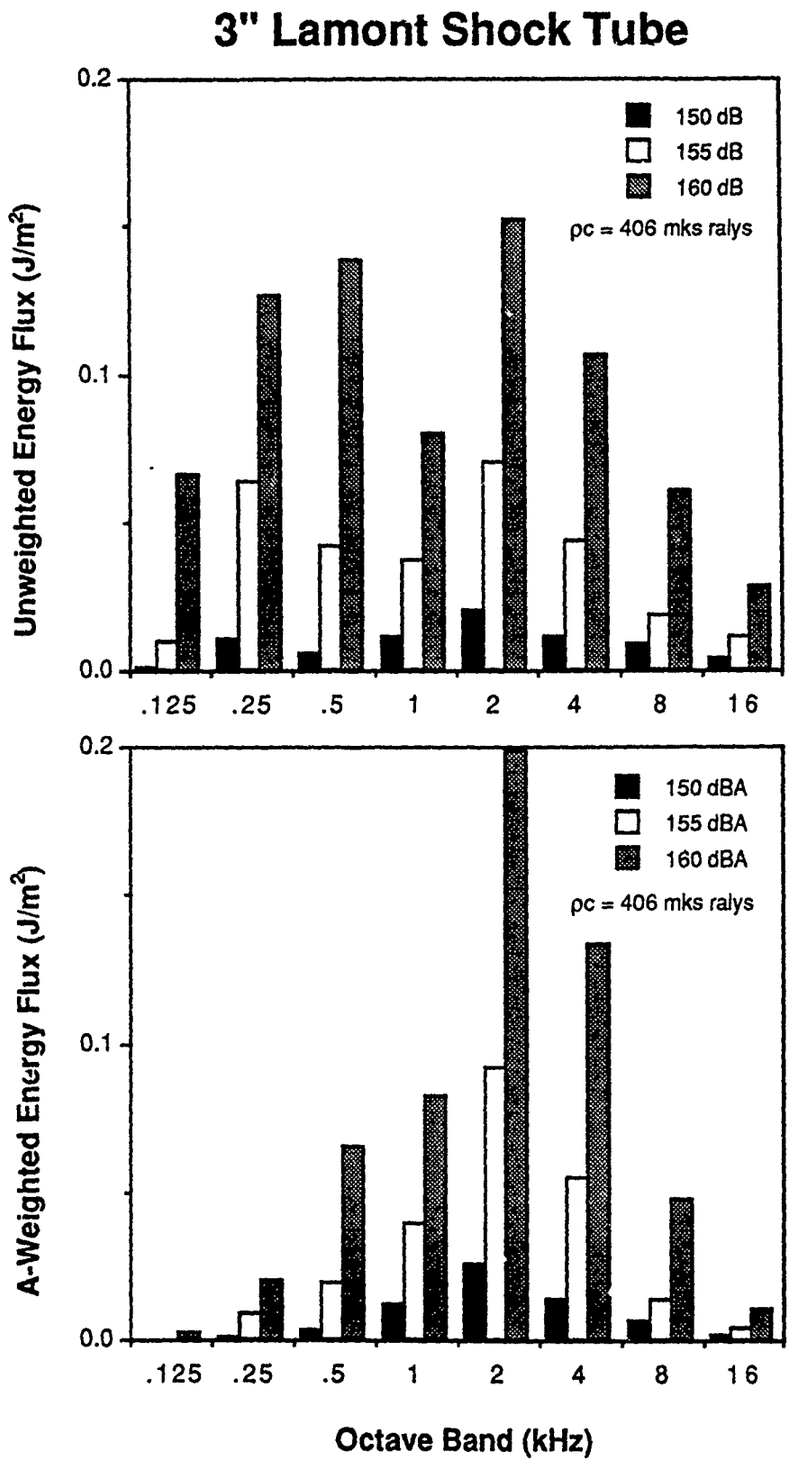

Figure 5. The unweighted (upper) and A-weighted (lower) octave band analysis of the three blast waves.

Table VII

Analysis of Variance Summary Table for Groups Exposed to 1 Impulse

Maximum Threshold Shift

Source of Variation	SS	df	MS	F	p
Peak	335.31	2	167.65	1.44	.276
Between Subjects	1400.74	12	116.73		
Frequency	529.75	2	264.88	4.55	.021
Peak x Frequency	116.91	4	29.23	.50	.734
Within Subjects	1395.93	24	58.16		

Permanent Threshold Shift

Source of Variation	SS	df	MS	F	p
Peak	65.45	2	32.72	1.41	.283
Between Subjects	279.37	12	23.28		
Frequency	108.15	6	18.02	.70	.649
Peak x Frequency	332.33	12	27.69	1.08	.391
Within Subjects	1848.41	72	25.67		

Percent Inner Hair Cell Loss

Source of Variation	SS	df	MS	F	p
Peak	5.10	2	2.55	.57	.579
Between Subjects	53.54	12	4.46		
Frequency	30.44	7	4.35	1.38	.224
Peak x Frequency	20.62	14	1.47	.47	.944
Within Subjects	264.33	84	3.15		

Percent Outer Hair Cell Loss

Source of Variation	SS	df	MS	F	p
Peak	43.21	2	21.60	.75	.493
Between Subjects	345.38	12	28.78		
Frequency	474.66	7	67.81	19.65	.000
Peak x Frequency	61.69	14	4.41	1.28	.239
Within Subjects	289.86	84	3.45		

Table VIII

Analysis of Variance Summary Table for Groups Exposed to 10 Impulses

Maximum Threshold Shift

Source of Variation	SS	df	MS	F	p
Peak	24596.35	2	12298.18	9.91	.000
Rate	2589.53	2	1294.76	1.04	.363
Peak x Rate	5741.71	4	1435.43	1.16	.346
Between Subjects	44687.59	36	1241.32		
Frequency	10295.79	2	5147.89	38.05	.000
Peak x Frequency	4179.08	4	1044.77	7.72	.000
Rate x Frequency	161.39	4	40.35	.30	.878
Peak x Rate x Frequency	352.69	8	44.09	.33	.954
Within Subjects	9741.45	72	135.30		

Permanent Threshold Shift

Source of Variation	SS	df	MS	F	p
Peak	4423.86	2	2211.93	4.80	.014
Rate	2272.54	2	1136.27	2.47	.099
Peak x Rate	580.11	4	145.03	.31	.866
Between Subjects	16583.81	36	460.66		
Frequency	4355.36	6	725.89	7.55	.000
Peak x Frequency	793.05	12	66.09	.69	.763
Rate x Frequency	929.19	12	77.43	.80	.645
Peak x Rate x Frequency	2124.46	24	88.52	.92	.574
Within Subjects	20777.30	216	96.19		

Percent Inner Hair Cell Loss

Source of Variation	SS	df	MS	F	p
Peak	434.71	2	217.36	2.65	.084
Rate	276.33	2	138.17	1.69	.200
Peak x Rate	221.36	4	55.34	.68	.613
Between Subjects	2950.20	36	81.95		
Frequency	737.64	7	105.38	3.90	.000
Peak x Frequency	670.77	14	47.91	1.78	.043
Rate x Frequency	718.25	14	51.30	1.90	.027
Peak x Rate x Frequency	431.00	28	15.39	.57	.962
Within Subjects	6800.41	252	26.99		

Percent Outer Hair Cell Loss

Source of Variation	SS	df	MS	F	p
Peak	19624.43	2	9812.22	5.25	.010
Rate	7972.67	2	3986.34	2.13	.133
Peak x Rate	5145.61	4	1286.40	.69	.605
Between Subjects	67296.89	36	1869.36		
Frequency	38475.53	7	5496.50	16.80	.000
Peak x Frequency	15888.46	14	1134.89	3.47	.000
Rate x Frequency	8730.46	14	623.60	1.91	.026
Peak x Rate x Frequency	9177.76	28	327.78	1.00	.467
Within Subjects	82439.51	252	327.14		

Table IX

Analysis of Variance Summary Table for Groups Exposed to 100 Impulses

Maximum Threshold Shift

Source of Variation	SS	df	MS	F	p
Peak	18459.07	2	9229.54	7.76	.002
Rate	5793.73	2	2896.87	2.43	.102
Peak x Rate	4033.78	4	1008.45	.85	.504
Between Subjects	42831.31	36	1189.76		
Frequency	6165.76	2	3082.88	21.03	.000
Peak x Frequency	1054.45	4	263.61	1.80	.139
Rate x Frequency	506.22	4	126.56	.86	.490
Peak x Rate x Frequency	1528.74	8	191.09	1.30	.256
Within Subjects	10556.69	72	146.62		

Permanent Threshold Shift

Source of Variation	SS	df	MS	F	p
Peak	44397.67	2	22198.84	12.70	.000
Rate	9354.97	2	4677.49	2.68	.082
Peak x Rate	3768.78	4	942.20	.54	.708
Between Subjects	62903.65	36	1747.32		
Frequency	5420.95	6	903.49	9.88	.000
Peak x Frequency	1297.14	12	108.10	1.18	.298
Rate x Frequency	4476.88	12	373.07	4.08	.000
Peak x Rate x Frequency	3075.66	24	128.15	1.40	.108
Within Subjects	19761.90	216	91.49		

Percent Inner Hair Cell Loss

Source of Variation	SS	df	MS	F	p
Peak	15172.04	2	7586.02	5.15	.011
Rate	2588.55	2	1294.27	.88	.424
Peak x Rate	5477.78	4	1369.44	.93	.458
Between Subjects	53022.07	36	1472.84		
Frequency	6668.61	7	952.66	3.55	.001
Peak x Frequency	8277.49	14	591.25	2.21	.008
Rate x Frequency	3255.56	14	232.54	.87	.595
Peak x Rate x Frequency	8292.46	28	296.16	1.10	.333
Within Subjects	67569.02	252	268.13		

Percent Outer Hair Cell Loss

Source of Variation	SS	df	MS	F	p
Peak	119178.12	2	59589.06	12.76	.000
Rate	39573.94	2	19786.97	4.24	.022
Peak x Rate	8286.82	4	2071.70	.44	.776
Between Subjects	168161.15	36	4671.14		
Frequency	83045.23	7	11863.60	30.08	.000
Peak x Frequency	30257.69	14	2161.26	5.48	.000
Rate x Frequency	12419.66	14	887.12	2.25	.007
Peak x Rate x Frequency	9340.99	28	333.61	.85	.693
Within Subjects	99397.74	252	394.44		

with peak, number and frequency as the independent variables (i.e., the data were collapsed across rate, see Table X).

Figures 6 through 12 present the audiometric and histological results for 1, 10 and 100 impulses. The error bars represent one standard error of the mean above and below the mean. If error bars are not present, the standard error was less than the size of the symbol representing the mean. The following is an interpretation of the results presented in these figures and tables.

1. Frequency: The main effect of frequency represents the only within-subjects independent variable. In general, the main effect of frequency was statistically significant for most of the analyses that are reported in this manuscript. A significant frequency main effect suggests that the audiometric or histological losses are different at the various audiometric test frequencies or locations along the basilar membrane. The statistically significant interactions of between-subjects independent variables and frequency indicate that the effect of the between-subjects variable depends on the frequency at which the dependent variable is measured. Thus, a statistically significant interaction between peak and frequency for percent outer hair cell loss tells us that the effect of impulse peak on the outer hair cell losses depended upon the frequency (i.e., place on the basilar membrane) that the losses were measured. From examining the figures, it is apparent that little losses were sustained at the locations on the basilar membrane associated with the very low frequencies, while the most severe effects of the impulse were in the mid-frequency region of the cochlea and to a lesser degree in the high-frequency region. Since a statistically significant main effect of frequency and interactions between frequency and other main effects are expected in this type of study, we will not discuss frequency effects or interactions at length throughout the remainder of this report. It is important to remember, however, that many of the statistically significant effects interact with the within-subjects frequency variable and thus the significance of the between-subjects main effects will be dependent upon the audiometric test frequency or the location in the cochlea.

2. Impulse Presentation Rate: The effect of rate was examined in two separate analyses (Tables VIII & IX) of the groups exposed to 10 impulses and those exposed to 100 impulses. The main effect of impulse presentation rate was statistically significant for only the percent outer hair cell losses for those groups exposed to 100 impulses ($F=4.24$, $df = 2/36$). In these groups, the faster impulse presentation rate caused the greatest amount of damage.

There were significant interactions between presentation rate and frequency. A significant interaction between rate and frequency in the percent outer hair cell losses in groups exposed to 100 impulses suggested that the faster rates (10/m) were more hazardous than the slower rates. However, a parallel analyses of the groups exposed to 10 impulses showed a different pattern of interactions. When examining these groups, it was apparent that the groups exposed to the intermediate rate of one impulse per minute either caused the greatest damage or was almost as damaging as the faster rate. The statistically significant interaction of rate and frequency for PTS in groups exposed to 100 impulses shows that the fastest rate cause the most hearing loss at the middle and upper frequency regions. Thus, in spite of the suggestion that the faster rates are more damaging, the inconsistent results from the higher two rates which extend across the three sources employed to date lead us to conclude that a systematic or consistent effect of impulse presentation rate could not be extracted from these data.

Table X

Analysis of Variance Summary Table for all Groups Collapsed Across the Rate Variable

Maximum Threshold Shift

Source of Variation	SS	df	MS	F	p
Peak	20677.33	2	10338.66	9.27	.000
Number	85883.27	2	42941.63	38.50	.000
Peak x Number	4431.86	4	1107.96	.99	.415
Between Subjects	107078.39	96	1115.40		
Frequency	9510.57	2	4755.28	37.66	.000
Peak x Frequency	2176.74	4	544.18	4.31	.002
Number x Frequency	1255.54	4	313.89	2.49	.045
Peak x Number x Frequency	1361.98	8	170.25	1.35	.222
Within Subjects	24243.11	192	126.27		

Permanent Threshold Shift

Source of Variation	SS	df	MS	F	p
Peak	15851.14	2	7925.57	7.95	.001
Number	52759.55	2	26379.77	26.45	.000
Peak x Number	15849.79	4	3962.45	3.97	.005
Between Subjects	95743.23	96	997.33		
Frequency	3491.71	6	581.95	6.33	.000
Peak x Frequency	1158.71	12	96.56	1.05	.401
Number x Frequency	2793.65	12	232.80	2.53	.003
Peak x Number x Frequency	896.17	24	37.34	.41	.995
Within Subjects	52993.81	576	92.00		

Percent Inner Hair Cell Loss

Source of Variation	SS	df	MS	F	p
Peak	3459.43	2	1729.72	2.57	.082
Number	22668.65	2	11334.33	16.85	.000
Peak x Number	7928.25	4	1982.06	2.95	.024
Between Subjects	64589.84	96	672.81		
Frequency	2100.96	7	300.14	2.31	.025
Peak x Frequency	1967.28	14	140.52	1.08	.371
Number x Frequency	2883.21	14	205.95	1.58	.078
Peak x Number x Frequency	4755.94	28	169.85	1.31	.135
Within Subjects	87331.03	672	129.96		

Percent Outer Hair Cell Loss

Source of Variation	SS	df	MS	F	p
Peak	42658.61	2	21329.30	6.90	.002
Number	136931.87	2	68465.94	22.15	.000
Peak x Number	44478.59	4	11119.65	3.60	.009
Between Subjects	296782.46	96	3091.48		
Frequency	37695.01	7	5385.00	16.32	.000
Peak x Frequency	15990.54	14	1142.18	3.46	.000
Number x Frequency	36819.65	14	2629.97	7.97	.000
Peak x Number x Frequency	12964.00	28	463.00	1.40	.082
Within Subjects	221795.98	672	330.05		

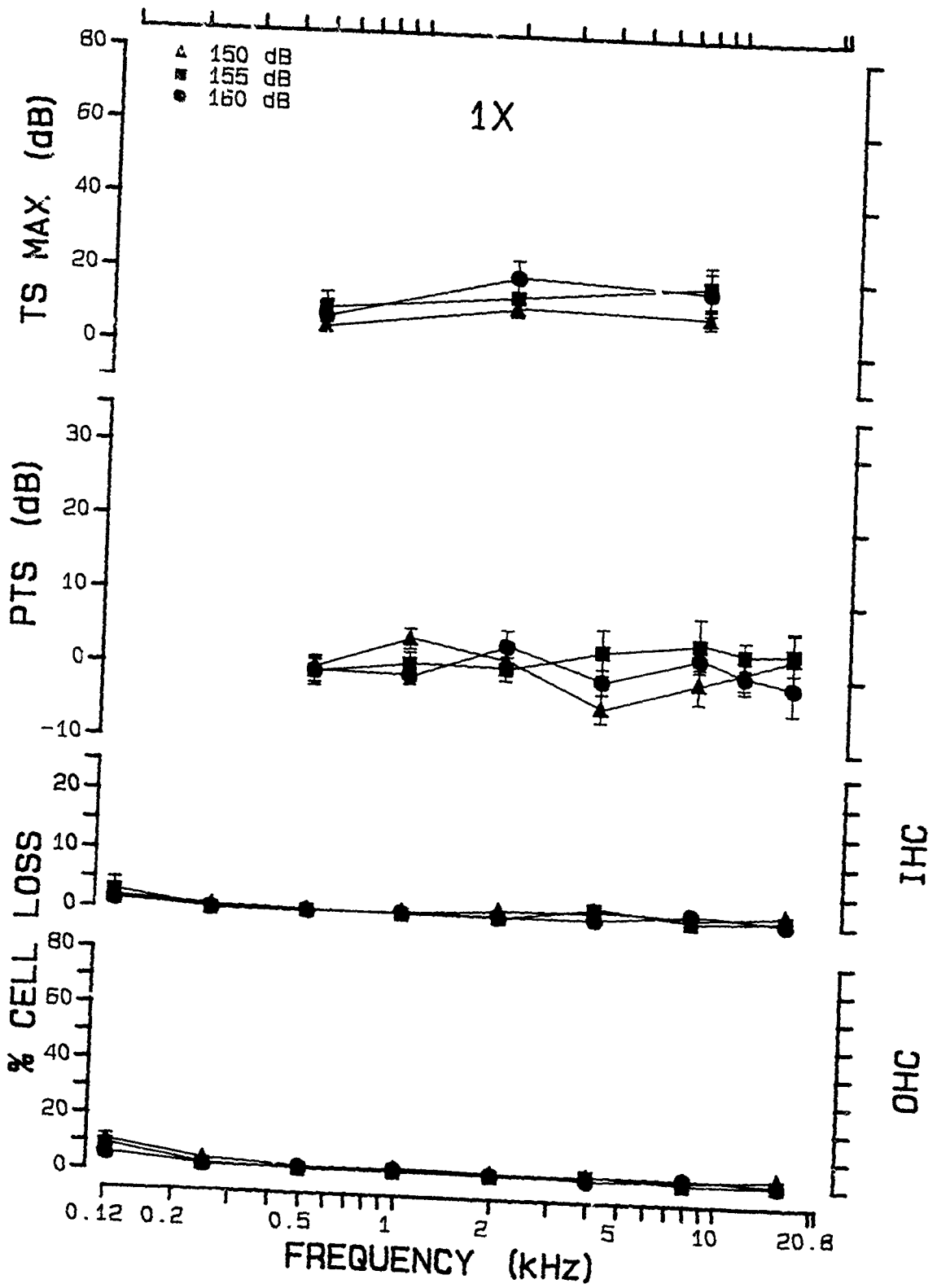

Figure 6. Mean TS_{max} (upper), PTS (middle) and sensory cell loss (lower) for groups of animals exposed to 1 blast wave

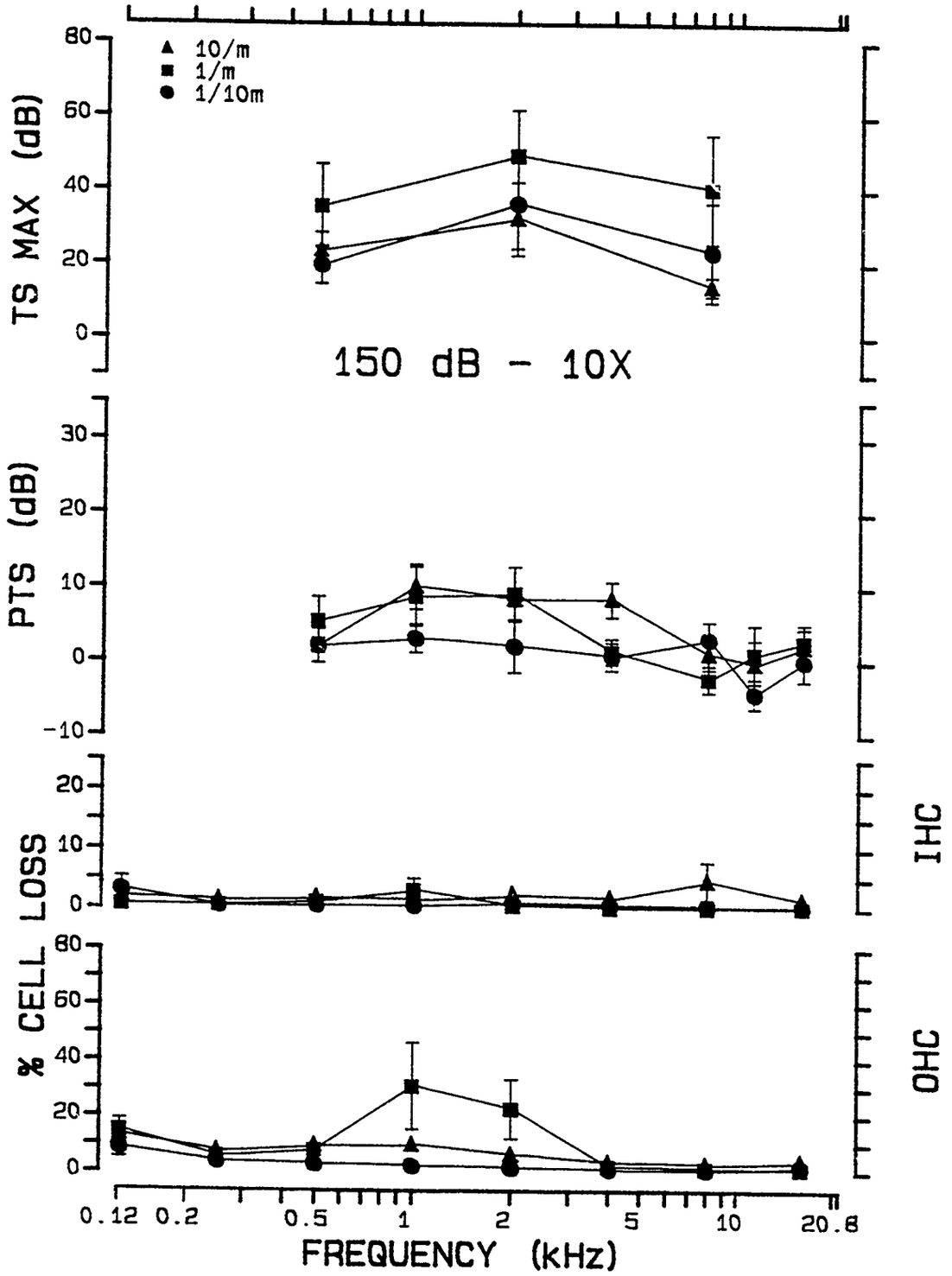

Figure 7. Mean TS_{max} (upper), PTS (middle) and sensory cell loss (lower) for groups of animals exposed to 10 blast waves at 150 dB peak SPL

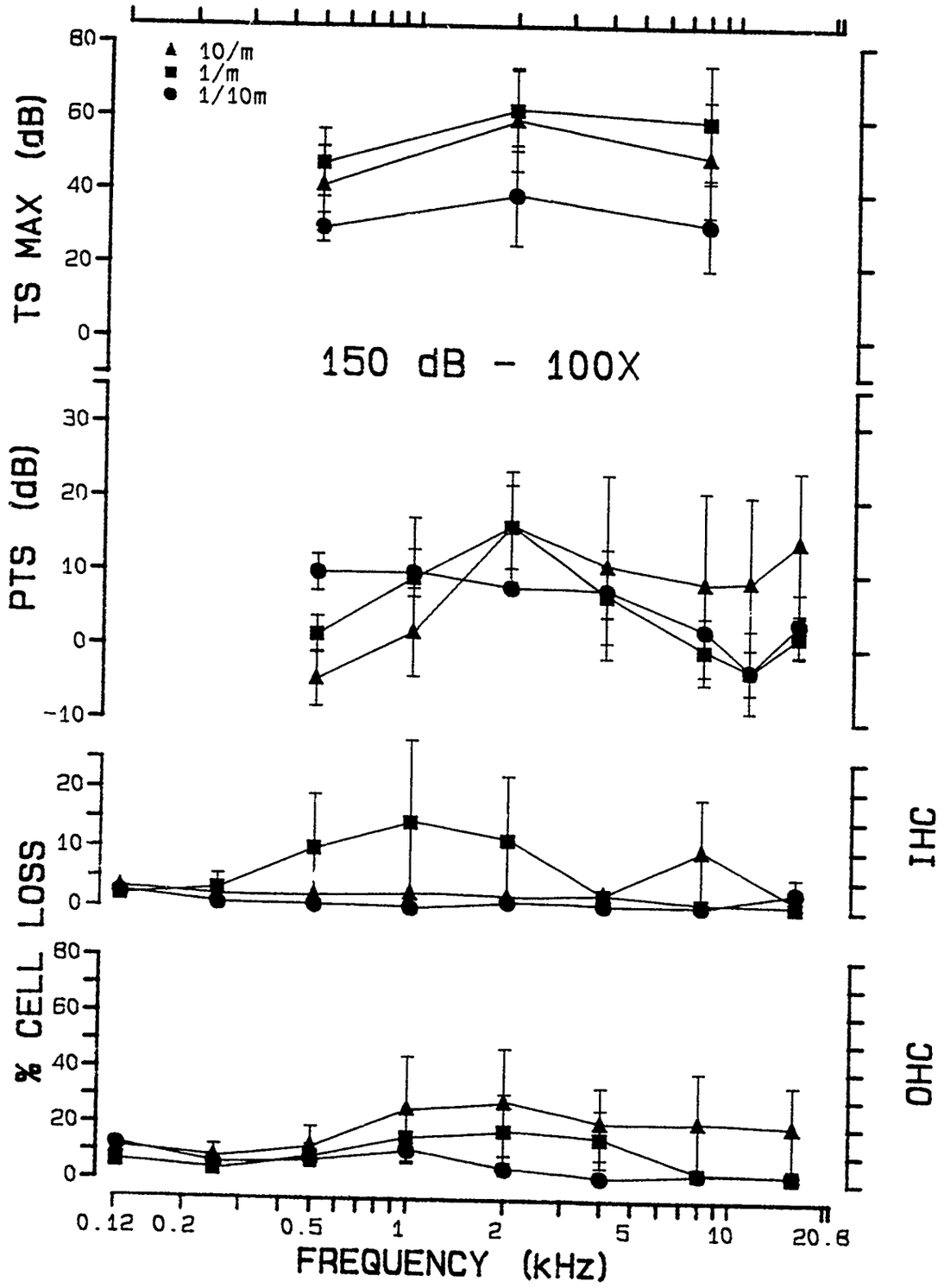

Figure 8. Mean TS_{max} (upper), PTS (middle) and sensory cell loss (lower) for groups of animals exposed to 100 blast waves at 150 dB peak SPL

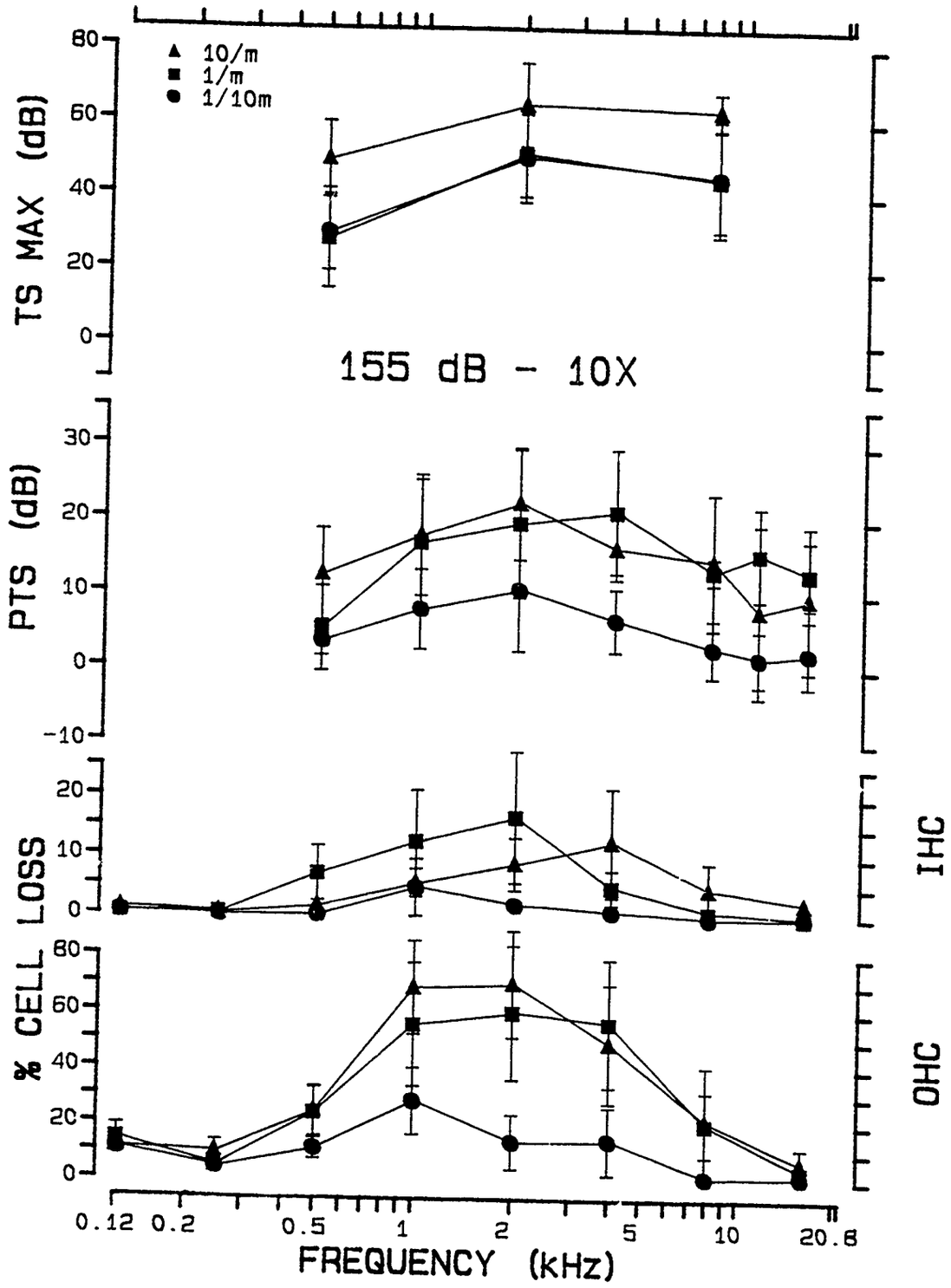

Figure 9. Mean TS_{max} (upper), PTS (middle) and sensory cell loss (lower) for groups of animals exposed to 10 blast waves at 155 dB peak SPL

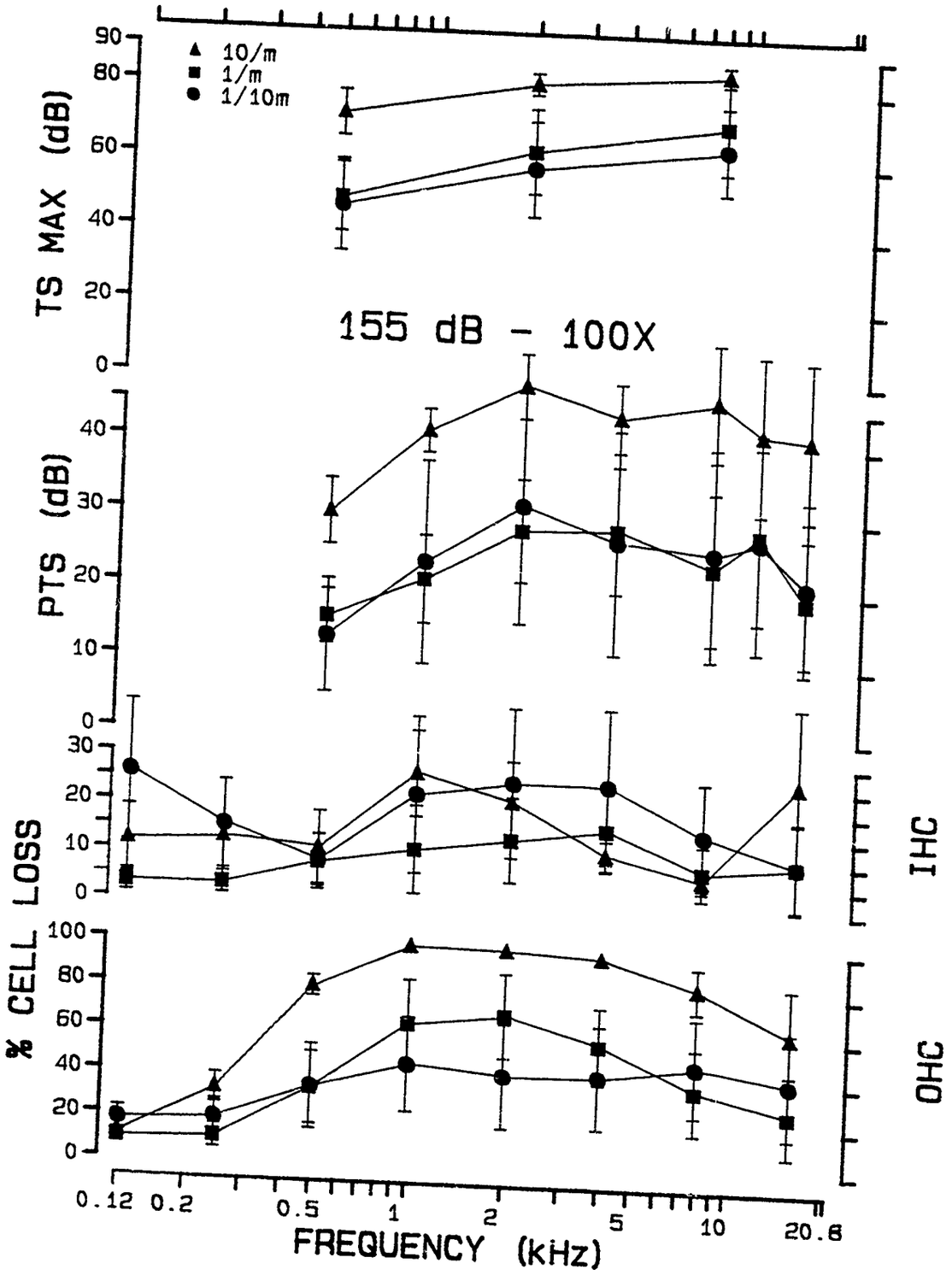

Figure 10. Mean TS_{max} (upper), PTS (middle) and sensory cell loss (lower) for groups of animals exposed to 100 blast waves at 155 dB peak SPL

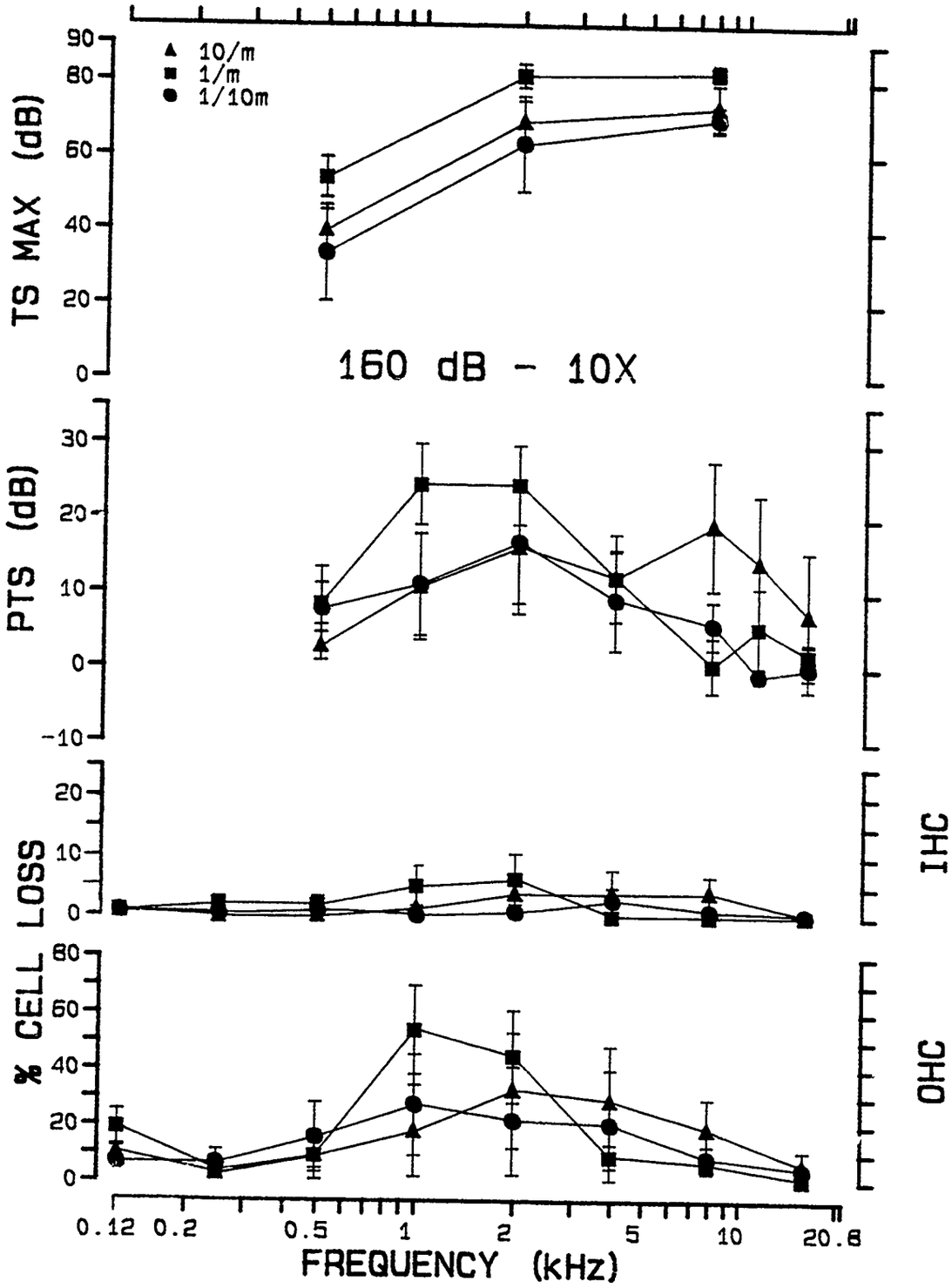

Figure 11. Mean TS_{max} (upper), PTS (middle) and sensory cell loss (lower) for groups of animals exposed to 10 blast waves at 160 dB peak SPL

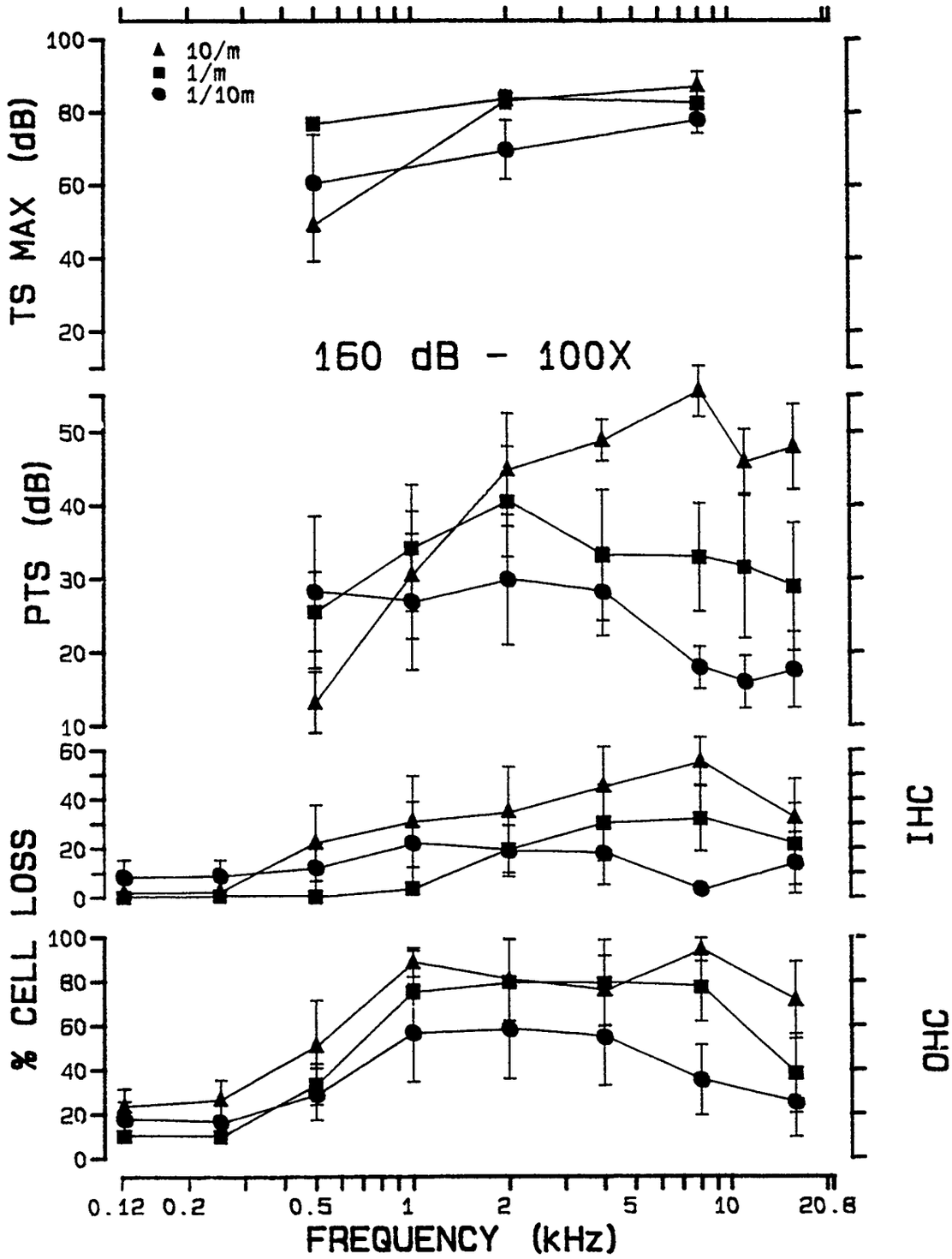

Figure 12. Mean TS_{max} (upper), PTS (middle) and sensory cell loss (lower) for groups of animals exposed to 100 blast waves at 160 dB peak SPL

In our earlier reports (ADA 206-180 and ADA 221-731), the data from Sources I and II (using impulses that had a lower frequency spectral distribution of energy) there was not a clear and consistent effect of impulse presentation rate upon the dependent variables. Tables XI and XII summarize the effects of impulse presentation rate on hearing trauma from the three sources. Table XI represents a subjective decision based upon the plotted postexposure data concerning which of the three rates caused the most hearing loss or cell loss. The term "mixed" indicates an exposure for which no clear determination of the most hazardous exposure could be made. Multiple contrasts may be performed on individual means to determine which groups showed the greatest losses at individual frequencies. However, visual inspection of Figures 6 through 12 also provides an indication of which groups are most severely damaged by the impulse noise exposures. The conclusions made from the visual inspection are unlikely to be appreciably different than those made using a large number of multiple contrasts.

Table XI

Summary evaluation of the exposure producing the greatest degree of trauma based upon the repetition rate of the impulse. Trauma is estimated on the basis of PTS or sensory cell loss.

	Source I	Source II	Source III
150 dB 10X	mixed	mixed	mixed
150 dB 100X	1/m	mixed	mixed
155 dB 10X	1/m	1/m	mixed
155 dB 100X	mixed	10/m	10/m
160 dB 10X	10/m	mixed	mixed
160 dB 100X	10/m	10/m	mixed

A more objective approach to determining which exposure rate causes the greatest trauma can be obtained by computing the mean PTS evaluated at 1, 2 and 4 kHz and comparing the means across the various groups exposed to different impulse rates. Table XII presents the summary of such an evaluation.

Table XII

Summary evaluation of the exposure producing the largest mean PTS evaluated at 1, 2 and 4 kHz, based upon repetition rate of the impulse

	Source I	Source II	Source III
150 dB 10X	1/m	1/10m	10/m
150 dB 100X	1/m	10/m	1/m
155 dB 10X	1/m	1/m	1/m
155 dB 100X	10/m	10/m	10/m
160 dB 10X	10/m	1/10m	1/m
160 dB 100X	10/m	10/m	10/m

The results shown in Table XI and XII differ for some exposure conditions because the sensory cell losses did not necessarily correlate perfectly with the PTS measure. Thus, based on the data from 12 groups of animals that were exposed to blast waves from Source III at different rates, we are still unable to make any conclusive statement regarding the systematic effects of rate on the hazard to hearing resulting from blast wave exposure, although the slowest rate (1 impulse every 10 minutes) of exposure from Source III seemed to cause the least damage. Examining all three sources together, however, gives the impression that the fastest rate (10 impulses per minute) is the most damaging, at least for the highest two energy conditions (155 and 160 dB peak SPL).

3. Number of Impulses: On the basis of the inconsistent effects of repetition rate on the dependent variables, we have collapsed the data across the rate variable to allow us to analyze the effects of number of impulses (since rate cannot be used as a variable for groups exposed to a single blast wave). By collapsing across rate, an analysis of variance can be performed using impulse peak pressure, number of impulses and frequency as the three main factors. In this analysis (see Table X), the main effect of number of impulses was statistically significant for both audiometric and both histological variables. Examining the figures it is clear that the single impulses caused the least amount of hearing and hair cell losses while the 100 impulses caused the greatest losses. The interaction of number of impulses and peak level was statistically significant for all but the TS_{max} dependent variable. Thus the effect of peak or number was dependent upon the level of the other variable. In this case, there appears to be no effect of impulse peak for the groups exposed to a single impulse, but an appreciable effect of peak between groups exposed to 10 or 100 impulses.

4. Impulse Peak Pressure: The main effect of impulse peak pressure was a factor in each of the four analyses reported above. The results of these analyses were consistent in that, in general, with the same number of impulses, the 150 dB impulses were less hazardous than the 160 dB impulses, with the 155 dB impulses causing a somewhat intermediate effect on the dependent variables. In the analysis which included impulse peak and number (Table X), the main effect of peak pressure was statistically significant for all but the TS_{max} variable.

The analyses of groups exposed to the same number of impulses show a similar effect of peak. In the groups exposed to a single impulse, the analysis show no statistically significant effect of peak. The main effect of peak pressure was statistically significant for seven of the other eight analyses summarized in Tables VIII and IX. The percent inner hair cell losses did not show a statistically significant effect for groups exposed to 10 impulses. The effect of impulse peak pressure was statistically significant for all other dependent variables (i.e., TS_{max}, PTS, %OHC) and groups (i.e., 100X). Thus, as one would expect, the higher peak pressure impulses caused more damage than did the lower peak pressure exposures.

An alternate presentation of these data is shown in Figure 13 where the mean PTS evaluated at 1, 2 and 4 kHz ($PTS_{1,2,4}$) (a) and the percent total inner (b) and outer hair cell loss (c) for each animal are shown plotted as a function of the total A-weighted sound exposure level. The sound exposure level is defined as:

$$10 \log_{10} \left[\frac{\int p^2(t)\,dt}{p_{ref}^2 \, \Delta t_{ref}} \right] \qquad \text{where } p_{ref} = 20\mu Pa \text{ and } \Delta t_{ref} = 1s.$$

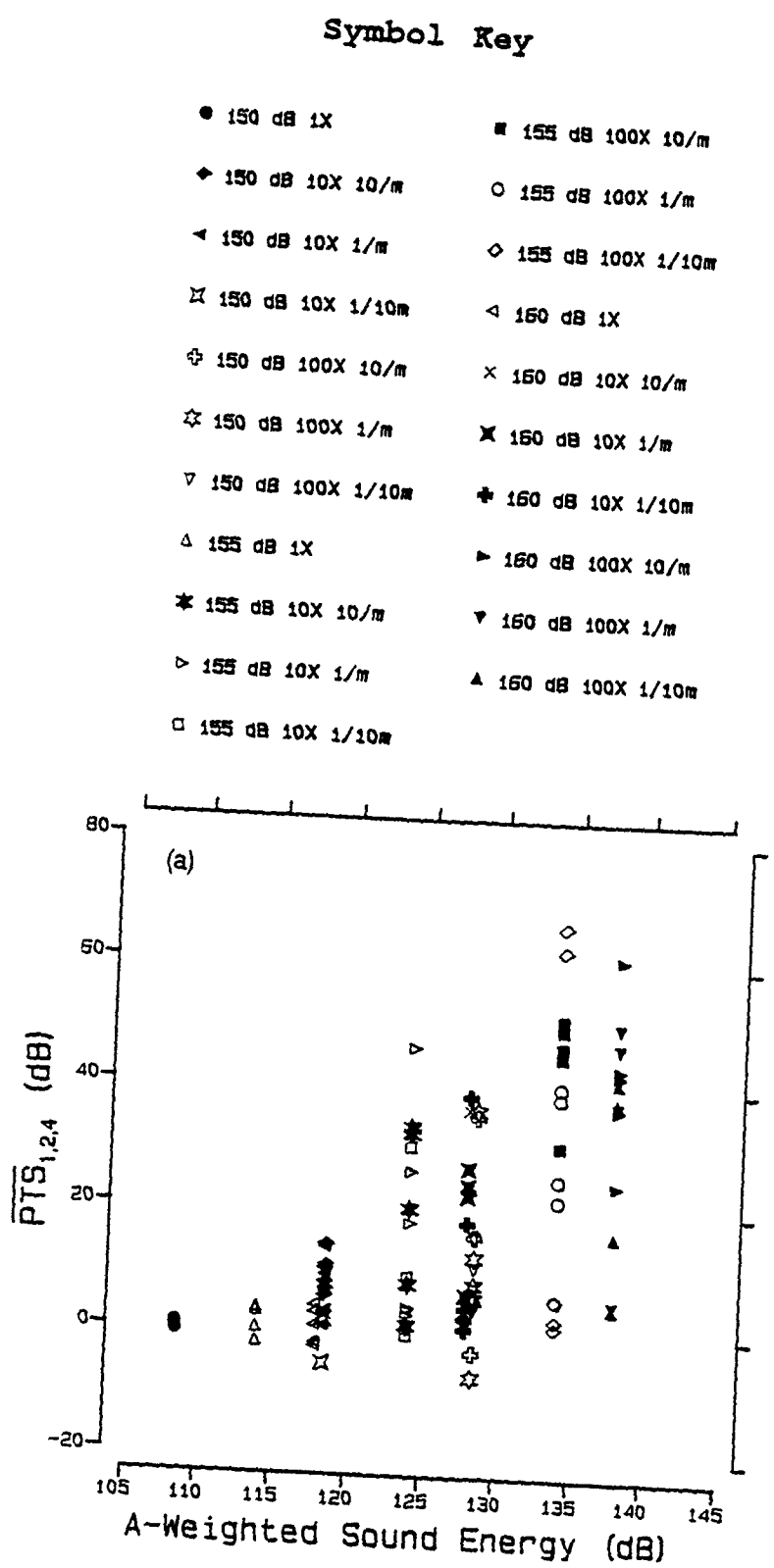

Figure 13. The distribution of mean $PTS_{1,2,4}$ and percent cell losses from individual animals (n=105) for all exposure conditions

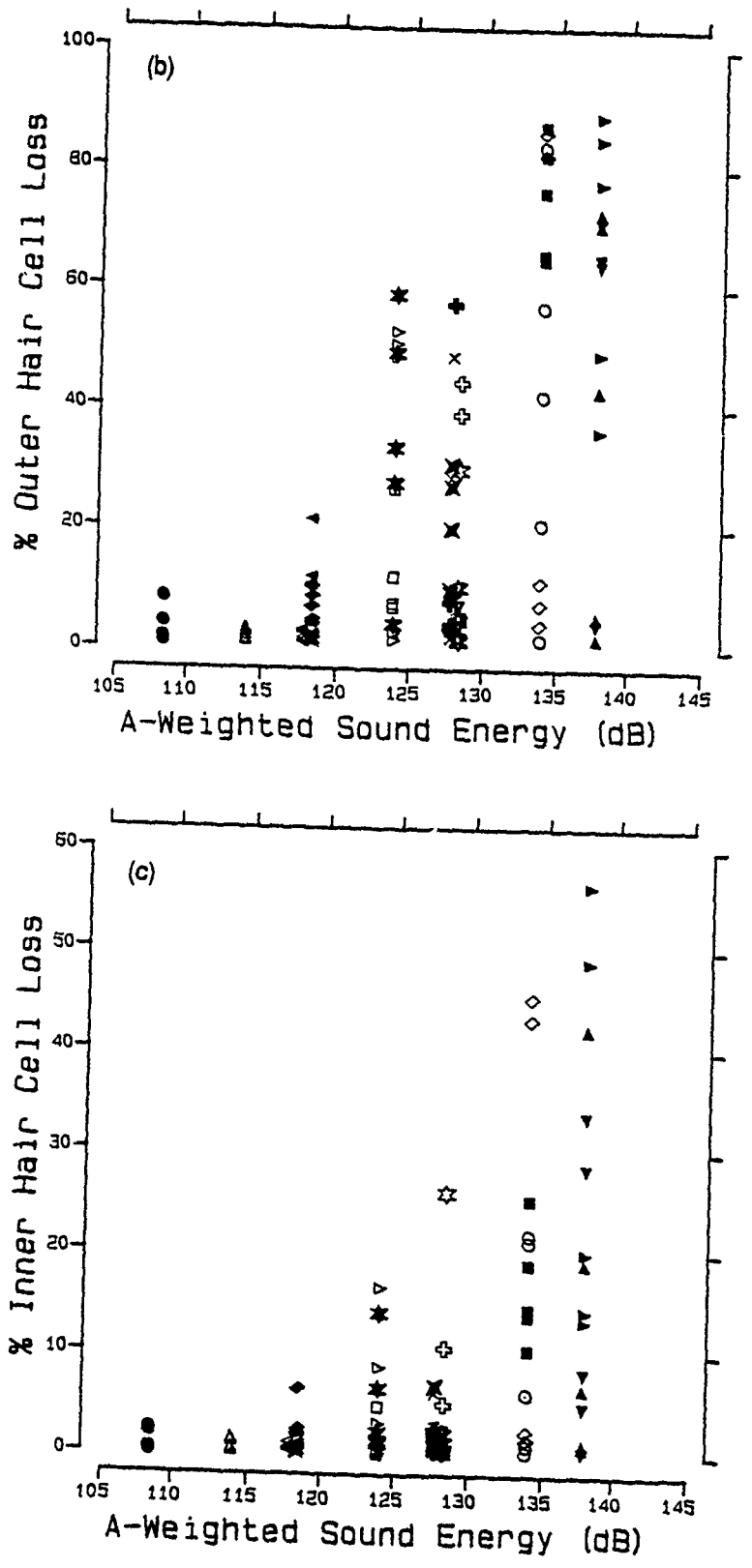

Figure 13. The distribution of mean $PTS_{1,2,4}$ and percent cell losses from individual animals (n=105) for all exposure conditions

The density of the data points makes it difficult to distinguish between the individual animals of each exposure group. Nevertheless, the presentation of data in Figure 13 clearly shows the increase in variability that occurs from exposure levels above about 120 dB. Consider, for example, the animals exposed to the more severe conditions (i.e., to A-weighted sound exposure levels above 130 dB). A number of animals show no PTS or sensory cell loss while others are severely traumatized. This degree of variability is a common observation following blast wave exposures and points out the need for an alternate approach to the data analysis. The addendum of this report illustrates one such alternate analysis that attempts a synthesis of the data from the previous two reports, portions of the data from the present report, and data obtained at the USAARL laboratories using a conventional electro-acoustic sound system.

C. CONCLUSIONS

The following preliminary conclusions can be made from the present data. (1) There was very little or no hearing loss or sensory cell loss for exposure to a single impulse at 150, 155 or 160 dB peak SPL. The variability among animals in these three groups was also relatively small. (2) There is a considerable increase in the variability or degree of susceptibility to trauma across animals as the severity of the exposure increases. The variability produces, in some extreme cases, a complete dichotomy in the results (i.e., within an experimental group of five animals, half the animals can show little or no effect of the exposure, while the remaining animals can be severely traumatized). Such a dichotomy makes it difficult to describe the data with conventional statistics. The only alternative seems to be to substantially increase the total number of animals in such exposure conditions. (3) With the above in mind a general, though not surprising trend in the data, is that as the peak levels and total energy increase permanent effects increase. These permanent effects seem to be dependent upon peak levels more than upon the total energy in the exposure stimulus (compare, for example, groups 6, 7 and 8 with groups 16, 17 and 18).

After completing exposures on 329 experimental subjects using three different sources, there appear to be several consistent trends emerging when the data are viewed in total. The first trend is that increases in variability correlated with increases in sound exposure level are consistent across the three sources. We have been developing alternative analytical procedures that show promise in their ability to adequately describe the results from the three sources and which may be used to predict the hazard posed by a variety of impulsive noise exposures (see Section IV). The second observation is that we have not seen an obvious difference in frequency (or location) of maximum losses as the frequency of the peak of the A-weighted spectrum increased from 0.25 kHz (Source I) to 2.0 kHz (Source III). Rather, at high sound exposure levels, each source appears to cause a broad hearing loss across the middle frequency region (i.e., 1 to 4 kHz). However, Source III, with an A-weighted spectral peak in the 2.0 kHz octave band, did cause some appreciable losses in the higher frequency regions. Finally, while the issue of blast wave presentation rate is still ambiguous, it appears, from examination of the results from all three sources (Tables XI and XII) that the faster repetition rates may be particularly hazardous, especially at high sound exposure levels.

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IV. ADDENDUM

An experimental basis for the estimation of auditory system hazard following exposure to impulse noise

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INTRODUCTION:

There are a number of different suggested standards for exposure to impulse/impact noise, e.g. Coles, et al. (1968), OSHA, (1974), Smoorenburg (1982), and Pfander (1980). Although each of these criteria has its proponents, none of them are in complete agreement with existing data (Smoorenburg, 1987). What is needed is a new criterion. Unfortunately, there is an extremely limited empirical data base upon which a new standard can be built. The difficulties associated with generating such a data base are compounded by the extremely broad range of high intensity noise transients that exist in various industrial and military environments. For example, in industry impacts with variable peak intensities and a reverberant character often occur. At the other extreme, the diversity of military weapon systems produce impulses which originate as the result of a process of shock wave formation and propagation following an explosive release of energy. These waves, which can have peak levels in excess of 180 dB, can be either reverberant or non-reverberant in nature depending upon the environment in which they are encountered. Trying to develop a single standard to cover this broad range of "acoustic" signals is a formidable task.

One of the features of the existing or proposed exposure criteria is the general lack of specific consideration that is given to the frequency domain representation of the impulse, a point frequently raised by Price (1979) and others. Some deference is, however, given to the spectrum in these criteria, but in a covert or indirect manner; e.g., through the use of A-weighting of the stimulus or through the handling of the A and B duration variables. A more direct spectral approach to the evaluation of impulses and impacts was proposed by Kryter (1970). His suggestions, while based upon sound reasoning, never really caught on. The Kryter approach appeared attractive in its ability to predict the amount of temporary threshold shift measured two minutes after exposure (TTS_2) to a noise transient provided that the TTS_2 was not very large or alternatively that the levels of the transient in any given frequency band were not excessive. Price (1979, 1983, 1986) to some extent has tried to build upon and extend the Kryter approach by considering the spectral transmission characteristics of the peripheral auditory system. Price's reasoning led to the following conclusions: (1) There is a species specific frequency, f_o, at which the cochlea is most vulnerable and that impulses whose spectrum peaks at f_o will be most damaging. This would appear to be true, according to Price, regardless of the distribution of energy above and below f_o. For man the suggested frequency is 3.0 kHz. (2) Relative to the threshold for damage at f_o, the threshold for damage should rise at 6 dB/octave for $f_p<f_o$ and at 18 dB/octave for $f_p>f_o$ where f_p = spectral peak of the impulse. Thus, a model for permanent damage was developed which is amenable to experimental testing. In subsequent studies Price (1983, 1986) has tried to relate, with varying degrees of success, experimental data obtained from the cat to the predictions of this model. More recently Hamernik et al. (1990) and Patterson et al. (1990) have reported on an extensive series of parametric studies in which the spectra of the impulses were varied. A review of the literature indicates that, except for the studies mentioned above, there are few other published results obtained from experiments specifically designed to study the effects of the spectrum of an impulse on hearing trauma.

This paper will present an analysis of the Patterson et al. (1990) data from which a spectral weighting function is derived. This weighting function will then be applied to the blast wave data of Hamernik et al. (1990) and to the synthetic impulses from Patterson et al. (1986) in order to develop a relation between the permanent threshold shift (PTS) and the sound exposure

level. Our intention is not to present a set of conclusive results, but rather to illustrate an approach to the analysis of this type of experimental data that appears to be somewhat different from that which has been attempted in the past. It is an approach which develops a direct relation between frequency specific measures of PTS and the frequency domain representation of the impulse. The results of this approach can be directly related to the Price (1983) model and can also be used to estimate the permanent effects of a traumatic impulse noise exposure in a manner similar to that proposed by Kryter (1970) for estimating TTS after an impulse noise exposure.

METHODS:

The noise-induced permanent threshold shift (NIPTS) data presented in this report were acquired from 475 chinchillas that were exposed to high levels of impulse noise. Audiometric data on each animal was obtained using either a shock avoidance procedure (Patterson et al., 1986) or measures of the auditory evoked potential (Henderson et al., 1983). Permanent threshold shifts were computed from the mean of three preexposure audiograms and at least three 30 day post exposure audiograms. The behaviorally trained animals were tested at octave intervals from .125 kHz through 8 kHz including the half octave points 1.4, 2.8 and 5.7 kHz. Evoked potential thresholds were measured at octave intervals from 0.5 to 16 kHz and at the 11.2 kHz point. For each animal measures of compound threshold shift, permanent threshold shift (PTS) and quantitative histology (cochleograms) were obtained. In the analysis that follows, only PTS data will be discussed.

Series I exposures: (N = 118) Animals were exposed at a normal incidence (i.e., the plane of the external canal is parallel to the speaker exit plane) to 100 impulses presented at the rate of 1/3s. This series of exposures consisted of 20 groups of animals with 5 to 7 animals/group. The stimuli were narrow band impulses produced by passing a digital impulse through a digital bandpass filter of the 4 pole Learner type (Gold and Rader, 1969). Following analog conversion, the signal was transduced through an Altex 515 B speaker in a model 815 enclosure. The filter bandwidth was independent of center frequency with steep attenuation outside the passband permitting the synthesis of equal energy impulses at a variety of center frequencies while assuring minimal spread of energy to other frequencies. The center frequencies of the six sets of impulses varied from 260 Hz through 3350 Hz. The bandwidth of the impulses was approximately 400 Hz. Impulse peaks were varied from 124 dB to 146 dB. For each of the exposure conditions listed in Table I the total sound exposure level, (SEL) was computed (Young, 1970) where

$$SEL = 10 \log_{10} \int_{-\infty}^{\infty} \frac{p^2(t)\,dt}{p_r^2\, t_r} \; ; \qquad t_r = 1s,\ p_r = 20\ \mu Pa.$$

Figure 1 illustrates an example of the pressure-time histories of the 775 Hz and 1350 Hz center frequency impulses along with their respective spectra.

Series II exposures: (N = 42) Animals were exposed at a normal incidence to 100 impulses presented at the rate of 1/3s. There were seven different exposure conditions (Table II) to which seven groups of animals were exposed. Each group contained six animals. Two types (low peak, high peak) of relatively broad band impulses with identically shaped amplitude spectra were digitally synthesized (Patterson et al., 1986). The peak SPL of the impulses was varied from 127 dB to 147 dB. Hearing threshold data was obtained using

the avoidance conditioning procedure. Figure 2 illustrates the pressure-time histories of typical high and low peak impulses along with their common spectrum.

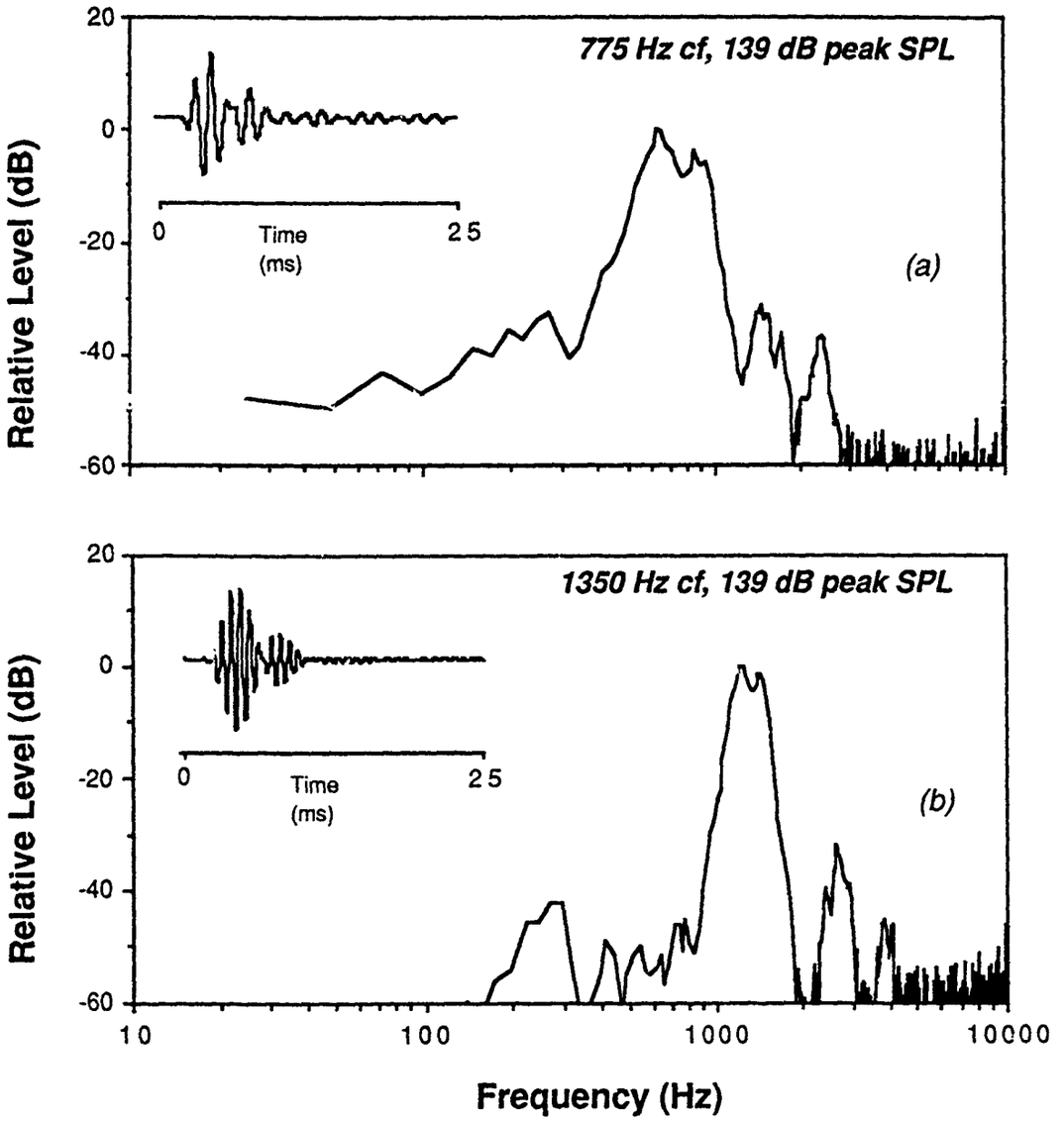

Figure 1. Examples of the 775 Hz and 1350 Hz center frequency impulses of the Series I exposures along with their respective spectra.

Series III exposures: (N = 315) Animals were exposed at a normal incidence to either 1, 10, or 100 impulses, presented at the rate of 1/s, 10/s or 1/10s at intensities of 150, 155, or 160 dB peak SPL. All of the above combinations of number, repetition rate, and peak yielded 21 different exposure groups with 5 animals/group. The impulses were generated by a compressed air driven shock tube. The above set of 21 exposures was repeated using waves generated by

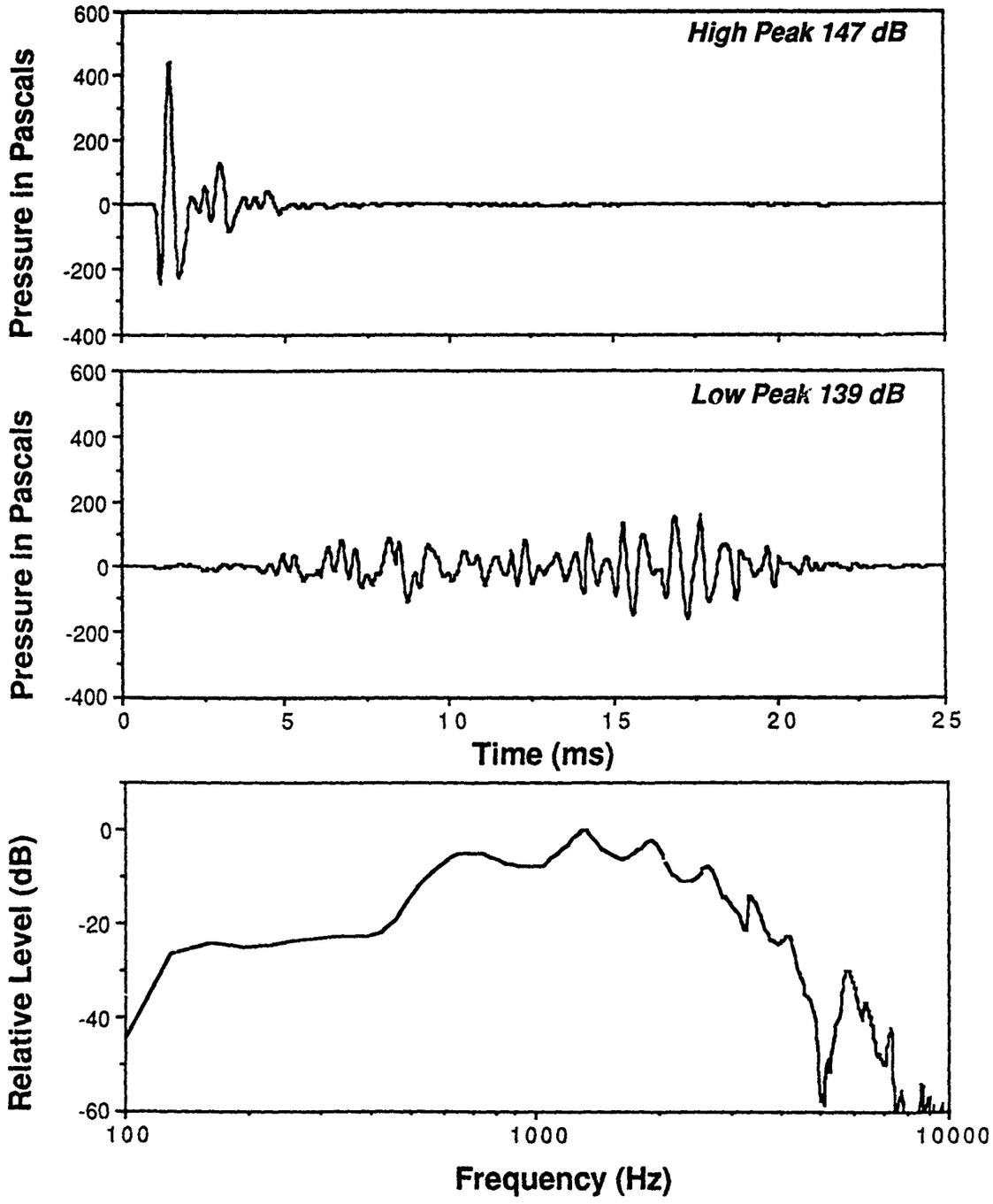

Figure 2. Examples of the Series II impulses and their common spectrum. (a) The high-peaked 147 dB peak SPL impulse; (b) The low-peaked 139 dB impulse; (c) The spectrum of each of the above, approximately equal energy, impulses.

three different diameter shock tubes which produced blastwaves whose spectrum peaked at three different locations of the audible spectrum. The pressure-time traces and spectral analysis of these waveforms are shown in Figure 3. In addition, the A-weighted octave band energies are shown in Figure 4 so that comparisons could be made for each wave from each source. Because of the high levels of very low frequency energy in these blast waves the resolution at the high frequencies is poor if unweighted energies are plotted. For further details see Hamernik and Hsueh (1990). Table III summarizes the conditions for the Series III exposures. Only the SELs for the 100X conditions are tabulated. Successive 10 dB adjustments need to be made to obtain the 10X and the 1X SEL values. All animals in this series were tested using the auditory evoked potential procedures.

RESULTS:

The results of each series of exposures will be presented separately, and the methods used to analyze the NIPTS data from each series will be explained.

Series I exposures: For each of the 20 groups of animals that were exposed to the narrow band impulses a mean permanent threshold shift evaluated at 1, 2, and 4 kHz ($\overline{PTS}_{1,2,4}$) was computed and the groups were compared on the basis of sound exposure level. This data set is shown in Figure 5. The group mean PTS from each set of the two to four groups of animals that comprise an intensity series for a specific CF impulse behaves in an orderly manner with $\overline{PTS}_{1,2,4}$ increasing in an approximately linear fashion with increasing SEL. The relative susceptibility to NIPTS is also seen to be a function of the impulse center frequency, with the lower frequency impulses producing relatively little NIPTS even at the higher SEL's. A relative frequency weighting function can be derived from the data presented in Figure 5 by shifting each frequency specific data set along the SEL axis the amount that is necessary to collapse the data into a single PTS/SEL function using one of the exposures as a "zero" reference. Such a data shifting process was carried out "by eye" to produce a best fit using the 1350 Hz series of data as the reference point. The amounts shifted were: 260 Hz CF impulses, -20 dB; 775 Hz CF impulses, -7.2 dB; 1025 Hz CF impulses, -4 dB; 1350 Hz CF impulses, 0 dB; 2450 Hz CF impulses, -4 dB; 3550 Hz CF impulses, +4 dB. The realignment of the data that such a shift produces is shown in Figure 6, and the weighting function, thus obtained, is shown plotted (solid line with symbols) in Figure 7 where it is compared to the conventional A-weighting function (solid line). The new empirical weighting function is referred to as P-weighting in the text that follows. A linear regression through the shifted data set showed a correlation coefficient of 0.89 with a slope of 2.6 dB PTS/dB P-weighted sound exposure level and a threshold for the onset of $\overline{PTS}_{1,2,4}$ of 116 dB P-SEL. The empirical function derived from the narrow band impulse data is seen to differ from the A-weighting function by as much as 10 dB at the low frequencies. Also, evident in this figure is the anomalous behavior of the data point produced by the exposures to the 2450 Hz, CF impulses.

Series II exposures: The detailed histological and audiometric results of this series of exposures have been published in Patterson, et al. (1985, 1986). The $\overline{PTS}_{1,2,4}$ data from this series of seven exposures is shown plotted as a function of the SEL and the P-weighted SEL in Figure 8. The latter was obtained by applying the empirical weighting function (Figure 7) to consecutive octave bands of the spectrum of the Series II exposures. Also included in this figure are the shifted (or P-weighted) data points from the Series I exposures. It is evident that the P-weighting function does not have the desired effect of increasing the degree of congruence between the Series I and II exposures. Since the Series II exposures had substantial energy in the

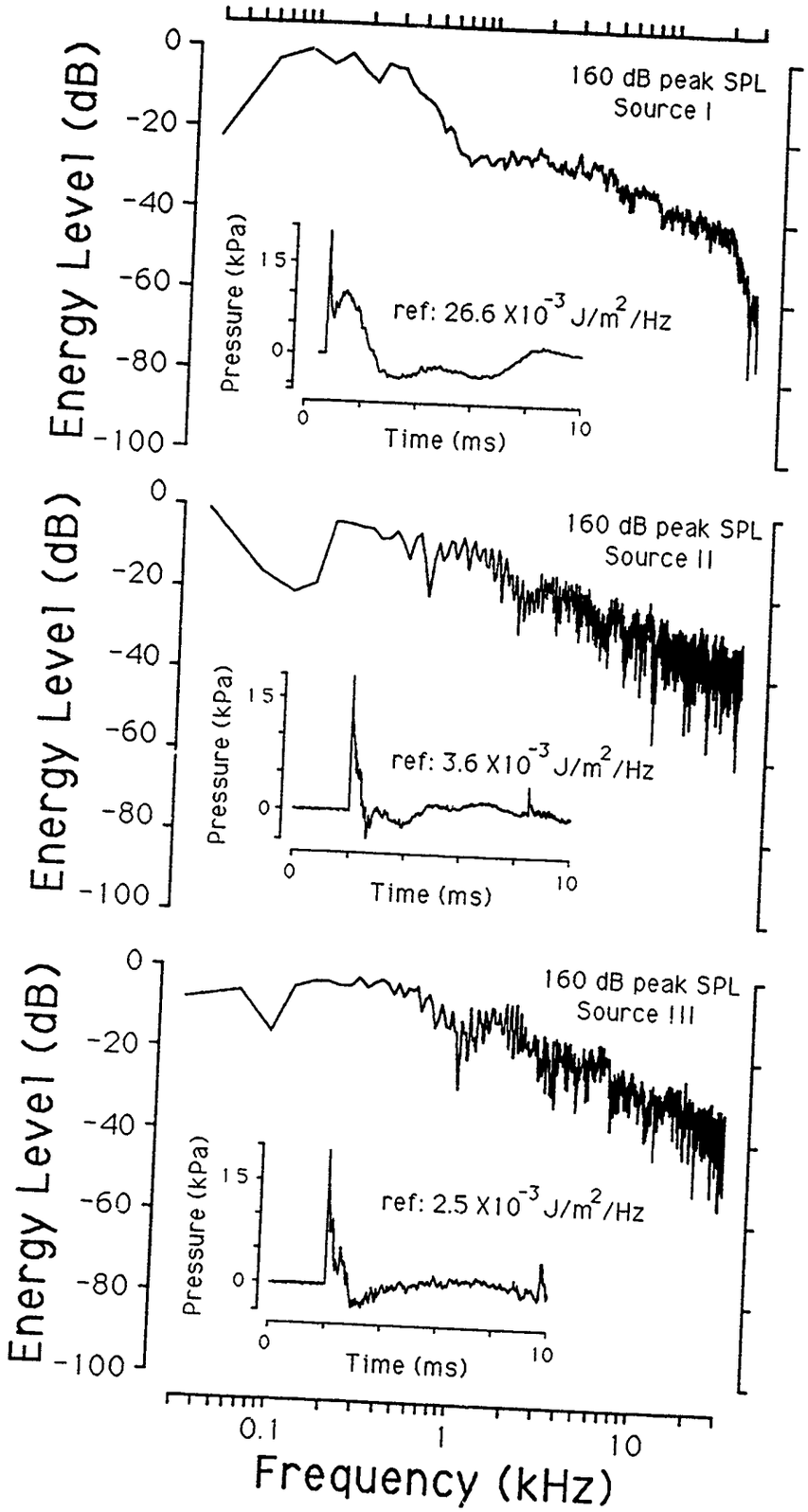

Figure 3. Examples of the 160 dB peak SPL impulses produced by the three different shock tubes and their respective spectra. These waves are typical of those used for the Series III exposures.

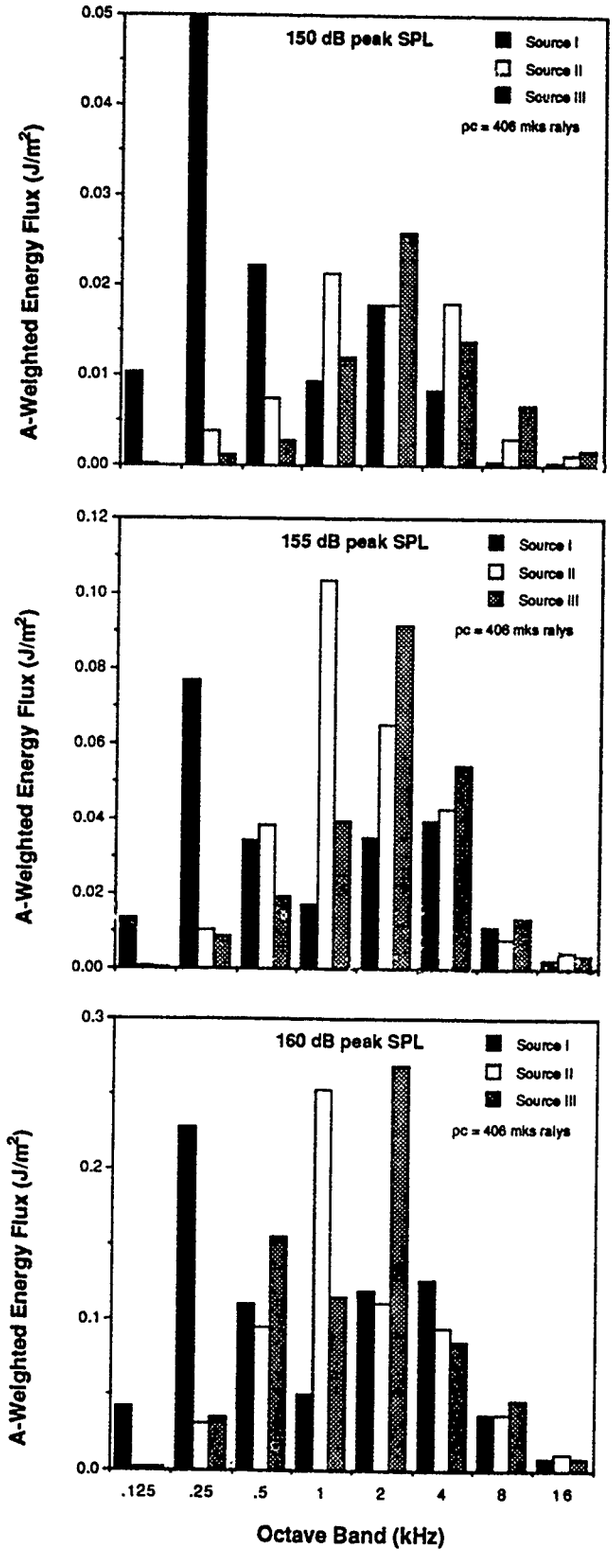

Figure 4. A-weighted octave band spectra of each of the waves that were used for the Series III exposures.

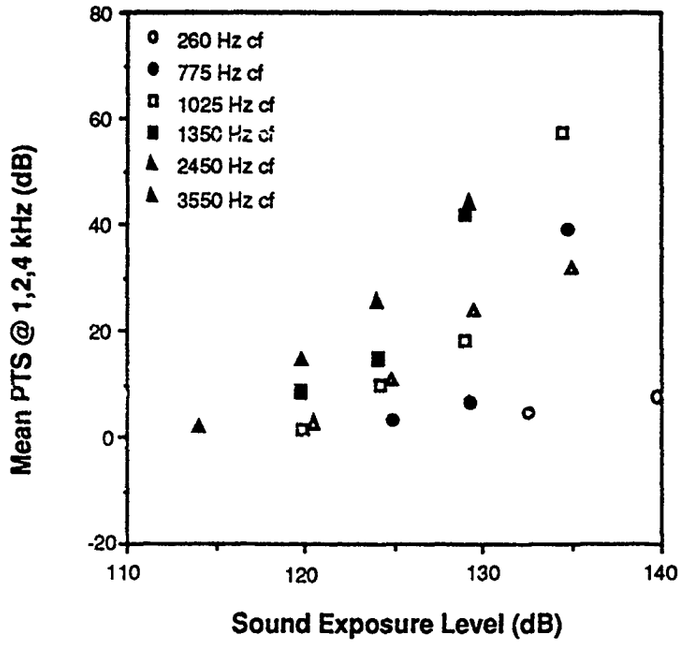

Figure 5. The group mean PTS evaluated at 1, 2, and 4 kHz $\overline{PTS_{1,2,4}}$ as a function of the total sound exposure level for the six groups exposed to the Series I narrow band impulses.

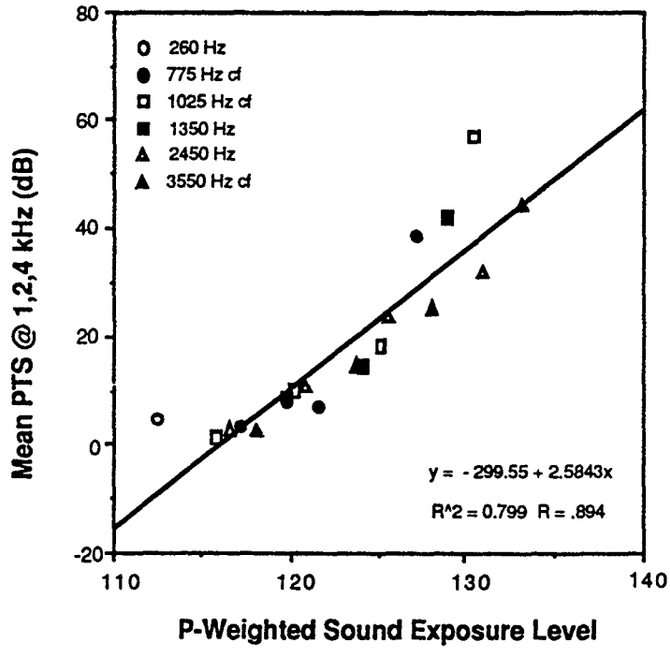

Figure 6. The $\overline{PTS_{1,2,4}}$ as a function of the empirically derived P-weighted sound exposure level for all the Series I exposures. The regression line has a slope of 2.6 and an x-intercept of 116 dB.

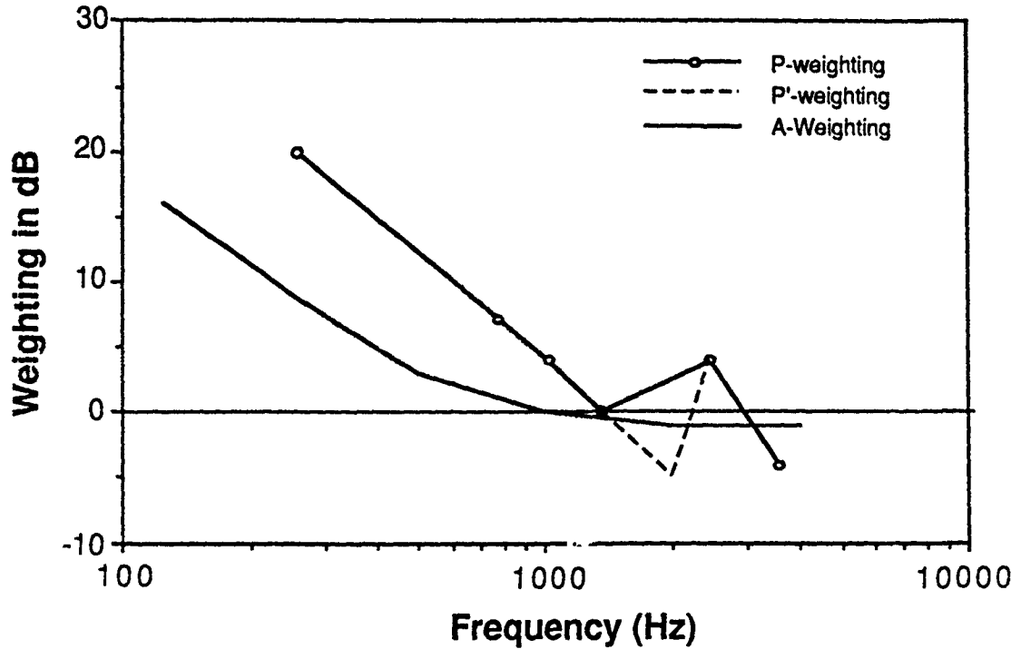

Figure 7. The empirical P-weighting function derived from the Series I exposures along with the conventional A-weighting function and the P'-weighting function inferred from the Series II and III experiments.

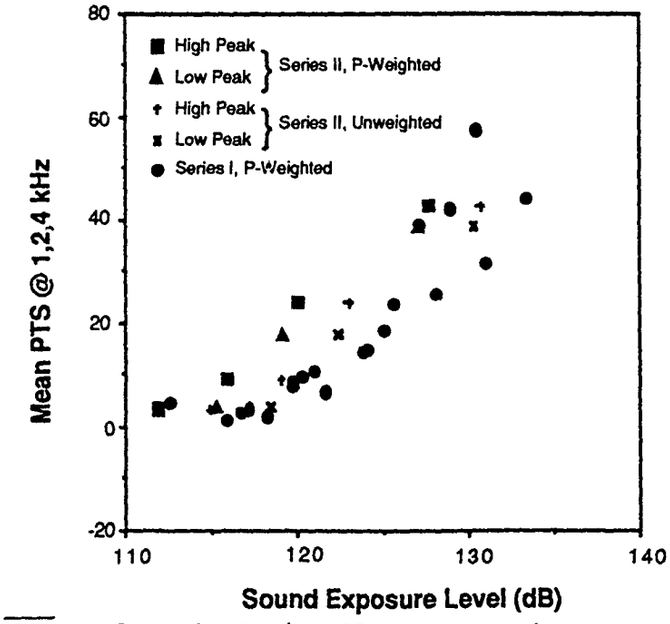

Figure 8. The $\overline{PTS_{1,2,4}}$ from the Series II exposures shown as a function of unweighted and P-weighted sound exposure level compared to the $\overline{PTS_{1,2,4}}$ versus P-weighted sound exposure level of the Series I exposures.

2 kHz region of the spectrum, it was apparent that the effect of applying the empirical weighting function to this region of the spectrum would shift the Series II data points in the wrong direction. However, if the empirical P-weighting function is extrapolated as shown by the dotted portion of the function shown in Figure 7, and then used to weight the Series II impulses, the agreement between the Series I and II data becomes quite good as seen in Figure 9. A linear regression analysis (solid line) of the entire data set from the Series I and II exposures shows a correlation coefficient of 0.91 a slope of 2.5 and an x-intercept of 116 dB. This modified weighting function is referred to as P'-weighting.

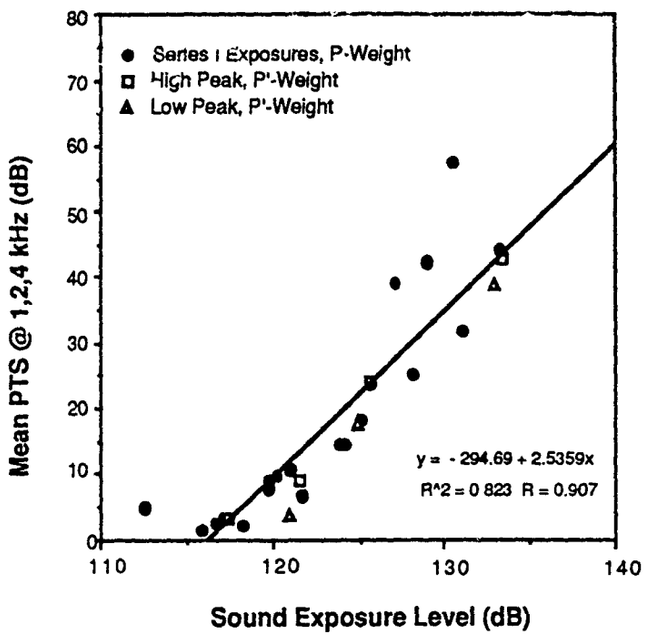

Figure 9. $\overline{PTS}_{1,2,4}$ obtained from the Series I and Series II exposures as a function of P and P'-weighted sound exposure level respectively. The linear regression line was computed using all the points shown plotted in this figure. (x-intercept = 116 dB; slope = 2.5)

Series III exposures: One of the problems that seems to characterize the measurement of PTS following exposure to these very high peak levels of impulse noise is that of extreme intersubject variability. A number of authors have commented on this problem in the past, e.g., Kryter and Garinther, (1965), Henderson and Hamernik, (1982). Price (1983, 1986) also experienced a severe intersubject variability when measuring threshold shifts in cats that had been exposed to blast waves that were similar to some of the impulses in the Series III exposures. Another problem is the excessive time necessary to run an experimental animal through a complete experimental paradigm of audiometric and histological protocols, thereby effectively limiting the number of animals in each experimental group and hence the statistical power. On the basis of a preliminary analysis of the PTS data (using analysis of variance) it was apparent that the effects on PTS of the different impact presentation rates was, at best, a marginal statistical effect. Thus, a decision was made to evaluate all the PTS data without regard for presentation rate. Also, since relations between PTS and the increasing energy of the stimulus were being sought, presentation rate did not affect the

independent variable. This effectively increased the number of animals at each sound exposure level to 15 except for the 1X exposure conditions. Total sound exposure or exposure level is increased by increasing the peak SPL or the number of impulse presentations.

For each audiometric test frequency a plot of the individual animal PTS at that frequency as a function of the total unweighted sound exposure level in the octave band centered on that test frequency was prepared for each animal. Two typical examples of this analysis at 2 kHz and 4 kHz for Source II are shown in Figure 10. For impact Sources I, II, and III, 105 individual data points for each source at each audiometric test frequency were plotted over approximately a 30 dB range of sound exposure level. The actual number of data points in each panel of Figure 10 is less than 105 since a number of animals had the same data coordinate. Using data sets such as those shown in Figure 10 the 90th percentile hearing loss (PTS_{90}) was computed for each sound exposure level at each octave frequency from 0.5 to 16 kHz. The PTS_{90} at any frequency was computed from:

$$PTS_{90} = \bar{x} + st_{.10}$$

where, $\bar{x}$ is the group mean PTS; $t_{.10}$ is the value of t below which 90% of the PTS data lies; s is the group standard deviation. This procedure yields 9 percentile points for each test frequency shown by the filled symbols in Figure 10, i.e., three peak levels for each of three numbers of impacts. This exercise was repeated for each of the six octave test frequencies and for each of the three sources.

From this set of frequency specific 90th percentile points a 90th percentile $\overline{PTS}_{1,2,4}$ was computed for each exposure group and plotted as a function of the P'-weighted sound exposure levels. These results are shown plotted in Figure 11. The P'-weighting has the effect of collapsing all the shock tube data into a reasonably cohesive pattern for which a linear regression produces a relation between $\overline{PTS}_{1,2,4}$ and P'-SEL whose correlation coefficient is 0.91. A threshold for the onset of $\overline{PTS}_{1,2,4}$ of 113 dB SEL and a slope of approximately 2 dB $\overline{PTS}_{1,2,4}$ for each dB P'-weighted SEL describes the equation of this regression line.

Figure 12 shows the entire data set from the Series I, II and III exposures plotted as a function of the P'weighted SEL. As a first approximation the P'weighting function has the desired effect of unifying the PTS/SEL relation following a very diverse series of impulse noise exposures. The correlation coefficient between the PTS and weighted SEL variables is approximately 0.9.

DISCUSSION:

In this paper we have presented a preliminary analysis of a large experimental data base obtained from 475 chinchillas that were exposed to a variety of impulse/blast wave noise transients. This analysis, while encouraging in its ability to unify the PTS data, is considered preliminary because only a portion of the data that will eventually be available have been analyzed. In addition to the results presented, the following data sets will ultimately be entered into the data base for a final analysis: (1) Non-reverberant, high frequency, Series III type impulses (N = 105); (2) A more detailed exploration of the 1-8 kHz region of the empirical weighting function using the Series I narrow band impulses (N = 50); (3) Highly reverberant

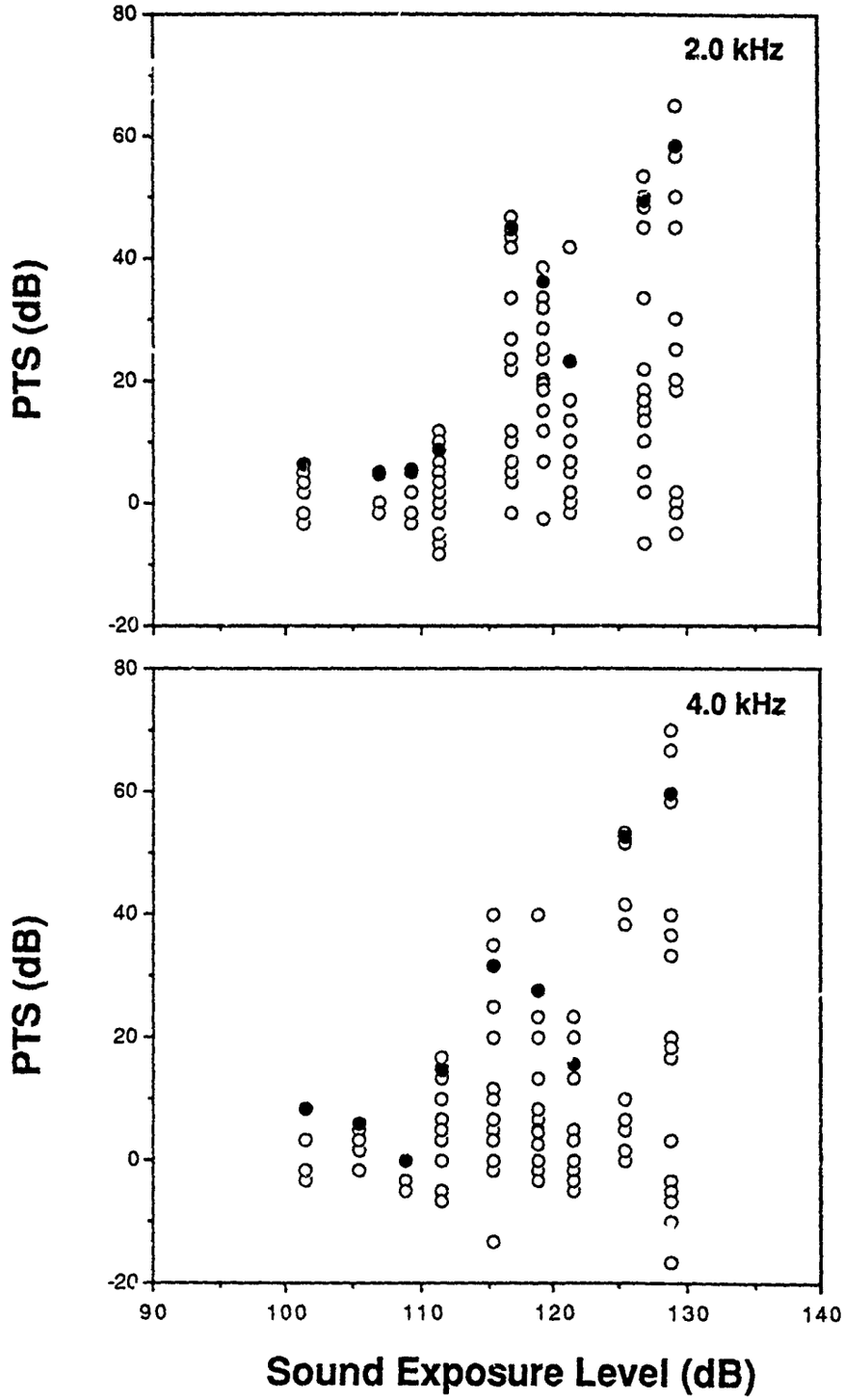

Figure 10. Two examples that illustrate the individual animal PTS values at 2 and 4 kHz following the Series III exposures to Source II. The solid symbols represent the 90th percentile values of the PTS at the various exposure energies.

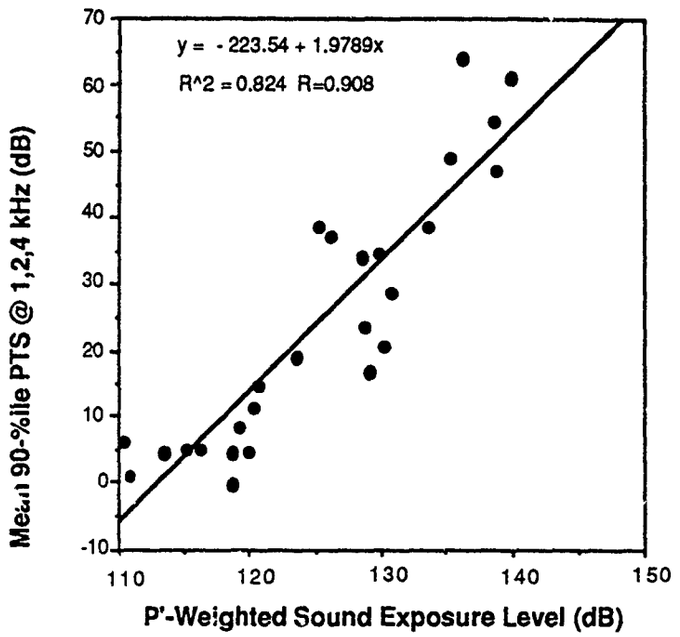

Figure 11. The mean of the 90th percentile PTS measured at 1, 2, and 4 kHz for all of the groups exposed to the Series III impulses as a function of the P'-weighted sound exposure level. A linear regression analysis (solid line) yields a slope of approximately 2.0 and an x-intercept of 113 dB.

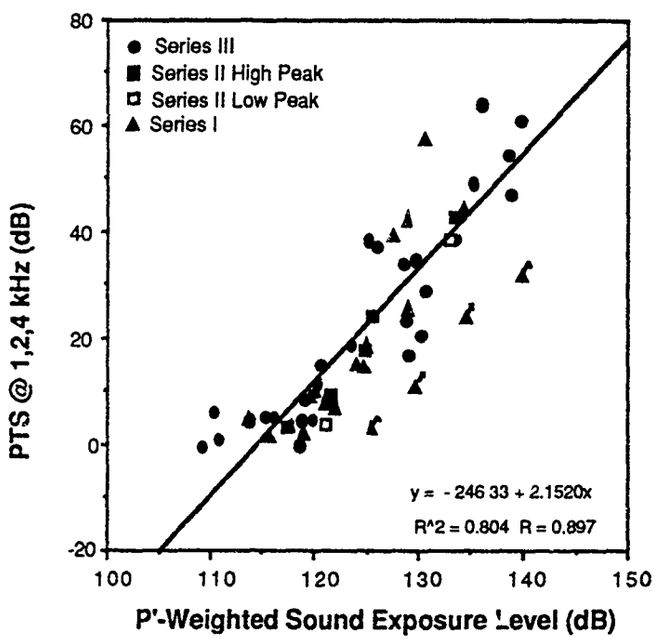

Figure 12. The mean PTS produced by exposures to the Series I, II and III impulses as a function of the P'-weighted sound exposure level. The equation for the linear regression line (solid line) is also given.

Series III type impulses (N = 300); (4) All sensory cell loss data from the above exposures.

The surprising order that is imposed upon the PTS data by the P'weighting function is encouraging and tends to lend some validity to the methods that were used in the analysis, i.e., the organization of group mean data averaged over several frequencies, and in the Series III exposures, the use of a 90th percentile PTS. The analysis presented would indicate that despite the problems and inconsistencies in some of the data obtained from high level impulse noise that have been described in the literature, the use of large samples and the systematic variation of exposure conditions can yield a data base that reflects some underlying order and can be useful in developing exposure criteria. These data have, in addition, shown that using electroacoustic methods and narrow band impulses, a weighting function appropriate for high level blast waves can be established. This weighting function may also be appropriate for use in the evaluation of industrial impact noise data.

The empirical P-weighting function that was presented in Figure 7 has a low frequency segment (i.e., below 1.5 kHz) with a slope of approximately 10 dB/octave which is greater that the low frequency slope of either the A-weighting function or the "relative susceptibility" curve presented by Price (1983). This results in a greater overall attenuation for the lower frequency components of the impulse noise spectrum. Above 1.5 kHz the A-weighting function is relatively flat while the Price susceptibility curve rises monotonically at about 18 dB/octave above 3 kHz. The unusual feature of the empirical P-weighting function is the 2450 Hz point. When the attenuation indicated by this point is applied to the 2 kHz octave band energy of the impulse of the Series II or Series III data the effect is to decrease the correlation coefficient between the $PTS_{1,2,4}$ and the P-weighted sound exposure level. (The actual attenuation used at the 2 kHz octave band is the value obtained by linear interpolation between the 1350 Hz and 2450 Hz data points.) Although the 2450 Hz point appears to be inconsistant with the rest of the P-weighting function, it should be noted that this point is the result of a consistant set of data that were obtained from four different exposure groups (N = 24). If however, the P'-weighting function is used, i.e., an attenuation factor of -5 dB is applied to the 2 kHz octave band energy of the Series II and III impulses the correlation coefficient between $PTS_{1,2,4}$ and the weighted exposure level increases to more than 0.9 (see Figures 9 and 11). This result seems to indicate that the appropriate weighting function to be applied to an impulse spectrum is not a simple monotonic function as implied by A-weighting or the Price susceptibility curve, but rather a more complex function (at least in the chinchilla) at frequencies above about 1 kHz. The data of von Bismarck (1967) on the external ear transfer function and the multifrequency impedance data of Henderson (personal communication), along with the intracochlear pressure measurements of Patterson et al. (1988), would indicate that such a non-monotonic behavior is to be expected.

In conclusion then, it would appear that if a suitable weighting function can be empirically established it could then be applied to the spectrum of an impulse to develop an energy based approach to the establishment of criteria for exposure to a wide variety of noise transients.

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