Noise and vibration interactions: Effects on hearing[a)]

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(Received 30 April 1989; accepted for publication 14 August 1989)

There is the suggestion in the literature that vibration may potentiate the effects of noise and may thus increase the risk of hearing loss in a variety of exposure situations. However, in human experimental studies, which, by necessity, are limited to low levels of exposure, the effects measured are relatively small. A very limited number of animal studies have also shown an enhanced noise-induced hearing loss in the presence of vibration, but the scope of these studies is limited. The animal studies (chinchilla) that form the basis of this report were performed using a 30-Hz, 3*g* rms and a 20-Hz, 1.3*g* rms cage vibration separately and in combination with continuous noise (95-dB, 0.5-kHz octave band) and impact noise (113, 119, or 125 dB peak SPL) exposure paradigms. All exposures lasted for 5 days. The impact noise exposures were designed to have approximately equal total energy. Temporary and permanent threshold shifts were measured using evoked potentials, and sensory cell loss was measured using surface preparation histology. The results obtained from some of the noise/vibration paradigms showed that such exposures can alter some of the dependent measures of hearing. This effect was statistically significant only for the stronger vibration exposure conditions and was evident primarily in the extent of the outer hair cell losses and in the shape of the PTS audiogram.

PACS numbers: 43.66.Ed, 43.50.Qp, 43.40.Ng, 43.66.Wv [NFV]

INTRODUCTION

There is a general concern that interactions among various ototraumatic agents may place a noise-exposed individual at a greater risk for acquiring hearing loss than would be anticipated on the basis of exposure to the noxious stimuli separately. Since the vestibular portion of the membraneous labyrinth contains the primary receptors for displacement and acceleration, and since the vestibular system is intimately associated with the cochlea, it is natural to inquire into the effects of vibration on hearing. Specifically, to what extent is the risk of hearing loss following a noise exposure changed if the noise is presented simultaneously with vibration? Although this study is limited to data acquired from an animal model, a brief overview of the limited database obtained from humans and some historical background will help place the contemporary animal data into perspective. Such a review will also provide a rationale for the animal experiments.

Concerns over interactions between noise and vibrations are not new, having been initiated during the first decade of this century. One of the first reviews and comprehensive discussions of the effects of noise and vibration on hearing was written by Temkin in 1933. From this monograph we learn that more than a decade earlier Wittmaack may have been among the first to propose that structurally borne vibrations might be an important factor in the etiology of noise-induced hearing loss (NIHL). His observations were made on the basis of studies of workmen exposed to noise and vibration during the work day. However, Wittmaack's ideas were not universally shared by his contemporaries.

Temkin's 1927 study (cited in Temkin, 1933) on workers exposed to noise and vibration may be the first clinically based epidemiological study in this area. Data were collected on two classes of workmen: one group exposed only to airborne noise, the other to both noise and vibration. The former population rarely showed any low-frequency hearing losses (32 and 64 Hz), while the noise- and vibration-exposed individuals showed both high- and low-frequency losses.

One of the earliest experiments involving the exposure of animals to noise and vibration was performed by Popow in 1928 (also cited in Temkin, 1933) using mice. Two cages of mice, one placed on the floor to receive both structurally borne vibrations and airborne sound, the other suspended so as to be isolated from any structurally-borne vibrations, were analyzed for cochlear pathologies. Popow showed that although the airborne sound contained spectral energy as low as 50 Hz, this low-frequency energy was not capable of producing cochlear damage. However, the mice that were exposed to the structurally borne vibrations, which contained energy as low as 30 Hz, exhibited damage in the apical

[a)] Portions of these data were presented at the 115th Meeting of the Acoustical Society of America, in Seattle, WA [Hamernik *et al.*, J. Acoust. Soc. Am. Suppl. 1 **83**, S21–S22 (1988)] and at the Third International Conference on the Combined Effects of Environmental Factors, July 1988, in Tampere, Finland.

 0001-4966/89/122129-09$00.80

portions of the cochlea. This structurally borne low-frequency energy could reach the cochlea via a bone conduction route. Thus, on the basis of these early observations on mice and humans, the evidence seemed to implicate vibration in the pathogenesis of NIHL.

A. Human studies

The most recent epidemiological studies have been reviewed by Manninen (1983) (e.g., Pokrovskij, 1968; Taniewski and Banaszkiewicz, 1973). These investigators conducted surveys of personnel in heavy industry and mining, respectively. Their results have implicated whole-body and segmental vibration in the etiology of NIHL. Similarly, in the forest and lumber industry, Pintér (1973) concluded that vibration does have an effect on the dynamics of NIHL. In particular, losses in the low frequencies seem to be exacerbated in workers with combined exposures. The effects were more pronounced in those workers suffering from vibration-induced Raynaud syndrome. A similar result was shown by Pyykko *et al.* (1981). Comparable epidemiological or demographic studies do not appear to have been conducted in the U.S. This is surprising considering that NIOSH (the National Institute for Occupational Safety and Health) has sponsored surveys on the number of workers exposed to various types of vibration (Wasserman *et al.*, 1978, 1980). Data presented in these reports show that approximately eight million nonmilitary American workers are exposed to vibration, most often in combination with some background noise.

Although the survey data indicate that vibration does affect NIHL, it is important to note that: (1) the industries studied represent those with some of the highest noise levels that are to be encountered; (2) many of these industries are also characterized by a non-Gaussian noise environment (i.e., random high-level impact noises superimposed upon a broadband continuous noise); and (3) the occupations represented are those also characterized by high levels of stress. Thus factoring out the effect of some of these "other" complicating variables is difficult. That is, it is difficult to establish to what extent vibration alone can increase the risk of developing a NIHL in such complex exposure environments.

The results of controlled experimental studies have been somewhat equivocal. Guignard and Coles (1965) subjected eight men to a 30-min exposure to one of the following: (1) a 15-Hz, 0.7*g* rms sinusoidal, whole-body vibration, (2) white noise at 100 dB SPL, or (3) a combination of the noise and vibration. Hearing thresholds were measured within 15 min following each exposure. No significant effect of vibration on temporary threshold shift (TTS) could be detected. In fact, at the 4-kHz test frequency, the group exposed to noise alone showed five decibels more TTS than did the group exposed to noise and vibration (12 vs 7 dB). Several other experimental studies using human subjects were performed during the next decade (e.g., Okada *et al.*, 1972 and Yokoyama *et al.*, 1974). In general, these laboratory studies seemed to indicate that if vibration did have an effect on NIHL, the effect would amount to only approximately 5 dB at the most affected frequencies. A brief overview and critique of a number of such studies can be found in Manninen (1983a,b) and in Humes (1984). Von Gierke (1980) also reviewed some of these human experimental studies and concluded, "there is no evidence of a significant synergistic effect on hearing loss by simultaneous steady-state noise and vibration exposure as long as the individual exposure levels are below those recommended for each modality" (p. 650).

Probably, the most extensive set of human experimental data was obtained by Manninen (1983a) using a wide variety of noise and vibration stimuli. Manninen showed an interaction effect at the 4- and 6-kHz audiometric test frequencies when a broadband noise was combined with 5-Hz vibration at 2–12 m/s^2. The exposure durations were less than 1 h. The statistically significant effects amounted to approximately 5-dB increases in TTS_2 (the TTS measured 2 min after exposure) when the combined stimulus was presented. These results were in general agreement with the bulk of the earlier studies.

B. Animal studies

Experimental studies on the effects of noise and vibration interactions in animals that would extend or parallel the human studies are relatively few in number. Jauhiainen *et al.* (1969) showed that vibration alone can have a direct effect on the physiological state of the guinea pig cochlea. They used the cochlear microphonic (CM) as their dependent variable and showed that modest levels (1.2*g* rms) of sinusoidal whole-body vibration at 10 Hz could increase the sensitivity of the cochlea to sound. In the linear portion of the CM input/output function, the effect amounted to a 5- to 7-dB increase in sensitivity. Whether this increased sensitivity caused an increased risk of NIHL was not under investigation.

There are only three recent experimental animal studies that bear directly on the issue of noise and vibration interactions. Two of these studies were performed in our own laboratories by Hamernik *et al.* (1980, 1981), and the third was recently presented by Byrne *et al.* (1988). Each of these studies used the chinchilla as an animal model.

In the first study, Hamernik *et al.* (1980), three groups of animals, with five animals per group, were used. Group A was exposed to 50 shock-tube-generated impulses (blast waves) at 155 dB peak SPL, at 1.5-ms A duration, and at one impulse per minute. Group B was exposed to 1 h of continuous sinusoidal, whole-body vibration at 30 Hz with an acceleration of 1.0*g* rms. The animals were restrained so that the head acceleration in the vertical plane was also approximately 1*g* rms. Group C was exposed to a combination of the noise and vibration exposures described above. Severe exposure conditions were purposely chosen in an effort to precipitate an interaction effect, if possible. The vibration exposure alone produced no threshold shift, while the impulse noise produced an initial threshold shift of approximately 35 dB, which recovered by 96 h after exposure. The group exposed to the combination of noise and vibration showed a median maximum threshold shift of about 55 dB, and after a 30-day recovery period, thresholds were still elevated by more than 20 dB. The threshold recovery curve for the animals exposed to the combined stimulus was often unusual in that thresh-

olds got worse after exposure before recovery began; i.e., there was a growth of threshold shift. This type of growth has been documented in a number of other studies (e.g., Hamernik *et al.*, 1988) and has been shown to be correlated with a developing cochlear lesion. Out of 35 different recovery curves (i.e., five animals × seven frequencies) measured in the course of this experiment, there were 15 "growth-type" functions in the animals exposed to noise and vibration, and only five in the group exposed to only the noise.

An analysis of the sensory cell losses confirmed the audiometric data in showing a strong influence of vibration on the outcome of a severe noise exposure. An interesting feature of the histological results is that, when the total loss of outer hair cells in the apical 10% of the cochlea was compared in the noise-alone and the combination groups, the latter had substantially larger losses. These apical losses are probably not reflected in the audiometric data which are limited to frequencies of 0.5 kHz and higher. Although these median data do implicate vibration as a factor in increasing the risk of NIHL, it should be noted that these results are based upon a very limited number of animals and that the individual animal data are quite variable. Also to be considered is the relatively unrealistic severity of the exposure conditions.

With this in mind, a second series of experiments was performed (Hamernik *et al.*, 1981). This series of exposures utilized much more realistic levels of stimulation over a more prolonged period of time. Again, three groups of chinchillas were used with five animals in each group. Animals in group A were exposed for 10 days to an impact noise having a peak SPL of 113 dB and a presentation rate of 1/s. The impacts were produced by an automated hammer impinging upon a steel plate. Group B was exposed to only whole body vibration for 10 days. The cage containing the animal was vibrated at 1.0*g* rms at 30 Hz, in the vertical plane. The acceleration at the animals head was approximately 0.3*g* rms. Animals in group C were exposed to the combination of the above two conditions. Each animal's threshold was tested daily at 0.5 and 8 kHz during the exposure and at regular intervals following exposure for 30 days. At the 8-kHz test frequency, the asymptotic threshold shift (ATS) over the 10-day period of exposure was higher for the combination exposure than for either separate exposure. During the 30-day recovery period, the animals that received the combination exposure were slower to recover and ended up with a greater PTS. The histological data confirmed the audiometric results by showing greater sensory cell losses in the animals exposed to the combined stimulus. However, traditional statistical tests on the data were not reported. As in the first experiment (Hamernik *et al.*, 1980), these combination animals also showed greater losses, on the average, in the apical 10% of the cochlea. Thus two rather different experimental paradigms using very different stimulus conditions suggested that the dynamics of NIHL might be influenced by the simultaneous addition of whole-body vibration.

Byrne *et al.* (1988), using a complex exposure paradigm, exposed four groups of chinchillas to one of the following conditions: (1) a vibration of 63–89 Hz at 0.1*g* rms (days 1–5) and 1*g* rms level (days 6–10) for 6 h/day for 10 days; (2) an octave band of noise (OBN) centered at 0.5 kHz at 100 dB SPL for 6 h/day for 20 days; (3) a combination of the 0.5-kHz OBN at 100 dB and a vibration of 14–20 Hz at the 0.1 and 1*g* rms levels; and (4) a combination of the 0.5-kHz OBN at 100 dB and a vibration of 63–89 Hz at the 0.1 and 1*g* rms levels. They concluded that there was a small (5 dB) effect of the vibration on TTS, which was dependent upon the frequency of the vibration, but independent of the level of vibration. No statistically significant effect on PTS measures was found for the combined noise/vibration exposures. These results were consistent with the human studies, which seemed to indicate that the TTS effects could be consistently measured, but that they were probably the result of systemic changes associated with the cardiovascular system rather than the result of direct stimulation of the cochlea.

The purpose of the present experiment was, in part, to replicate the second set of data referred to above (Hamernik *et al.*, 1981) and also to investigate the role of the various exposure parameters while keeping the total acoustic energy of the exposure approximately constant.

I. METHOD

Subjects: Eighty-two chinchillas were used in this study. Each animal was anesthetized [IM injection of Ketamine (12.86 mg/kg), Acepromazine (0.43 mg/kg), and Xylazine (2.57 mg/kg)] and made monaural by the surgical destruction of the left cochlea. A chronic electrode was implanted near the inferior colliculus for single-ended nearfield recording of the evoked potential (Henderson *et al.*, 1973; Salvi *et al.*, 1982). The animals were allowed to recover for at least a week before evoked potential testing began.

A. Pre-exposure testing

Hearing thresholds were estimated on each animal using the auditory-evoked potential (AEP). The animals were awake during testing and restrained in a yokelike apparatus to maintain the animal's head in a constant position within the calibrated sound field. AEPs were collected to 20-ms tone bursts (5-ms rise/fall time) presented at a rate of 10/s. Each sampled waveform was analyzed for large-amplitude artifacts, 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 kHz and at the half-octave frequency of 11.2 kHz. Threshold was defined to be one-half step size (2.5 dB) below the lowest intensity that showed a "response" consistent with the responses seen at higher intensities. The average of at least three separate threshold determinations at each frequency obtained on different days was used to obtain the mean pre-exposure audiogram for each animal.

B. Noise and vibration exposures

All animals were exposed for 5 days to one of the six control or eight noise-vibration combination conditions listed in Table I. Two control groups were exposed to vibration alone at either 30-Hz, 3.0*g* rms or 20-Hz, 1.3*g* rms. Another

TABLE I. Pre-exposure threshold means ($\bar{X}$) and standard deviations (s.d.) for each group.

Exposure condition	N	Test frequency (kHz) 0.5	1.0	2.0	4.0	8.0	11.2	16.0	
1.3*g* rms 20 Hz	5	21.2	2.8	11.2	2.2	12.8	12.8	22.8	$\bar{X}$
Cage vibration (CV)		2.2	4.8	7.2	3.4	6.5	6.1	5.4	s.d.
3.0*g* rms 30 Hz (CV)	5	21.5	6.5	11.2	4.2	18.8	14.2	25.2	$\bar{X}$
		5.1	5.2	4.3	3.1	5.7	4.9	6.5	s.d.
0.5-kHz OBN at 95 dB	6	13.9	7.8	13.9	4.3	17.5	24.2	27.8	$\bar{X}$
		4.4	4.9	3.6	3.3	4.8	7.3	6.4	s.d.
113-dB impact 1/1 s	5	15.3	8.2	4.9	2.9	10.6	···	13.1	$\bar{X}$
		6.0	4.8	3.4	4.9	5.3	···	13.2	s.d.
119-dB impact1/4 s	7	13.9	9.4	9.1	− 1.5	15.3	···	24.0	$\bar{X}$
		9.0	8.4	6.8	5.1	10.0	···	8.9	s.d.
125-dB impact 1/16 s	11	19.0	8.3	9.6	0.5	10.8	···	17.2	$\bar{X}$
		8.6	4.7	7.9	6.7	9.3	···	7.2	s.d.
0.5-kHz OBN at 95 dB	6	17.2	− 0.8	3.1	− 7.8	10.6	9.4	16.4	$\bar{X}$
and 1.3*g* rms 20-Hz CV		4.4	3.5	5.7	8.8	3.6	5.5	7.2	s.d.
113-dB impact 1/1 s	6	20.6	1.9	5.6	− 0.3	9.2	10.6	21.4	$\bar{X}$
and 1.3*g* rms 20-Hz CV		2.7	4.0	5.8	8.1	4.3	4.4	5.2	s.d.
119-dB impact 1/4 s	5	19.2	4.2	3.8	3.2	11.5	9.2	18.8	$\bar{X}$
and 1.3*g* rms 20-Hz CV		2.6	3.3	3.4	10.0	7.8	7.1	5.1	s.d.
125-dB impact 1/16 s	5	21.2	3.8	10.8	5.5	13.5	12.2	19.5	$\bar{X}$
and 1.3*g* rms 20-Hz CV		1.8	3.6	6.8	6.6	4.8	6.5	8.0	s.d.
0.5-kHz OBN 8 95 dB	6	19.4	4.4	8.1	3.3	17.2	18.3	24.7	$\bar{X}$
and 3.0*g* rms 30-Hz CV		3.2	4.8	5.7	6.3	6.4	7.4	10.7	s.d.
113-dB impact 1/1 s	5	15.8	0.8	7.5	− 2.5	14.5	13.5	19.5	$\bar{X}$
and 3.0*g* rms 30-Hz CV		2.0	2.0	3.7	4.3	5.6	6.3	10.4	s.d.
119-dB impact 1/4 s	5	19.2	5.5	7.2	2.2	9.5	10.5	20.2	$\bar{X}$
and 3.0*g* rms 30-Hz CV		4.9	6.8	5.1	8.1	6.5	4.0	7.7	s.d.
125-dB impact 1/16 s	5	15.8	0.5	7.5	− 0.2	19.5	21.2	24.8	$\bar{X}$
and 3.0*g* rms 30-Hz CV		2.6	3.0	2.6	3.5	4.3	7.6	8.0	s.d.

control group was exposed to an octave band of continuous noise centered at 0.5 kHz and presented at 95 dB SPL. Three other control groups were exposed to one of the following impact noise paradigms: 113 dB peak SPL at a rate of one impact every second, 119 dB peak SPL once every 4 s, and 125 dB Peak SPL once every 16 s. By adjusting the repetition rate and the intensity of the impacts, the impact noise exposures could be adjusted so that each exposure had approximately equal total energy. Combinations of each of the two vibrations with each of the four noise exposures comprised the eight experimental or combination groups.

The vibration exposures were produced by mounting two small (12-in. length × 6-in. width × 8-in. height) cages rigidly to a Brüel & Kjær accelerator body (type 4805) with general purpose head (type 4812). The accelerator body was controlled by the sinusoidal input from a Wavetek model 193, 20-MHz sweep/modulation generator through a Brüel & Kjær power amplifier (type 2707). A Brüel & Kjær accelerometer (type 4374) was mounted on the cage platform. A second accelerometer (PCB model 303A11) was attached to a post cemented to the skull of approximately half of the animals in each of the vibration exposure groups, and the head vibration in the vertical plane was measured with the animal sitting quietly in the vibrating cage. The 20-Hz, 1.3*g* cage vibration resulted in a mean 0.3*g* vibration measured at the skull. The 30-Hz, 3.0*g* cage vibration similarly resulted in a mean head vibration of 0.4*g*. The acceleration at the head roughly followed the sinusoidal input to the accelerator body.

The octave band of noise was produced by filtering a broadband noise. The impacts were produced by passing a broadband noise through a set of active filters and multiplying this waveform by an exponential decay (constructed using a resistance-capacitance circuit) with a custom-built multiplier circuit. The noises were mixed electronically, and the mixed electrical signal was amplified by a McIntosh MC2100 power amplifier that drove a JBL 2445J driver with JBL model 2360H exponential horn.

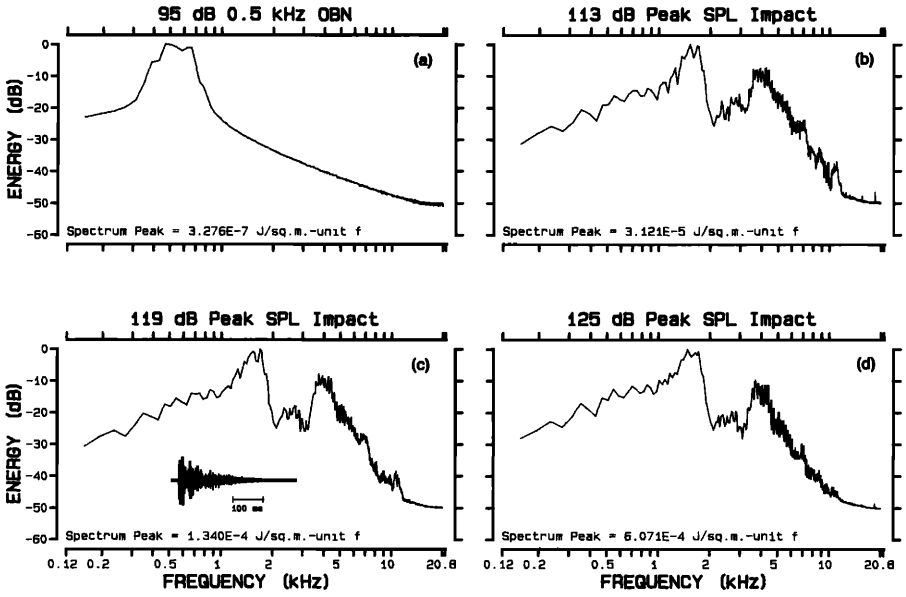

FIG. 1. Frequency spectra and pressure-time waveforms for the noise exposures employed in this study. Data were sampled at 40 kHz. The spectra are computed using an FFT over 4096 points (102.4 ms): (a) 0.5-kHz octave-band noise at 95 dB SPL; (b) 113-dB peak SPL impact; (c) 119-dB peak SPL impact; and (d) 125-dB peak SPL impact.

The frequency spectra for the 0.5-kHz octave-band noise and the three impact noises are presented in Fig. 1. An example of the pressure-time trace of a typical impulse is also shown. The other two impulses had the same basic shape and only differed in their peaks. The spectrum of the impact is not appreciably altered by increasing the peak intensity from 113 to 125 dB. Note that the peak of the impact noise spectrum lies between 1 and 2 kHz, with a pronounced depression in the 2- to 3-kHz region. Table II presents the total energy (J/m^2) contained in each of the four noise control conditions over the 5-day exposure period. The energy calculations were performed using a specific acoustic impedance of 406 mks rayls ($N\ s/m^3$).

Thresholds were measured daily at 0.5, 2, and 8 kHz throughout the 5-day exposure and the asymptotic threshold shift (ATS) was computed as the difference between the threshold during exposure (averaged over 5 days) and the pre-exposure threshold.

C. Post-exposure testing

After the 5-day exposure was complete, threshold recovery functions were measured at 0.5, 2, and 8 kHz at 0, 2, 8, 24, and 240 h after removal from the noise (using the same method as described for pre-exposure testing). After at least 30 days, final audiograms were constructed using the average of three separate threshold determinations at each of the seven pre-exposure frequencies. Permanent threshold shift (PTS) was defined as the difference between the post- and pre-exposure thresholds at each individual test frequency.

TABLE II. Total acoustic energy in each of the noise control exposures.

Exposure group	J/m^2
0.5-kHz OBN at 95 dB	1346
113-dB peak SPL, 1/1 s	884
119-dB peak SPL, 1/4 s	812
125-dB peak SPL, 1/16 s	985

D. Cochlear histology

Following threshold testing, the animals were killed by decapitation, and their cochleas were immediately removed and fixed. The cochleas were dissected, and the status of the sensory cell population was evaluated using conventional surface preparation histology. [See Hamernik *et al.* (1987) for a more complete description.] The status of sensory and supporting cells were evaluated with Nomarski differential interference contrast microscopy and entered into a database on a laboratory computer (Macintosh II). Standard cochleograms were then constructed by computing the percent sensory cell loss across the length of the cochlea in 0.24-mm steps. These cell-loss figures were then converted into percent loss averaged over octave bands centered at the audiometric test frequencies along the length of the cochlea and correlated with the frequency-place map for the chinchilla constructed by Eldredge *et al.* (1981).

II. RESULTS

A. Pre-exposure thresholds

The mean pre-exposure thresholds in dB SPL for the 14 groups of animals are presented in Table I. The mean pre-exposure thresholds from the present study are, in general, lower than Miller's (1970) threshold data when the Miller data are adjusted for the effects of temporal integration (Henderson, 1969). In most cases, however, these differences did not exceed the resolution of the threshold testing procedure (i.e., 5 dB). The pre-exposure group mean thresholds for each of the 14 experimental and control groups were relatively homogeneous, and only two of the mean thresholds fell outside of a 95% confidence interval constructed around Miller's threshold data that were corrected for the effects of temporal integration.

B. Threshold shift and sensory cell analysis

Figure 2 illustrates the mean ATS, PTS, and percent sensory cell loss for the two vibration-alone groups. The probability of a type-I error (α) for all the analyses presented was set at 0.05. An analysis of variance indicated that there were no statistically significant differences between the ATS caused by the two different vibration conditions ($F = 0.231$, $df = 1/8$). There was essentially no ATS or PTS caused by either of the two vibration conditions. It also is clear that the vibration alone caused very little or no sensory cell damage. There was a statistically significant frequency effect for the outer hair cell losses ($F = 12.395$, $df = 7/56$), which reflects the slightly higher cell losses (approximately 7%) in the apical portion of the cochlea for these animals.

The results of the three equal-energy impact noise exposures are illustrated in Fig. 3. The impact noise control exposures caused a mean ATS of approximately 30–60 dB at the three test frequencies. There was a statistically significant frequency effect ($F = 41.224$, $df = 2/40$) in which more ATS was caused at the higher than at the lower test frequencies. However, the frequency by impact interaction was not statistically significant ($F = 0.825$, $df = 4/40$), indicating that the frequency effect was the same for each impact-alone group. Thus the three impact-alone groups that were exposed to noises of approximately equal total energy had statistically equivalent ATS levels.

The mean PTS levels ranged from 4–18 dB with the greater hearing losses at the higher test frequencies. As was the case with the ATS measure, there were no statistical differences among the PTSs caused by the three impact noise exposures presented alone ($F = 0.244$, $df = 2/20$). The frequency by group interaction for the PTS-dependent measure did not reach statistical significance ($F = 0.872$, $df = 10/100$). Thus the equal total energy impact noise exposures produce similar levels of PTS.

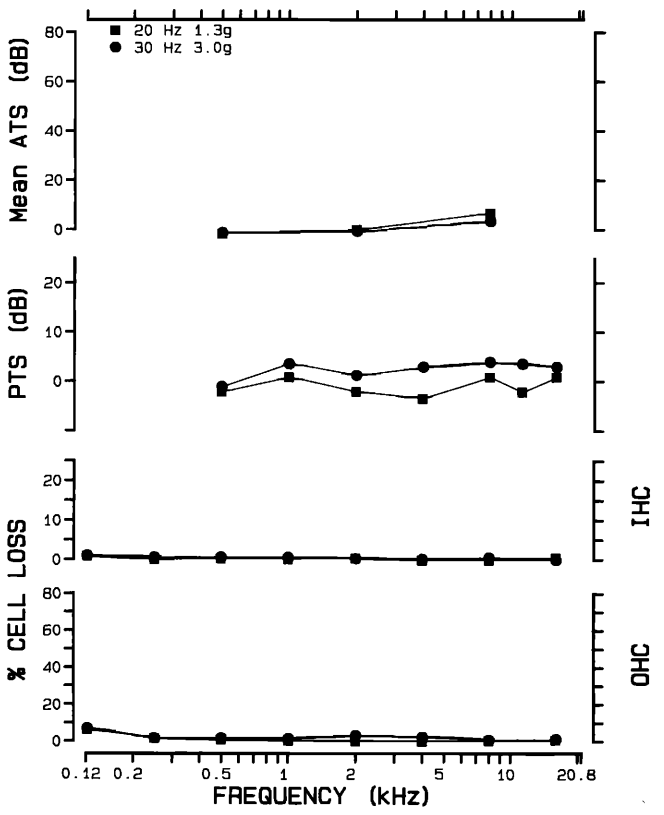

FIG. 2. Mean asymptotic threshold shift, permanent threshold shift, and percent sensory cell losses for the vibration-alone groups.

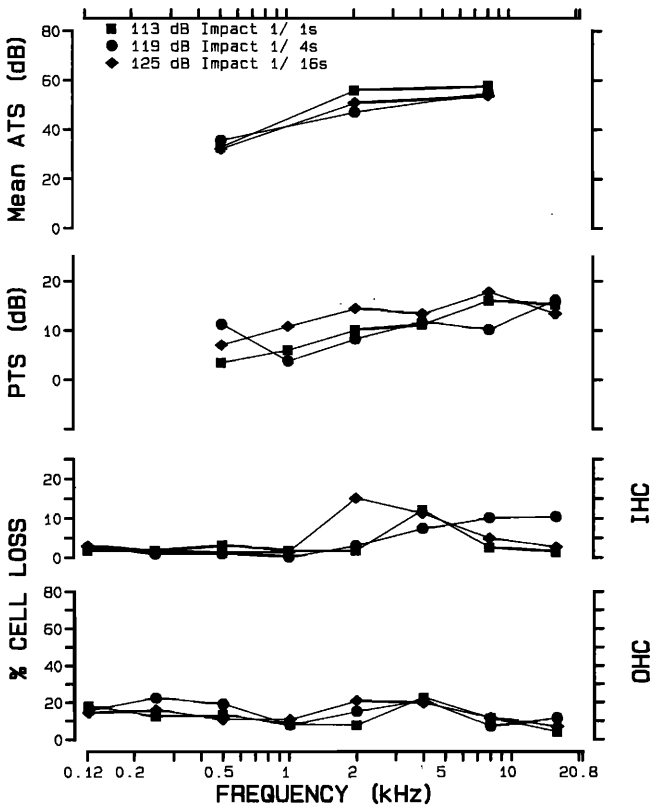

FIG. 3. Mean asymptotic threshold shift, permanent threshold shift and percent sensory cell losses for the impact-alone groups.

There was little to distinguish the impact-alone groups on the basis of percent sensory cell loss, as was the case with the mean ATS and PTS measures. There was a statistically significant frequency effect for both percent inner ($F = 2.436$, $df = 7/140$) and percent outer ($F = 3.609$, $df = 7/140$) hair cell losses, which reflects greater sensory cell losses in the midfrequency region of the cochlea where the majority of the acoustic energy of the impacts was located.

Figure 4 shows the mean ATS, PTS, and percent sensory cell losses for the three groups (noise alone and noise plus vibration) exposed to the 95-dB, 0.5-kHz octave band of noise. There were no statistically significant differences between the three groups (i.e, the noise control and the two interaction groups) for both mean ATS and PTS measures (ATS: $F = 1.919$, $df = 2/15$; PTS: $F = 0.462$, $df = 2/14$). The slightly lower ATS thresholds at 8 kHz are reflected in the statistical analysis by a significant frequency effect ($F = 10.115$, $df = 2/30$). However, the absence of a significant interaction term (frequency by group) demonstrates that there was no effect of vibration on the ATS ($F = 1.940$,

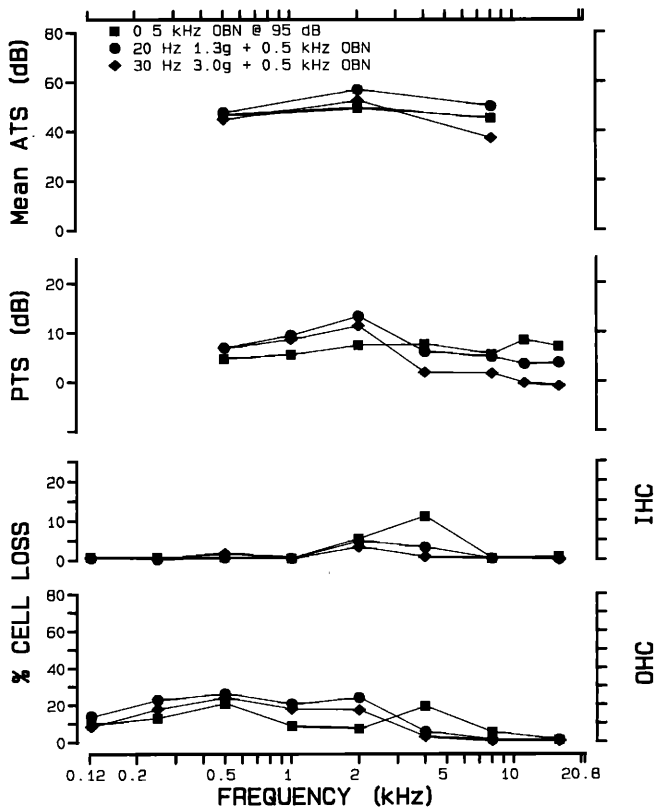

FIG. 4. Mean asymptotic threshold shift, permanent threshold shift, and percent sensory cell losses for the groups exposed to the 0.5-kHz octave band of noise presented at 95 dB SPL, alone and in combination with the two vibration conditions.

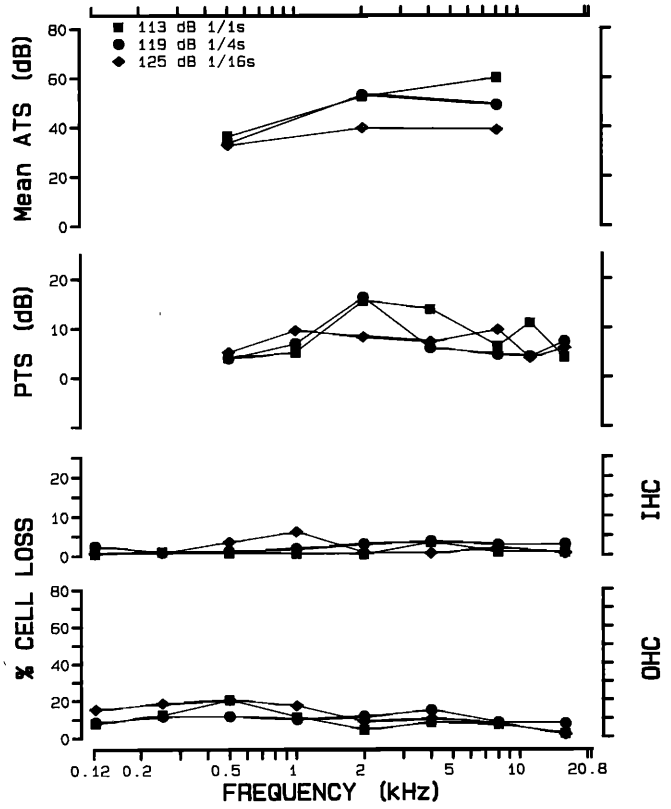

FIG. 5. Mean asymptotic threshold shift, permanent threshold shift, and percent sensory cell losses for the groups exposed to the equal-energy impact exposures in the presence of 20-Hz, 1.3g rms cage vibration.

$df = 4/30$) or PTS ($F = 0.886$, $df = 10/70$) caused by the 0.5-kHz octave band of noise.

There were no statistically significant group main or interaction effects in the percent IHC or percent OHC loss analysis, indicating that the addition of the vibration to the noise exposure did not alter the sensory cell loss relative to that measured following the octave-band noise exposure alone. The main effect of frequency was statistically significant (OHC: $F = 6.672$, $df = 7/105$; IHC: $F = 3.979$, $df = 7/105$) with an increase in percent OHC loss in the midfrequency region, the region corresponding most closely with the maximum energy of the noise exposure. The increase in percent IHC loss was located in the midfrequency region of the cochlea, but exceeded 5% loss for only one frequency in the noise control (i.e., no vibration) condition.

Figure 5 illustrates the mean ATS, PTS, and percent sensory cell losses for the three groups exposed to the equal-energy impact conditions in the presence of 20-Hz, 1.3g cage vibration. There was no statistically significant effect of the presence of the 20-Hz, 1.3g vibration on the mean ATS and PTS caused by the equal-energy impact exposures (ATS: $F = 0.620$, $df = 1/33$; PTS: $F = 1.640$, $df = 1/33$). Furthermore, none of the interactions with impact group was statistically significant for either dependent measure. There was a statistically significant frequency effect (ATS: $F = 58.444$, $df = 2/66$; PTS: $F = 3.462$, $df = 5/165$) for both dependent variables, which could be anticipated from the impact-alone group results. A statistically significant vibration by frequency interaction in the PTS analysis ($F = 2.929$, $df = 5/165$) reflects the fact that the PTS at the higher test frequencies is affected by the presence of the 20-Hz, 1.3g vibration. In the absence of vibration, the PTS rises approximately monotonically with log frequency (Fig. 3). In the presence of vibration, however, the PTS does not change and appears to be generally the same across all frequencies, or at least it does not show the same pattern as is observed for the impact-alone groups. Specifically, at and above the 8-kHz test frequencies, there was significantly less PTS in the groups exposed to impact noise and vibration than in the groups exposed to the impacts alone.

There were no statistically significant effects in the percent sensory cell losses except for the main effect of frequency for the percent OHC losses ($F = 4.042$, $df = 7/231$). The percent OHC losses show different amounts of damage throughout the cochlea with a predominance of the cell loss through the apical half of the basilar membrane. However, these losses seldom exceeded 20% of the outer hair cells.

Figure 6 shows the mean ATS, PTS, and sensory cell losses for the three groups exposed to the equal-energy impact conditions in the presence of 30-Hz, 3.0g cage vibration. The statistical decisions made from the analyses of the 30-Hz, 3.0g cage vibration were similar to those made from the analyses of the 20-Hz, 1.3g cage vibration conditions. In fact, there are no threshold shift results that seem to differentiate between the two cage vibration conditions except, perhaps, an elevated PTS at the 16-kHz test frequency in two of the 30-Hz vibration groups relative to the 20-Hz vibration

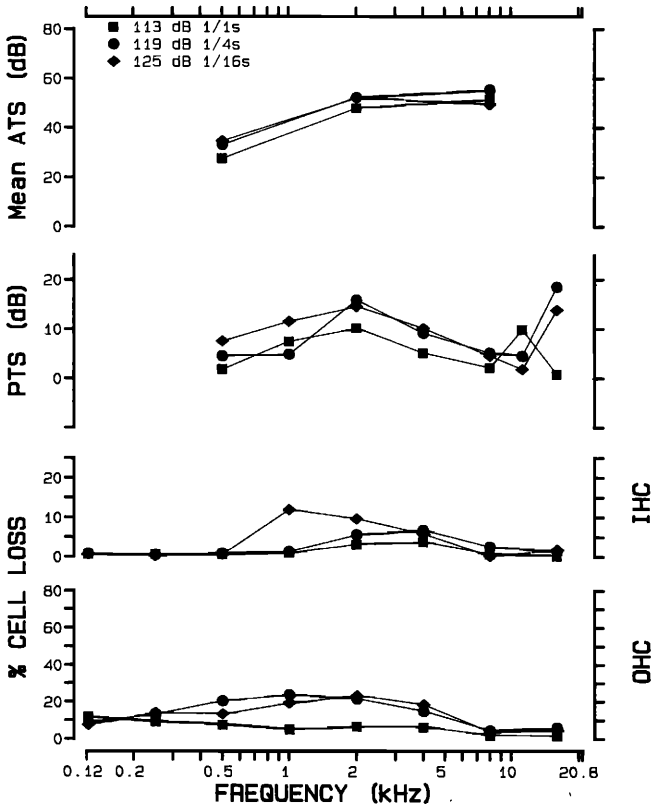

FIG. 6. Mean asymptotic threshold shift, permanent threshold shift, and percent sensory cell losses for the groups exposed to the equal-energy impact exposures in the presence of 30-Hz, 3.0*g* rms cage vibration.

groups. In the percent sensory cell loss analysis for these groups, the main effect of frequency was statistically significant for both percent IHC and percent OHC losses (IHC: $F = 2.439$, $df = 7/224$; OHC: $F = 3.902$, $df = 7/224$). The mean losses, while not exceeding 15% (IHC) and 25% (OHC) were in the midfrequency region of the cochlea. Once again, this is the region that contained the bulk of the energy from the three impact exposures. The percent OHC impact by frequency interaction was statistically significant ($F = 2.430$, $df = 7/224$) and is illustrated by greater midfrequency losses for the groups exposed to the combination of vibration and impact noise than to the impact noises alone. All other main effects and interactions were not statistically significant.

III. DISCUSSION

The following points can be made from these data. (1) The two vibration-alone exposures resulted in no significant audiometric changes in either the mean ATS, PTS, or percent sensory cell loss measures. (2) The three impact noise exposure conditions, which were balanced to produce approximately equal total energy exposures, produced the same levels of mean ATS and no statistically different levels of PTS or percent sensory cell losses. (3) The addition of the vibration to either the continuous noise or the three different impact noises did alter some of the dependent measures of hearing employed. There were two statistically significant effects of vibration on the outcome of a noise exposure: (a) a change in the profile of the PTS audiogram for each of the vibration/impulse noise exposure conditions where less PTS was produced by the combination exposures than the impact-alone exposure at the higher test frequencies and (b) an increased midfrequency percent OHC loss for the 30-Hz vibration plus 119- and 125-dB peak SPL impacts.

These experimental results should have confirmed the data of Hamernik *et al.* (1981), but did not. In the previous study, the ATS data for the combination exposures fell above the range of the control animals. In addition, the median PTS data for the combination group was well above the PTS levels for the control group at some frequencies. The present study did not show similar clear differences in the ATS or PTS measures. The PTS as a result of the impact noise exposures at the 8- and 11.2-kHz test frequencies was lower in the 30-Hz, 3.0*g* combination groups than the control groups. On the other hand, there did appear to be a small potentiation of outer hair cell loss in the midfrequency range of the cochlea caused by the addition of the 30-Hz, 3.0*g* cage vibration to the impact noise exposures. Although this effect was statistically significant, the differences were small and any conclusions from the OHC results should be made with caution. The major difference between these two sets of data is that one series of exposures lasted for 5 days and the other lasted for 10 days. This difference in the duration of the exposure might account for the difference in results. Considering that the epidemiological data were acquired from individuals that were exposed for many years, it is conceivable that our relatively short–duration exposures are too short to provoke a noticeable interaction effect unless unrealistically high stimulus levels are used as in the Hamernik *et al.* (1980) report. On the other hand, the epidemiological data may need to be more critically analyzed in order that we may be certain that the effects ascribed to vibration are indeed the result of vibration and not a result of a variety of other factors known to confound this type of data.

Laboratory studies using human subjects are, by necessity, limited to levels of stimulation that will not produce any permanent changes in hearing. Thus, if vibration can affect the dynamics of NIHL, it must be an effect that is demonstrable in the TTS domain if human experimentation is to be of any value. While animal experiments do not have this limitation, we are instead faced with another set of problems, namely, the trade-off among the severity and duration of an exposure (both of which may be unrealistic or impractical) and ethical considerations associated with prolonged animal discomfort. Given these caveats, we are left with the impression from the literature that consistent low-level effects in humans can be measured, although the effects are small; in animals, it may be necessary to use relatively long exposure times or severe conditions of exposure before effects of an interaction begin to manifest themselves to a degree that is amenable to experimental study. Considering the relatively small and inconsistent effects on hearing and sensory cell populations that we are reporting in the chinchilla (an animal known to be more susceptible to NIHL than humans) from combinations of noise and vibration, an increased risk of NIHL from vibration in the industrial population is probably relatively small.

ACKNOWLEDGMENTS

This work was supported by the National Institute for Occupational Safety and Health Grant 2 R01 OH02317. All research has been carried out in full compliance with the National Institutes of Health Guidelines for the Use of Experimental Animals and has been approved by the Institutional Animal Care and Use Committee.

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