

THE INFLUENCE OF WHOLE BODY VIBRATION ON
NOISE-INDUCED HEARING LOSS: A REVIEW OF ANIMAL
EXPERIMENTS

Hamernik R.P., Ahroon W.A. and Davis R.I.

Auditory Research Laboratory, State University of New York,
Plattsburgh, NY 12901

ABSTRACT

There is the suggestion in the literature that vibration may potentate the effects of noise and may thus increase the risk of hearing loss. However in human experimental studies, which, by necessity, are limited to low levels of TTS, the effects measured while generally consistent, are relatively small. A very limited number of animal studies have also shown an enhanced hearing loss in the presence of vibration, but the scope of these studies is limited. Also the large intersubject variability and small number of subjects in some of these studies makes the data difficult to interpret with any degree of confidence. Furthermore, the high levels of stimulation that were used in some of these animal experiments were not typical of realistic exposure situations. Our recent animal studies (chinchilla) have used a 30 Hz, 3g rms and a 20 Hz, 2g rms cage vibration 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 five days. The impact noise exposures were designed to have equal total energy. Temporary (and combined) and permanent threshold shifts were measured using evoked potentials. The results obtained from each of the above paradigms were consistent in showing that the presence of vibration did not have a statistically significant effect on hearing thresholds.

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 either noxious stimulus separately. Since the vestibular portion of the membranous labyrinth contains the primary receptors for displacement and acceleration, and 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 changed following the simultaneous exposure of an individual to noise and vibration. Although this review is to be limited to data acquired from animal models, a brief overview of the limited data base 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 recent 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. This monograph summarizes some of the earliest experimental studies in this area. From this monograph we learn that Wittmaack more than a decade earlier 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 vibrations 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 air borne noise the other to both noise and vibrations. The former population rarely showed any low frequency hearing losses (32 & 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 1929 (cited in Temkin, 1933) using mice. Two cages of mice, one placed on the floor to receive both structurally borne vibrations and air borne 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 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.

More recent epidemiological studies have been reviewed by Manninen (1983) [e.g. Pokrovskij (1968) and Taniewski and Banaszkievicz (1973)]. These investigators conducted surveys of personnel in heavy industry and mining respectively. Their results also have implicated whole body and segmental vibration in the etiology of NIHL. Similarly in the forest and lumber industry Pinter (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 studies do not appear to have been conducted in the U.S. This is rather surprising considering the NIOSH-sponsored (National Institute of Occupational Safety and Health) survey of workers exposed to vibration (Wasserman et al., 1978). Data presented in this report shows that approximately 8 million non-military American workers are exposed to vibrations most often in combination with some background noise.

Although the survey data referred to above indicate that vibration does alter the dynamics of 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) that many of these industries are also characterized by a non-Gaussian noise environment (i.e. random high level impact noises superimposed upon a broad band 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 if not impossible. That is, it is difficult to establish to what extent vibration alone can increase the risk of developing NIHL in such complex exposure environments.

The results of recent controlled experimental studies have been somewhat equivocal. Guignard and Coles (1965) subjected eight men to one of the following: (1) a 30 min exposure to a 15 Hz, 0.7g 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 5 dB more TTS than did the group exposed to noise and vibration (12 dB vs 7 dB). Several other experimental studies using human subjects followed during the next decade. In general, these laboratory studies seemed to indicate that if vibration did in fact 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 (1983) and 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."

Probably the most extensive set of human experimental data was obtained by Manninen (1983) using a wide variety of noise and vibration stimuli. Manninen showed an interaction effect

at the 4 & 6 kHz audiometric test frequencies when a broad band noise was combined with 5 Hz vibration at $2-12 \text{ M/s}^2$. The exposure durations were less than 1 hr. 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.

REVIEW OF ANIMAL STUDIES

Experimental studies on the effects of noise and vibration interactions in animals which 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 (1.2g 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 sensitivity increase.

A literature search has uncovered only two recent experimental animal studies which bear directly on the issue of noise and vibration interactions, (Hamernik et al., 1980, 1981) both of which were performed in our own laboratories. Given the evidence which exists in the literature this dearth of experimental animal data is surprising. The following is a brief summary of the two studies referred to above. In all of the following results the experimental animal was the chinchilla. All animals were monaural, and hearing function was measured using the evoked auditory response (EAR) recorded from a chronic electrode implanted in the inferior colliculus. Pure tone thresholds were obtained on each animal prior to exposure and at regular intervals following exposure. Thirty days after exposure final threshold estimates were made and the animal was killed for surface preparation histology.

In the first study (Hamernik et al., 1980) three groups of animals, with 5 animals/group, were used. Group A was exposed

to 50 shock tube-generated impulses (blast waves) at 155 dB peak SPL; 1.5ms A-duration; 1/min. Group B was exposed to 1 hr of continuous sinusoidal, whole body vibration at 30 Hz with an acceleration of 1.0g rms. The 30 Hz vibration stimulus was chosen because a great deal of the energy in the impulse was centered around 30 Hz. The animals were restrained so that the head acceleration in the vertical plane was also approximately 1g rms. Group C was exposed to a combination of the two exposures described above. Severe exposure conditions were purposely chosen in an effort to precipitate an interaction effect, if possible. Figure 1 illustrates the threshold recovery curves measured at 0.5 kHz for each of the three experimental groups. 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 hours 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 thirty 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 there was a growth of threshold shift after exposure. This type of growth has been documented in a number of other studies (Hamernik et al. 1988) and has been shown to be correlated with a developing cochlear lesion. Out of 35 different recovery curves measured in the course of this experiment there were 15 "growth type" functions in the animals exposed to noise and vibration and only 5 in the group exposed to only the noise. Figure 2 shows the permanent threshold shift (PTS) for each group measured at each test frequency. While the combination group in general had more PTS at all test frequencies, the greatest effect was found at the 0.5 kHz frequency where the animals exposed to both the noise and vibration sustained 20 dB more PTS than did either the noise alone or vibration alone groups. 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

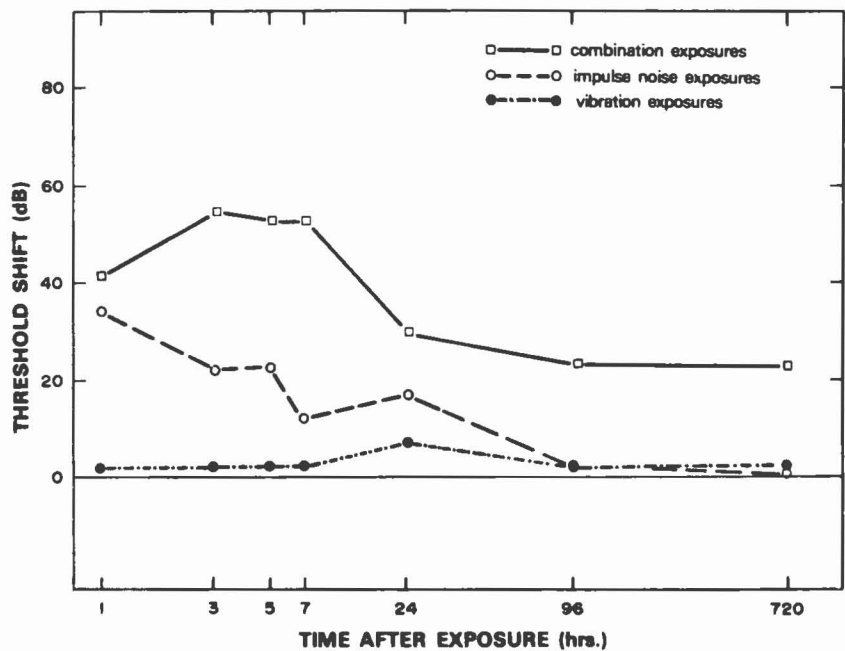


Figure 1. Median threshold recovery functions at 0.5 kHz for the three experimental groups.

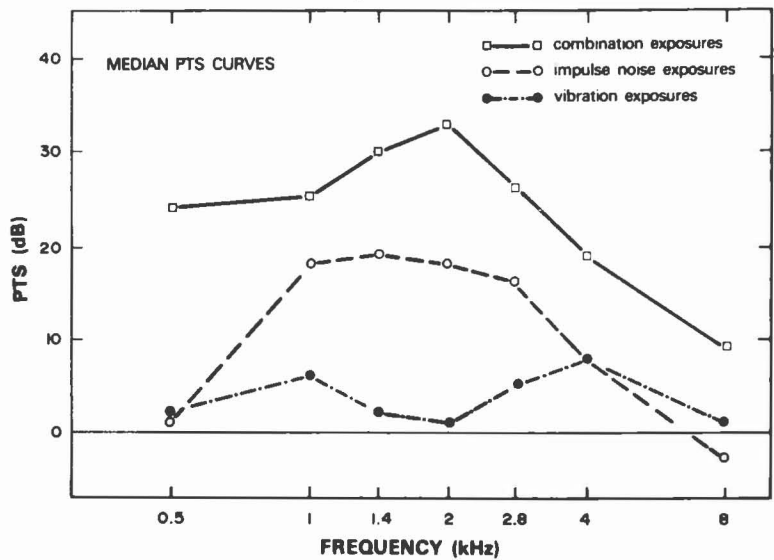


Figure 2. Median PTS for the three experimental groups.

cochlea is compared in the noise alone and the combination group the latter have substantially larger losses. These apical losses are probably not reflected in the audiometric data which is 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 were 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; 5 animals per 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. The details of the impact can be found in Blakeslee et al., (1978). Group B was exposed to only whole body vibration for 10 days. The cage containing the animal was vibrated at 1g rms at 30 Hz, in the vertical plane. The acceleration at the animals head was approximately 0.3g 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 8.0 kHz during the exposure and at regular intervals following exposure for 30 days. Figure 3 illustrates the median results at 8.0 kHz. The animals exposed to only vibration showed essentially no threshold shifts and their data has not been plotted. The range of thresholds for the animals exposed to just the impacts is shown as the solid pair of lines while the individual animals that were exposed to the combination stimulus are shown as symbols. At the 8.0 kHz test frequency the asymptomatic threshold shift (ATS) over the 10 day period of exposure is generally higher for the combination exposure. Similarly during the 30-day recovery period the combination exposure was slower to recover and yielded greater PTS. Figure 4 shows the PTS audiograms for each group. Across most of the test

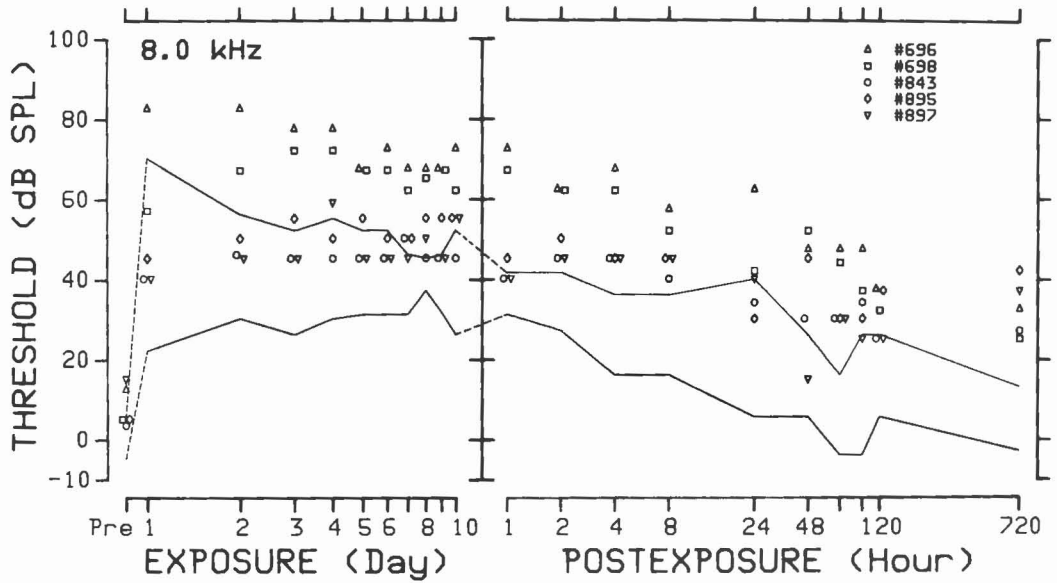


Figure 3. Hearing thresholds at 8.0 kHz for the animals exposed to noise alone (range shown by solid lines) and to noise and vibration (symbols).

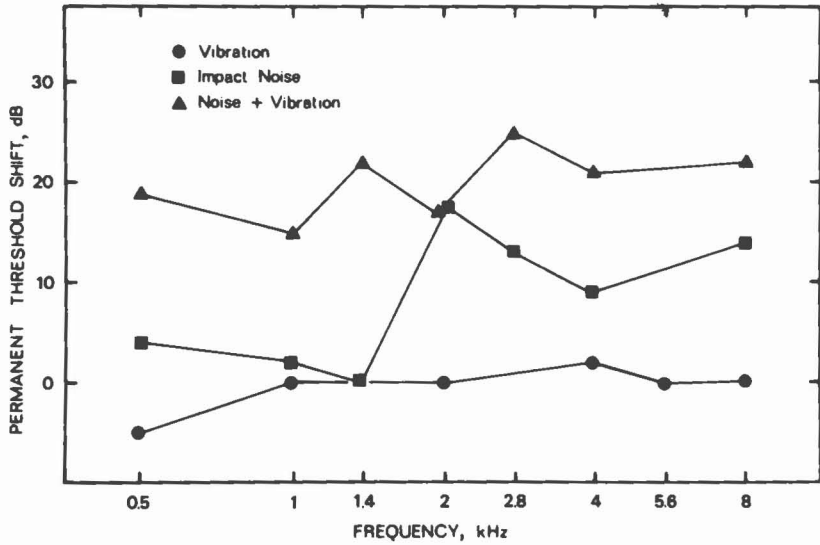


Figure 4. Median PTS for the three experimental groups from the 10-day exposure paradigm.

frequency range the animals exposed to the noise and vibration stimulus showed substantially greater PTS than those exposed to the control stimuli. The histological data confirmed these audiometric results by showing greater sensory cell losses in the animals exposed to the combination stimulus. As in the first experiment that was reviewed, 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 indicated that the dynamics of NIHL can be influenced by the simultaneous addition of whole body vibration.

The final and most extensive set of experiments that will be discussed have not been published previously. These experiments were undertaken in order to replicate the second set of data referred to above and to investigate the role of the various exposure parameters while keeping the total energy of the exposure the same.

Each group contained a minimum of 5 chinchillas, and all exposures lasted for 5 days. The combination exposures consisted of pairs of each of the noise and vibration exposures shown in Table 1; thus there were 6 control exposure conditions and 8 noise/vibration interaction paradigms. All animals were monaural, and all threshold data was acquired using the EAR. Details of the experimental protocol and noise analysis can be found in Roberto et al., (1985). Figures 5 through 8 summarize the basic audiometric results from this series of experiments. In each figure the upper panel illustrates the ATS measured at

Table 1. The exposure conditions used in the 5-day noise and vibration experiments.

<u>Continuous Noise</u>	<u>Impact Noise</u>	<u>Vibration</u>
95 dB; 0.5 kHz OBN	113 dB peak SPL 1/1s	20 Hz; 1.3 g rms
	119 dB peak SPL 1/4s	(0.3 g @ Head)
	125 dB peak SPL 1/16s	30 Hz; 3.0 g rms (0.4 g @ Head)

0.5, 2.0 and 8.0 kHz for each exposure condition; the lower panel shows the PTS audiogram for each exposure condition. The following points can be made from these figures: (1) The two

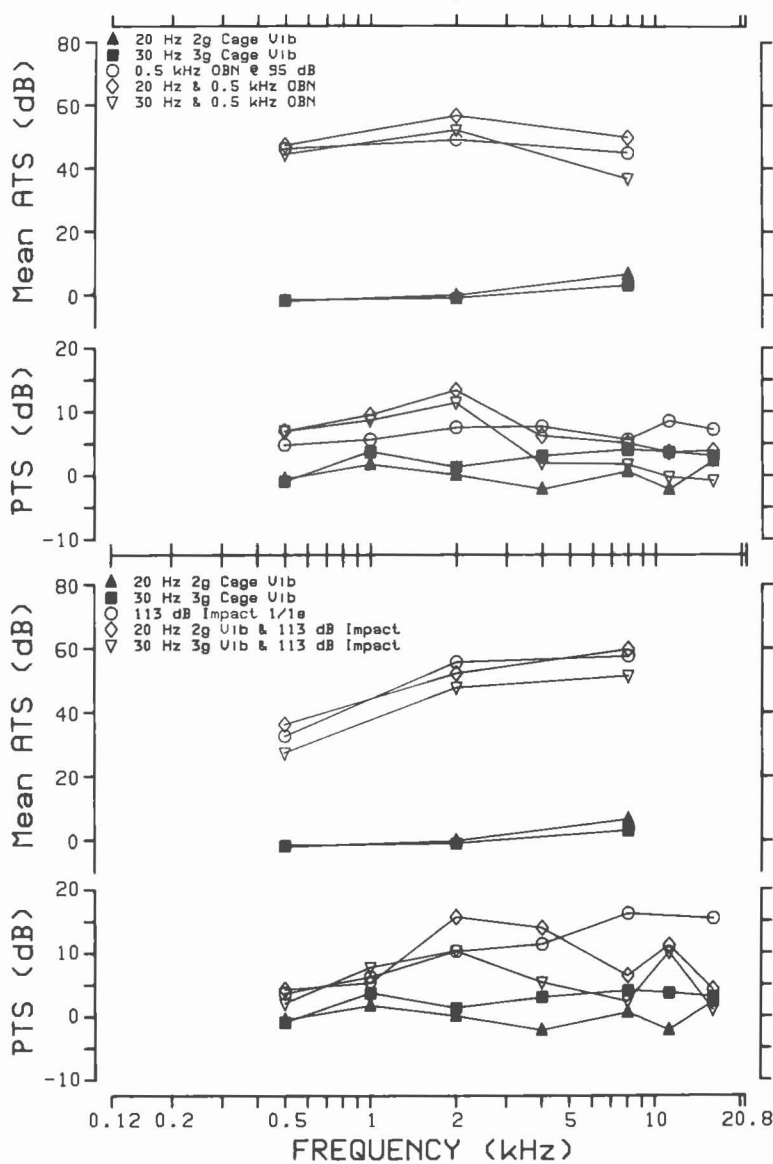


Figure 5. Mean ATS and PTS (upper pair) measures in the 95 dB SPL paradigm.

Figure 6. Mean ATS and PTS (lower pair) measures in the 113 dB peak SPL paradigm.

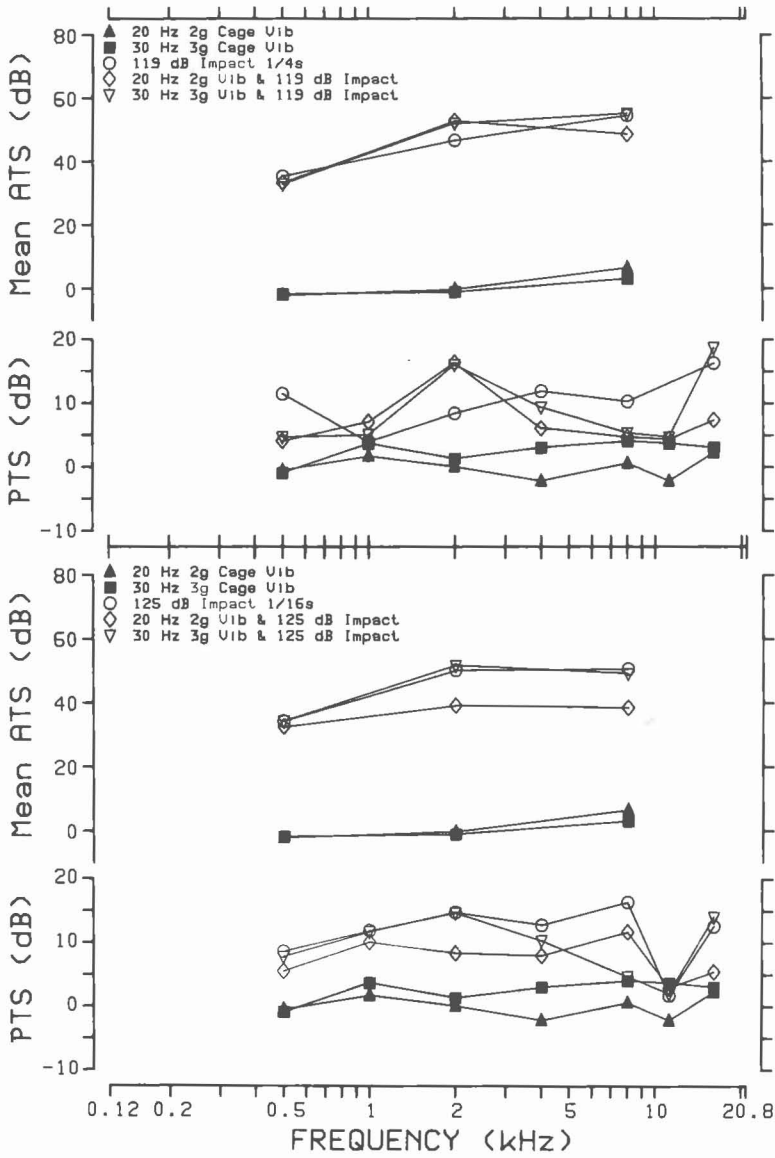


Figure 7. Mean ATS and PTS (upper pair) measures in the 119 dB peak SPL paradigm.

Figure 8. Mean ATS and PTS (lower pair) measures in the 125 dB peak SPL paradigm.

vibration exposures showed no significant audiometric response in either the ATS or PTS measures. (2) The three impact noise exposure conditions, which were balanced to produce approximately equal total energy exposures, produced the same levels of ATS and no statistically different levels of PTS i.e. under these exposure conditions the equal total energy concept applies. (3) The addition of the vibration to either the continuous noise or the three different impact noises did not alter the degree of ATS or PTS in any of the exposure paradigms. The histological analysis of these animals has not been completed. Thus although there are no substantial effects seen in the audiometric data the histological results especially in the apical 20% of the cochlea (i.e. below 0.5 kHz) may alter the final conclusions that can be drawn from these experiments.

The above audiometric results should have confirmed the data of Hamernik et al., (1981) but did not. 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 quite conceivable that our relatively short duration exposures are too short to provoke an interaction effect. 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.

CONCLUSIONS

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, i.e. the trade off between the severity (perhaps unrealistic severity) of an exposure and the duration of exposure that is practical. The animal experiments which were reviewed above are very limited and studies such as the Hamernik et al., (1980) need to be replicated. Given these caveats we do get the impression that consistent low level effects in humans can be measured although the effect is small; in animals, the effects if real, need relatively long exposure times or severe conditions of exposure to manifest themselves. Thus in conclusion, we must still agree with Humes (1984) and Von Gierke (1980) that more research is necessary before we can be sure of the role of vibration in the insidious process of NIHL.

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DISCUSSION

Comment from Ward: I just wanted to reinforce what Dr Hamernik has just indicated: namely, the lack of correlation between hair cell destruction and permanent threshold shifts in the individual chinchillas. All laboratories studying both phenomena report this discrepancy. So although both measures are valid indicators of auditory damage, it must be concluded that they are largely independent.

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PREFACE

This book is based on the scientific material that was presented in the Third International Conference on the Combined Effects of Environmental Factors (ICCEF 88) held in Tampere, Finland, 15-18 August 1988. The reports are of particular scientific significance and contain a wide range of useful information on the problems researchers in our field are currently concerned with. The conference was arranged under the auspices of The International Society of Complex Environmental Studies - ISCES.

The financial and material support of several enterprises, ministries, communities, individuals and the City of Tampere made it possible to arrange the conference and to edit and publish this material. It is not an overstatement to say that only with this support it was possible to create the warm, inspired and enlightening scientific and social programme to suit the exceptional scientific merits and international esteem of the participants. The high quality and friendly personnel of Hotel Ilves also made their contribution to the fruitful atmosphere of the conference. In addition to the conference itself most of the participants also attended to a post-conference tour to Heinola. The time at Heinola will remain in our memories like an old saga and remind us of the hospitality, the pure nature, and the air of poetry and adventure we experienced there.

A local Organizing Committee with representatives of different fields was established to take care of the preparations. The practical arrangements, in turn, were entrusted to a Board of Secretaries composed of the members of my research team and my friends. All these people together with the conference participants contributed to the fine success of the conference.

Olavi Manninen
Docent
ICCEF 88 President