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

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Annual Report

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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 waves. 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 variety 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 while Source III results were summarized in report ADA 228-368. This report summarizes the results from Source IV, an electrical spark discharge device which generates a blast wave in anechoic (free-field) surroundings whose peak of the A-weighted energy spectrum is in the 4 kHz octave band.

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.

Four previous progress reports (ADA 206-180, ADA 203-854, ADA 221-731 and ADA 228-368) 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 and fourth reports 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 and from 105 chinchillas exposed to non-reverberant blast waves (Source III) with an A-weighted energy spectral peak in the 2.0 kHz octave band, respectively. This report documents the results of parametric experiments performed on 104 chinchillas exposed to non-reverberant blast waves produced by an electrial spark discharge (Source IV) whose peak of the A-weighted energy spectrum is in the 4.0 kHz octave band.

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, II and III. However, the four 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. 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 nere 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 to the contract officer's representative (COR).

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 in 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 was 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 Sources II and III are documented in reports ADA 221-731 and ADA 228-368 respectively. This present report, which documents the audiometric and histological results of exposure to the impulses produced by Source IV, follows a pattern of data presentation that parallels the presentation format of reports ADA 206-180, ADA 221-731 and ADA 228-368. In addition to the importance of the parameters mentioned above 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. This issue will be addressed in the final report from this contract. An appendix of all the audiometric and histological data from Source IV will be made available at the termination of this contract to the COR. [Note: The format of this report follows essentially the same format used for the presentation of the results from Sources I, II 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 report ADA 206-180. Briefly, the basic experimental protocol that is common to all of the experiments consists of the following steps: (1) Preexposure evoked potential 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 euthanized 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 four (104) 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 62 chinchillas (i.e., 3 from each group) from which 372 preexposure TC's and 372 postexposure TC's were obtained. All the individual animal data is tabulated in a data appendix which will be submitted to the COR at the termination of this contract. The results of an analysis of a portion of the 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 included in the final report.

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 diagram of the equipment used to generate the high-energy electrical spark discharge is illustrated in Figure 1. The SPL of the blast wave can be controlled by systematically adjusting the voltage to the spark gap electrodes, the distance between the electrodes and the distance of the subject from the electrodes. The pressure-time history of the blast wave was recorded using a transducer located on the center line at a variable distance from the spark gap. 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, $\rho c = 406$ 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, 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 spark gap electrodes. 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 104 animals were used to complete this experimental paradigm. (Note: one animal was lost in Group 5 due to otitis interna.)

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	4	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	104			

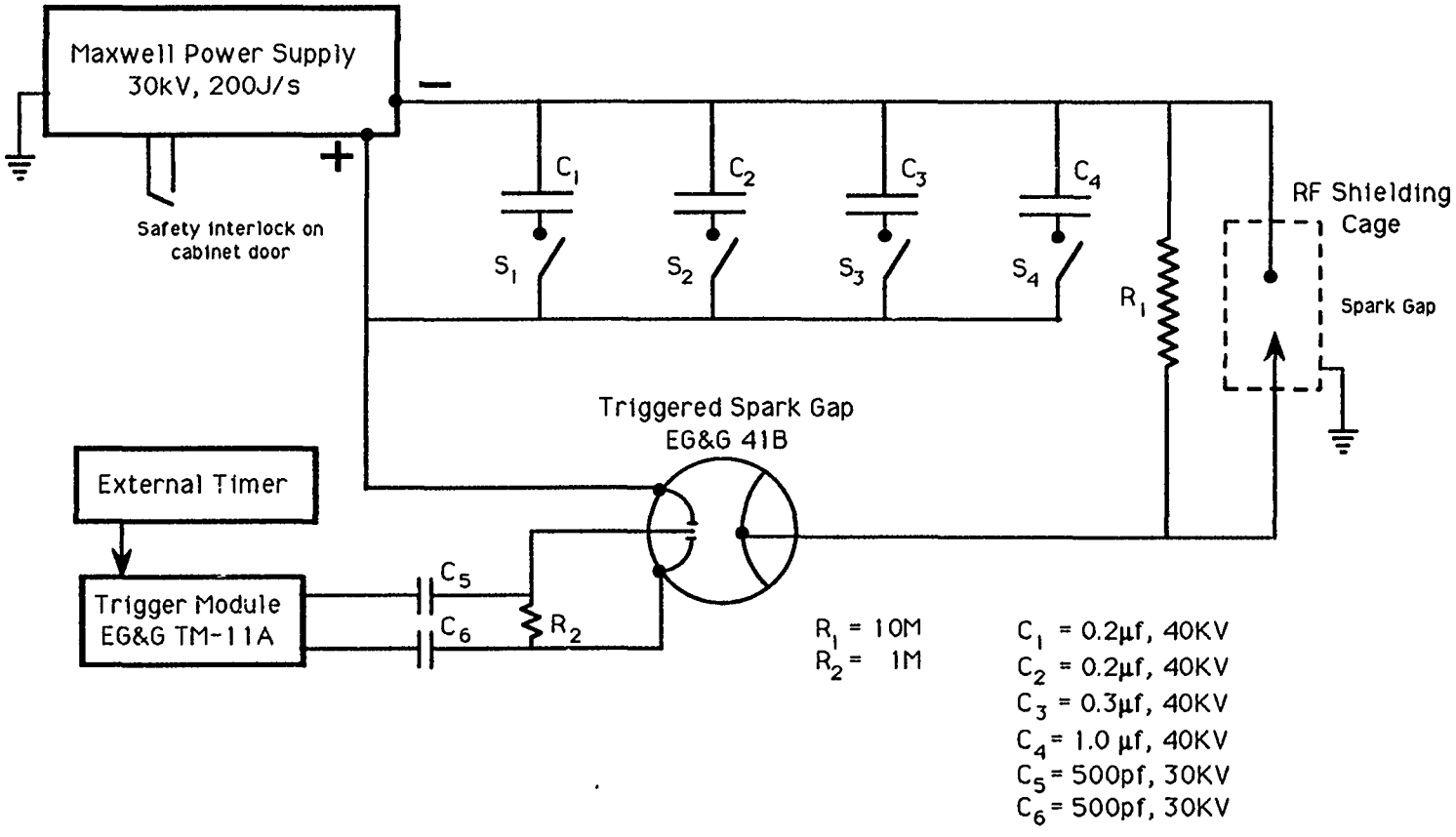

Figure 1. Schematic of the spark discharge impulse noise source.

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 euthanized 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 (Release 4) 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 104 animals are reported in Table II and plotted in Figure 2 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. 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 104 subjects are summarized in Table III.

Table II

Summary of Mean Preexposure Thresholds (dB) for All Animals (N = 104) Compared to Published Norms

	Test Frequency (kHz)							
Present study	0.5	1.0	2.0	4.0	8.0	11.2	16.0	
	19.6	2.5	8.8	-2.5	14.0	12.4	21.8	$\bar{X}$
	5.2	5.1	5.7	6.6	6.6	7.3	7.3	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	$\bar{X}$

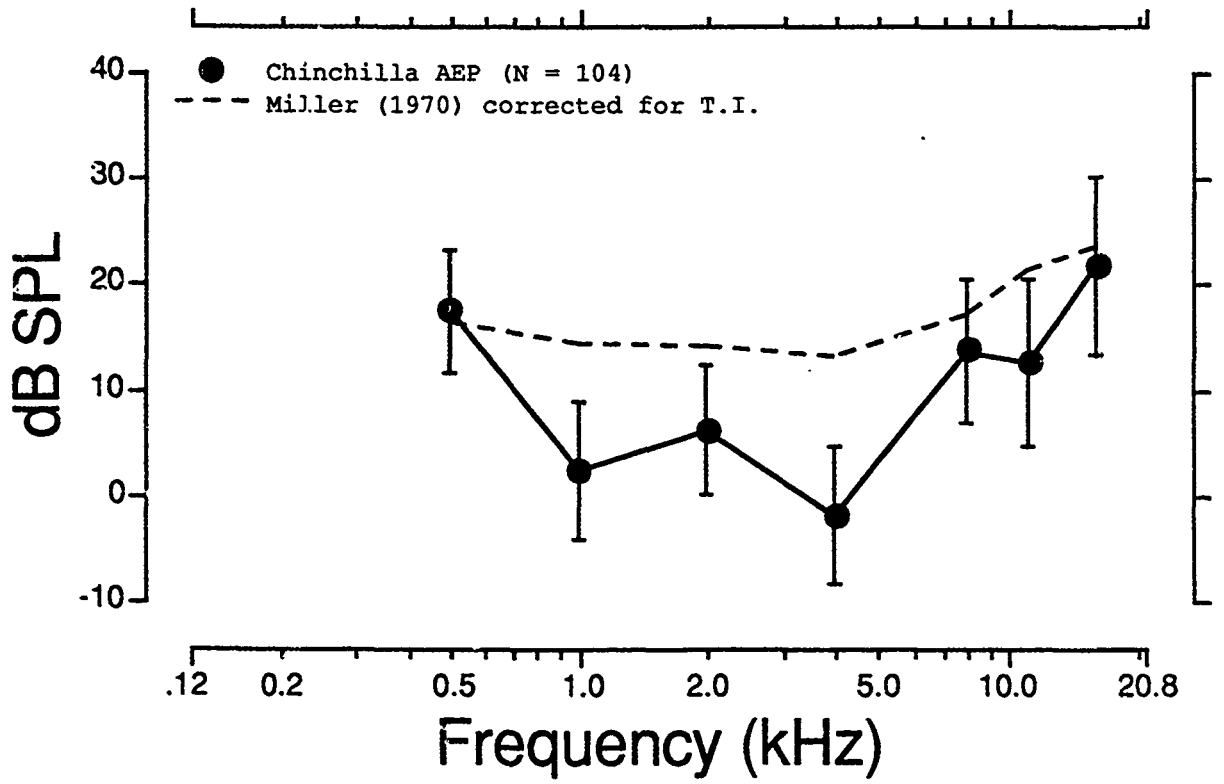

Figure 2. Mean preexposure thresholds for 104 chinchillas.

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	17.2	0.8	4.8	-0.5	15.2	11.2	20.5	$\bar{X}$
				4.8	3.7	4.5	7.4	3.0	6.4	4.5	s
150 dB	10	10/m	5	17.5	1.8	3.8	-1.5	16.2	15.8	24.5	$\bar{X}$
				5.7	10.1	2.2	6.7	5.6	7.7	9.7	s
150 dB	10	1/m	5	17.2	3.2	7.8	-4.5	12.2	7.5	20.2	$\bar{X}$
				4.0	2.8	5.9	7.4	8.6	7.2	11.0	s
150 dB	10	1/10m	5	17.5	0.8	7.5	-2.8	7.5	9.8	16.5	$\bar{X}$
				7.4	5.9	3.9	8.4	5.9	6.5	6.3	s
150 dB	100	10/m	4	18.8	1.7	15.8	1.7	15.0	15.4	22.9	$\bar{X}$
				4.2	4.4	2.7	5.2	8.4	2.1	5.5	s
150 dB	100	1/m	5	19.8	-0.2	10.5	-6.2	15.8	13.5	20.2	$\bar{X}$
				5.1	6.6	2.2	7.8	5.7	11.0	7.0	s
150 dB	100	1/10m	5	18.5	3.2	7.8	-5.5	7.5	5.8	18.8	$\bar{X}$
				6.7	5.1	9.7	5.5	5.4	8.7	2.5	s
155 dB	1		5	23.5	2.5	8.5	-3.5	14.2	11.5	23.5	$\bar{X}$
				5.1	6.1	6.3	5.8	7.5	7.1	9.9	s
155 dB	10	10/m	5	21.5	4.5	11.8	-2.2	18.2	13.2	23.2	$\bar{X}$
				1.5	4.8	5.7	6.2	6.9	8.6	4.7	s
155 dB	10	1/m	5	20.2	3.5	8.2	-6.5	14.5	21.8	23.8	$\bar{X}$
				8.6	7.5	7.5	5.8	5.3	10.3	6.8	s
155 dB	10	1/10m	5	19.2	2.5	12.5	-0.5	19.8	14.5	25.8	$\bar{X}$
				3.1	3.9	3.1	7.3	4.5	6.4	5.8	s
155 dB	100	10/m	5	19.5	3.5	10.2	-4.5	9.5	10.8	18.2	$\bar{X}$
				6.3	3.8	2.5	5.3	3.8	5.4	5.7	s
155 dB	100	1/m	5	18.2	1.2	8.5	-6.8	10.8	13.5	23.2	$\bar{X}$
				1.5	3.6	3.7	7.7	7.7	2.5	2.5	s
155 dB	100	1/10m	5	19.5	2.8	10.2	-2.5	13.2	5.8	14.8	$\bar{X}$
				4.0	8.9	10.8	6.1	8.0	5.5	0.9	s
160 dB	1		5	20.2	4.8	11.2	2.2	14.5	13.8	21.2	$\bar{X}$
				3.5	1.9	4.1	2.2	4.0	5.2	7.7	s
160 dB	10	10/m	5	21.8	4.5	12.2	1.8	11.5	14.5	24.2	$\bar{X}$
				3.5	4.3	6.2	10.6	7.7	2.2	6.3	s
160 dB	10	1/m	5	23.2	0.5	4.2	-3.8	14.8	10.2	16.2	$\bar{X}$
				6.1	3.8	3.5	3.6	8.9	9.2	5.1	s
160 dB	10	1/10m	5	22.5	5.5	11.2	2.8	15.8	14.2	23.5	$\bar{X}$
				7.0	5.8	2.7	5.9	8.6	5.4	6.9	s
160 dB	100	10/m	5	19.5	2.2	9.2	-4.2	15.2	8.5	27.8	$\bar{X}$
				5.2	4.0	4.9	7.5	2.5	8.1	15.7	s
160 dB	100	1/m	5	20.2	-1.2	3.5	-2.8	12.2	16.5	20.8	$\bar{X}$
				4.0	3.2	4.0	4.0	5.9	5.6	5.1	s
160 dB	100	1/10m	5	16.8	3.5	7.2	-2.2	20.2	13.5	27.5	$\bar{X}$
				8.9	3.0	5.2	7.8	3.7	5.7	5.9	s

The audiological dependent variables of the study reported are maximum threshold shift (TS_{max}) and 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 ($F = 1.27$, $df = 20/83$). There was a statistically significant frequency main effect ($F = 258.28$, $df = 6/498$) 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 ($F = 1.11$, $df = 120/498$). 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 IV are shown in Figures 3 (a-c). A time record over a period of 2 ms is shown. The Fourier energy spectrum for these same three waveforms over the entire 8.2 ms window is also shown in Figure 3 (a-c). The total energy flux for each exposure condition is presented in Table V in relative dB levels as well as in J/m^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. A similar tabulation of octave band sound exposures levels (SEL) (Young, 1970) is presented in Table VII where:

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

The data presented in Table VI is also shown plotted as a bar graph in Figure 4 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 octave band analysis clearly shows that the peak of the energy spectrum lies in the 4 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 IX and X). 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 VIII). 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 with peak,number and frequency as the independent variables (i.e., the data were collapsed across rate, see Table XIII).

Table IV

Analysis of Variance Summary Table
of Preexposure Thresholds

Source of Variation	SS	df	MS	F	p
Groups	2194.59	20	109.73	1.27	.223
Between Subjects	7176.65	83	86.47		
Frequency	47777.96	6	7962.99	258.28	.000
Groups x Frequency	4103.71	120	34.20	1.11	.225
Within Subjects	15353.49	498	30.83		

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: $1 J/m^2$		
SPL (dB)		1X	10X	100X	1X	10X	100X
150	None	0.03	0.31	3.15	-15.02	-5.02	4.98
	A	0.03	0.30	2.99	-15.24	-5.24	4.76
155	None	0.08	0.83	8.28	-10.82	-0.82	9.18
	A	0.08	0.76	7.64	-11.17	-1.17	8.83
160	None	0.24	2.42	24.23	-6.16	3.84	13.84
	A	0.23	2.29	22.95	-6.39	3.61	13.61

Table VI

Octave Band Unweighted and A-Weighted Energy Flux (J/m^2)
for a Single Impulse Generated by the Spark Gap Source.

	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	0.0018	0.0018	0.0003	0.0003	0.0026	0.0026
0.125	0.0003	0.0000	0.0000	0.0000	0.0001	0.0000
0.25	0.0001	0.0000	0.0000	0.0000	0.0000	0.0000
0.5	0.0000	0.0000	0.0002	0.0001	0.0004	0.0002
1.0	0.0002	0.0002	0.0008	0.0009	0.0019	0.0020
2.0	0.0016	0.0021	0.0036	0.0049	0.0105	0.0140
4.0	0.0105	0.0127	0.0264	0.0315	0.0811	0.0975
8.0	0.0119	0.0095	0.0375	0.0299	0.1030	0.0841
16.0	0.0024	0.0009	0.0073	0.0023	0.0207	0.0073
> 16.0	0.0026	0.0026	0.0065	0.0065	0.0219	0.0219
Total	0.0315	0.0299	0.0828	0.0764	0.2423	0.2295

Table VII

Octave Band Unweighted and A-Weighted Sound Exposure Levels (dB) for a Single Impulse Generated by the Spark Gap Source.

	150 dB Peak SPL		155 dB Peak SPL		160 dB Peak SPL	
Octave Band CF (kHz)	Unwtg. SEL	A-Wtg. SEL	Unwtg. SEL	A-Wtg. SEL	Unwtg. SEL	A-Wtg. SEL
< 0.125	92.6	92.6	84.9	84.9	94.1	94.1
0.125	84.6	68.0	74.3	57.8	78.5	62.0
0.25	79.3	70.4	58.5	49.7	75.7	66.9
0.5	75.6	71.9	82.6	78.7	86.3	82.9
1.0	83.4	83.4	89.3	89.5	92.8	92.9
2.0	91.9	93.2	95.6	96.9	100.2	101.4
4.0	100.2	101.0	104.2	105.0	109.1	109.9
8.0	100.8	99.8	105.7	104.8	110.1	109.2
16.0	93.9	89.6	98.7	93.6	103.2	98.6
> 16.0	94.2	94.2	98.1	98.1	103.4	103.4
Total	105.0	104.8	109.2	108.8	113.8	113.6

Figures 5 through 11 present the group mean 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.

. 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 indicates 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.

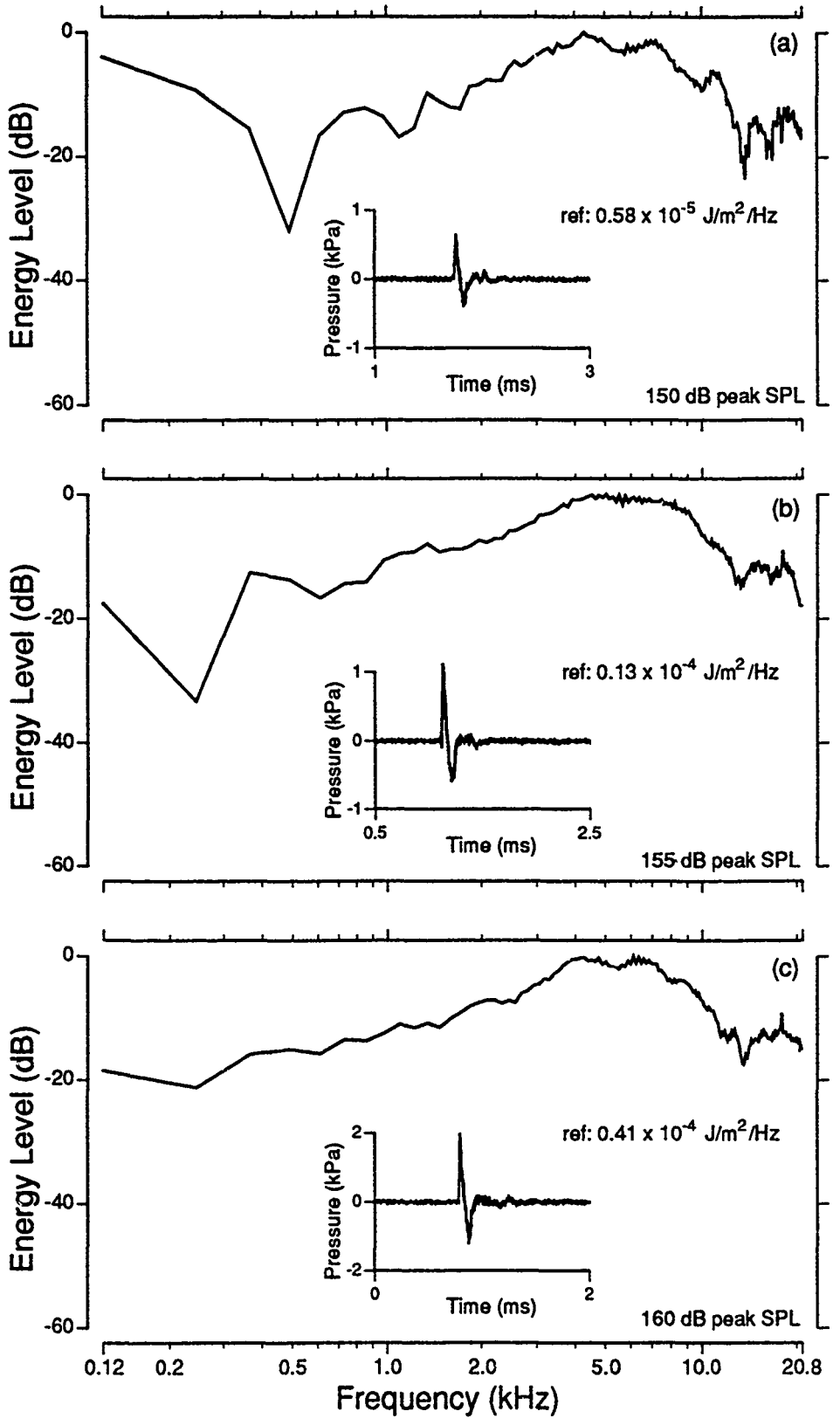

Figure 3. Amplitude spectra and pressure-time waveforms for the blast waves from the electrical spark gap.

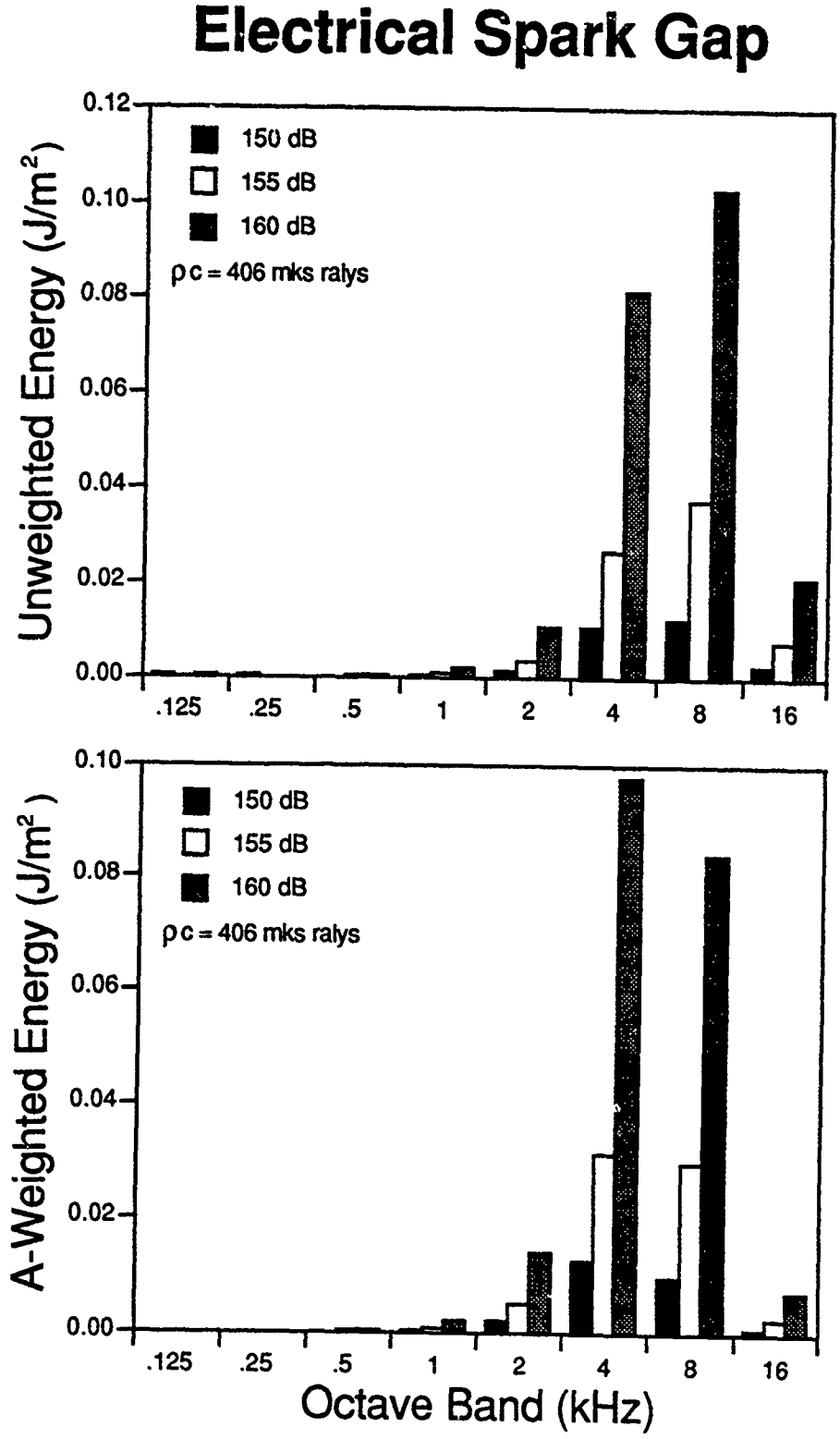

Figure 4. *The unweighted (upper) and A-weighted (lower) octave band analysis of the three blast waves.*

Table VIII

Analysis of Variance Summary Table for Groups Exposed to 1 Impulse

Maximum Threshold Shift

Source of Variation	SS	df	MS	F	p
Peak	2.35	2	1.17	.02	.979
Between Subjects	647.41	12	53.95		
Frequency	557.16	2	278.58	7.34	.003
Peak x Frequency	157.28	4	39.32	1.04	.409
Within Subjects	911.48	24	37.98		

Permanent Threshold Shift

Source of Variation	SS	df	MS	F	p
Peak	24.92	2	12.46	.25	.780
Between Subjects	588.73	12	49.06		
Frequency	246.61	6	41.10	1.92	.089
Peak x Frequency	152.12	12	12.68	.59	.841
Within Subjects	1540.16	72	21.39		

Percent Inner Hair Cell Loss

Source of Variation	SS	df	MS	F	p
Peak	2.27	2	1.13	1.19	.337
Between Subjects	11.40	12	.95		
Frequency	6.98	7	1.00	2.23	.040
Peak x Frequency	6.86	14	.49	1.10	.374
Within Subjects	37.59	84	.45		

Percent Outer Hair Cell Loss

Source of Variation	SS	df	MS	F	p
Peak	6.32	2	3.16	.25	.782
Between Subjects	150.79	12	12.57		
Frequency	527.23	7	75.32	23.83	.000
Peak x Frequency	75.71	14	5.41	1.71	.068
Within Subjects	265.46	84	3.16		

Table IX

Analysis of Variance Summary Table for Groups Exposed to 10 Impulses

Maximum Threshold Shift

Source of Variation	SS	df	MS	F	p
Peak	8671.63	2	4335.81	5.84	.006
Rate	1260.20	2	630.10	.85	.437
Peak x Rate	588.12	4	147.03	.20	.938
Between Subjects	26745.33	36	742.93		
Frequency	17168.38	2	8584.19	60.33	.000
Peak x Frequency	2100.18	4	525.05	3.69	.009
Rate x Frequency	187.69	4	46.92	.33	.857
Peak x Rate x Frequency	313.03	8	39.13	.27	.972
Within Subjects	10244.65	72	142.29		

Permanent Threshold Shift

Source of Variation	SS	df	MS	F	p
Peak	2401.48	2	1200.74	3.99	.027
Rate	124.66	2	62.33	.21	.814
Peak x Rate	33.86	4	8.47	.03	.998
Between Subjects	10843.18	36	301.20		
Frequency	1914.92	6	319.15	4.46	.000
Peak x Frequency	651.85	12	54.32	.76	.692
Rate x Frequency	625.34	12	52.11	.73	.723
Peak x Rate x Frequency	1255.03	24	52.29	.73	.817
Within Subjects	15451.27	216	71.53		

Percent Inner Hair Cell Loss

Source of Variation	SS	df	MS	F	p
Peak	95.46	2	47.73	.60	.556
Rate	20.49	2	10.24	.13	.880
Peak x Rate	288.75	4	72.19	.90	.473
Between Subjects	2880.11	36	80.00		
Frequency	203.36	7	29.05	1.02	.417
Peak x Frequency	711.24	14	50.80	1.79	.041
Rate x Frequency	324.49	14	23.18	.81	.653
Peak x Rate x Frequency	522.25	28	18.65	.66	.909
Within Subjects	7169.07	252	28.45		

Percent Outer Hair Cell Loss

Source of Variation	SS	df	MS	F	p
Peak	1922.61	2	961.30	2.40	.105
Rate	35.74	2	17.87	.04	.956
Peak x Rate	1062.29	4	265.57	.66	.622
Between Subjects	14412.49	36	400.35		
Frequency	1879.70	7	268.53	2.39	.022
Peak x Frequency	2930.64	14	209.33	1.86	.031
Rate x Frequency	1161.36	14	82.95	.74	.735
Peak x Rate x Frequency	1431.81	28	51.14	.45	.993
Within Subjects	28322.85	252	112.39		

Table X

Analysis of Variance Summary Table for Groups Exposed to 100 Impulses

Maximum Threshold Shift

Source of Variation	SS	df	MS	F	p
Peak	2102.71	2	1051.36	1.13	.334
Rate	8024.77	2	4012.38	4.31	.021
Peak x Rate	1390.64	4	347.66	.37	.826
Between Subjects	32546.11	35	929.89		
Frequency	42760.47	2	21380.23	96.32	.000
Peak x Frequency	353.20	4	88.30	.40	.810
Rate x Frequency	307.84	4	76.96	.35	.845
Peak x Rate x Frequency	599.50	8	74.94	.34	.948
Within Subjects	15538.25	70	221.98		

Permanent Threshold Shift

Source of Variation	SS	df	MS	F	p
Peak	842.97	2	421.49	.47	.627
Rate	18078.81	2	9039.40	10.13	.000
Peak x Rate	7070.00	4	1767.50	1.98	.119
Between Subjects	31226.67	35	892.19		
Frequency	14937.01	6	2489.50	23.36	.000
Peak x Frequency	1852.41	12	154.37	1.45	.146
Rate x Frequency	4925.91	12	410.49	3.85	.000
Peak x Rate x Frequency	2537.79	24	105.74	.99	.478
Within Subjects	22381.66	210	106.58		

Percent Inner Hair Cell Loss

Source of Variation	SS	df	MS	F	p
Peak	340.48	2	170.24	.30	.742
Rate	4982.75	2	2491.38	4.40	.020
Peak x Rate	5652.47	4	1413.12	2.49	.061
Between Subjects	19839.35	35	566.84		
Frequency	11290.69	7	1612.96	10.30	.000
Peak x Frequency	2095.17	14	149.66	.96	.500
Rate x Frequency	6756.49	14	482.61	3.08	.000
Peak x Rate x Frequency	8682.40	28	310.09	1.98	.003
Within Subjects	38370.61	245	156.61		

Percent Outer Hair Cell Loss

Source of Variation	SS	df	MS	F	p
Peak	1952.13	2	976.07	.55	.583
Rate	23847.68	2	11923.84	6.70	.003
Peak x Rate	18131.86	4	4532.96	2.55	.056
Between Subjects	62261.00	35	1778.89		
Frequency	54375.35	7	7767.91	23.92	.000
Peak x Frequency	3202.19	14	228.73	.70	.769
Rate x Frequency	19631.98	14	1402.28	4.32	.000
Peak x Rate x Frequency	14835.75	28	529.85	1.63	.027
Within Subjects	79551.59	245	324.70		

2. Impulse Presentation Rate: The effect of rate was examined in two separate analyses (Tables IX & X) of the groups exposed to 10 impulses and those exposed to 100 impulses. The main effect of impulse presentation rate was not statistically significant for any of the analyses for subjects exposed to 10 impulses, but was statistically significant for all audiometric and histological variables for groups exposed to 100 impulses. In these groups, the faster impulse presentation rate caused the greatest amount of damage.

There were also significant interactions between presentation rate and frequency for the groups exposed to 100 impulses. A significant interaction between rate and frequency for PTS and both histological variables suggested that the faster rates (10/m) were more hazardous than the slower rates. There were no statistically significant interactions involving rate in any of the analyses of groups exposed to 10 impulses.

In our earlier reports (ADA 206-180, ADA 221-731 and ADA 228-368), the data from Sources I, II and III (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 all four 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 5 through 11 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 and/or sensory cell loss.

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

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.

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 IV 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 IV seemed to cause the least damage. Examining all four 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).

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	Source IV
150 dB 10X	1/m	1/10m	10/m	1/10m
150 dB 100X	1/m	10/m	1/m	10/m
155 dB 10X	1/m	1/m	1/m	1/m
155 dB 100X	10/m	10/m	10/m	10/m
160 dB 10X	10/m	1/10m	1/m	1/m
160 dB 100X	10/m	10/m	10/m	10/m

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 XIII), 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 also was statistically significant for all dependent variables. 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.

The analyses of groups exposed to the same number of impulses show an 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 only the audiometric variables from those groups exposed to 10 impulses. However, there were a number of statistically significant interactions of peak and frequency or between peak with frequency and rate, indicating that the effect of peak depended upon the audiometric frequency or place on the basilar membrane. In the analysis which included impulse peak and number (Table XIII), the main effect of peak pressure was not

Table XIII

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	3809.86	2	1904.93	2.54	.084
Number	67403.49	2	33701.75	44.98	.000
Peak x Number	2970.31	4	742.58	.99	.416
Between Subjects	71187.17	95	749.34		
Frequency	28651.29	2	14325.64	96.81	.000
Peak x Frequency	557.62	4	139.41	.94	.441
Number x Frequency	8176.85	4	2044.21	13.81	.000
Peak x Number x Frequency	1611.19	8	201.40	1.36	.216
Within Subjects	28116.02	190	147.98		

Permanent Threshold Shift

Source of Variation	SS	df	MS	F	p
Peak	1165.43	2	582.72	.80	.450
Number	24846.70	2	12423.35	17.15	.000
Peak x Number	771.92	4	192.98	.27	.899
Between Subjects	68814.18	95	724.36		
Frequency	5298.01	6	883.00	10.31	.000
Peak x Frequency	618.24	12	51.52	.60	.842
Number x Frequency	5999.82	12	499.98	5.84	.000
Peak x Number x Frequency	1743.47	24	72.64	.85	.675
Within Subjects	48837.95	570	85.68		

Percent Inner Hair Cell Loss

Source of Variation	SS	df	MS	F	p
Peak	151.97	2	75.99	.21	.810
Number	4940.90	2	2470.45	6.88	.002
Peak x Number	123.56	4	30.89	.09	.987
Between Subjects	34105.58	95	359.01		
Frequency	2856.11	7	408.02	4.35	.000
Peak x Frequency	702.16	14	50.15	.53	.913
Number x Frequency	5634.59	14	402.47	4.29	.000
Peak x Number x Frequency	1441.29	28	51.47	.55	.973
Within Subjects	62385.68	665	93.81		

Percent Outer Hair Cell Loss

Source of Variation	SS	df	MS	F	p
Peak	1737.67	2	868.84	.68	.508
Number	33172.29	2	16586.14	13.02	.000
Peak x Number	597.05	4	149.26	.12	.976
Between Subjects	121043.56	95	1274.14		
Frequency	13360.05	7	1908.58	8.71	.000
Peak x Frequency	1493.70	14	106.69	.49	.941
Number x Frequency	27351.43	14	1953.67	8.92	.000
Peak x Number x Frequency	3405.91	28	121.64	.56	.971
Within Subjects	145712.26	665	219.12		

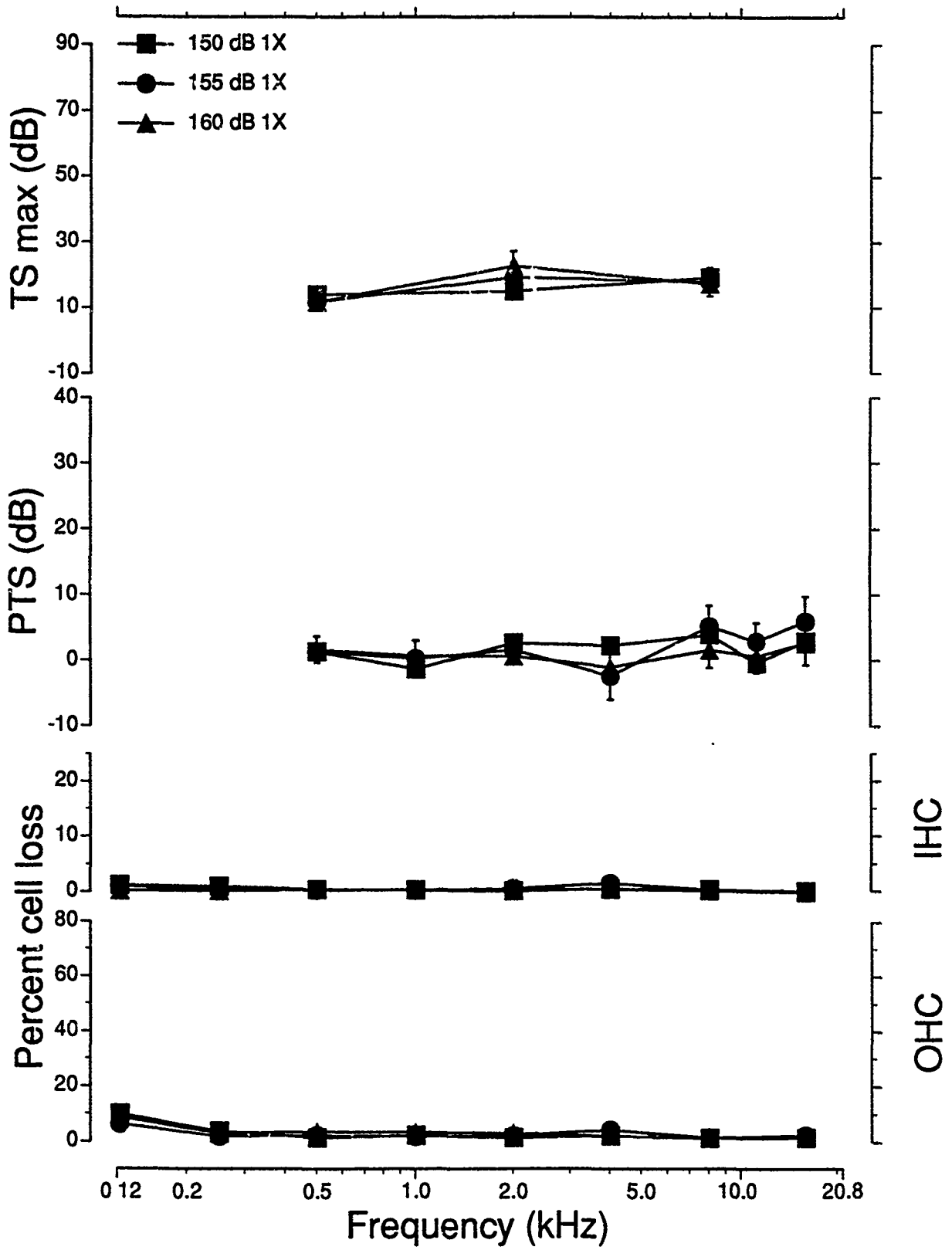

Figure 5. Mean TS_{max} (upper), PTS (middle) and sensory cell loss (lower) for groups of animals exposed to 1 blast wave

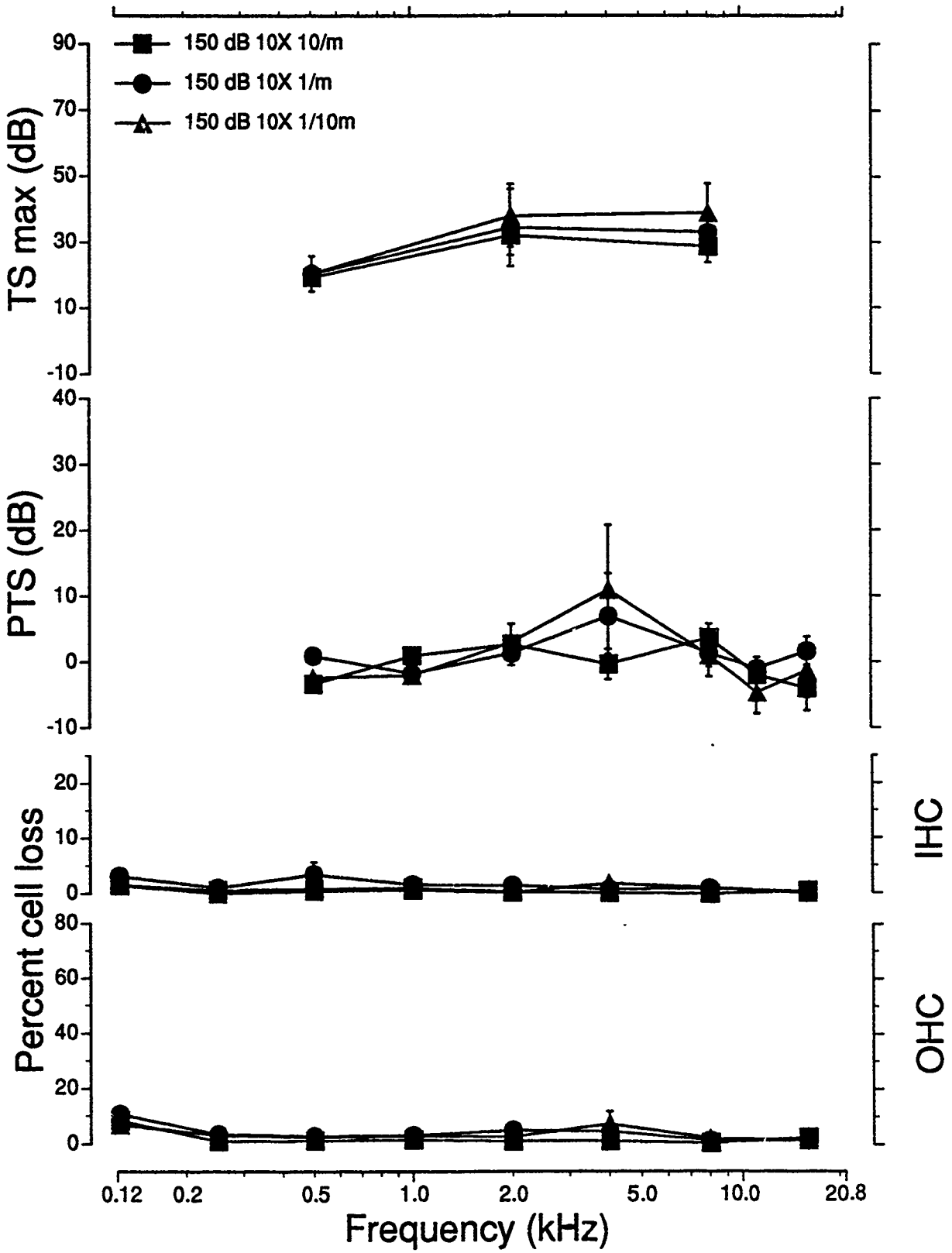

Figure 6. Mean TS_{max} (upper), PTS (middle) and sensory cell loss (lower) for groups of animals exposed to 10 blast waves at 150 dB peak SPL

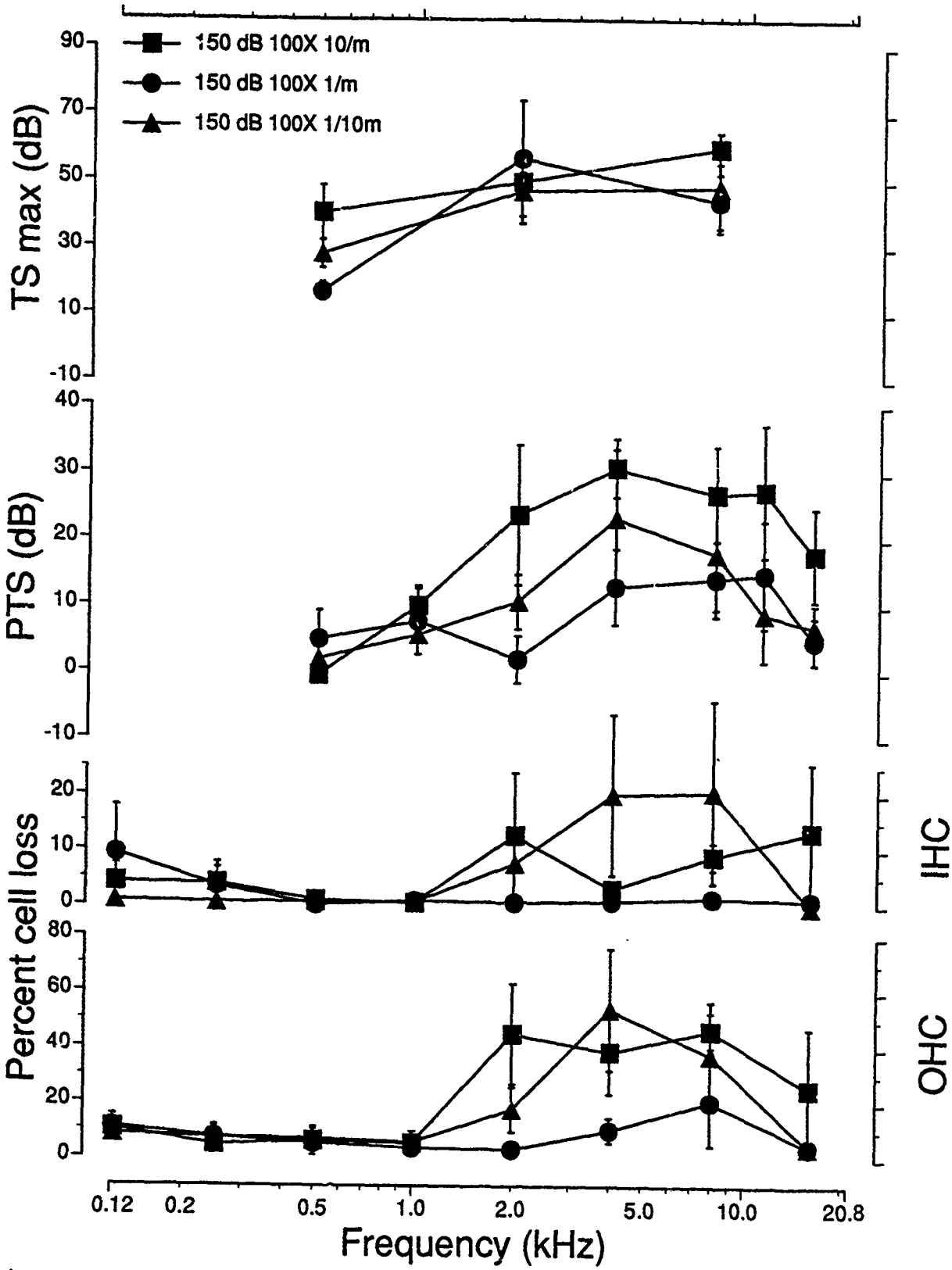

Figure 7. *Mean TS_{max} (upper), PTS (middle) and sensory cell loss (lower) for groups of animals exposed to 100 blast waves at 150 dB peak SPL*

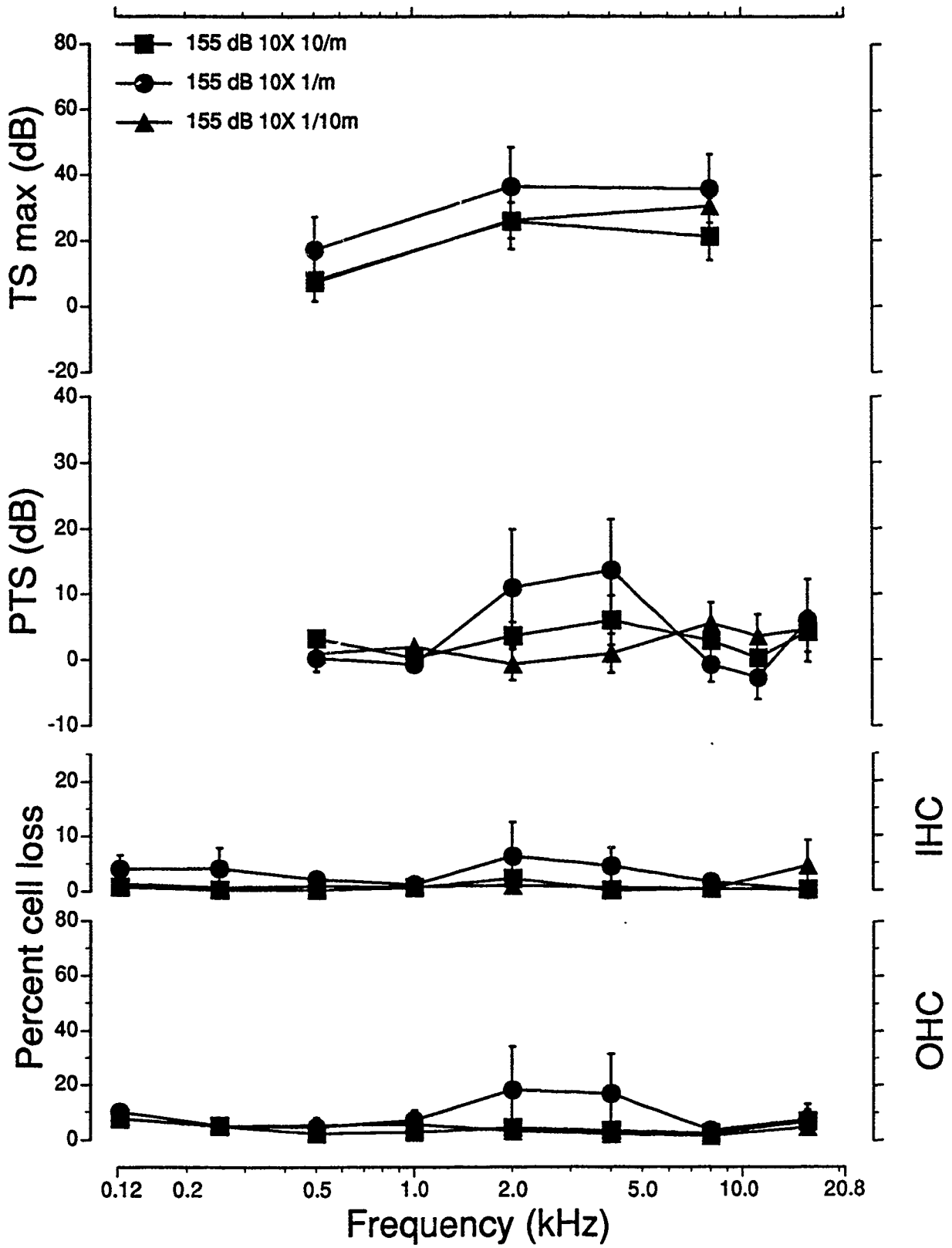

Figure 8. Mean TS_{max} (upper), PTS (middle) and sensory cell loss (lower) for groups of animals exposed to 10 blast waves at 155 dB peak SPL

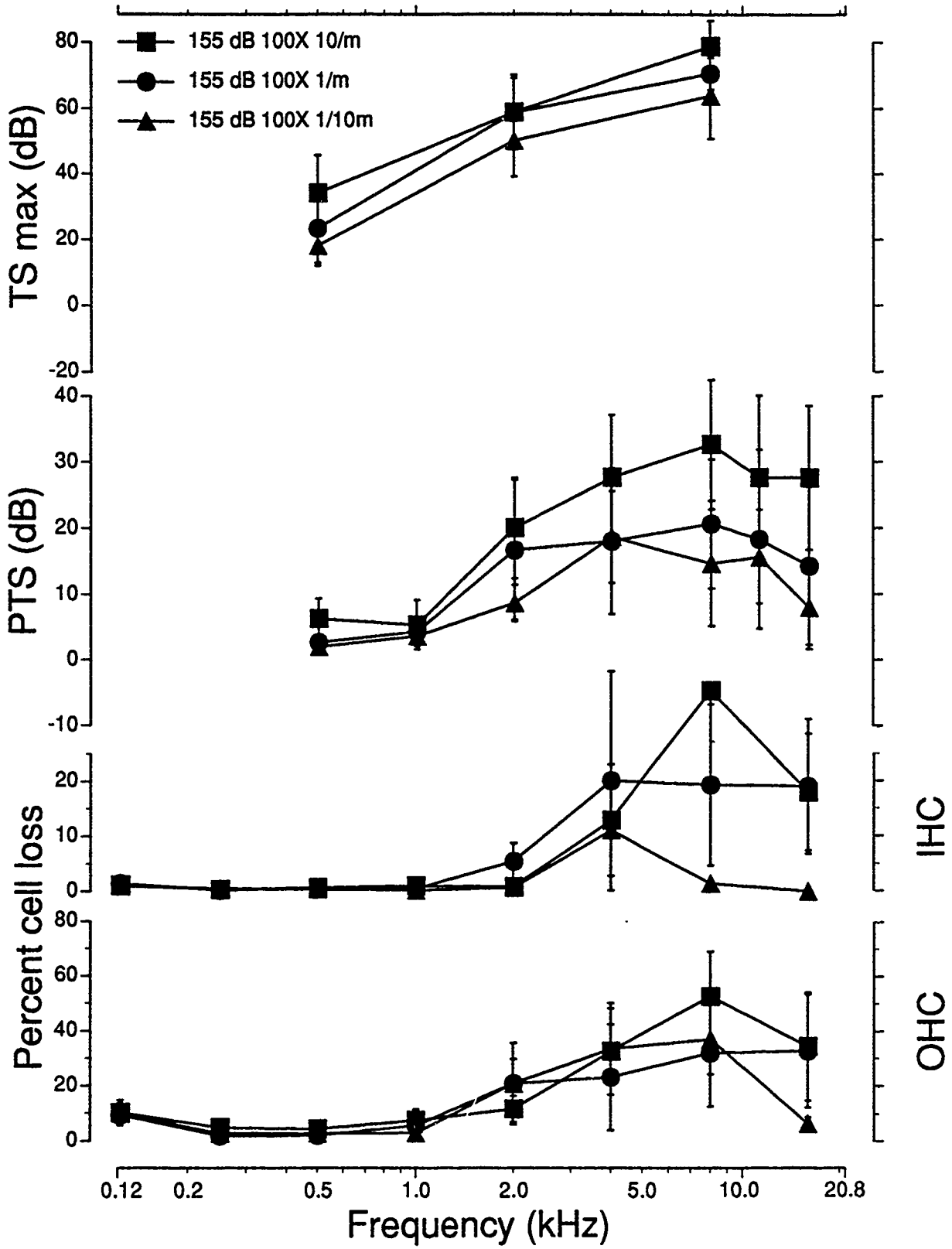

Figure 9. *Mean TS_{max} (upper), PTS (middle) and sensory cell loss (lower) for groups of animals exposed to 100 blast waves at 155 dB peak SPL*

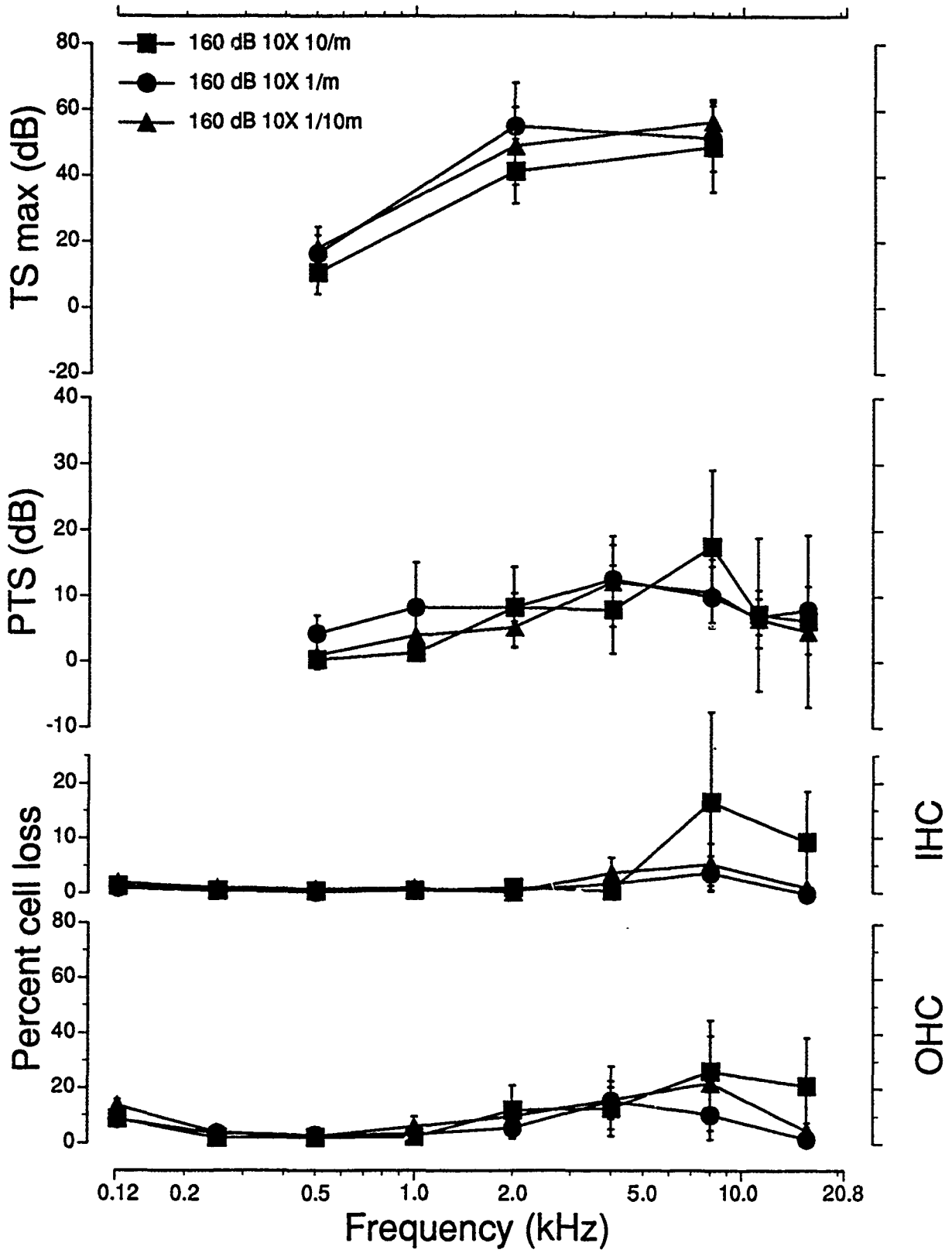

Figure 10. Mean TS_{max} (upper), PTS (middle) and sensory cell loss (lower) for groups of animals exposed to 10 blast waves at 160 dB peak SPL

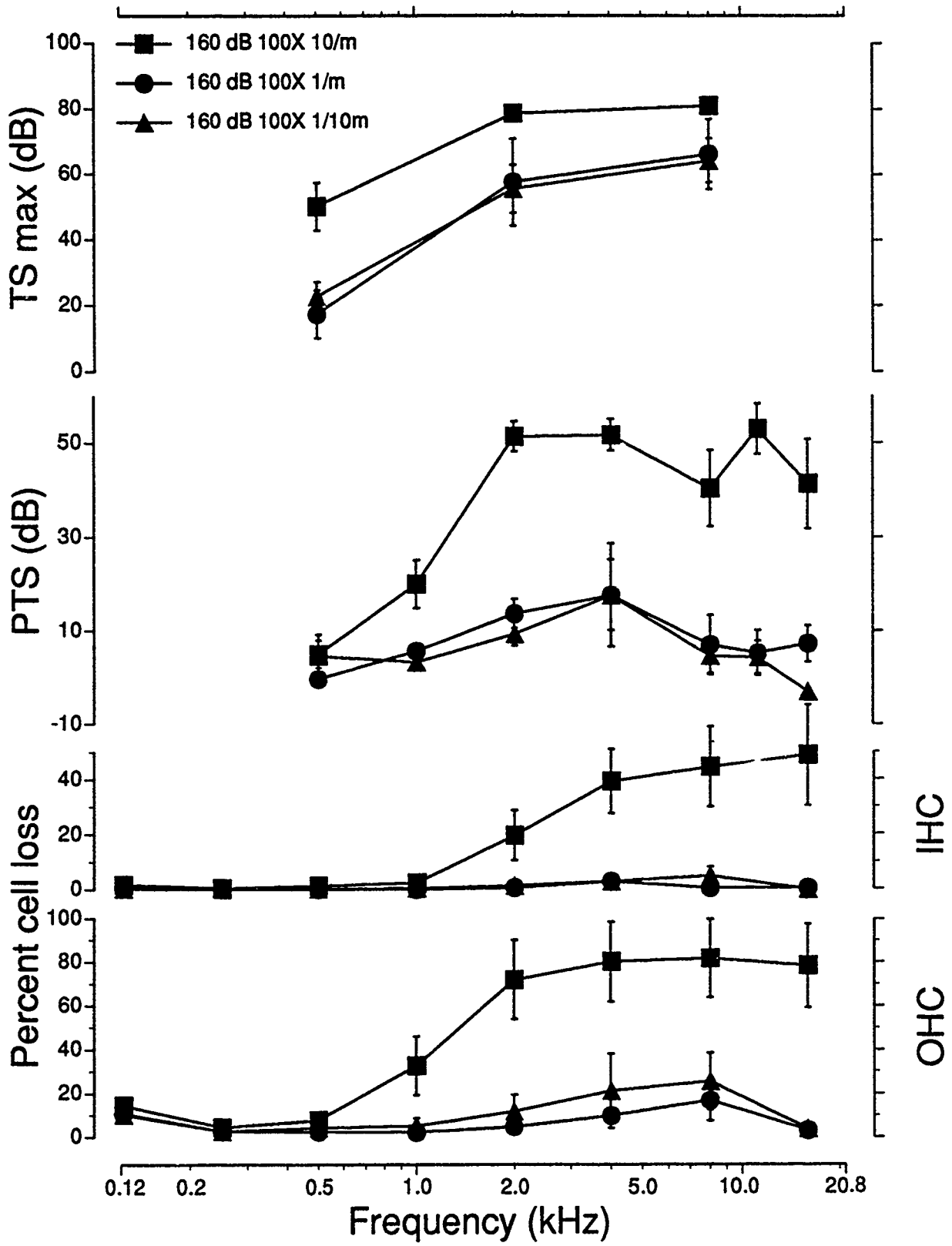

Figure 11. Mean TS_{max} (upper), PTS (middle) and sensory cell loss (lower) for groups of animals exposed to 100 blast waves at 160 dB peak SPL

significant nor were any of the two-way interactions that included the rate variable. However, the three-way interaction of rate with peak pressure and frequency was statistically significant for both histological dependent variables for groups exposed to 100 impulses, indicating an effect of rate that depended upon the peak pressure and the location on the basilar membrane at which the loss was measured. In the analysis which included impulse peak and number (Table XIII), the main effect of peak pressure was not statistically significant for any of the dependent variables, nor were any of the interactions of the other independent variables with peak. Therefore, 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 12 where the mean PTS evaluated at 1, 2 and 4 kHz ($PTS_{1,2,4}$), the percent total inner and outer hair cell loss for each animal are shown plotted as a function of the total sound exposure level. 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 12 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 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.

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.

After completing exposures on 433 experimental subjects using four 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 four sources. We have been developing alternative analytical procedures that show promise in their ability to adequately describe the results from the four sources and which may be used to predict the hazard posed by a variety of impulsive noise exposures (Hamernik et al., 1991). The second observation is that there is an increase in the 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 4.0 kHz (Source IV). In addition, at high sound exposure levels, each source causes a broad hearing loss across a three octave range. However, the most severe sensory cell lesions produced by Source IV do not extend as far into the low frequencies as do the lesions produced by the other three sources. Finally, while the issue of blast wave presentation rate is still ambiguous, it appears, from examination of the

results from all four sources (Tables XI and XII) that the faster repetition rates may be particularly hazardous, especially at high sound exposure levels.

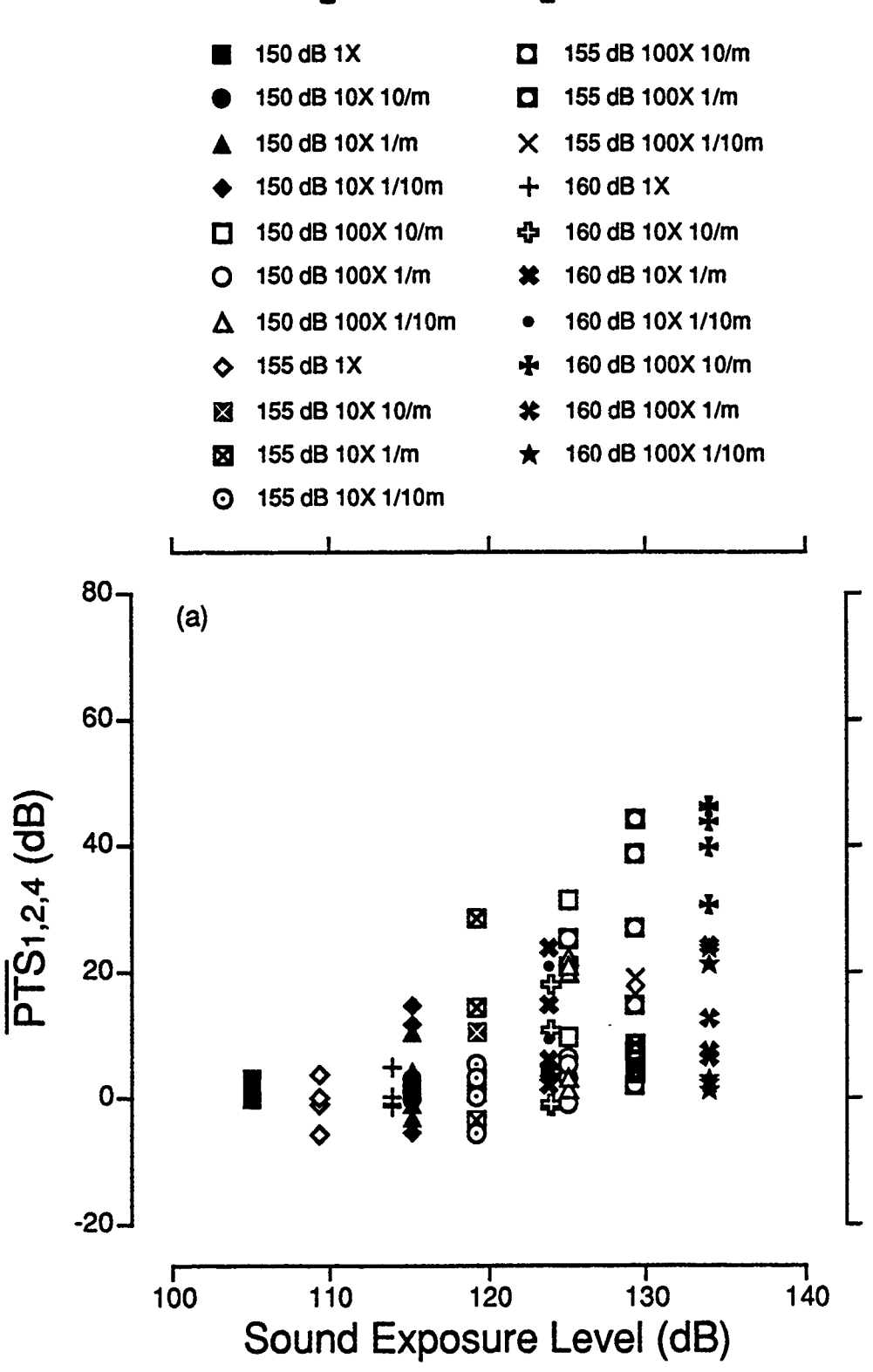

Figure 12a. The distribution of mean $PTS_{1,2,4}$ and percent cell losses from individual animals (N=104) for all exposure conditions

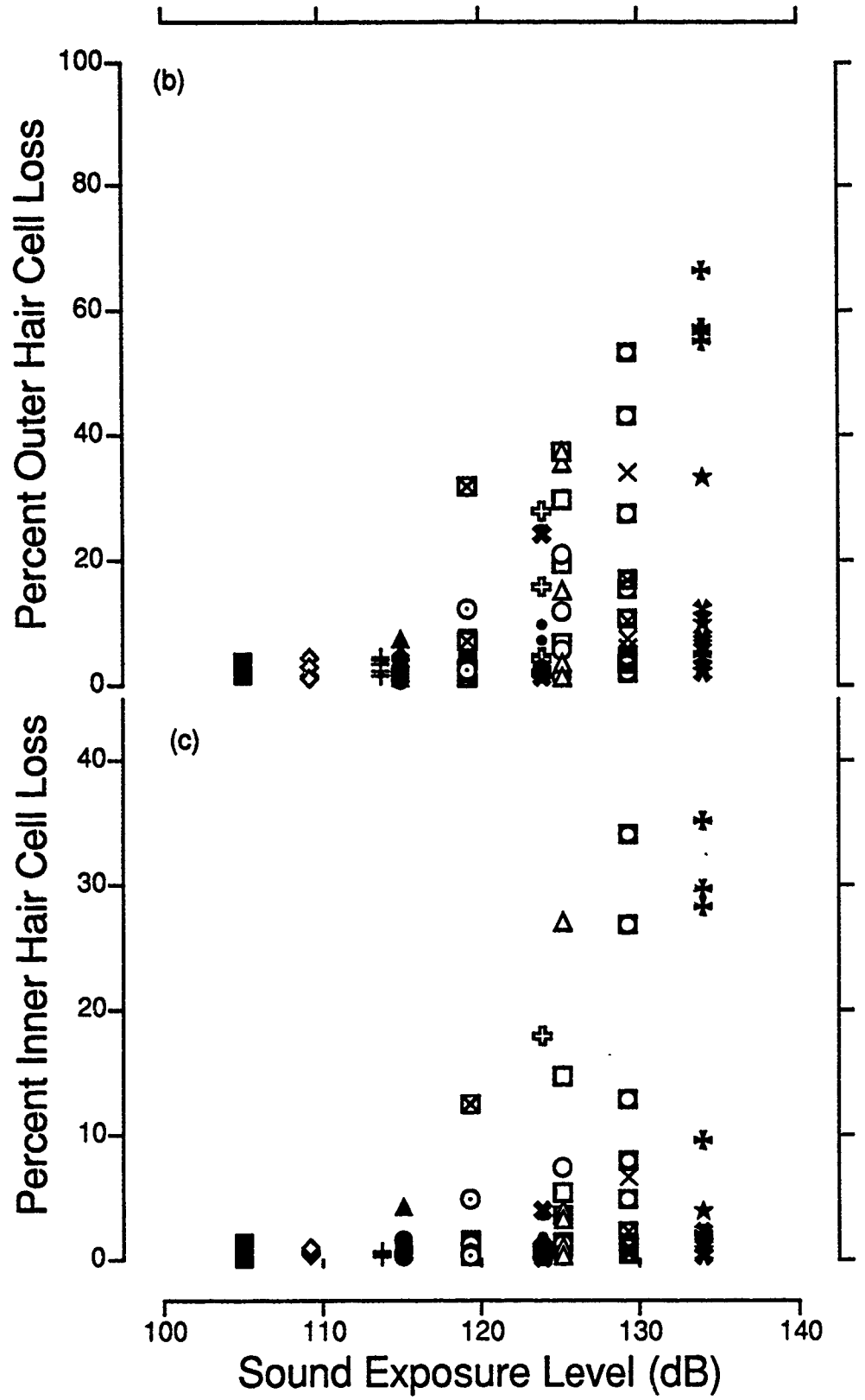

Figure 12b,c. The distribution of mean $PTS_{1,2,4}$ and percent cell losses from individual animals (N=104) for all exposure conditions

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