Energy-independent factors influencing noise-induced hearing loss in the chinchilla model

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The effects on hearing and the sensory cell population of four continuous, non-Gaussian noise exposures each having an A-weighted $L_{eq} = 100$ dB SPL were compared to the effects of an energy-equivalent Gaussian noise. The non-Gaussian noise conditions were characterized by the statistical metric, kurtosis (β), computed on the unfiltered, $\beta(t)$, and the filtered, $\beta(f)$, time-domain signals. The chinchilla ($n = 58$) was used as the animal model. Hearing thresholds were estimated using auditory-evoked potentials (AEP) recorded from the inferior colliculus and sensory cell populations were obtained from surface preparation histology. Despite equivalent exposure energies, the four non-Gaussian conditions produced considerably greater hearing and sensory cell loss than did the Gaussian condition. The magnitude of this excess trauma produced by the non-Gaussian noise was dependent on the frequency content, but not on the average energy content of the impacts which gave the noise its non-Gaussian character. These results indicate that $\beta(t)$ is an appropriate index of the increased hazard of exposure to non-Gaussian noises and that $\beta(f)$ may be useful in the prediction of the place-specific additional outer hair cell loss produced by non-Gaussian exposures. The results also suggest that energy-based metrics, while necessary for the prediction of noise-induced hearing loss, are not sufficient. *© 2001 Acoustical Society of America.* [DOI: 10.1121/1.1414707]

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I. INTRODUCTION

Contemporary noise standards (e.g., ISO-1999, 1990; ANSI S3.44, 1996) are based on the assumption that an energy metric such as the equivalent noise level (L_{eq}) is sufficient for estimating the potential of a noise stimulus to cause noise-induced hearing loss (NIHL). While the L_{eq} may be an adequate index for estimating the hazard associated with exposure to Gaussian, steady-state noise exposures, such exposures are more typical of controlled laboratory experiments rather than industrial noise environments. The latter are often non-Gaussian, with a sound-pressure level (SPL) that varies over the course of the workday and is punctuated by high-level impulsive components or other type of noise transients. Available data, from laboratory-based experiments (Lei *et al.*, 1994; Dunn *et al.*, 1991), as well as epidemiological studies (Passchier-Vermeer, 1983; Sulkowski *et al.*, 1983; Thiery and Meyer-Bisch, 1988) indicate that while an energy metric may be necessary, it is not sufficient for the prediction of NIHL. Other variables of the noise exposure must be considered. Unfortunately there are a large number of stimulus variables that can be used to characterize a complex noise environment.

Experimental studies such as those of Clark *et al.* (1987) and others have shown the importance of the temporal structure of an exposure on the threshold shift (TS) dynamics following exposures to various interrupted noises. Since temporal variables do not affect an energy metric and since there are an infinite number of very different noise exposures characterized by the same L_{eq}, it seems reasonable that a metric that would incorporate both temporal and level variables might be a useful adjunct to the L_{eq} metric. One such metric is the kurtosis, β, of a sample distribution which is defined as the ratio of the fourth-order central moment to the squared second-order central moment of the distribution. Erdreich (1986) was perhaps the first to suggest the use of kurtosis to identify/characterize impulsive noise environments for application to hearing conservation strategies. This statistic, used to estimate the deviation of a distribution from the Gaussian, can be computed on the unfiltered, $\beta(t)$, and the filtered, $\beta(f)$, time-domain signal. With non-Gaussian signals, $\beta(f)$ can serve as a useful adjunct to conventional spectral analysis (Dwyer, 1984).

The potential of the β metrics for identifying hazardous noise environments was demonstrated by Lei *et al.* (1994). They hypothesized that for the same total energy and spectrum a high-kurtosis noise exposure is more hazardous to hearing than a Gaussian exposure, and that this effect is frequency dependent. Using a very limited set of exposure parameters, $\beta(t)$ and $\beta(f)$ were shown: (i) to rank order the degree of hearing trauma and (ii) to reflect the frequency specificity of trauma.

The data presented in this paper extend the results of Lei *et al.* (1994) by considering more generalized non-Gaussian signals as well as spectral influences.

II. METHODS

Fifty-eight chinchillas were used as subjects. Each animal was made monaural by surgical destruction, under anes-

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thesia, 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 auditory-evoked potentials (AEP). The AEP was used to estimate pure-tone thresholds, and surface preparations of the organ of Corti were used to estimate the inner- and outer hair cell (IHC, OHC) populations. Additional details of the experimental methods, beyond those presented below, may be found in Ahroon *et al.* (1993).

A. Experimental protocol

The animals were randomly assigned to one of five experimental groups with 11 or 12 animals/group. Following a 2-week postsurgical recovery, three AEP preexposure audiograms were obtained (on different days) on each animal at octave intervals between 0.5 and 16.0 kHz. If the mean of the three audiograms, at two or more frequencies, fell beyond one standard deviation of laboratory norms (Hamernik and Qiu, 2000), in the direction of poorer thresholds, the animal was rejected from the group. Two animals out of 60 were rejected because preexposure thresholds did not meet the criterion.

The animals were exposed to one of the noise conditions described below, five or six at a time, 24 h/day, for 5 consecutive days. Animals were given free access to food and water and rotated through the bank of six cages daily. The SPLs, across cages, in the middle of each cage, varied within ±1 dB. During the exposure, animals were removed daily for less than 0.5 h for AEP testing. The mean of the five audiograms thus obtained defined asymptotic threshold shift (ATS). Thirty days following the last exposure day, three more audiograms were collected on different days and the mean used to define permanent threshold shift (PTS). Following audiometric testing the animals were killed under anesthesia. Their right cochlea was removed and prepared for surface preparation histology from which sensory cell populations along the length of the basilar membrane were determined.

B. Noise measurement and analyses

During the 5-day exposures the noise field was monitored with a Larson Davis 814 sound-level meter equipped with a 1/2-in. microphone. The noise was maintained at an L_{eq} of 100 dB(A) SPL. The acoustic signal produced by the Electro-Voice Xi-1152/94 speaker system was transduced by a Brüel and Kjær 1/2-in. microphone (model 4134), amplified by a Brüel & Kjær (model 2610) measuring amplifier and fed to a Windows PC-based analysis system. The design and digital generation of the acoustic signal is detailed in Hsueh and Hamernik (1990, 1991). The signal was sampled at 48 kHz with a recording duration of 5.5 min. $\beta(t)$ was computed over 40 s of the digitized temporal waveform. Similarly, $\beta(f)$ was computed over a 40-s interval on octave bands of the digitally filtered temporal samples. Center frequencies of the octave bands were the same as the audiometric test frequencies. The samples of every window were convolved with the impulse response of the octave bandwidth filter. The filter was designed as an infinite impulse response digital filter in which the coefficients of the impulse response were obtained from the Signal Processing toolbox of MATLAB 5.3 (The Math Works Inc.). The filtering process was performed repeatedly to obtain $\beta(f)$ over successive octave bands.

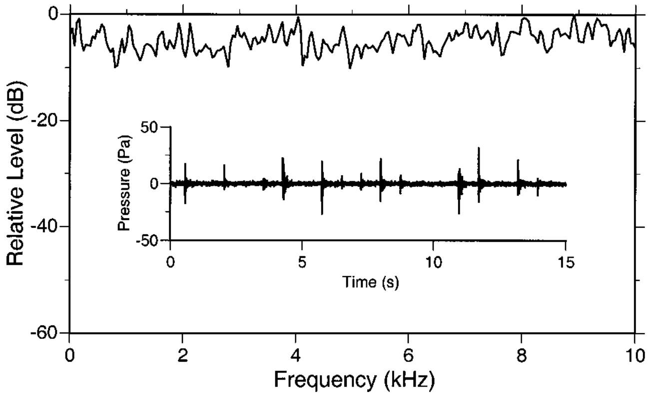

FIG. 1. The average spectrum obtained from a 40-s sample of the digitized waveform. The spectrum was the same for each of the five noise exposures. The inset shows a 15-s sample of the pressure–time waveform of a non-Gaussian exposure. Impact peak SPLs and interimpact intervals were randomly varied.

C. Noise exposures

The animals were exposed for 5 consecutive days (24 h/day) to one of the following five exposure conditions, identified by group number.

G-43	Gaussian noise, $\beta(t)=3$. Reference condition.
G-44	Non-Gaussian noise, $\beta(t)=25$. The impact peak SPLs varied randomly between 115 and 128 dB. The impact was created from three 400-Hz bands of energy centered on 1.0, 2.0, and 4.0 kHz. The level of the background Gaussian noise was kept at 95 dB(A) SPL.
G-49	Non-Gaussian noise, $\beta(t)=33$. The impact peak SPLs varied randomly between 115 and 129 dB. The impact was created from the band of energy between 0.710 and 5.680 kHz. The level of the background Gaussian noise was kept at 92 dB(A) SPL.
G-50	Non-Gaussian noise, $\beta(t)=21$. The impact peak SPLs varied randomly between 114 and 128 dB. The impact was created from a single 400-Hz band of energy centered at 2.0 kHz. The level of the background Gaussian noise was kept at 95 dB(A) SPL.
G-55	Non-Gaussian noise, $\beta(t)=25$. The impact peak SPLs varied randomly between 117 and 129 dB. The impact was created from a broad band of energy between 0.125 and 10.0 kHz. The level of the background Gaussian noise was kept at 95 dB(A) SPL.

Each exposure had in common the same flat spectrum between 0.125 and 10.0 kHz shown in Fig. 1 and was presented at an $L_{eq}=100\,\text{dB(A)}$. The five exposures differed only in their temporal structure, which was designed to produce one Gaussian and four different non-Gaussian exposure

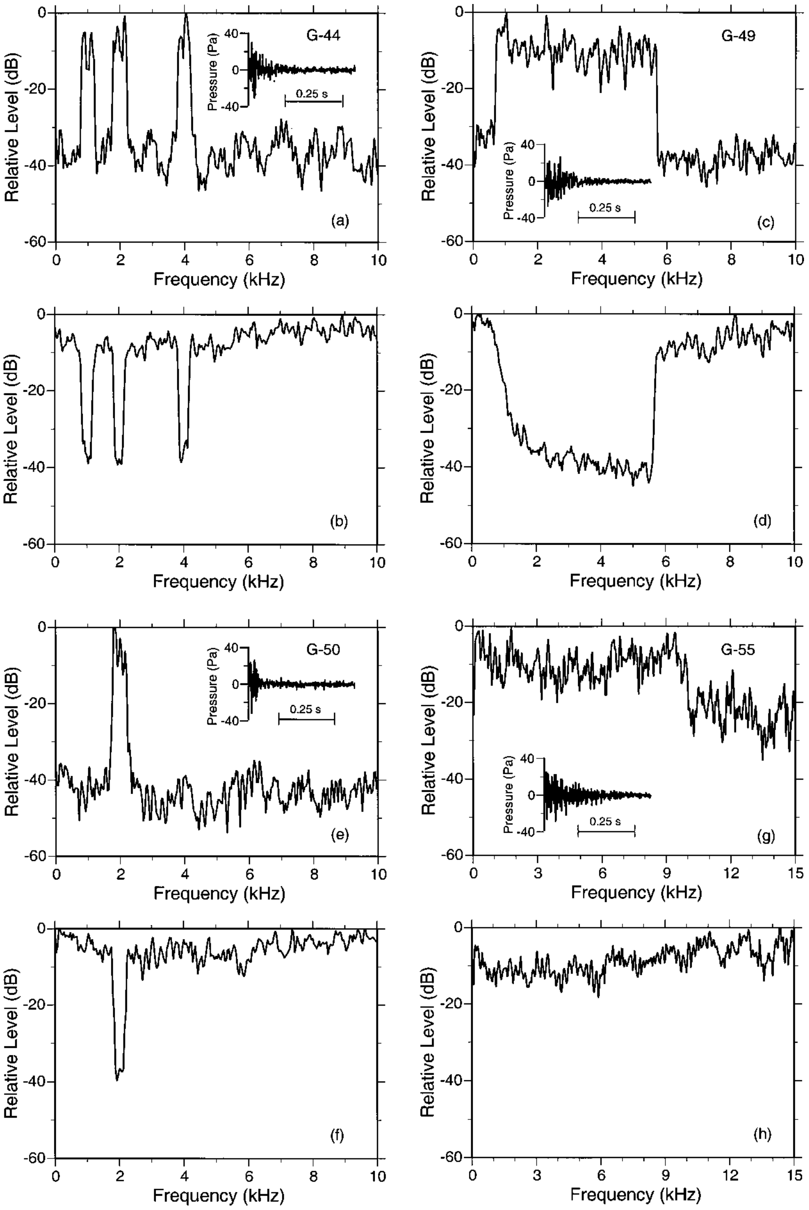

FIG. 2. The upper panels (a), (c), (e), and (g) show the spectrum of the impact that was used to create the character of each of the indicated non-Gaussian noise exposures. The inset shows a typical impact waveform. The lower panels (b), (d), (f), and (h) show the respective spectra of the Gaussian noises that were mixed with the impact stimuli.

conditions, three of which (G-44, G-50, and G-55) had similar values of $\beta(t)$. The non-Gaussian conditions were designed in the frequency domain as described by Hsueh and Hamernik (1990, 1991) and were the result of inserting impacts, whose spectra were complementary to the background Gaussian noise, into the otherwise Gaussian signal. The impact peak levels were randomly varied between the limits indicated above and the probability of an impact occurring in a 750-ms window was set at 0.6. The inset in Fig. 1 shows a 15-s sample of the non-Gaussian waveform. Variation in $\beta(f)$ was achieved by varying the spectrum of the impacts.

Figures 2(a)–(h) illustrate the waveform and spectrum of each of the non-Gaussian noises. Each of the top panels in Fig. 2 shows the spectrum of the impacts along with a pressure–time waveform of one of the impacts. The lower panels in Fig. 2 show the spectrum of the continuous background Gaussian noise that was combined with the impacts to produce the non-Gaussian signals. The Gaussian component of the non-Gaussian exposure had a spectrum that was complementary to that of the impact and a level (L_b) that was dependent on the value of $\beta(t)$. Generally, if the impact peak and interval histograms were kept approximately the same, $\beta(t)$ could be increased by decreasing L_b.

For exposure conditions G-44, -50, and -55, not only was the overall L_{eq} the same but the L_{eq} of the impact component (i.e., the L_{eq} of the exposure with the Gaussian noise component filtered out) of these exposures was also the same [i.e., $L_{eq} = 98$ dB(A)]. Table I gives the 1/3-octave band levels of each noise exposure. Values shown are the mean values obtained from eight 40-s samples of the digitized waveform.

III. RESULTS

In the following figures data points are given with standard error (se) bars. When no error bar is present the se is less than the size of the datum symbol. The mean preexposure AEP audiogram for all 58 animals along with individual group means are shown in Fig. 3, where they are compared

TABLE I. Mean total and third-octave-band sound-pressure levels (dB SPL) over 5-day exposure period for all experimental groups.

Third-octave band cf (kHz)	G-43	G-44	G-49	G-50	G-55
0.25	84	74	78	81	87
0.32	84	76	79	81	89
0.40	84	76	78	81	88
0.50	84	76	79	81	89
0.63	85	76	81	82	89
0.80	84	86	90	80	88
1.00	85	91	91	82	89
1.25	84	85	89	79	86
1.60	83	82	89	87	85
2.00	83	94	89	97	84
2.50	82	84	90	85	83
3.15	85	84	91	82	87
4.00	86	94	91	83	86
5.00	88	84	92	85	88
6.30	91	88	85	89	90
8.00	94	90	88	92	92
10.00	96	92	89	93	93
12.50	95	92	90	91	90
16.00	94	91	89	91	90
Mean L_{eq}	103	101	102	101	102
Mean L_{eqA}	100	100	101	100	100
s.d.	0.04	0.26	0.65	0.23	0.77

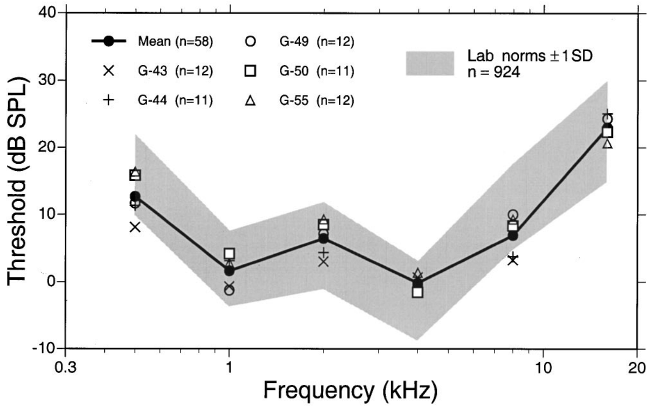

FIG. 3. The mean AEP audiograms for the entire group of animals and for the five individual groups compared to the laboratory norms based on a population of 924 animals.

to laboratory norms. The overall se was very small and the five group means at all test frequencies were within ±4 dB of each other. Figures 4(a)–(d) show the PTS and the percent OHC loss for the four non-Gaussian exposure conditions compared to the Gaussian G-43 noise exposure. It is clear from this figure that for the two indices of trauma, PTS and OHC loss, the four non-Gaussian exposure conditions (G-44, G-49, G-50, and G-55) produced more severe permanent changes in the auditory system than did the Gaussian exposure. In the most extreme cases (G-49 and G-55) there is as much as 40 to 50 dB more PTS and 8 to 9 times the mean OHC loss at the most affected frequencies than in the Gaussian exposure, despite the same L_{eq} and spectrum in each of the exposures. For clarity, IHC losses are not shown. IHC losses were, as is usually the case in NIHL, much less than

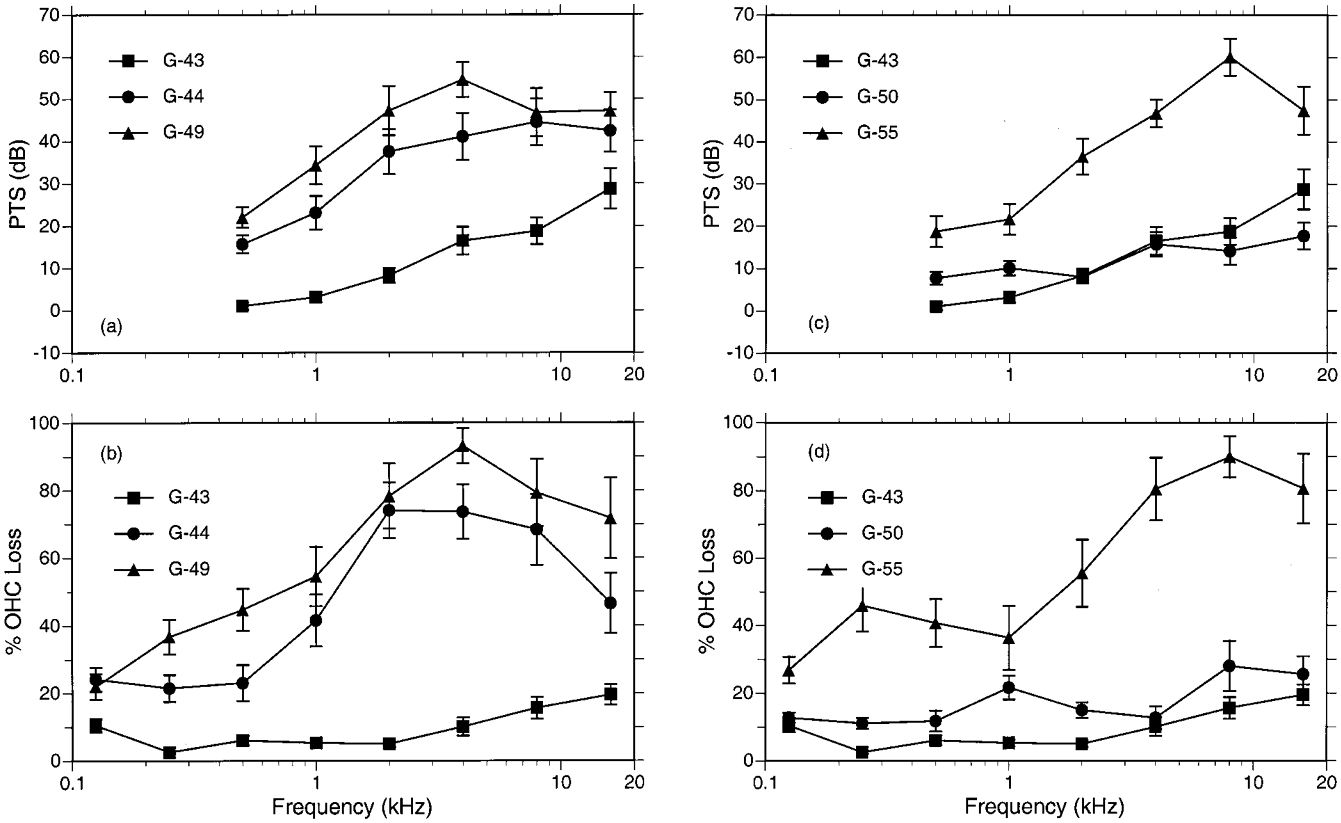

FIG. 4. Each of the upper panels (a) and (c) compares the mean PTS measured in the four groups exposed to the indicated non-Gaussian noise with the group exposed to the energy-equivalent Gaussian noise. The lower panels (b) and (d) show the corresponding mean percent OHC loss computed over adjacent octave band lengths of the basilar membrane in these groups.

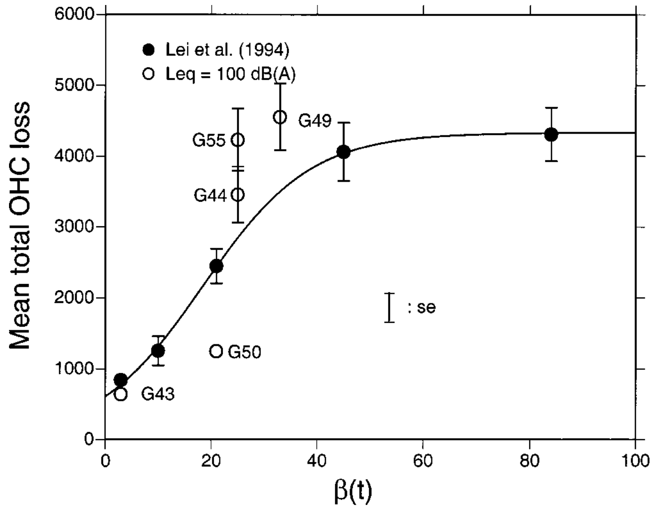

FIG. 5. The group mean total OHC loss in each of the five experimental groups as a function of $\beta(t)$ compared to similar data taken from Lei *et al.* (1994).

OHC losses for each group, but were greater in each of the non-Gaussian exposures than in the Gaussian.

A slightly different perspective on these data is shown in Fig. 5, where the total group mean OHC loss is plotted as a function of $\beta(t)$ and compared to the results of Lei *et al.* (1994). Seen in this figure, for similar values of $\beta(t)$, is the steady increase in sensory cell loss in groups G-50, G-44, and G-55 as the spectrum of the impact transient in the non-Gaussian noise is widened. The OHC loss for the G-50 exposure, having only 400-Hz bandwidth transients, approaches the loss seen in the $\beta(t)=3$, Gaussian condition, but is significantly larger (t-test, $\alpha=0.05$). Despite the different bandwidths of the impacts the total energy of only the transients in the G-44, G-50, and G-55 non-Gaussian conditions was approximately the same $[\mathrm{L}_{\mathrm{eq}}=98\,\mathrm{dB(A)}]$ but the cell loss increased nevertheless.

In order to increase $\beta(t)$ for exposure group G-49, L_b was reduced to 92 dB(A). The other exposure variables were similar to those of group G-55. Although the increase in $\beta(t)$ was modest $[\beta(t)=33$ vs $\beta(t)=25]$, there was a small increase in the mean OHC loss consistent with the trends in the Lei *et al.* (1994) data. This difference was, however, not statistically significant.

The relation between the OHC loss in consecutive octave bands along the basilar membrane and the variable $\beta(f)$ computed on the filtered octave band noise signal is shown in Fig. 6. In this figure the percent OHC loss data represents the difference in the percent loss, in consecutive octave band lengths of the basilar membrane, between the indicated non-Gaussian group and the Gaussian group. There is a clear suggestion that, for those exposures that did produce substantial sensory cell loss, the profile of OHC loss difference and $\beta(f)$ are somewhat congruent, with the cell loss profile shifted about an octave to the high frequencies relative to the $\beta(f)$ profile. This is in agreement with the results of Lei *et al.* (1994). For group G-50 there is comparatively little difference in octave band percent OHC loss and it is scattered along the length of the basilar membrane. This profile of cell loss difference bears no resemblance to $\beta(f)$.

IV. DISCUSSION

The equal energy hypothesis (EEH) had its origins in the retrospective studies of populations exposed to industrial noise (Burns and Robinson, 1970; Robinson, 1976). Originally applied to steady-state noise exposures, the EEH was extended to encompass industrial impact noise by Martin (1976). The EEH now forms the foundation of the current international noise exposure standard (ISO-1999, 1990). A number of studies both experimental and demographic (see, e.g., Lei *et al.*, 1994) indicate that the EEH is not an adequate predictor of NIHL. The results of Lei *et al.* showed that in addition to an energy metric, the kurtosis of an exposure stimulus which is a statistical metric incorporating both the temporal and amplitude characteristics of the noise needs to be considered in the prediction of the hazards of an exposure. The data presented in this paper are an extension of the results of Lei *et al.* (1994) and are in agreement with their results. These data show that for non-Gaussian noises the kurtosis of the amplitude distribution computed on the filtered and unfiltered temporal signal provides information on

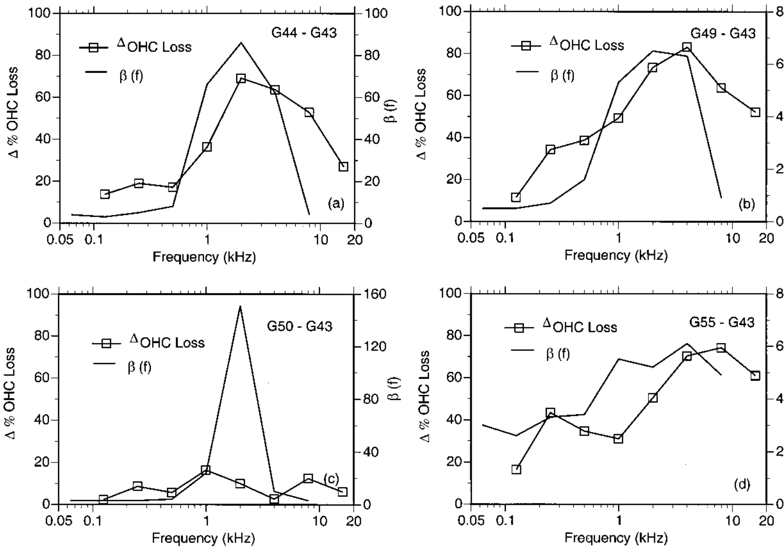

FIG. 6. The difference between the percent OHC loss produced by the indicated non-Gaussian and Gaussian exposure conditions computed over octave band lengths of the basilar membrane is shown and compared with the frequency specific kurtosis, $\beta(f)$, profile.

hazardous noise exposures that cannot be obtained from the L_{eq} metric. The exposures used by Lei *et al.* had an overall $L_{eq} = 100$ dB; those in the present study had an $L_{eq} = 100$ dB(A), a relatively minor difference which is emphasized by the same OHC loss following exposure to the two reference $\beta(t) = 3$ conditions shown in Fig. 5. The other eight data points in this figure represent the results of exposures that all had approximately the same L_{eq}. Each of these exposures produced more severe OHC loss than the Gaussian noise conditions. Clearly L_{eq} is not sufficient for the estimation of the hazard of exposure to this limited set of non-Gaussian noise exposures.

These data also address the proposition of Martin (1976) and others who advocate the application of an L_{eq} metric for the evaluation of impact noise. Consider in Fig. 5 the four data points associated with the $20 < \beta(t) < 25$ exposures. The L_{eq} of just the transients in each of these exposures is roughly the same [97–98 dB(A)]. The transients in three of the exposures (G-50, G-44, and G-55) are impacts, while in the Lei *et al.* (1994) data point they are broadband Gaussian noise bursts. The three impact-containing exposures differ appreciably only in the frequency content of the impacts; nevertheless, each of these exposures produces a different level of OHC loss which systematically increases as the bandwidth of the impacts increase. Similarly, the exposure G-55 and the Lei *et al.* (1994), $\beta(t) = 21$ exposure differ only in the nature of the transient, i.e., impulses in the former and noise bursts in the latter. In both cases, while the OHC loss is greater than in the Gaussian reference condition, there is a significant difference between the two non-Gaussian data points with the impact containing stimulus causing much greater OHC loss.

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