The value of a kurtosis metric in estimating the hazard to hearing of complex industrial noise exposures

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A series of Gaussian and non-Gaussian equal energy noise exposures were designed with the objective of establishing the extent to which the kurtosis statistic could be used to grade the severity of noise trauma produced by the exposures. Here, 225 chinchillas distributed in 29 groups, with 6 to 8 animals per group, were exposed at 97 dB SPL. The equal energy exposures were presented either continuously for 5 d or on an interrupted schedule for 19 d. The non-Gaussian noises all differed in the level of the kurtosis statistic or in the temporal structure of the noise, where the latter was defined by different peak, interval, and duration histograms of the impact noise transients embedded in the noise signal. Noise-induced trauma was estimated from auditory evoked potential hearing thresholds and surface preparation histology that quantified sensory cell loss. Results indicated that the equal energy hypothesis is a valid unifying principle for estimating the consequences of an exposure if and only if the equivalent energy exposures had the same kurtosis. Furthermore, for the same level of kurtosis the detailed temporal structure of an exposure does not have a strong effect on trauma.
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I. INTRODUCTION

Industrial noise is often non-Gaussian (nonG) in character. That is, it consists of a steady or non-steady state Gaussian (G) noise punctuated by high level transients, either impacts or noise bursts. The transients typically have variable peak intensities, durations and rates of occurrence making the noise environment difficult to quantify or to characterize for hearing conservation purposes. There is considerable data available indicating that such complex noise exposures pose a greater hazard to hearing than does a purely G noise exposure of equivalent energy (Lataye and Campo, 1996; Thiery and Meyer-Bisch, 1988; Passchier-Vermeer, 1983; Sulkowski, 1983; Ahroon *et al.*, 1993; Dunn *et al.*, 1991). Current international standards for exposure to noise (ISO-1999, 1990) rely solely on an energy metric and may thus not protect large numbers of workers employed in complex noise environments from acquiring a noise-induced hearing loss (NIHL).

The statistical metric kurtosis (β), an index of the extent to which the distribution of a variable deviates from the Gaussian, is defined as the ratio of the fourth-order central moment to the squared second-order central moment of a distribution. The kurtosis [β(t)] can be computed on the amplitude distribution of the temporal waveform of the noise presented to the subject or by filtering the waveform, a frequency specific kurtosis [β(f)] can be computed on the resultant time-domain signal. Experimental animal model data using the chinchilla have shown that for a fixed energy of exposure, hearing and sensory cell loss increase as β(t) of the amplitude distribution of the noise stimulus increases (Hamernik and Qiu, 2001; Hamernik *et al.*, 2003b), while β(f) has been shown to be related to the frequency specific sensory cell loss caused by a high β(t) noise exposure (Hamernik *et al.*, 2003b). These and other experimental results (Zhao *et al.*, 2010) have suggested that the kurtosis metric in combination with an energy metric may be a better index of the potential of a noise exposure to cause hearing loss than is an energy based metric alone.

A nonG noise, $\beta(t) > 3$, can be effectively modeled as a combination of a G noise, $\beta(t) = 3$, with a variety of high level transients superimposed. The transients may be impacts or noise bursts of varying peak intensities, inter-transient intervals and durations. The distribution of the high level transient peaks, inter-transient intervals and transient durations are all known to affect the outcome of an exposure. One way of quantifying the complex temporal structure of a nonG noise is to measure the peak (P), interval (I), and duration (D) histograms of the transients in the noise signal. The kurtosis is sensitive to, and to a large extent is determined by, these three primary variables. It also has the advantage that the temporal structure of a complex noise can be incorporated into a single easily computed number (Erdreich, 1986). Different industries can have the same noise levels but have very different temporal noise profiles and thus different I, P, and D histograms. For a given exposure energy, there is an infinite number of combinations of I, P, and D histograms that will yield the same value of the kurtosis. A kurtosis metric can therefore be useful if and only if, at a given exposure energy, spectrum and value of kurtosis, hearing loss and cochlear pathology are reasonably independent of the detailed temporal structure of the complex noise, i.e., independent of the combinations of the I, P, and D histograms. The experiments described below were designed to test this hypothesis. However, the experimental design also

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allowed for (a) a replication or reinforcement of earlier results that showed increased trauma produced by nonG exposures relative to equivalent energy G exposures (Hamernik *et al.*, 2003b; Hamernik and Qiu, 2001; Lei *et al.*, 1994); (b) an evaluation of the equal energy principle as it applies to interrupted/intermittent noise exposures (Qiu *et al.*, 2007; Hamernik *et al.*, 2007); and (c) the effect of toughening/conditioning (Miller *et al.*, 1963; Clark *et al.*, 1987; Canlon *et al.*, 1988; Hamernik *et al.*, 2003a, 1994; Hamernik and Ahroon, 1999) on the resultant hearing and sensory cell loss.

II. MATERIALS AND METHODS

A. Auditory evoked potential

In this study, 225 chinchillas, obtained from a local breeder, were used as subjects. Animals were between 1 and 6 years old and weighed between 450 and 850 g; 79% were males. Each animal was anesthetized [IM injection of Ketamine (35 mg/kg) and Xylazine (1 mg/kg)] and made monaural by the surgical destruction of the left cochlea. During this procedure a bipolar electrode was implanted, under stereotaxic control, into the left inferior colliculus and the electrode plug cemented to the skull for the recording of the auditory evoked potential (AEP) (Henderson *et al.*, 1973; Salvi *et al.*, 1982). The AEP was used to estimate pure tone thresholds. The animals were awake during testing and restrained in a yoke-like apparatus to maintain the animal's head in a fixed position within the calibrated sound field. AEPs were collected to 20 ms tone bursts (5 ms nonlinear rise/fall time) presented at a rate of 10/s. Each sampled AEP waveform was analyzed for large-amplitude artifact, and if present, the sample was rejected from the average and another sample taken. Averaged AEPs were obtained from 250 presentations of the 20 ms signal. Thresholds were measured using an intensity series with 5 dB steps at octave intervals from 0.5 to 16.0 kHz. Threshold was defined to be one-half step size (2.5 dB) below the lowest intensity that showed a response consistent in amplitude and latency with the responses seen at higher intensities. Additional details of the AEP experimental methods may be found in Ahroon *et al.* (1993).

B. Histology

Following the last AEP test protocol, each animal was euthanized under anesthesia and the right auditory bulla removed and opened to gain access to the cochlea for perfusion. Fixation solution consisting of 2.5% glutaraldehyde in veronal acetate buffer (final pH = 7.3) was perfused through the cochlea. After 24 h of fixation the cochlea was postfixed in 1% OsO_4 in veronal acetate buffer. Surface preparation mounts of the entire organ of Corti were prepared (Engstrom *et al.*, 1966) and inner hair cell (IHC) and outer hair cell (OHC) populations were plotted as a function of frequency and location using the frequency-place map of Eldredge *et al.* (1981). Missing cells were identified by the presence of a characteristic phalangeal scar. For purposes of this presentation, sensory cell population data are presented as group averages (in percent missing) taken over octave band lengths of the cochlea centered on the primary AEP test frequencies and as the group mean total number of IHCs and OHCs missing.

C. Noise exposures

The experimental approach was designed to determine to what extent auditory system trauma, as quantified by AEP threshold shifts and sensory cell loss, is affected by differences in the temporal structure of various nonG noise exposures all having the same spectral energy and value of kurtosis. All noise exposures had in common the same approximately flat spectrum between 0.125 and 8.0 kHz and were presented at an overall SPL of 97 dB(A). The equal energy noise exposures were presented either continuously for 5 days (24 h/d) or on an interrupted schedule for 19 day. Both G and nonG noises were used. The 19 days exposures were structured to model an idealized 3-week work shift. Each daily exposure consisted of two 4.25 h periods with an hour break in between. Each 4.25 h exposure was interrupted for 15 min and each 5 day sequence was separated by a 2 day break. Two different I, P, and D histograms, shown in Fig. 1, were used to create and define the nonG noises. Each of the eight different nonG exposure groups that were exposed to one of the three fixed $\beta(t)$ conditions (Table I) is identified by the unique combination of $I_xP_xD_x$ where the subscript identifies the specific histogram shown in Fig. 1 that was used. Each of the eight groups of animals within each set of exposures at $\beta(t) = 25$, 50, or 100 was exposed to a different nonG noise characterized by one of the eight possible combinations of the two different I, P and D histograms. Animals in the 19 day exposures were returned to the colony immediately following each daily exposure unless AEP thresholds were to first be measured.

D. Noise measurement and analyses

The acoustic signal produced by the Electro-Voice Xi-1152/94 speaker system was initially calibrated by a Bruel and Kjær 1/2 in. microphone (Model 4134), amplified by a Bruel and Kjær (Model 2610) measuring amplifier and fed to a Windows PC-based analysis system. The signal was sampled at 48 kHz with a recording duration of 5.5 min. The design and digital generation of the acoustic signal is detailed in Hsueh and Hamernik (1990, 1991) and Hamernik *et al.* (2007). During the exposures the noise field was monitored with a Larson Davis 814 sound level meter equipped with a 1/2 in. microphone. For all exposures a mean value of $\beta(t)$ was obtained by averaging $\beta(t)$ computed on consecutive 40 s windows (Hamernik *et al.*, 2003b) over a 5.5 min sample of the exposure waveform using MATLAB (MathWorks) software. Similarly, $\beta(f)$ was obtained by first filtering the acoustic signal over consecutive octave bands centered on the AEP test frequencies and computing the value of $\beta(f)$ on the temporal waveform of the filtered signals.

E. Experimental protocol

Following a 2-week post surgical recovery, three AEP preexposure audiograms were obtained (on different days)

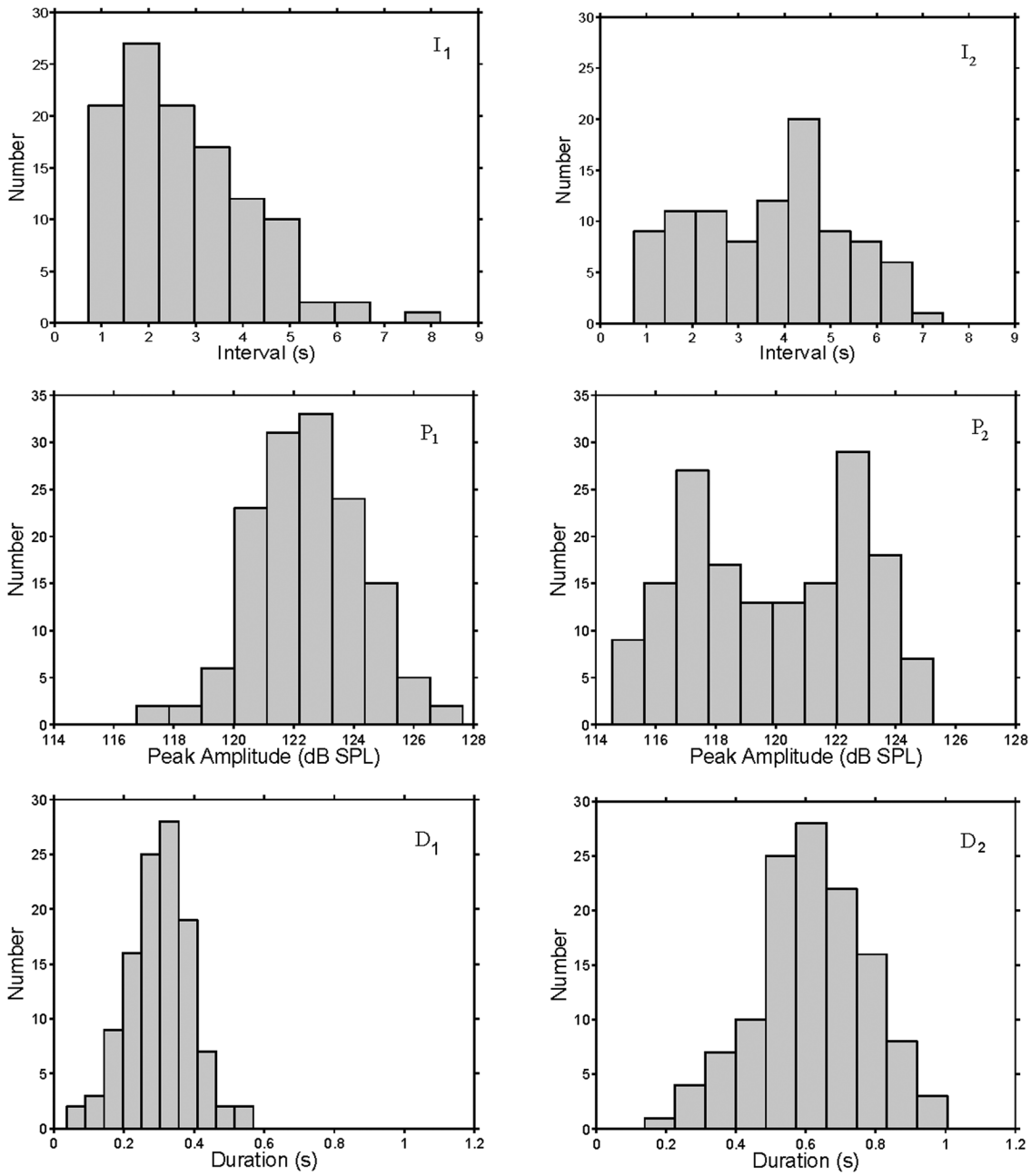

FIG. 1. The impact interval histograms (I_1 and I_2), peak amplitude histograms (P_1 and P_2) and duration histograms (D_1 and D_2) computed over a 5.5 min digitized segment of the non-Gaussian noise.

on each animal at octave intervals between 0.5 and 16.0 kHz. If the mean of the three audiograms fell beyond one standard deviation of laboratory norms (Hamernik and Qiu, 2001), in the direction of poorer thresholds at more than one test frequency, the animal was rejected. The animals were randomly assigned to one of 29 experimental groups with 6 to 8 animals/group as summarized below and in Table I.

(a) Two groups were exposed to either a 5 day continuous (24 h/d) G noise or to a 19 day interrupted G noise. These two groups served as G noise exposed control/reference groups.

(b) Three groups were exposed for 5 day (24 h/d) to one of three continuous nonG noises having $\beta(t) = 25$, 50, or 100. These groups served as 5 day, nonG control/reference groups.

(c) Twenty-four groups were exposed to a nonG, interrupted noise for 19 days. Of these, three sets of eight groups were exposed to a noise having either a $\beta(t) = 25$, 50 or 100. Within each of the three sets of fixed $\beta(t)$ exposures, each group was exposed to one of eight different nonG noises created from all combinations of the two different I, P, and D histograms (Fig. 1).

The animals were exposed four at a time in individual open grill cages (10 in. × 17 in. × 11 in.) that allowed the animal some limited degree of movement. During exposure, animals were given free access to food and water and were

TABLE I. Outline of the 29 groups of animals exposed to the various broadband, 97 dB SPL noise exposures (Σn = 225).

Gaussian $\beta(t) = 3$ exposures					
5-day (n = 8)				19-day (n = 6)	
NonGaussian 5-day exposures ($I_1P_1D_1$)[a]					
$\beta(t) = 25$ (n = 8)		$\beta(t) = 50$ (n = 8)		$\beta(t) = 100$ (n = 7)	
NonGaussian 19-day exposures					
$\beta(t) = 25$ Σn = 62		$\beta(t) = 50$ Σn = 63		$\beta(t) = 100$ Σn = 63	
n	n	n	n	n	n
$I_1P_1D_1$ 8	$I_2P_1D_1$ 8	$I_1P_1D_1$ 8	$I_2P_1D_1$ 8	$I_1P_1D_1$ 8	$I_2P_1D_1$ 8
$I_1P_1D_2$ 8	$I_2P_1D_2$ 8	$I_1P_1D_2$ 8	$I_2P_1D_2$ 8	$I_1P_1D_2$ 8	$I_2P_1D_2$ 8
$I_1P_2D_1$ 8	$I_2P_2D_1$ 6	$I_1P_2D_1$ 8	$I_2P_2D_1$ 7	$I_1P_2D_1$ 7	$I_2P_2D_1$ 8
$I_1P_2D_2$ 8	$I_2P_2D_2$ 8	$I_1P_2D_2$ 8	$I_2P_2D_2$ 8	$I_1P_2D_2$ 8	$I_2P_2D_2$ 8

[a]I, the interval histogram; P, the peak SPL histogram; D, the transient duration histogram. The subscript for each parameter identifies one of the two different distributions (Fig. 1) that were used. (n = subjects/group, $\beta(t)$ = kurtosis value).

rotated through a bank of four individual cages daily. The SPLs, across cages, in the middle of each cage, varied within less than ±1 dB. For the 5 day continuous (24 h/day) reference/control exposures, animals were removed daily at the same time for less than 0.5 h for AEP testing. The mean of the five audiograms thus obtained defined an asymptotic threshold or an asymptotic threshold shift (ATS) (Mills, 1976; Blakeslee *et al.*, 1978). For the interrupted 19 day exposures the animals were tested at the end of the exposure on day 1, 17, 18, and 19. The difference between the threshold measured following the first day (T_1) and the mean of the thresholds measured following the last 3 days ($T_{17–19}$) of the exposure was accepted as an estimate of threshold recovery in noise or toughening (Tr) [i.e., Tr = (T_1) − ($T_{17–19}$)] (Miller *et al.*, 1963; Clark *et al.*, 1987; Hamernik *et al.*, 1994). Thirty days following the last exposure day for all exposure paradigms, three more AEP audiograms were collected on different days and the mean used to define permanently shifted thresholds or permanent threshold shift (PTS).

In conducting the research described in this study, the investigators adhered to the guidelines specified by the National Research Council (1996). All research protocols were reviewed yearly by the University IACUC.

F. Statistical analysis

The thresholds and sensory cell loss data were compared among the groups of noise exposed animals using a two-way, mixed model analysis of variance (ANOVA) with repeated measures on one factor (frequency). The probability of a type 1 error was set at 0.05. Statistically significant main effects of frequency were expected and found in all of the following analyses because of the frequency-specific nature of the audibility curve of the chinchilla and the noise exposure stimulus. For this reason main effects of frequency are not addressed in the presentation of the results. Where appropriate, *post hoc*, pair-wise comparisons among the groups were made using the Tukey/Kramer test. In the graphical presentation of the data, bars for the standard error of the mean (s.e.) are shown. No bar is shown when the s.e. was smaller than the datum symbol.

III. RESULTS

A. Preexposure thresholds

The mean preexposure thresholds for all animals (n = 225) are shown in Fig. 2 and compared with laboratory norms based on a sample of 1572 chinchillas (shaded area). These data are consistent with published thresholds for the chinchilla (Fay, 1988). There were no significant differences in preexposure thresholds among the experimental groups.

B. The acoustic stimulus

Figure 3(a) shows the average long term spectrum of the G noise along with a representative example of the spectrum and waveform of the $\beta(t) = 50$ nonG noise [Fig. 3(b)] and the high level noise impacts [Fig. 3(c)] used to create the nonG noise. The Leq [dB and dB(A)] computed over octave band widths of the signal for all exposure conditions is shown in Fig. 4(a). Between 1.0 and 8.0 kHz the Leq is relatively flat. The mean values of $\beta(f)$ for all exposure conditions is shown in Fig. 4(b) as a function of the center frequency of the octave band filtered noise stimulus from which it was calculated. Error bars are not shown since the standard error of the mean was less than the size of the symbol.

C. For a fixed kurtosis and energy spectrum, is noise-induced trauma independent of the detailed temporal structure of the noise, i.e., independent of the combinations of the I, P, and D histograms?

A summary of the group mean PTS, IHC, and OHC loss for the three sets of eight groups of animals exposed to the $\beta(t) = 25$, 50, or 100 nonG noise at 97 dB SPL is shown in Figs. 5–7, respectively. The eight groups within each of the three sets differed only in the I, P, and D histograms that were used to create the nonG noise (Table I). Within each

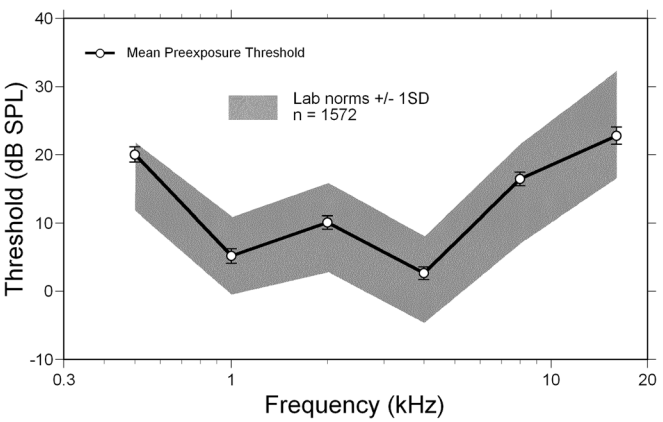

FIG. 2. Mean preexposure evoked potential thresholds for all (n = 225) chinchillas compared to laboratory norms (shaded area) based on a sample of 1572 chinchillas.

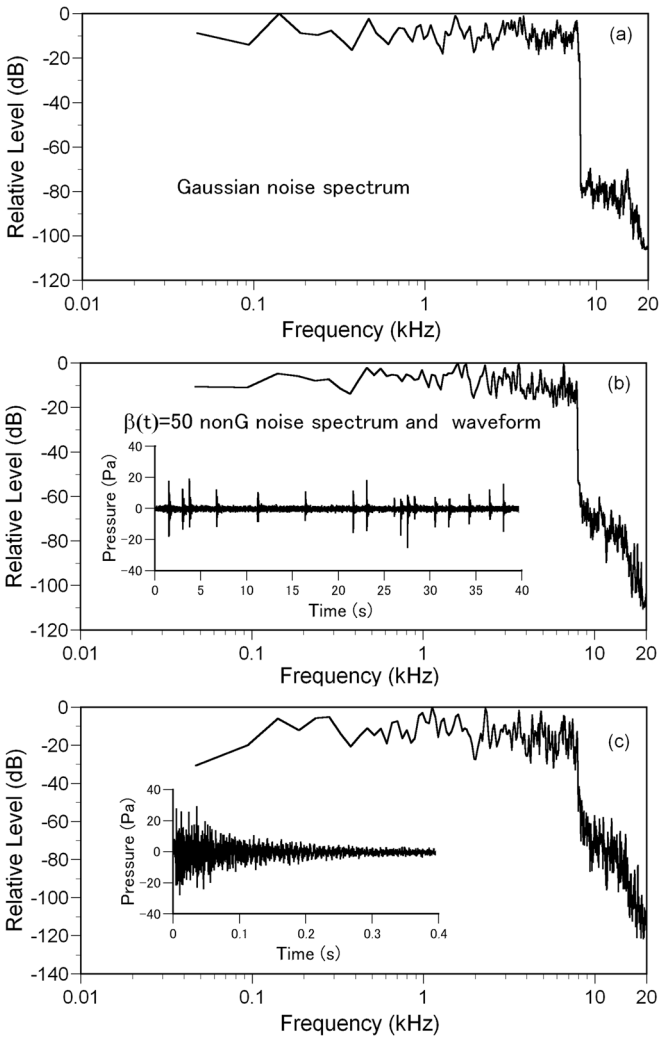

FIG. 3. (a) The relative spectral level of the 97 dB SPL Gaussian noise. (b) The relative spectral level of the 97 dB SPL nonG, $\beta(t) = 50$, noise. The insert shows a representative 40 s sample of the nonG waveform. Both spectra were computed over a 5.5 min sample of the digitized waveform. (c) The relative spectral level of a representative impact noise transient along with the temporal waveform. All the synthesized impacts had similar spectra. The impacts were designed using an exponential decay function. The duration of the impact was taken as the time from the initial peak to the 20 dB down point.

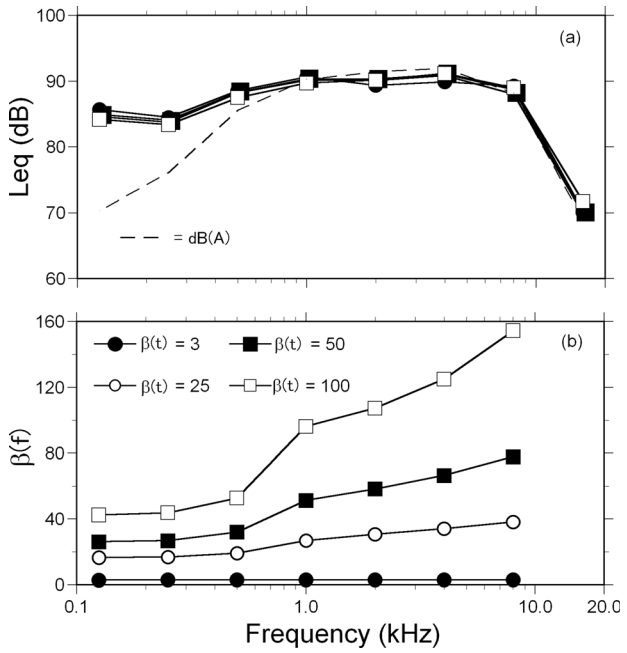

FIG. 4. (a) The octave band Leq of the four $\beta(t)$ classes of noise used in the exposures. The dotted line represents the A weighted Leq. The data points were computed over a 5.5 min sample of the digitized waveform. (b) The frequency specific kurtosis, $\beta(f)$, computed on consecutive octave bands of the filtered noise signal for each of the four classes of noise. Each data point represents a value of $\beta(f)$ computed on consecutive 40 s bins of the filtered 5.5 min waveform and then averaged.

constant $\beta(t)$ set of exposures, the frequency specific profile of PTS and sensory cell loss is similar. Also evident is the increase in trauma as $\beta(t)$ increases.

An ANOVA analysis of the $\beta(t) = 25$ set of exposures (Fig. 5) indicated that for PTS there was no main effect of group ($F = 1.57$, $df = 7$, $p = 0.166$) but there was an interaction between group and frequency ($F = 1.97$, $df = 35$, $p = 0.0015$). For IHC and OHC loss there was no main effect of group [IHC: ($F = 0.430$, $df = 7$, $p = 0.873$); OHC: ($F = 0.820$, $df = 7$, $p = 0.575$)] and no interaction between group and frequency.

For the $\beta(t) = 50$ set of exposures (Fig. 6) the ANOVA analysis of PTS showed no main effect of group ($F = 2.11$, $df = 7$, $p = 0.058$). The group effect, however, approached significance as a result of the statistically significant (Tukey test) reduced PTS measured in group $I_2P_1D_2$ [solid squares in Fig. 6(a)]. At the 0.05 level there was statistically less PTS in group $I_2P_1D_2$ than in groups $I_1P_1D_1$, $I_1P_1D_2$, $I_1P_2D_1$ and $I_1P_2D_2$.There was also an interaction between group and frequency ($F = 2.025$, $df = 35$, $p = 0.001$). For IHC and

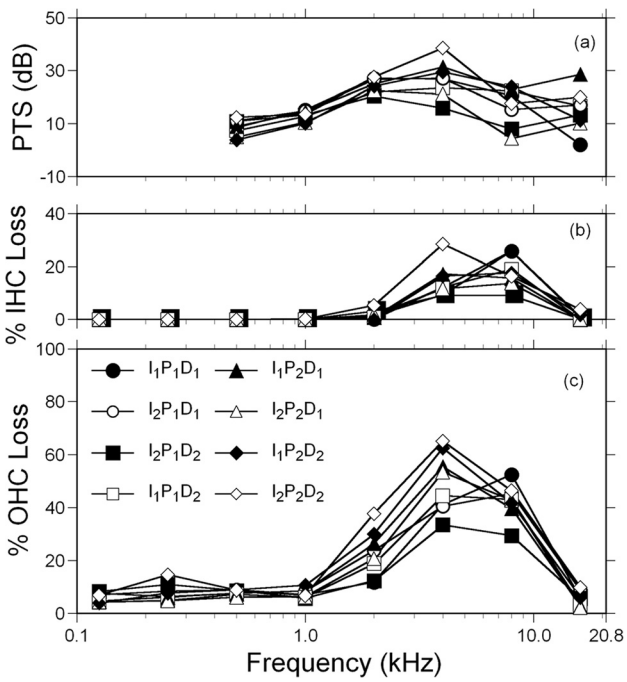

FIG. 5. Group mean (a) permanent threshold shift (PTS), (b) percent inner hair cell loss (%IHC), and (c) percent outer hair cell loss (%OHC) for the eight groups of animals exposed to the interrupted $\beta(t) = 25$ nonG noise at 97 dB SPL for 19 d. Symbols refer to the different combinations of the impact noise interval (I), peak (P), and duration (D) histograms used to create the nonG noise (Table I). The exposure for each group differed only in the I, P, and D histograms used to create the nonG noise.

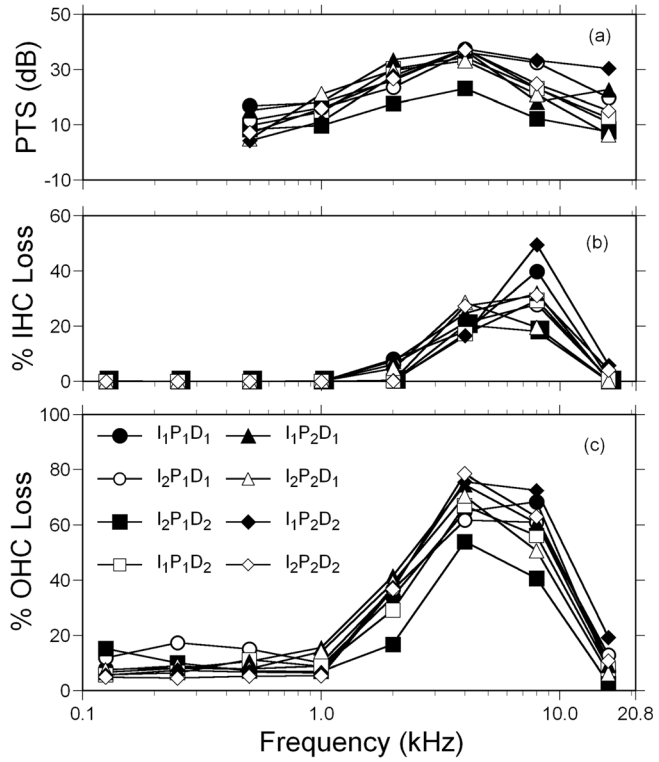

FIG. 6. Group mean (a) permanent threshold shift (PTS), (b) percent inner hair cell loss (%IHC) and (c) percent outer hair cell loss (%OHC) for the eight groups of animals exposed to the interrupted $\beta(t) = 50$ nonG noise at 97 dB SPL for 19 d. Symbols refer to the different combinations of the impact noise interval (I), peak (P) and duration (D) histograms used to create the nonG noise (Table I). The exposure for each group differed only in the I, P, and D histograms used to create the nonG noise.

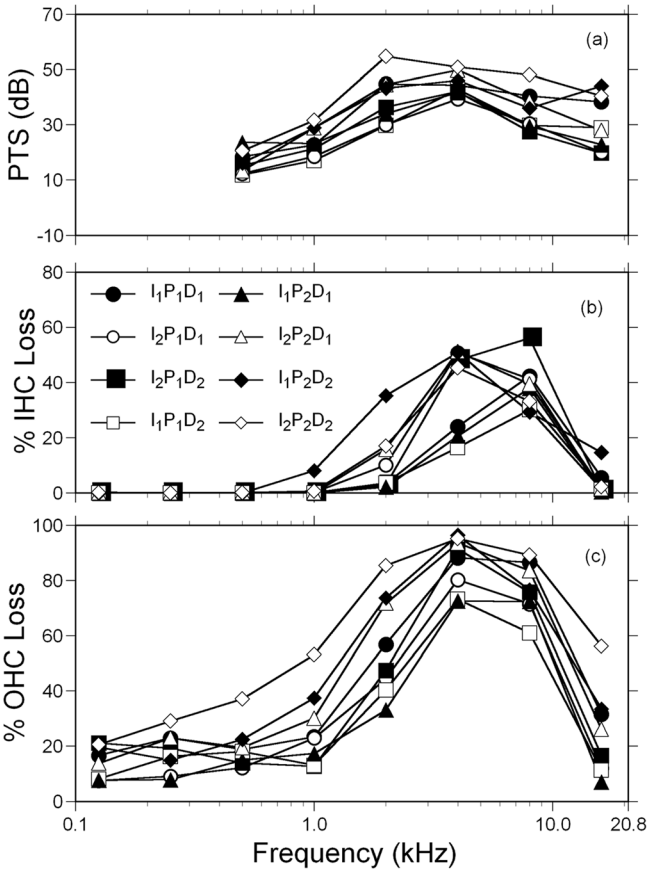

FIG. 7. Group mean (a) permanent threshold shift (PTS), (b) percent inner hair cell loss (%IHC), and (c) percent outer hair cell loss (%OHC) for the eight groups of animals exposed to the interrupted $\beta(t) = 100$ nonG noise at 97 dB SPL for 19 d. Symbols refer to the different combinations of the impact noise interval (I), peak (P), and duration (D) histograms used to create the nonG noise (Table I). The exposure for each group differed only in the I, P, and D histograms used to create the nonG noise.

OHC loss, there were no statistically significant differences among the groups [IHC: ($F = 0.504$, $df = 7$, $p = 0.827$); OHC: ($F = 0.971$, $df = 7$, $p = 0.462$)], and there were no significant group by frequency interactions.

For the $\beta(t) = 100$ set of exposures (Fig. 7) there was a significant main effect of group for PTS ($F = 2.987$, $df = 7$, $p = 0.01$) and an interaction between group and frequency. For IHC loss there was no statistically significant difference among the groups ($F = 1.687$, $df = 7$, $p = 0.1313$) but there was a significant group effect for the OHC loss ($F = 3.459$, $df = 7$, $p = 0.0039$). For both the IHCs and OHCs there was a significant group by frequency interaction. Based on the results of the Tukey test, group $I_2P_2D_2$ was an outlier (open diamond symbols) that affected the statistical results. Repeating the ANOVA without the $I_2P_2D_2$ group resulted in there not being any statistically significant main effect of group for any dependent variable [PTS: ($F = 2.014$, $df = 6$, $p = 0.0821$); OHC: ($F = 1.984$, $df = 6$, $p = 0.0865$); IHC: ($F = 2.025$, $df = 6$, $p = 0.0804$)]. An interaction between group and frequency was found for PTS and IHC loss but not for OHC loss.

Based on the above analysis, it appears that for the same spectral energy and fixed value of kurtosis, complex noise-induced trauma, as quantified by PTS and sensory cell loss, is reasonably independent of the detailed temporal structure of a nonG noise as defined by the I, P, and D histograms.

D. What is the effect of the kurtosis on noise-induced trauma?

The mean PTS, IHC loss, and OHC loss across all the groups exposed to the 19 day complex noise at $\beta(t) = 25$ ($n = 62$), $\beta(t) = 50$ ($n = 63$), and $\beta(t) = 100$ ($n = 63$) is shown in Fig. 8 along with the $\beta(t) = 3$ ($n = 6$), 19 day reference group for comparison. An ANOVA was performed on the three nonG data sets. The $\beta(t) = 3$ was not included in the analysis because of the small sample size compared to the nonG groups. For all measures of noise-induced trauma there was a significant effect of group [PTS: ($F = 36.55$, $df = 2$, $p < 0.0001$); IHC: ($F = 21.94$, $df = 2$, $p < 0.0001$); OHC: ($F = 50.29$, $df = 2$, $p < 0.0001$)] and an interaction between group and frequency.

Data from the 19 d $\beta(t) = 3$, G exposure shown in Fig. 8 has been replotted and compared to the three higher $\beta(t)$ groups having the same temporal structure ($I_1P_1D_1$) and similar sample sizes ($n = 8$) in Fig. 9. The results of the ANOVA were similar to the overall mean data presented in Fig. 8 in showing that there was a significant main effect of group for all dependent variables [PTS: ($F = 17.00$, $df = 3$, $p < 0.0001$); IHC: ($F = 4.877$, $df = 3$, $p < 0.0080$); OHC: ($F = 27.00$, $df = 3$, $p < 0.0001$)] and a group by frequency

interaction. Clearly, for the same exposure energy, PTS and sensory cell loss increase with increasing $\beta(t)$.

The relation between the loss of sensory cells within an octave band length of the basilar membrane and the kurtosis metric computed on that octave band was estimated using the values of $\beta(f)$ shown in Fig. 4(b). For a constant Leq [Fig. 4(a)] between approximately 1.0 and 8.0 kHz, Fig. 4(b) shows that $\beta(f)$ steadily increases as the frequency band over which it is computed increases with a sharp increase for the $\beta(t) = 100$ exposures. Figure 10 shows the percent OHC and IHC loss in the 1.0, 2.0, 4.0, and 8.0 kHz octave bands from the 19 day exposures averaged over all eight groups within a fixed $\beta(t)$ exposure set for the $\beta(t) = 25$, 50, and 100 plotted as a function of $\beta(f)$. Also included is the cell loss data for the 19 day G exposure condition. Despite the relatively constant Leq between 1.0 and 8.0 kHz for all exposures, the lesion increases slowly in the 1.0 kHz octave band region as $\beta(f)$ increases and more sharply in the higher octave band regions with a maximum $\beta(f)$ related loss in the 4.0 kHz octave band. The relation between $\beta(f)$ and OHC loss reflects the profile of OHC loss seen in Figs. 5–7 with a peak loss in the 4 kHz octave band followed by progressively smaller losses at and above 8 and below 2 kHz.

Figure 11 shows a set of PTS, OHC and IHC data from the groups exposed for 5 uninterrupted days that parallel the 19 day data shown in Fig. 8. The G and nonG noise exposure energy and spectrum were the same as for the 19 day exposures and the data for the three nonG exposures at $\beta(t) = 25$, 50, and 100 were from the exposure groups that used the $I_1P_1D_1$ histograms. The results of the 5 day exposures were qualitatively similar to the 19 day exposures, i.e., trauma increased with increasing $\beta(t)$. There was a significant main effect of group for PTS, IHC loss and OHC loss [PTS: ($F = 5.919$, $df = 3$, $p = 0.003$); IHC: ($F = 9.798$, $df = 3$, $p = 0.0002$); OHC: ($F = 13.00$, $df = 3$, $p = < 0.0001$)]. For PTS, there was no interaction of group and frequency, but there was for IHC and OHC loss.

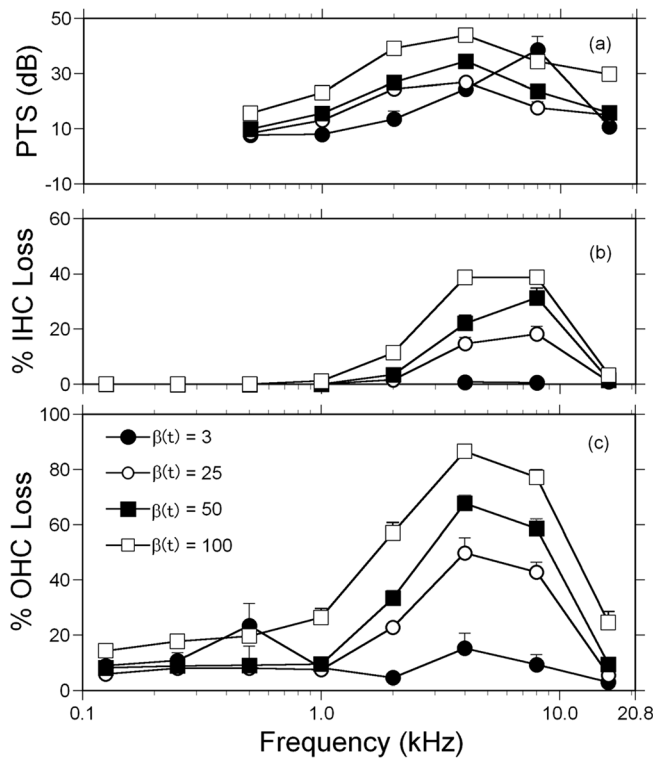

FIG. 8. The averaged (a) permanent threshold shift (PTS), (b) percent inner hair cell loss (%IHC), and (c) percent outer hair cell loss (%OHC) of all groups exposed to the $\beta(t) = 25$ ($n = 62$), 50 ($n = 63$), or 100 ($n = 63$) nonG noise at 97 dB SPL for 19 d regardless of the temporal structure of the noise exposure. The mean data ($n = 6$) from the group exposed to the equivalent energy 19 d Gaussian [$\beta(t) = 3$] noise is shown in each panel for comparison.

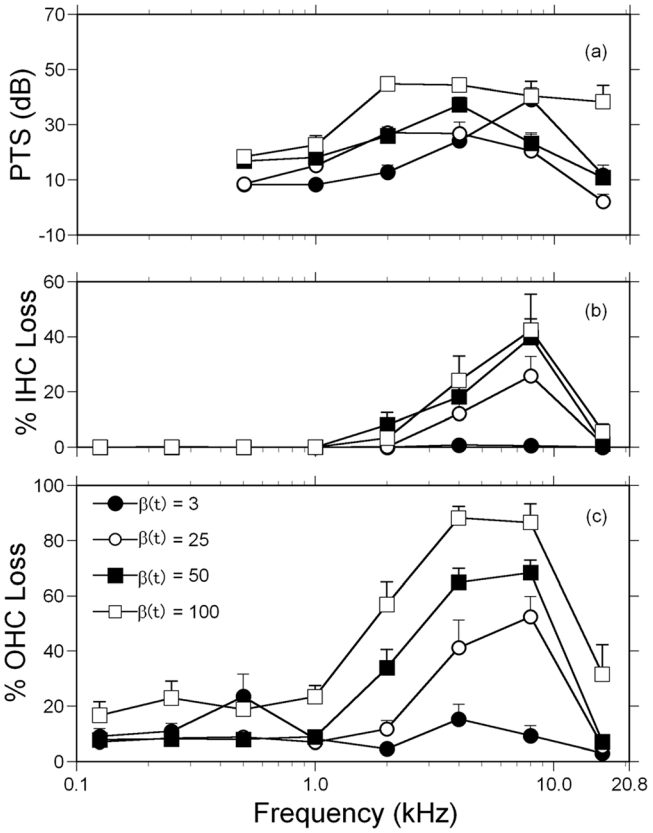

FIG. 9. The group mean (a) permanent threshold shift (PTS), (b) percent inner hair cell loss (%IHC), and (c) percent outer hair cell loss (%OHC) for groups exposed to the $\beta(t) = 25$ ($n = 8$), 50 ($n = 8$), or 100 ($n = 8$) nonG noise at 97 dB SPL for 19 d. All three groups were exposed to the nonG noise having the same $I_1P_1D_1$ temporal structure. The mean data ($n = 6$) from the group exposed to the equivalent energy 19 d Gaussian [$\beta(t) = 3$] noise is shown in each panel for comparison.

The group mean threshold measured daily over the 5 d of the uninterrupted exposures provided an estimate of ATS shown in Fig. 12. Each animal in the 5 day exposure was removed at the same time each day for approximately 20 min for AEP threshold testing. An ANOVA showed a statistically significant effect of group. This effect was caused by the $\beta(t) = 100$ group that showed an approximately 10 dB greater ATS at the lower (<4 kHz) AEP test frequencies. Repeating the ANOVA on the three lower $\beta(t)$ groups showed no significant difference between the groups. A comparison of the data from the three lower $\beta(t)$ groups in Figs. 11 and 12 indicates that while ATS across all tested frequencies was similar, there was a large difference in OHC loss between the G and the two nonG groups [i.e., between the $\beta(t) = 3$ and $\beta(t) = 25$ and 50 groups]. While metrics such as ATS have relatively little significance for workers

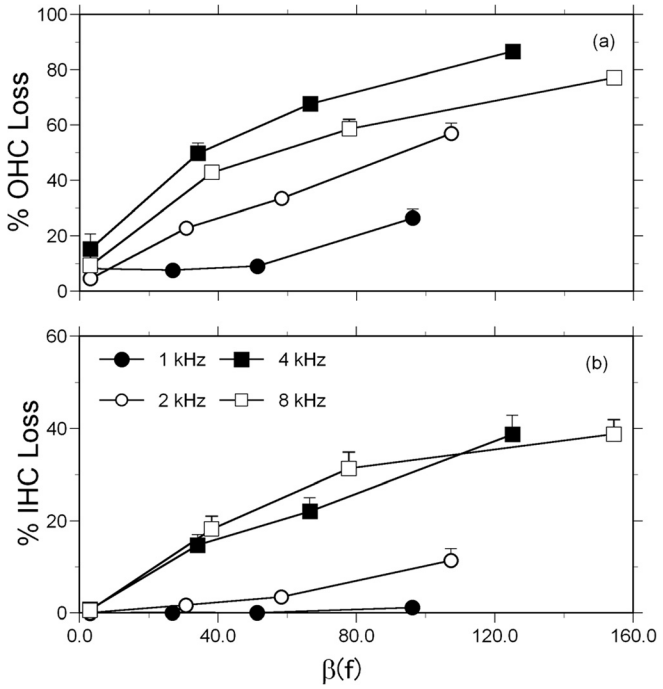

FIG. 10. The mean percent (a) outer hair cells (OHC) and (b) inner hair cells (IHC) lost within the 1.0, 2.0, 4.0, and 8.0 kHz octave band region of the basilar membrane for all animals exposed at a fixed $\beta(t)$ to the 97 dB SPL noise for 19 interrupted days as a function of the frequency specific kurtosis $\beta(f)$ computed on that octave band [see Fig. 4(b)].

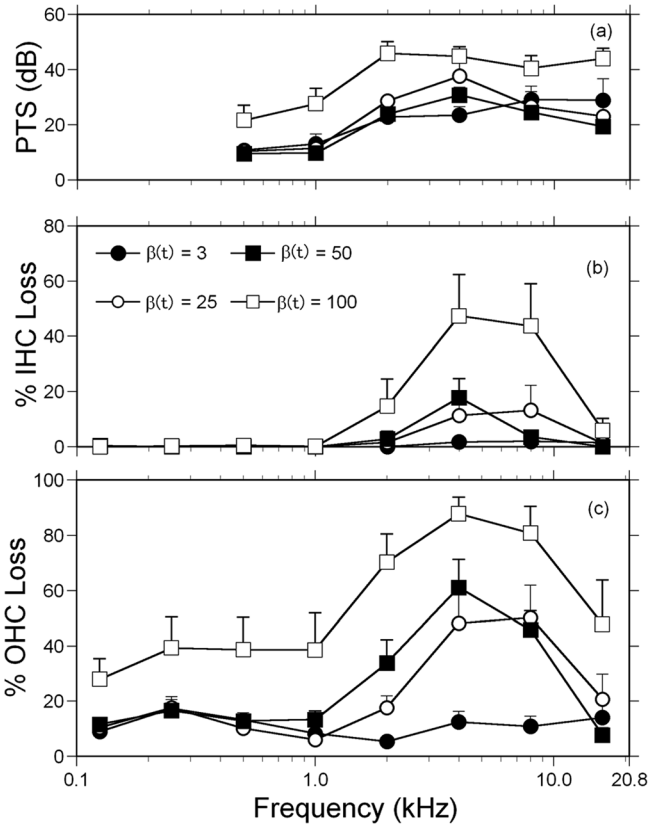

FIG. 11. The group mean (a) permanent threshold shift (PTS), (b) percent inner hair cell loss (%IHC), and (c) percent outer hair cell loss (%OHC) for the groups exposed to the $\beta(t) = 3$, 25, 50, and 100 noise at 97 dB SPL for 5 continuous days. All three nonG groups were exposed to the noise having the same $I_1P_1D_1$ temporal structure.

exposed in the industrial noise environment the above result emphasizes the often-observed lack of correlation between various measures of threshold having a recoverable component (i.e., compound threshold shifts) and permanent threshold shifts.

E. For the same noise energy and exposure parameters what is the effect on noise-induced trauma of interrupting the exposure?

Interrupting a noise exposure for a significant period of time over the course of a chronic exposure should lead to a reduced traumatic effect either as a consequence of the partial or complete recovery of thresholds during the silent periods, or as a result of the toughening effect (Miller *et al.*, 1963; Clark *et al.*, 1987; and others), or under some conditions of exposure, the conditioning effect (Canlon *et al.*, 1988). The above statement can be tested by comparing the results of the 5 day continuous exposures and the 19 day interrupted exposures, i.e., a comparison between pairs of G and nonG groups shown in Figs. 9 and 11 having the same $\beta(t)$, the same $I_1P_1D_1$ histograms and similar group subject numbers. An ANOVA analysis of the PTS and cell loss between pairs of groups having the same $\beta(t)$ produced the following results:

(a) For $\beta(t) = 3$, there was no main effect of group for any of the dependent variables [PTS: ($F = 2.034$, $df = 1$, $p = 0.1793$); IHC: ($F = 1.151$, $df = 1$, $p = 0.2856$); OHC: ($F = 0.094$, $df = 1$, $p = 0.7644$)];

(b) For $\beta(t) = 25$, there was no main effect of group for any of the dependent variables [PTS: ($F = 2.307$, $df = 1$, $p = 0.1511$); IHC: ($F = 0.518$, $df = 1$, $p = 0.4731$); OHC: ($F = 1.106$, $df = 1$, $p = 0.3108$)];

(c) For $\beta(t) = 50$, there was no main effect of group for PTS and OHC loss [PTS: ($F = 1.954$, $df = 1$, $p = 0.1839$); OHC: ($F = 0.024$, $df = 1$, $p = 0.8790$)]. There was, however, a significant group effect for IHC loss ($F = 12.0$, $df = 1$, $p = 0.0033$).

(d) For $\beta(t) = 100$, there was no main effect of group for any of the dependent variables [PTS: ($F = 0.086$, $df = 1$, $p = 0.7738$); IHC: ($F = 1.145$, $df = 1$, $p = 0.3040$); OHC: ($F = 1.407$, $df = 1$, $p = 0.2568$)].

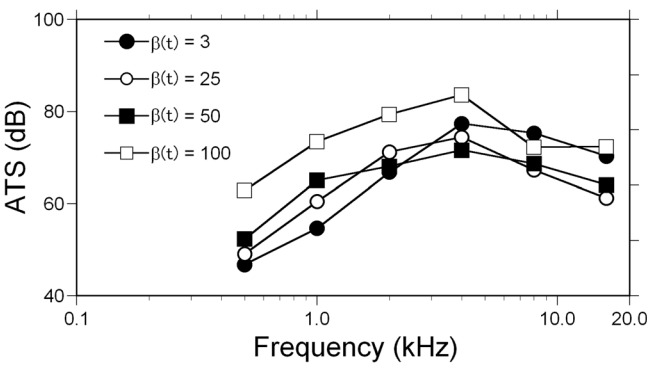

FIG. 12. The group mean asymptotic threshold shift (ATS) for the groups exposed to the $\beta(t) = 3$, 25, 50, or 100 nonG noise at 97 dB SPL for 5 continuous days. All three nonG groups were exposed to the noise having the same $I_1P_1D_1$ temporal structure.

Based on the above results, there were no substantial differences between the continuous and interrupted exposure paradigms.

A slightly different perspective on the sensory cell loss figures presented in Fig. 8 is shown in Fig. 13 where, for all the 5 day and 19 day exposure groups, the mean total loss of OHCs and IHCs over the entire basilar membrane is shown as a function of β(t). There is a systematic increase in sensory cell loss for both sensory cell populations as β(t) increases. Considering the finite number of IHCs and OHCs on the basilar membrane the exponential sigmoid function shown in Fig. 13 was chosen to fit the cell loss data. The exponential curve fits, with high correlation coefficients, provide a rough estimate of the asymptotic limit to the absolute number of cells that will be lost with a continuing exposure. For the OHC loss the asymptotic value for the 5 day groups of 3900 lost cells is greater than the 3090 loss estimate from the 19 day exposures. This difference, however, is likely not significant since the standard error of the 5 day data points is quite large especially for the $\beta(t) = 100$ data point and the sample size very small compared to the 19 day data points. For the IHC loss the asymptotic value for the 5 day and 19 day exposures is 244 and 255 cells lost, respectively. The above results suggest that despite the interrupted exposure paradigm of the 19 day exposures there is not an appreciable difference in trauma between the 5 and 19 day exposures.

The toughening effect shown in Fig. 14, often found using an interrupted exposure paradigm, was estimated for all the 19 day interrupted exposures. In this figure, Tr is shown as a function of the test frequency for the G and nonG groups. The mean data for the nonG groups was obtained

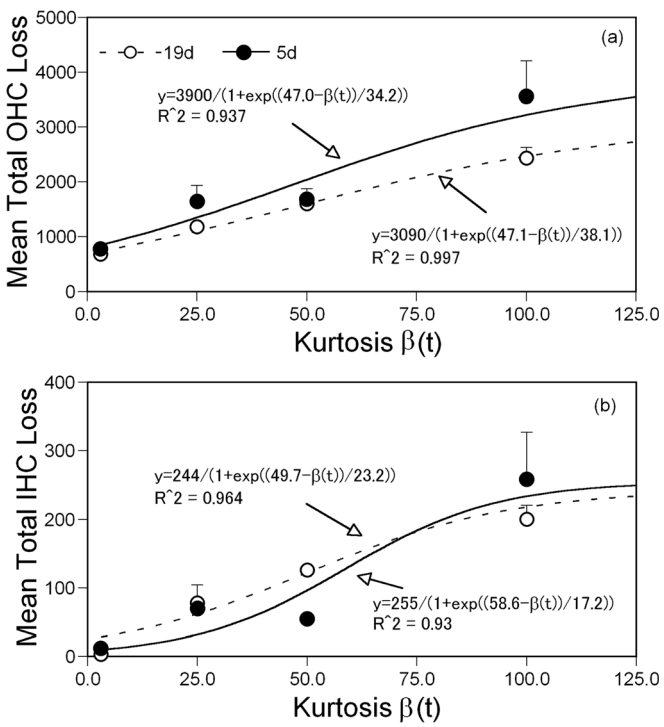

FIG. 13. The mean total number of (a) outer hair cells (OHC) and (b) inner hair cells (IHC) lost for all animals exposed to the 97 dB SPL noise for 19 interrupted days and for 5 continuous days is shown as a function of the kurtosis β(t).

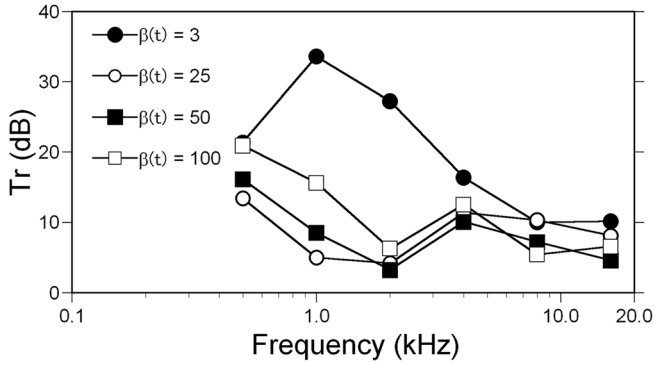

FIG. 14. The mean threshold recovery (Tr) for the groups exposed to the $\beta(t) = 25$, 50, or 100 the interrupted nonG noise at 97 dB SPL for 19 days regardless of the temporal structure of the noise exposure. The mean Tr data ($n = 6$) from the group exposed to the equivalent energy Gaussian [$\beta(t) = 3$] noise is also shown for comparison. Note: Tr is the difference between the threshold measured following the first day (T_1) and the mean of the thresholds measured following the last three days ($T_{17–19}$) of the interrupted exposure [$Tr = (T_1) - (T_{17–19})$].

from all 8 exposure conditions of the $\beta(t) = 25$, 50, and 100 groups with $n > 60$ while the $\beta(t) = 3$ group mean is based on $n = 6$ animals. All the nonG groups showed up to 20 dB toughening effects at the lower (0.5 and 1.0 kHz) test frequencies while the G noise exposed group showed toughening in excess of 30 dB at 1 kHz and 10 to 25 dB at the other test frequencies. Despite these toughening effects, the data presented above show that there were no substantive differences in PTS and cell loss between the interrupted 19 day and non-interrupted, 5 day noise exposed groups.

In summary, Secs. III C, III D, and III E of the results suggest that the equal energy approach to the evaluation of noise exposure hazard has merit but only for noise exposures having the same kurtosis value. Thus both an energy metric and a kurtosis metric are necessary for the evaluation of the potential for a noise to produce hearing loss. A kurtosis metric may add a level of refinement to our ability to predict the consequences to hearing of any noise exposure and can identify frequency specific regions of the cochlea at risk of sensory cell loss.

IV. DISCUSSION

Hearing loss from impact noise exposure is dependent on the rate of presentation, the duration of the impact and the peak level (Henderson *et al.*, 1991; Ward, 1962). The duration of the impact has also been considered in hearing risk assessment (Coles *et al.*, 1967). Laboratory studies such as these and others have the luxury of studying the effect of each parameter of an exposure in isolation. In an industrial setting the situation is far more complex with a variety of manufacturing processes producing not only impact peaks, rates of occurrence and impact durations that vary during the work shift but also a variable background noise level.

An examination of the epidemiological data that was incorporated into ISO 1999 reveals hearing losses from presumably comparable exposures that span more than 70 dB at most audiometric test frequencies making it virtually impossible to estimate with confidence the consequences of a

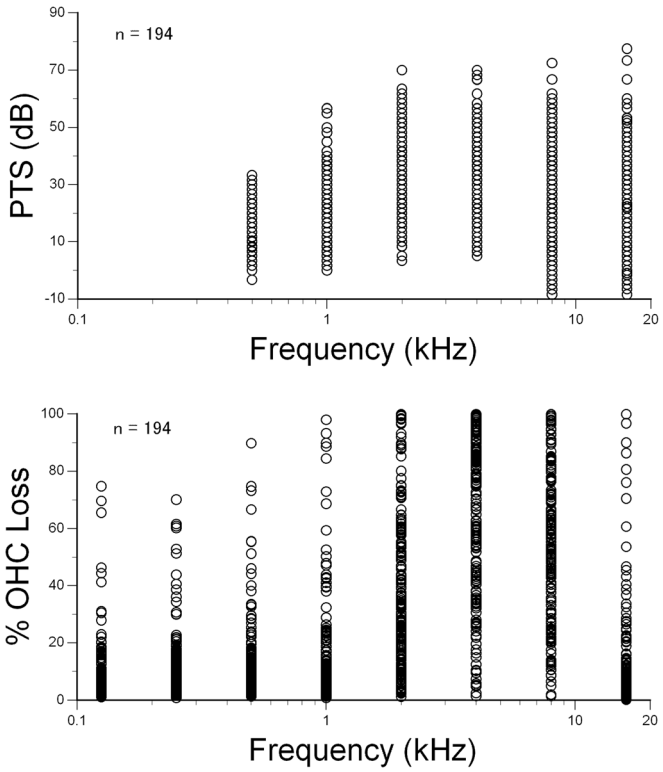

FIG. 15. Scatter plots of the individual animal (n = 194) PTS and OHC loss from all the 19 day exposures.

long-term exposure (Burns and Robinson, 1970; Martin, 1976; Passchier-Vermeer, 1977; Mills *et al.*, 1996). The chinchilla data presented here were derived from realistically modeled and controlled exposure conditions all having the same exposure energy and thus the same potential for producing hearing loss. Figure 15 shows scatterplots of the individual animal (n = 194) PTS and OHC loss from all the 19 day exposures. Although the exposure energy and spectrum were the same for all subjects the variability is large; across the AEP range of test frequencies PTS varied from −10 to over 70 dB, with corresponding OHC losses ranging from 0% to 100%. However organizing the data into sets of fixed β(t) as shown in Figs. 5–7 yields a more tractable data set. It is interesting to compare the spread in the animal data with the human demographic data that is also extremely variable and was used to from the basis of current noise exposure criteria.

Having shown that for a fixed energy and β(t), PTS and cell loss are reasonably independent of the temporal structure of a noise, including a kurtosis metric in the evaluation of an exposure has the advantage of reducing all the temporal variables that quantify or characterize a complex noise into a single numeric metric. Results such as these highlight the potential usefulness of both kurtosis and energy in the evaluation of hazardous noise exposure as opposed to an energy metric alone.

V. CONCLUSIONS

(1) Over a broad range of values of the kurtosis and for the same spectral energy, the data are generally consistent in showing that noise-induced trauma is not appreciably affected by different combinations of the I, P, and D variables that define a complex (nonG) noise. This raises the prospect that a kurtosis metric can be used to grade equivalent energy exposures for their potential to cause trauma.

(2) The data also reinforce previous work showing the following.

   (a) The equal energy hypothesis, that states that equivalent exposure energy will produce equivalent hearing trauma, is not valid for high β(t) exposures. The data, however, do suggest that the equal energy hypothesis is valid provided the exposures have the same spectral energy and value of β(t).
   (b) For noise exposures having the same spectral energy hearing trauma increases with increasing β(t).
   (c) For a nonG noise exposure the kurtosis, β(f), computed on the filtered wave form can identify cochlear regions that will sustain excess OHC loss relative to G exposures.
   (d) The toughening effect, often found following interrupted exposures, does not affect the extent of noise-induced trauma when compared to an uninterrupted noise exposure of the same spectral energy.
   (e) There was no consistent relation between ATS and the subsequent noise-induced trauma in the 5 day uninterrupted noise exposures.

ACKNOWLEDGMENTS

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