

Chapter 37

Noise-Induced Hearing Loss: Implication for Tinnitus

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Keypoints

1. Noise-induced hearing loss (NIHL) is often associated with tinnitus.
2. The shape and depth of the audiogram in patients with NIHL varies considerably.
3. Characteristics of tinnitus (sensation level, pulsatile versus continuous, perceived pitch) also vary widely across individuals.
4. The relationship between the pattern of hearing loss and the characteristics of the tinnitus is complex and a relevant topic of research.
5. This chapter focuses on three topics relevant to NIHL and tinnitus:
 - (a) The relationship between the parameters of a noise exposure and the resulting hearing loss.
 - (b) The cochlear pathologies underlying permanent hearing loss and temporary hearing loss and how they differ.
 - (c) Noise-induced tinnitus and the animal modeling of tinnitus used to study the relationship between noise and tinnitus.

Keywords Temporary threshold shift • Permanent threshold shift • Kurtosis • Noise interactions • Tinnitus

Abbreviations

ATS	Asymptotic threshold shift
CNS	Central nervous system

EAM	External auditory meatus
GPIAS	Gap-prepulse inhibition of the acoustic startle
IHC	Inner hair cell
NBN	Narrow band noise
NIHL	Noise-induced hearing loss
NIOSH	National Institute for Occupational Safety and Health
OHC	Outer hair cell
OSHA	Occupational Safety and Health Administration
PTS	Permanent threshold shift
TTS	Temporary threshold shift

Introduction

Hearing loss from exposure to noise can either be temporary or permanent, depending on the level or duration of the exposure. The audiological symptoms associated with both noise-induced temporary threshold shift (TTS) and permanent threshold shift (PTS) include an elevation in hearing thresholds with particular vulnerability in the 3–6 kHz region; decreased frequency resolution and increased vulnerability to masking; abnormal growth of loudness; compromised temporal processing (i.e. decreased temporal summation of acoustic power and increased forward masking); and, of course, tinnitus (see review by Henderson et al. [1]).

There have been scores of studies on the relationship between noise exposure, the resultant hearing loss, changes in cochlear tuning, and the pathological basis for the corresponding audiometric symptoms (see Review articles by Saunders et al., Lieberman, Henderson and Hamernik [2–4]). However, our understanding of the biological basis of tinnitus is not as well understood (see review by McFadden and Wightman [5]).

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Tinnitus is a particularly interesting problem because noise exposures primarily damage the auditory periphery (cochlea) while evidence of tinnitus is often clearly central in origin. A fundamental question is what the changes in the operation of the cochlea that leads to a phantom perception generated in the central nervous system (CNS).

Acoustic parameters of Noise-Induced Hearing Loss (NIHL)

A review of the relationship between the parameters of noise exposure and temporary or permanent hearing loss is a reasonable place to begin an examination of the relation between noise exposure and tinnitus.

Temporary Threshold Shift (TTS)

Exposure to loud sound can lead to acute TTS, or if the noise is loud enough or long enough the hearing loss

can be PTS. The most comprehensive study of TTS was done in the 1940s and 1950s by Hallowell Davis [6] and his distinguished colleagues. They systematically studied the relationship between the acoustic variables of *frequency*, *intensity*, and *duration* and the perceptual correlates of loudness changes, pitch coding, and tinnitus.

A summary of their findings is schematically illustrated in Fig. 37.1 and includes the following results: (1) Exposure to pure tones or noise above 90 dB SPL can shift an individual's hearing threshold; (2) The magnitude of the hearing loss caused by a specific tone depends on the frequency of the tone, i.e. high frequencies such as 2,000 and 4,000 Hz caused a larger threshold shift than low frequencies (500 Hz). Note that the 500 Hz tone caused a broad hearing loss that was roughly equal in magnitude to the 2,000 and 4,000 Hz tones, but that the 500 Hz tone required a 32-min exposure while the 2,000 and 4,000 Hz tones required only 4-min exposures to elicit the same threshold shifts (Fig. 37.1a); (3) The peak of threshold shift in the audiogram was typically 1/2 to 1 octave above the frequency of the exposure (Fig. 37.1a-c); (4) The magnitude of TTS grew

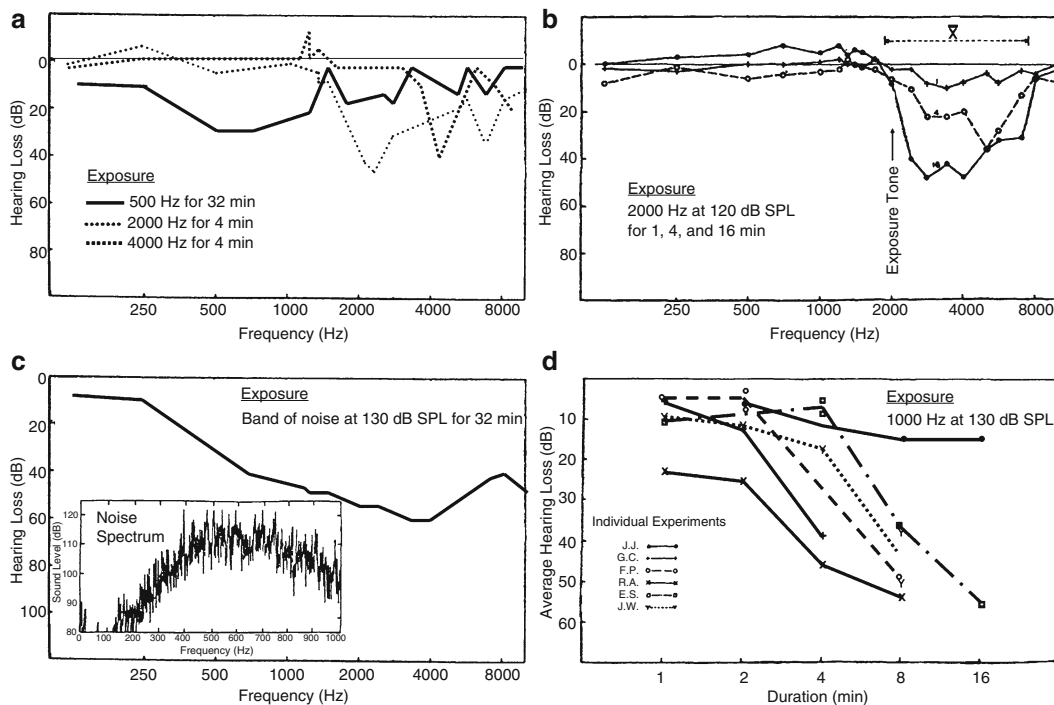


Fig. 37.1 Pattern of TTS from exposure to tones and noise. (a) Average TTS following exposure to either 500, 2,000, or 4,000 Hz; (b) growth of hearing loss for 2000 Hz tone at 120 dB SPL for 1, 4,

or 16 min; (c) average hearing loss from exposure to band of noise (*insert*) at 130 dB SPL for 32 min; (d) individual subject's exposure to 1000 Hz at 130 dB SPL. Adapted from Davis et al. [6]

with duration of exposure; (5) There was substantial inter- and intra-subject variability (Fig. 37.1b). One subject develops less than 15 dB of TTS after 16 min while another subject develops 50 dB after only an 8-min exposure to the 1,000 Hz tone. The variability across subjects is especially puzzling given that they all had the same pre-exposure audiogram and received exactly the same noise exposure; (6) Wide band noise caused a pattern of hearing loss with a “notch” or peak ranging between 3 and 6 kHz. Since the external auditory meatus (EAM) acts like a $\frac{1}{4}$ wave resonator, the actual location of the notch (3, 4, or 6 kHz) partially depends on the length of the subject’s EAM. Larger subjects with longer EAMs tend to have notches at lower frequencies, while smaller subjects with shorter EAMs tend to have notches at higher frequencies.

The authors used binaural loudness balancing techniques to compare the loudness between an exposed and non-exposed ear. They reported a change in loudness with TTS (i.e. the degree of loudness shift is greater at low sensation levels, but the difference is reduced or disappears at high levels of stimulation). This phenomenon has been termed ‘recruitment’ [7]. With regard to frequency coding, they reported a diplacusis (i.e. for the same stimulus, the normal and ear with TTS develop different pitches). Finally, without analyzing the observation, they reported that a number of the subjects developed a buzzing or ringing in their ears which has become known as tinnitus. The tinnitus following a pure tone exposure was reported to have a much more consistent and defined pitch than the tinnitus following a noise exposure. Most of the subjects completely recovered. However, several were left with a permanent hearing loss. The results of Davis et al. [6] on the development of TTS have been expanded and confirmed by a number of investigators (series summary by Ward [8]). The early collection of research raises several questions. What is the relation between TTS and PTS in cases of more extreme exposures? What are the underlying changes in cochlear anatomy and physiology that lead to the constellation of symptoms associated with TTS and tinnitus?

Given that the audiological symptoms are essentially the same for both the TTS and PTS, it is reasonable to assume that the underlying changes in the cochlea are similar between the two conditions. However, this assumption ultimately proves to be too difficult to confirm or deny. An interesting perspective on TTS and PTS is provided in the literature on asymptotic threshold

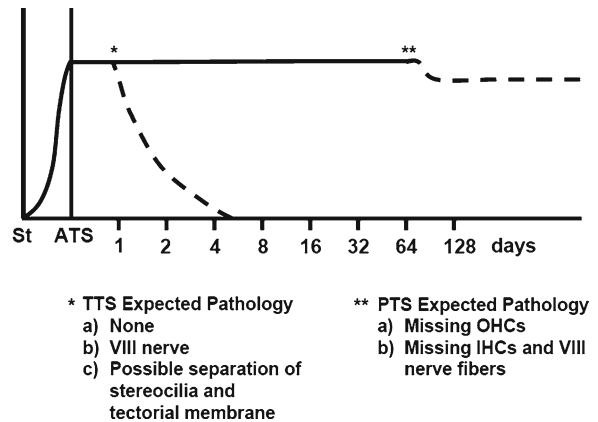


Fig. 37.2 Model of transition from temporary and permanent threshold shift using ATS paradigm

shift (ATS) [9–11]. ATS refers to the phenomenon where hearing loss grows over the first 8–24 h of a noise exposure. Hearing then stabilizes at an asymptotic level and remains at the same level for weeks or months of a constant noise exposure. If subjects are studied at different time points (i.e. 24-h exposure to 60 days), an interesting trend emerges (see Fig. 37.2). Both the 24 h subjects and the 60 day subjects have the same magnitude of threshold shift. When the 24-h subjects are removed from the noise, they begin to recover to normal sensitivity and suffer no PTS or cochlear damage. However, for subjects exposed to 60 days of noise, even though they have the same magnitude of threshold shift, when they are removed from noise they recover slowly and only partially [12]. The transition from TTS to PTS illustrates how the conditions produce the same apparent threshold shift on the audiogram but with significant differences in the underlying pathology.

Pathology of TTS

The term “TTS” suggests the pathological changes might be insignificant. However, in cases of TTS the cochlea can suffer a fairly wide spectrum of possible anatomical changes, from substantial temporary pathological damage to subtle, non-symptomatic pathological changes.

Nordmann et al. [13] have shown that TTS exposure can be associated with a disconnection between the tallest outer hair cell (OHC), stereocilia, and the tectorial membrane due to changes in the structure of the organ of Corti. The assumption is partial recovery

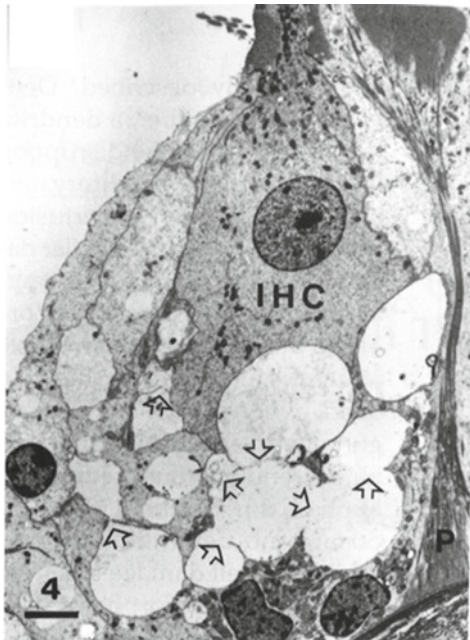


Fig. 37.3 Inner hair cell after noise exposure. Note *arrows* identify swollen VIII nerve dendrites

results from structural recovery of the supporting cells and eventual reattachment of the stereocilia to the tectorial membrane. Also, the VIII nerve dendrites under the inner hair cells (IHCs) suffer excitotoxicity [14, 15], leading to de-afferentation of the IHC (Fig. 37.3). However, studies of IHC/VIII nerve fiber excitotoxicity with kainic acid (which mimics the effects of noise) show that the swollen VIII nerve dendrites recover and become functional again [16]. Consequently, part of TTS is likely due to the repairable excitotoxicity. Finally, the cochlea can sustain permanent losses of OHCs that are not sufficient in number to impair threshold detection. Collectively, the pathology associated with TTS may be repairable, or the permanent changes are too minor to be detected with typical audiological measures, so PTS would not be observed audiometrically.

Permanent Threshold Shift (PTS)

The relationship between the parameter of a noise and PTS are similar to TTS, but the levels required to cause PTS are higher or the durations are longer.

For humans the threshold for causing PTS with years of daily repeated noise exposures is approximately 85 dBA [17]. The assumption is that repeated daily exposure for 5–10 years will lead to PTS. The predictive course of NIHL prepared by ISO1999 is associated with large degrees of variability, consequently making the prediction for an individual questionable. The underlying assumption of the ISO1999 procedure is that the degree of HL is related to the total energy of the exposure. The U.S. Occupational Health and Safety Administration (OHSa) considers 85 dB(A) to be the “action” level where workers are monitored and 90 dB(A) is permissible for 8 h. For each 5 dB increase in level, there is a halving of the duration (for example 95 dB(A) for 4 h is equivalent to 100 dB(A) for 2 h). In 1995, the National Institute for Occupational Safety and Health (NIOSH) prepared a recommendation for a noise standard that has a maximum tolerable exposure of 85 dB for 8 h and a 3 dB trading ratio (88 dB(A) for 2 h equals 91 dB(A) for 1 h), but the NIOSH amendment has not been enacted into law.

The effects of continuous and impulse/impact noise are different. For example, in the relationship between the noise level and ATS or PTS, for either laboratory studies or in large demographic studies, hearing loss grows at the rate of 1.7 dB for each dB of noise above the threshold for damage [18]. The relationship between the noise level and hearing loss changes dramatically with exposure to impulse, impact, and high level bursts of continuous noise. To illustrate, chinchillas were exposed to impact noises of equal energy, i.e. the impacts’ peak levels \times the number of repetitions were counterbalanced so that each group had equal amounts of acoustic energy (102–135 dB SPL) (Fig. 37.4) [19]. As seen in Fig. 37.4, the hearing loss was approximately the same for exposure to impacts of 102–119. However, above 119 dB, the hearing loss increased dramatically in spite of the equal energy that each exposure had. The interpretation of these results is that for the lower levels 99–119 dBA, the impact noises caused the same cochlear damage and HL because the ear was responding to the total energy of the exposure. However, at higher levels the hearing loss is more related to the peak level of the impact. This can suggest direct mechanical damage. This and a number of experiments with high level exposure [11, 20] lead/led to the formulation of the “critical level” hypothesis [21]

Fig. 37.4 Average PTS at 4,000 Hz for chinchilla exposed to impact noise ranging from 107 to 143 dB peak SPL at either rate of 1/s (a) or 4/s (b). From Henderson and Hamernik [19]

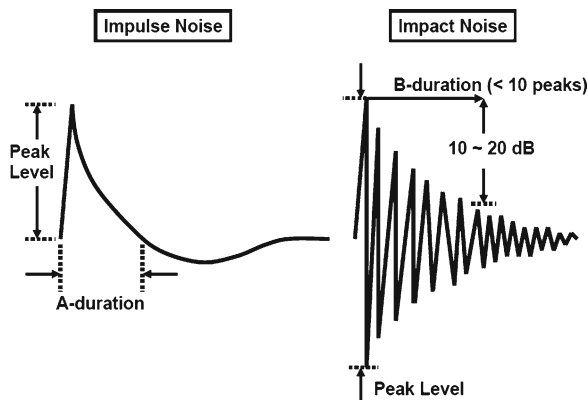
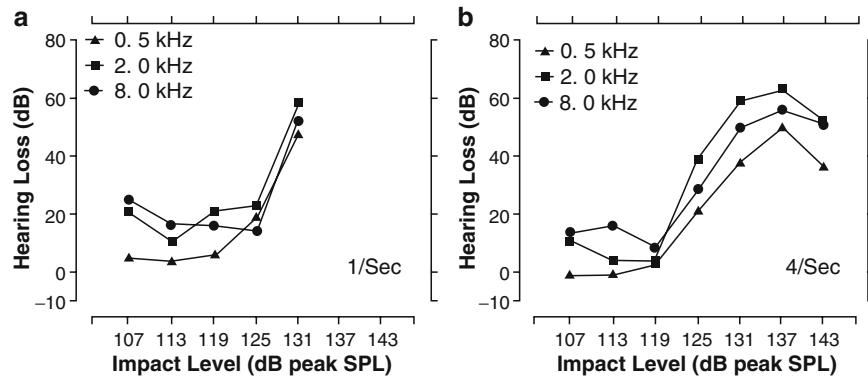


Fig. 37.5 Schematic of impulse and impact noise. Impulses are created by explosive phenomenon while impacts are consequence of hard object colliding. From Henderson and Hamernik [22]

which assumes that high level exposures damage the ear causing direct mechanical failure.

The threshold of direct mechanical failure or “critical level” depends on the duration of the exposure. For example, for gunfire with peak levels of approximately 140–165 dB pSPL and impulse durations of approximately 1 ms, the “critical level” is between 150 and 155 dB pSPL peak level. For impact noise with duration of 200 ms, the “critical level” for mechanical failure is approximately 120 dBA. Short duration impulse and impact noises are shown in Fig. 37.5 [22].

When a noise exceeds the “critical level”, damage to the cochlea is immediate and direct, as seen Fig. 37.6. This figure illustrates a number of pathologies associated with exposure to “gunfire” and the resultant mechanical failures. These failures range from dramatic damage as seen in Fig. 37.6a [23], where the organ of Corti is ripped from the basilar membrane

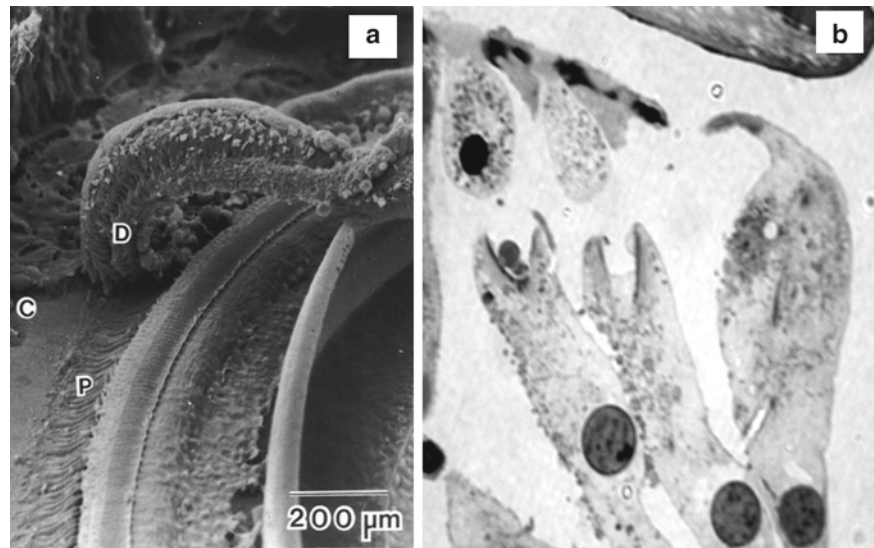
(Note the split of the cuticular plate between first and second row of OHC; this type of damage allows endolymph to bathe the OHCs and cause their death), to a more subtle damage where OHCs are separated from their Deiters’ cups (Fig. 37.6b).

When a continuous noise exposure is terminated, recovery of function proceeds almost immediately in the affected cochlear region and hearing sensitivity recovers to baseline or to a stable level of PTS. However, with exposure to high level impact/impulse noise, the time course of recovery may be complicated and tri-phasic. For example, there is a rapid recovery for 15 min–1 h, a rebound where hearing loss increases over a 2–6 h period, and then finally a slower recovery to a stable level of hearing or hearing loss.

NIHL and Tinnitus

There is no question about the strong correlation between NIHL and tinnitus. In the review of the clinical and experimental literature on noise and tinnitus for the military, it is stated, “... noise doses associated with hearing loss are likely to be associated with tinnitus”. However, they were not specific about the exact relationship between HL and tinnitus, i.e. the percentage of people with hearing loss that suffer tinnitus or the magnitude of the hearing loss and tinnitus. They did report that exposure to impulse noise is more likely to produce tinnitus than exposure to continuous noise. More recently, a study evaluating soldiers exposed to blast trauma in Iraq and Afghanistan found that 49% of combat personnel exposed to blasts developed tinnitus. Moreover, tinnitus ranked as the chief audiologic complaint. These new findings provide direct evidence

Fig. 37.6 Chinchilla exposed to impulse noise of 155 dB peak SPL. (a) Mechanical damage when organ of Corti (d) is ripped from basilar membrane (c) From Hamernik et al. [23]; (b) OHC separated from Dieter's cells



of noise overexposure and subsequent tinnitus. However, more studies are needed to characterize the persistence and features of this tinnitus.

The correlation between NIHL and tinnitus remains far from perfect. There is still a large gap in our knowledge on how peripheral damage (by noise) in the cochlea leads to abnormal neural activity in the brain and the false perception of tinnitus. Two possible causes of tinnitus may be secondary neural degeneration (i.e. VIII nerve to cochlear nucleus, etc.) in the CNS or changes in the balance of excitation and inhibition in auditory pathways. Morest and colleagues [24] have reported neural degeneration in the auditory system secondary to cochlear degeneration caused by noise. The implication of the noise-induced CNS degeneration for perception is not clear. TTS, which presumably does not cause CNS degeneration, can also cause tinnitus. The alternative hypothesis for tinnitus and NIHL is a change in balance of excitation-inhibition. Salvi and colleagues [25] have experimental data showing rapid changes in the inferior colliculus and auditory cortex after NIHL. After traumatic noise exposure, the spontaneous activity of the VIII nerve remains normal, but the spontaneous activity of the cochlear nucleus can increase with “bursts” of neural responses [26]. In addition, studies of evoked potentials (inferior colliculus, auditory cortex) after acute noise exposure show an elevation of threshold as well as enhancement of the amplitude of the evoked potential [25, 27]. These findings suggest that the hearing

loss caused a release of inhibition. With the development of animal models of tinnitus, we can expect more information for the relationship between cochlear pathology, changes in neural firing patterns, and tinnitus.

Animal Models of Tinnitus

Jastreboff was the first to develop an animal model of tinnitus over 20 years ago. The initial studies looked at the effects of high doses of sodium salicylate and the development of transient tinnitus in rats. The model used a creative and straightforward lick-suppression paradigm that required discrimination between real sound and quiet. When the animals are exposed to high-dose salicylate, they fail to discriminate between quiet conditions and audible sound conditions. Since the animals failed to perceive the quiet intervals they continued to drink in the presence of a quiet/calm state. The inability to perceive the quiet state is interpreted to mean that the animals are experiencing tinnitus induced by the salicylate.

A similar technique was developed by Heffner. However, there were a number of notable differences. Heffner used an operant food-reinforced behavioral technique whereby gerbils could avoid shock if they refrained from responding during quiet intervals. Responding was allowed during sound. More importantly, Heffner used

varying levels of unilateral tone trauma (10 kHz, 124 or 127 dB SPL, for 0.5, 1, 2, or 4 h) to induce tinnitus. The key findings were threefold. First, regardless of sound intensity or duration not all animals developed tinnitus, highlighting individual differences in susceptibility of tinnitus. Second, the probability of tinnitus increased as a function of sound intensity. Finally, only long duration, high intensity tone trauma resulted in tinnitus. Tinnitus was seldom reported for low intensity or short duration trauma. Thus, there is a direct relationship between the trauma duration or level and the probability of developing tinnitus.

The most recent animal model of tinnitus relies on the acoustic startle response to a brief startling broadband or band-pass noise. Presentation of this stimulus reliably induces a large motor startle in rats that can be measured on a pressure sensitive plate. However, when a brief low intensity signal is presented before the startling sound, a significant reduction in startle amplitude is observed. This is known as pre-pulse inhibition. The acoustic signal preceding a startling sound that is audible serves to reduce the startle response. Another way of inhibiting the startle is by presenting a silent gap in a low-level continuous background noise before a startling stimulus. In this paradigm, there is always a background band-pass noise running throughout the session. At random intervals, startling sounds are presented and elicit large startle responses. On some trials, silent gaps are embedded in the continuous noise 100 ms before the startle sound. If these are detected, the amplitude of the startle response is decreased. This is known as gap-prepulse inhibition.

We have performed a number of preliminary studies to evaluate the suitability of the gap-prepulse inhibition of the acoustic startle (GPIAS) model on detecting the presence of noise induced tinnitus. When we pooled the results across a number of preliminary studies we found a direct correlation with the level of noise trauma and the probability of chronic tinnitus. When animals were exposed to a 123 dB SPL (12 kHz, NBN, BW=100 Hz, 2 h) noise exposure (Fig. 37.7), approximately 33% showed evidence of tinnitus. Raising the noise exposure level to 126 dB SPL increased the percentage of animals with evidence of tinnitus to 75%. In contrast, when salicylate was used to induce transient tinnitus the incident level was 100%. As not all animals were tracked long term, the data from noise exposure is related to evidence of tinnitus of 2–15 days post noise. Further studies are

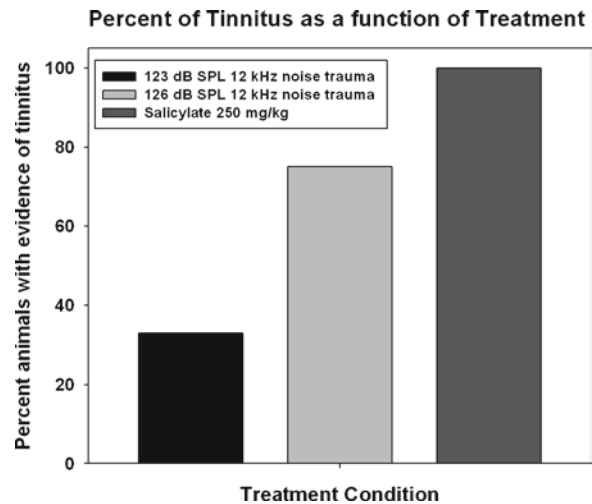


Fig. 37.7 The percentage of animals with evidence of tinnitus after unilateral noise trauma increased from 33% at 123 dB SPL (NBN centered at 12 kHz, BW = 100 Hz, duration of 1 h) to 75% at 126 dB SPL (NBN centered at 12 kHz, BW = 100 Hz, duration 1 h). Pharmacologically-induced transient tinnitus with a high dose of sodium salicylate (250 mg/kg, 1 h pre-session, i.p.) yielded evidence of tinnitus in all the animals tested. Group sizes were 12, 12, and 24 rats (Harlan SASCO Sprague Dawley, adult males, mean body weight 375 g)

needed to determine the percentage of animals that develop long-term chronic tinnitus.

In addition to the duration of tinnitus, we were also interested in the pitch of noise-induced tinnitus. Evidence from human studies suggests that there is a relationship between the frequency of the maximal hearing loss and the pitch of the tinnitus. When animals were unilaterally exposed to 12 kHz noise at 123 dB SPL, tinnitus was observed between 12 and 16 kHz (Fig. 37.8). Immediately after the noise exposure, however, animals failed to detect gaps at multiple frequencies. This effect disappeared within 24 h, but evidence of tinnitus remained in the 12–16 kHz region. Increasing the level of the unilateral 12 kHz NB noise to 126 dB SPL led to a nearly complete loss of gap-induced prepulse inhibition at 16 kHz (Fig. 37.9). Changing the center frequency of the noise from 12 to 16 kHz resulted in the maximum loss of gap-induced prepulse inhibition occurring at 20 kHz instead of 16 kHz (Fig. 37.10). Audiometrically, these changes in the “pitch” of the tinnitus would seem to be related to a shift in the location of maximal OHC trauma in the cochlea, but that relationship has yet to be confirmed anatomically.

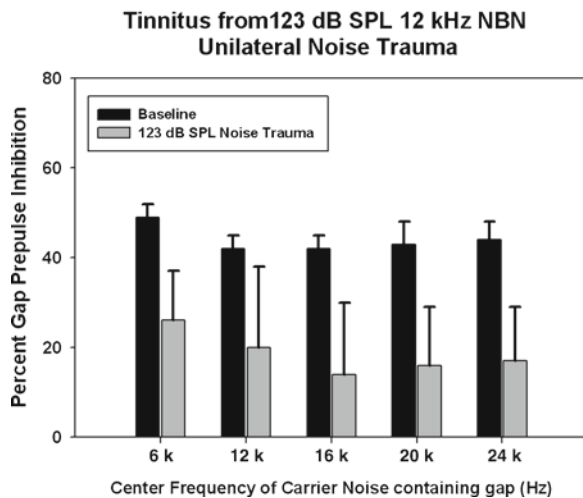


Fig. 37.8 The percentage gap prepulse inhibition before and after 123 dB SPL unilateral noise trauma (NBN centered at 12 kHz, BW=100 Hz, duration of 1 h). Baseline shows robust inhibition (40–50%) of the startle response when a gap is presented before the startling stimulus (100 ms gap, 100 ms before a 115 dB SPL, 20 ms Band-pass noise 5–10 kHz). In contrast, post exposure gap prepulse inhibition decreases by more than 50% with gaps in 16 kHz carrier NBN showing the largest drop. The decrease in the ability to detect the gap was interpreted as evidence of tinnitus

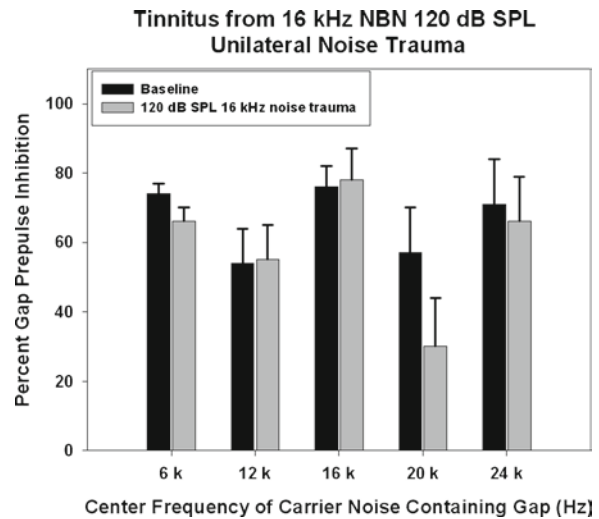


Fig. 37.10 The percentage gap prepulse inhibition before and after 120 dB SPL unilateral noise trauma (NBN centered at 16 kHz, BW=100 Hz, duration of 1 h). Post exposure gap prepulse inhibition decreases by more than 50% at 20 kHz. The decrease in the ability to detect the gap was interpreted as evidence of tinnitus centered primarily around 20 kHz

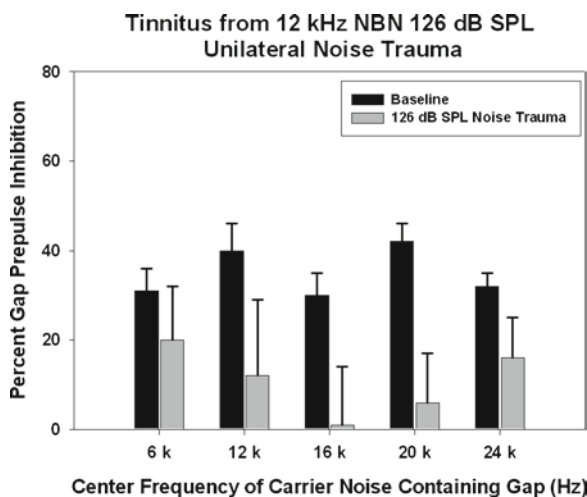


Fig. 37.9 The percentage gap prepulse inhibition before and after 126 dB SPL unilateral noise trauma (NBN centered at 12 kHz, BW=100 Hz, duration of 1 h). Baseline shows significant inhibition (30–40%) of the startle response when a gap is presented before the startling stimulus (100 ms gap, 100 ms before a 115 dB SPL, 20 ms Bandpass noise 5–10 kHz). In contrast, post exposure gap prepulse inhibition decreases by more than 50%, with gaps in the 16 kHz carrier NBN showing the largest drop resulting in them being virtually indistinguishable from trials with no gaps. The decrease in the ability to detect the gap was interpreted as evidence of tinnitus centered primarily around 16 kHz

One limitation of the GPIAS model is that the startle response is dependent on binaural hearing. If the unilateral acoustic trauma is excessive, the startle stimulus is less effective at producing a strong startle response. Because of this, it is advantageous to limit the hearing loss to the high frequencies. There is also clinical value of limiting the NIHL as tinnitus induced by noise tends to be perceived at higher frequencies. The startle stimulus can also be moved so that it is a band-pass noise within the audible range of even the exposed ear. This can increase the effectiveness of the startle stimulus following the exposure.

Despite the gaps of knowledge that still exist regarding the biological basis for tinnitus and the basis for tinnitus susceptibility, a number of research groups have been steadily narrowing the gaps. Progress is likely to accelerate as animal models continue to be developed and act as a platform for basic science and pre-clinical drug therapy models. However, challenges still remain for understanding tinnitus. However, NIHL is known to be one of the key catalysts for the development of chronic tinnitus. A concerted effort using animal models, human and animal imaging studies, physiological, behavioral, and pharmacological studies will likely enhance our knowledge base and move us closer to providing strategies to reduce the impact of tinnitus.

Conclusions

The hearing loss caused by exposure to noise can be either temporary or permanent. In addition to a loss of hearing sensitivity, traumatic noise exposure degrades signal detection in background noise, reduces the dynamic range of loudness, and can induce tinnitus. The deleterious effects of noise are related to each of the primary dimensions of sound: frequency, intensity, and duration of exposure. Our current noise standards are over 40 years old (from 1968), and do not reflect modern scientific research or our understanding of the effects of noise. For example, research has shown that certain types of noise exposure (combinations of continuous noise with impulse/impact noise) or noise combined with ototoxic solvents pose an increased risk to hearing compared with simple continuous noise exposures. Since the initial noise legislation of 1968, much has been learned about the mechanisms through which noise causes hearing loss, and in the last 10 years, much progress has been made in unraveling the mystery of noise-induced tinnitus.

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