The kurtosis metric as an adjunct to energy in the prediction of trauma from continuous, nonGaussian noise exposures

Wei Qiu, Roger P. Hamernik, and Bob Davis

Citation: The Journal of the Acoustical Society of America **120**, 3901 (2006); doi: 10.1121/1.2372455
View online: https://doi.org/10.1121/1.2372455
View Table of Contents: http://asa.scitation.org/toc/jas/120/6
Published by the Acoustical Society of America

The kurtosis metric as an adjunct to energy in the prediction of trauma from continuous, nonGaussian noise exposures

Wei Qiu,[a)] Roger P. Hamernik, and Bob Davis
Auditory Research Laboratory, State University of New York, 107 Beaumont Hall, Plattsburgh, New York 12901

(Received 16 June 2006; revised 21 September 2006; accepted 26 September 2006)

Data from an earlier study [Hamernik *et al.* (2003). J. Acoust. Soc. Am. **114**, 386–395] were consistent in showing that, for equivalent energy [L_{eq}=100 dB(A)] and spectra, exposure to a continuous, nonGaussian (nonG) noise could produce substantially greater hearing and sensory cell loss in the chinchilla model than a Gaussian (G) noise exposure and that the statistical metric, kurtosis, computed on the amplitude distribution of the noise could order the extent of the trauma. This paper extends these results to L_{eq}=90 and 110 dB(A), and to nonG noises that are generated using broadband noise bursts, and band limited impacts within a continuous G background noise. Data from nine new experimental groups with 11 or 12 chinchillas/group is presented. Evoked response audiometry established hearing thresholds and surface preparation histology quantified sensory cell loss. At the lowest level [L_{eq}=90 dB(A)] there were no differences in the trauma produced by G and nonG exposures. For L_{eq}>90 dB(A) nonG exposures produced increased trauma relative to equivalent G exposures. Removing energy from the impacts by limiting their bandwidth reduced trauma. The use of noise bursts to produce the nonG noise instead of impacts also reduced the amount of trauma.
[DOI: 10.1121/1.2372455]

PACS number(s): 43.66.Ed, 43.50.Pn [BLM] Pages: 3901–3906

I. INTRODUCTION

High-level nonGaussian (nonG) noise exposures are very common in industrial and military environments and clearly pose a hazard to hearing for large numbers of the exposed population. Over the past several decades a number of published papers have shown, in an animal model, that exposure to nonG noise produces more hearing and sensory cell loss than does an equivalent energy Gaussian (G) exposure (e.g., Dunn *et al.*, 1991; Lei *et al.*, 1994; Lataye and Campo, 1996; Hamernik and Qiu, 2001; Harding and Bohne, 2004). These results along with similar findings from human epidemiologic data (Sulkowski and Lipowczan, 1982; Taylor *et al.*, 1984; Thiery and Meyer-Bisch, 1988) challenge the use of the equal energy hypothesis (EEH) that forms the basis of current criteria for human exposure to noise (e.g., ISO 1999, 1990). One consequence of categorizing a temporally diverse and complex set of exposures with a single metric such as energy may be the appearance of large variability in the hearing threshold levels that is typically found in epidemiologic studies. This large variability (Mills *et al.*, 1996) precludes meaningful comparisons across exposure conditions as well as estimates of individual risk of hearing loss.

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 Leq, it seems reasonable that a metric that would incorporate both temporal and level variables might be a useful adjunct to the Leq metric. One such metric is the kurtosis of a sample distribution that is defined as the ratio of the fourth-order central moment to the squared second-order moment of the amplitude distribution. This statistic, used to estimate the deviation of a distribution from the Gaussian, can be computed on the unfiltered and the filtered time-domain signal. All the variables that characterize a nonG noise such as transient peaks, intertransient intervals, transient durations, crest factor, etc., have an effect on the kurtosis. While the effect on kurtosis of any one of these variables can be predicted, the effect of them all acting in unison is less predictable.

Several years ago data were presented from a number of different exposures (Hamernik *et al.*, 2003) that were consistent with the above referenced work in showing that, for equivalent energy [L_{eq}=100 dB(A)] and spectra, exposure to a continuous, nonG noise could produce substantially greater hearing and sensory cell loss in the chinchilla model than a G noise exposure and that the statistical metric, kurtosis [$\beta(t)$], computed on the amplitude distribution of the noise, could order the extent of the trauma. This presentation extends these results to L_{eq}=90 and 110 dB(A) and to nonG noises that are generated using broadband noise bursts, and band-limited impacts. A metric based on some combination of kurtosis and energy may be useful in the assessment of industrial noise environments for hearing conservation purposes.

II. METHODS

The experimental design and methods were identical to those used in the Hamernik *et al.* (2003) paper. One hundred and six chinchillas, divided into nine experimental groups, were used as subjects. Briefly, each animal was made mon-

a)author to whom correspondence should be addressed; electronic mail: wei.qiu@plattsburgh.edu

TABLE I. Summary of exposure conditions

L_{eq} dB(A)	$\beta(t)$	Noise type[a]	Group No.	N	Peak range dB SPL	Impulse probability	L_b-background dB(A)
90	3	(1)	47	12	⋯	⋯	⋯
90	32	(2)	48	11	[104, 118]	0.6	84
90	35	(3)	56	12	[105, 120]	0.6	83.5
100	21	(4)	50	11	[114, 128]	0.6	95.5
100	27	(5)	70(*)	12	[105, 115](**)	0.6	91
100	33	(3)	77	12	[115, 129]	0.22–0.35	91.5
100	45	(5)	82	12	[106, 117](**)	0.6	92
110	3	(1)	42	12	⋯	⋯	⋯
110	20	(2)	45	12	[129, 134]	0.6	104
110	27	(3)	78	12	[132, 137]	0.6	104

[a]Noise type: (1) Broadband Gaussian. (2) NonGaussian noise, with a Gaussian background component defined by L_b. The transients which provide the nonGaussian character of the noise were impacts created from three 400 Hz bands of energy centered at 1, 2, and 4 kHz. (3) Same as in (2) except the transients were broadband (710–5680 kHz). (4) Same as in (2) except the transients were narrow band (1800–2200 Hz). (5) For these exposures the transients were broadband (710–5680 Hz) noise bursts.

(*)Data of group 70 is taken from Hamernik *et al.* (2003).

(**)Levels represent the rms SPL of the noise burst.

aural by the surgical destruction, under anesthesia, 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.

A. Experimental protocol

The animals were randomly assigned to one of nine experimental groups with 11 or 12 animals/group. Following a two-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 more than one test frequency, fell beyond one standard deviation of laboratory norms (Hamernik and Qiu, 2001) in the direction of poorer thresholds the animal was rejected.

The animals, confined to individual cages ($10\times 11 \times 16$ in.) with free access to food and water, were exposed three or four at a time to one of the noise conditions summarized in Table I. Exposures lasted 24 h/day for five days and were interrupted once daily for approximately 20–30 min for AEP testing. The five-day exposures produced an asymptotic threshold shift. Thirty days following the last exposure day, three more audiograms were collected on different days and the mean used to define the postexposure threshold from which permanent threshold shift (PTS) was obtained.

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 12–24 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 and IHC and 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 or as the group mean total number of IHCs or OHCs missing.

C. Noise measurement and analyses

The design and digital generation of the acoustic signal is detailed in Hsueh and Hamernik (1990, 1991) and Hamernik *et al.* (2003). The noise was created using an Electro-Voice Xi-1152/94 speaker and amplifiers (Model P1200 and P2000). During exposure the sound level of noise field was monitored with a Larson Davis 814 sound level meter equipped with a 1/2 in. microphone. The sound field was recorded using a Bruel and Kjaer 1/2 in. microphone (Model 4134), amplified by a Bruel and Kjaer (Model 2610) measuring amplifier and digitalized by an analog-to-digital/digital-to-analog converter (Model PCI-6221, National Instrument Inc.). The signal was sampled at 48 kHz in 16 bits with a recording duration of 5.5 min. Several segments of the signal were recorded at each cage and saved on a hard disk for off line analysis. The sound preasure level (SPL) and spectral data on both impact and background noise were obtained from these recordings using programs developed using MATLAB. The SPLs, across cages, in the middle of each cage, varied within less than ±1 dB.

D. Noise exposures

Each exposure had in common approximately the same broadband (0.125–20 kHz) spectrum that was reasonably

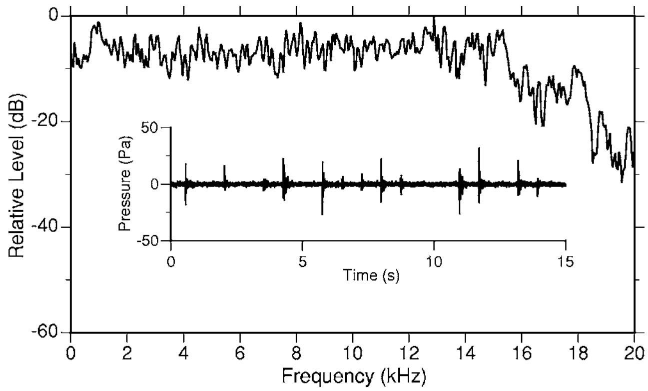

FIG. 1. The average spectrum of a 40 s sample of a nonG noise incorporating broadband impacts. This long-term spectrum was common to all the exposures. The inset shows a 15 s sample of one of the nonG wave forms.

flat between 0.125 and 10.0 kHz as shown in Fig. 1. It was this flat portion of the spectrum that was manipulated to produce the various types of nonG noises. The noise was presented at an $L_{\mathrm{eq}}=90$, 100, or 110 dB(A) SPL. The nonG noises were created by inserting either impacts or noise bursts into the continuous G noise. The impact or noise burst transients were created from several different regions of the energy spectrum. The probability of a transient occurring in a 750 ms window was set at 0.6 for each exposure except for experimental group 77 where it was randomly varied between 0.225 and 0.35. For group 50, impact transients were created from the energy in a single 400 Hz wide band centered at 2 kHz; for groups 45 and 48 the energy in the impacts was derived from three, 400 Hz wide bands centered at 1, 2, and 4 kHz and for groups 56, 70, 78, and 82 the energy for either the impacts or the noise bursts was derived from the 710–5680 Hz region of the spectrum, i.e., broadband transients. The spectrum of the continuous G background noise (Fig. 2) consisted of the frequencies that were not used to produce the transients. The background noise level given as L_b in Table I was measured from digital recordings of the noise signal between impacts. The overall L_{eq} of the exposure was adjusted in an iterative fashion by adjusting the impact variables and the L_b to obtain the required L_{eq}.

III. RESULTS AND DISCUSSION

Summary data for the groups defined in Table I are shown as the symbols in Fig. 3 where they are identified by group numbers. Panel (a) shows the group mean total number of OHCs lost while panel (b) presents the group mean PTS averaged over the 2, 4, and 8 kHz ($\mathrm{PTS}_{2,4,8}$) test frequencies as a function of the kurtosis, $\beta(t)$. Bars on the data points represent the standard error (s.e.). If a bar is not present the s.e. was less than the size of the symbol. For reference purposes, the total number of OHCs in the chinchilla cochlea is approximately 7300 (Bohne *et al.*, 1982 and Hamernik *et al.*, 1988). The two solid lines numbered (ii) and (iii) were taken from the Hamernik *et al.*, (2003) paper and represent the best fit of the function shown to the data that were obtained from a series of exposures extending from $\beta(t)=3$ to $\beta(t)>100$ at an $L_{\mathrm{eq}}=100$ dB(A). Broadband impacts were used in the nonG exposures that produced curve

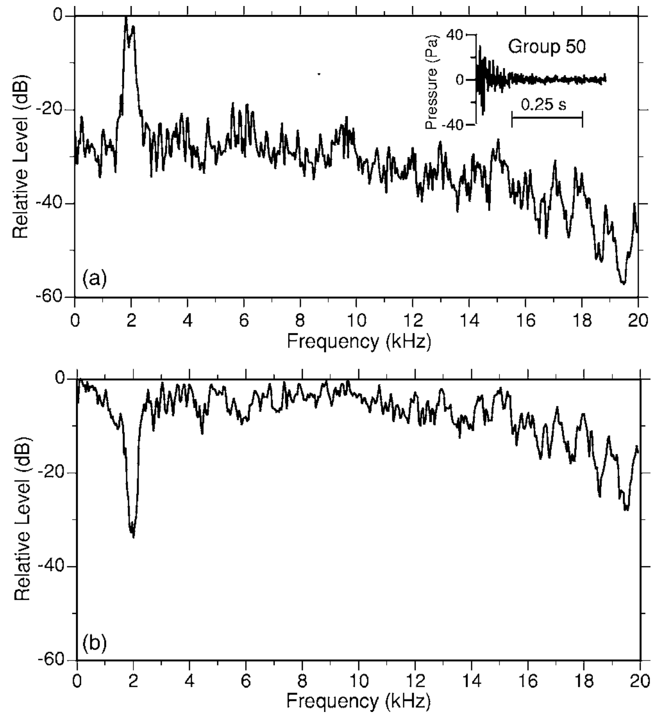

FIG. 2. The upper panel (a) shows the spectrum and wave form of the narrow band (1.8–2.2 kHz) impulse transient used in the exposure of group 50. The peak SPL was randomly varied between 114 and 128 dB and the probability of an impact occurring in a 750 ms window was set at 0.6. The lower panel (b) shows the complementary spectrum of the G background noise that was mixed with the impacts to create the nonG continuous noise.

(ii) while the impacts used in the nonG exposures that produced curve (iii) were created from the energy in three 400 Hz wide bands centered at 1, 2, and 4 kHz. These two pairs of curves show that the nonG exposures produced more OHC loss and PTS than did the energy and spectrally equivalent G noise, i.e., the exposures at a $\beta(t)=3$. For the nonG exposure containing broadband impacts [curve (ii)] the upper limit of OHC loss and PTS is 4515 missing OHCs and 49 dB PTS, respectively, while for the equivalent G exposure there are ~660 missing OHCs and a PTS of about 14 dB. IHC losses are not presented since they are much smaller than OHC losses and typically much less susceptible to noise damage than are the OHCs. IHC losses, however, did follow the trends presented for the OHCs.

Curve (iii) shows that as energy is removed from the impact and incorporated into the G background noise the degree of trauma is reduced considerably. The number of OHCs lost at the asymptotic value of this function drops to about 3000 and the PTS to about 33 dB, but this is still greater than for the $\beta(t)=3$, G exposure. This result could be anticipated since in the limit as the impact energy approaches zero the exposure approaches the Gaussian condition $\beta(t)=3$. These curves are used as a reference for the additional exposure conditions reported here. The same function will be used to relate the higher and lower level exposures [curves (i) and (iv) in Fig. 3] to this earlier data set.

The effect of increasing the L_{eq} to 110 dB(A) (groups 42 and 78) and fitting the same function as used for curve (ii) through the pair of data points (42 and 78) shifts the curves, as expected, upward. The G exposure (group 42) produced

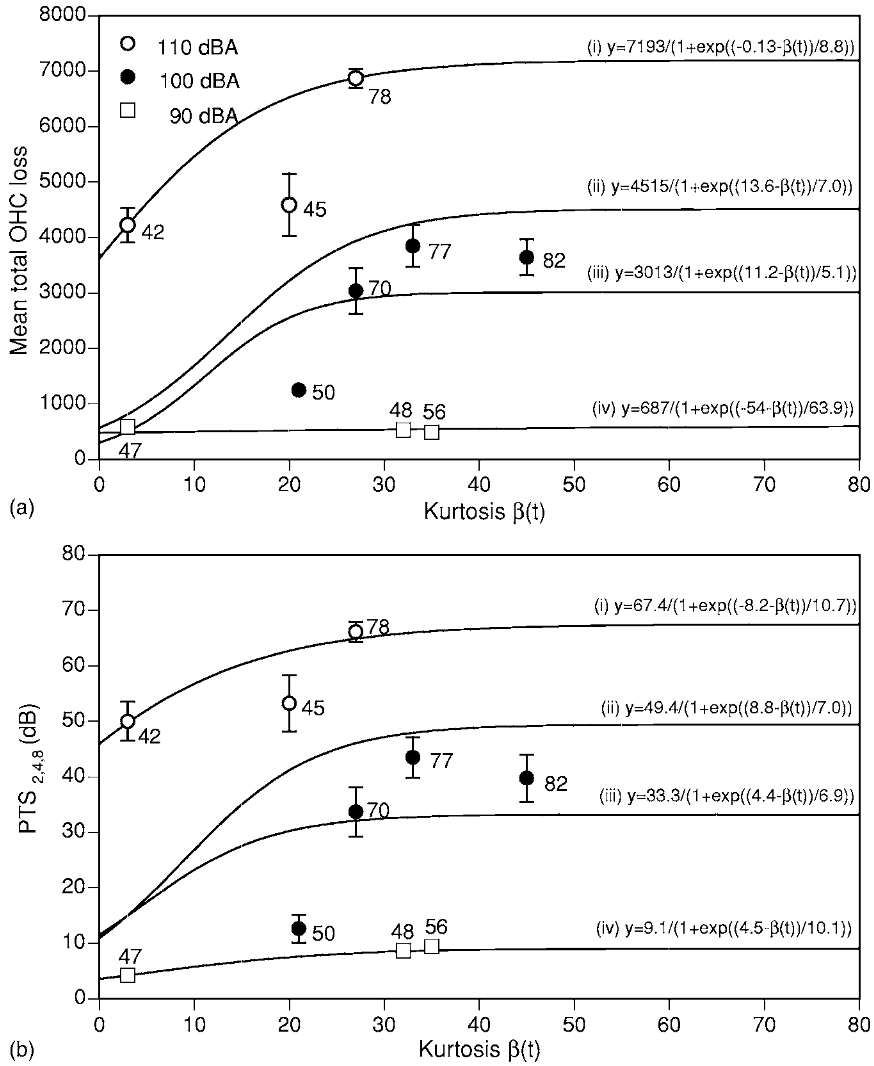

FIG. 3. (a) The group mean total outer hair cell (OHC) loss for the ten groups of animals exposed to the equal energy and spectrum Gaussian $[\beta(t)=3]$ and nonGaussian $[\beta(t)>3]$ noises. (b) The group mean permanent threshold shift averaged over the 2.0, 4.0 and 8.0 kHz AEP test frequencies ($PTS_{2,4,8}$). Numbers adjacent to data symbols identify the experimental groups outlined in Table I. Curves (ii) and (iii) represent the curve fit to data taken from Hamernik *et al.* (2003).

4230 missing OHCs and a $PTS_{2,4,8}=50$ dB while the nonG exposure (group 78) containing broadband impacts produced 6875 missing OHCs and a $PTS_{2,4,8}=66$ dB. Thus, at the higher L_{eq} the nonG exposure still produced considerably more trauma than did the equivalent G exposure. Removing energy from the impact by limiting its bandwidth and increasing the energy in the G component by a corresponding amount would be expected to cause a lessening of trauma. This is seen in the results from group 45. At an L_{eq} $=110$ dB(A) the total cell loss and mean PTS for group 45, an exposure that used the three, 400 Hz narrow bands of energy centered at 1, 2, and 4 kHz to produce the impacts, was statistically similar to the G exposure, condition 42. However, it is instructive to look at the frequency specific data when comparing the nonG and G groups 45 and 42, respectively.

Figure 4 compares the PTS and percent OHC loss for groups 42, 45, and 78 exposed to the 110 dB(A) noises. There are large differences in cell loss and PTS at the lower frequencies (0.5, 1.0, and 2.0 kHz) between groups 42 and 45 that are not reflected in the summary data shown in Fig. 3. Focusing on the high frequency mean $PTS_{2,4,8}$ and the group mean total OHC loss obscures the increased low frequency trauma produced by introducing nonG components into the exposure. The increased low frequency OHC loss in group 45 is reflected in the 12–15 dB increase in PTS at 0.5 and 1.0 kHz over the PTS produced by the G exposure (group 42). There is a group mean loss of 2031 OHCs in the 0.5 and 1.0 kHz octave band lengths of the basilar membrane following the nonG exposure of group 45 compared to 1164 missing as a result of the G exposure (group 42). Thus, the nonG exposure, incorporating the bandlimited impacts, also produced greater trauma than the equivalent G exposure but less than the group 78 that had more energy in the impacts. The series of curves in Fig. 4 clearly illustrate the increased trauma associated with high $\beta(t)$ exposures at 110 dB as well as the progression of the trauma toward the lower audiometric test frequencies as a greater fraction of the total energy is put into the impacts. At this level for the broadband impact condition a ceiling effect is beginning to appear. There is 100% OHC loss over a large part of the cochlea.

Group 50 was exposed to a nonG noise with $\beta(t)=21$ at 100 dB(A) in which the impacts were produced from a single 400 Hz wide band of the spectrum centered at 2 kHz. Using the points on curves (ii) and (iii) in Fig. 3 at the corresponding $\beta(t)=21$ as a reference it is clear that removing additional energy from the impacts, as in this one-band

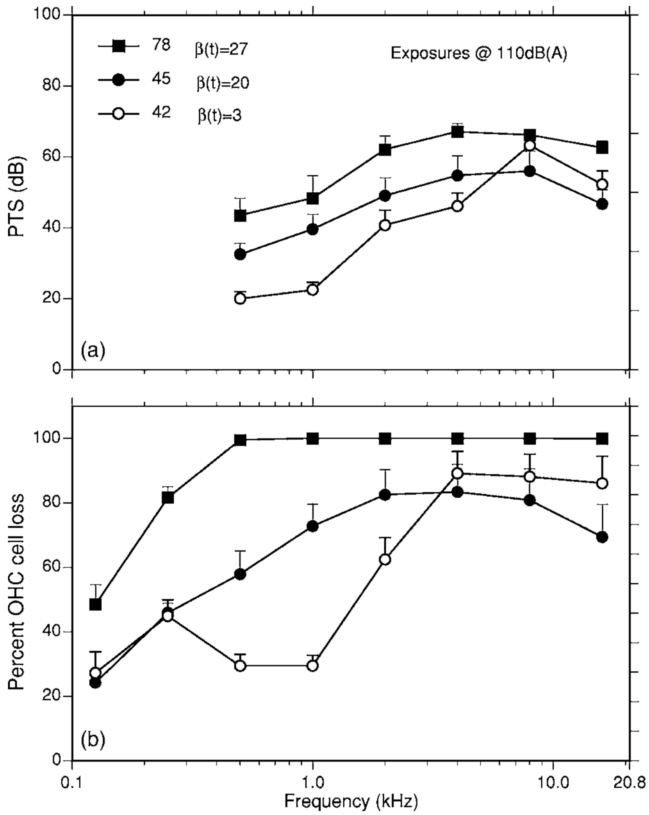

FIG. 4. (a) The frequency specific group mean permanent threshold shift (PTS) and (b) the group mean percent outer hair cell loss (OHC) for groups 42, 45, and 78 exposed to the 110 dB(A) Gaussian (42) and nonGaussian (45 and 78) noises. OHC data points represent average losses taken over octave band lengths of the basilar membrane at the indicated center frequencies.

exposure condition, causes a still further reduction in the trauma despite the presence of impact peaks that varied between 114 and 128 dB peak SPL. The impacts used in the exposures that produced curves (ii) and (iii) had approximately the same range of peak SPLs as those used in the exposure of group 50. The lessening of trauma from lower energy impacts having the same peaks suggests that the energy in the impact is more important in the production of hearing loss than is the impact peak SPL. Finally, it should be noted that despite the lower energy in the impacts the cell loss but not the $PTS_{2,4,8}$ for group 50 is statistically greater than the equivalent G exposure [i.e., the $\beta(t)=3$ point on curves (ii) and (iii) in Figs 3(a) and 3(b)]. The group mean total number of OHCs lost was 1250. This loss for group 50 was distributed across the entire basilar membrane and varied from 10% to 15% loss at the lower frequencies to 25% to 30% loss at 8 kHz and above. As with the OHC loss the PTS was evenly distributed across the test frequencies varying between 10 and 15 dB across the 0.5 through 16 kHz range.

The probability (P) of a transient occurring in a 750 ms window of the noise was set at 0.6 for all nonG exposures except group 77. For group 77, P was randomly varied such that $0.22 < P < 0.35$. The reduction of P resulted in less sensory cell loss and $PTS_{2,4,8}$ than for the $P=0.6$ conditions; curve (iii). This reduction in trauma agrees with a similar reduction reported in the Hamernik *et al.* (2003) paper when $P=0.1$ was used for several nonG exposures. Using broadband (710–5680 Hz) noise bursts instead of impacts to produce a nonG exposure condition as in groups 70 and 82 also had the effect of reducing the level of trauma at both values of $\beta(t)$ but the exposure still produced more trauma than the $\beta(t)=3$ exposure. The rms level of the noise bursts varied between 105 and 117 dB.

Groups 47, 48, and 56 received an $L_{eq}=90$ dB(A) exposure. At this Leq, both nonG groups (48 and 56), regardless of the bandwidth of the transients, produced the same degree of trauma as the G noise exposure group 47 (Fig. 3). While exposure conditions for the $L_{eq}=90$ dB are limited, the available data suggest that for exposures that produce relatively small amounts of PTS and sensory cell loss the EEH holds regardless of the value of the kurtosis. A similar conclusion was reached by Hamernik *et al.* (1981) using lower levels of nonG noise and a different experimental paradigm.

IV. CONCLUSIONS

The results presented above complement and extend our earlier results (Hamernik *et al.*, 2003) in showing that for noise exposures that have the potential for producing hearing loss a high kurtosis exposure will exacerbate the loss. Under such conditions the EEH does not apply. Temporally complex noise exposures having equivalent energy and spectrum if characterized by only an L_{eq} can result in hearing loss data showing extreme variability across exposed subjects. The kurtosis metric, which incorporates a number of variables that have in the past been examined individually such as impact peaks, inter-impact intervals, impact duration and crest factor can, however, separate exposed subjects into similarly exposed groups in a quantitative way and reduce variability. The kurtosis metric and the L_{eq} coupled with an examination of the transients in a complex exposure in terms of their range of peaks and spectrum may prove useful in the development of occupational noise exposure criteria for the protection of hearing.

ACKNOWLEDGMENTS

This work was supported by Grant No. 1-R01-OH02317 from the National Institute for Occupational Safety and Health. The technical assistance of George A. Turrentine, Ann Johnson, and Adam Bouchard is greatly appreciated. In conducting this research 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].

Bohne, B., Kenworthy, A., and Carr, C. D. (**1982**). "Density of myelinated nerve fibers in the chinchilla cochlea," J. Acoust. Soc. Am. **72**, 102–107.
Dunn, D. E., Davis, R. R., Merry, C. J., and Franks, J. R. (**1991**). "Hearing loss in the chinchilla from impact and continuous noise exposure," J. Acoust. Soc. Am. **90**, 1979–1985.
Eldredge, D. H., Miller, J. D., and Bohne, B. A. (**1981**). "A frequency-position map for the chinchilla cochlea," J. Acoust. Soc. Am. **69**, 1091–1095.
Hamernik, R. P., Henderson, D., and Salvi, R. (**1981**). "Potential for interaction of low-level impulse and continuous noise," AFAMRL-TR-80-68,

Air Force Aerospace Medical Research Laboratory, Wright-Patterson Air Force Base, Ohio 45433.

Hamernik, R. P., Patterson, J. H., Turrentine, G. A., and Ahroon, W. A. (**1988**). “The quantitative relation between sensory cell loss and hearing thresholds,” Hear. Res. **38**, 199–212.

Hamernik, R. P., and Qiu, W. (**2001**). “Energy-independent factors influencing noise-induced hearing loss in the chinchilla model,” J. Acoust. Soc. Am. **110**, 3163–3168.

Hamernik, R. P., Qiu, W., and Davis, B. (**2003**). “The effects of the amplitude distribution of equal energy exposures on noise-induced hearing loss: The kurtosis metric,” J. Acoust. Soc. Am. **114**, 386–395.

Harding, G. W., and Bohne, B. A. (**2004**). “Noise-induced hair-cell loss and total exposure energy: Analysis of a large data set,” J. Acoust. Soc. Am. **115**, 2207–2220.

Hsueh, K. D., and Hamernik, R. P. (**1990**). “A generalized approach to random noise synthesis: Theory and computer simulation,” J. Acoust. Soc. Am. **87**, 1207–1217.

Hsueh, K. D., and Hamernik, R. P. (**1991**). “Performance characteristics of a phase domain approach to random noise synthesis,” Noise Control Eng. J. **36**, 18–32.

International Standard ISO 1999 (**1990**). “Acoustics-determination of occupational noise exposure and estimation of noise-induced hearing impairment,” Technical Comm. ISO/TC43-Acoustics, International Organization for Standardization, Geneva, Switzerland.

Lei, S-F., Ahroon, W. A., and Hamernik, R. P. (**1994**). “The application of frequency and time domain kurtosis to the assessment of hazardous noise exposures,” J. Acoust. Soc. Am. **96**, 1435–1444.

Lataye, R., and Campo, P. (**1996**). “Applicability of the L_{eq} as a damage-risk criterion: An animal experiment,” J. Acoust. Soc. Am. **99**, 1621–1632.

Mills, J. H., Lee, F. S., Dubno, J. R., and Boettcher, F. A. (**1996**). “Interactions between age-related and noise-induced hearing loss,” in *Scientific Basis of Noise-Induced Hearing Loss*, edited by A. Axelsson, H. Borchgrevink, R. P. Hamernik, P. A. Hellstrom, D. Henderson, and R. J. Salvi (Thieme, New York), pp. 193–121.

Sulkowski, W. J., and Lipowczan, A. (**1982**). “Impulse noise-induced hearing loss in drop forge operators and the energy concept,” Noise Control Eng. **18**, 24–29.

Taylor, W., Lempert, B., Pelmear, P., Hemstock, I., and Kershaw, J. (**1984**). “Noise levels and hearing threshold in the drop forging industry,” J. Acoust. Soc. Am. **76**, 807–819.

Thiery, L., and Meyer-Bisch, C. (**1988**). “Hearing loss due to partly impulsive industrial noise exposure at levels between 87 and 90 dB (A),” J. Acoust. Soc. Am. **84**, 651–659.