Chapter 161: Auditory Dysfunction from Excessive Sound Stimulation

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Along with the hearing loss caused by aging, noise-induced hearing loss (NIHL) is one of the most common adult problems treated by the typical otolaryngologist - head and neck surgeon. Recently, it was estimated that approximately one third of the 28 million Americans exhibiting some degree of deafness have hearing losses caused by exposure to high-level sounds (Office of Medical Applications of Research, 1990). Future predictions of the growth of NIHL are even more unsettling given the estimated 20 million individuals in the USA alone (that is, about 25% of the work force (Suter and von Gierke, 1987) who are said to be habitually exposed to potentially hazardous sounds.

Because of its prevalence, exposure to loud sound has many far-reaching effects. These influences range from a personal loss of communication abilities that leads to a reduction in the quality of life and social well-being, to an economic burden involving costly compensatory payments that affects society as a whole. Because the industrialized world's accepted standard of living is based on mechanization, it is certain that noisy environments will persist for many years. Although the most common source of the damaging agent is found in the workplace, the increasingly noisy sounds of everyday life encountered in the home and during leisure-time activities make NIHL a predicament that affects all members of society, regardless of age, gender, or ethnic group. This chapter presents and discusses the most recent perspectives on the effects of noise on hearing that address the scientific and practical aspects of the affliction.

Measurement of Noise

The term *noise* is often used to designate an annoying or unwanted sound. In the hearing sciences, the expression has come to mean any excessively loud sound that has the potential to harm hearing. The temporal patterns of environmental noise are typically described as continuous, fluctuating, intermittent, or impulsive (OSHA, 1981). Continuous or steady-state noise remains relatively constant, whereas fluctuating noise rises and falls in level over time, and intermittent sounds are interrupted for varying time periods. Impulsive or impact noises caused by explosive or metal-on-metal mechanical events have rapidly changing pressure characteristics consisting of intense, short-lasting (that is, msec) wave fronts, followed by much smaller reverberations and echoes that can last for many seconds. The amount of noise, usually referred to as the *sound pressure level (SPL),* is conventionally measured by a sound-level meter in decibel (dB) units using a weighting formula called the *A-scale.* The A-scale measure of sound level essentially mimics the threshold-sensitivity curve for the human ear, so that low- and high-frequency components are given less emphasis as auditory hazards. Standard sound-level meters have electronic networks designed to measure noise magnitude automatically in decibels weighted by either the A-scale (dBA) or other, less-used scales, such as the B- and C-scales, which simulate equal-loudness contours for human ears at medium and high SPLs, respectively, or the linear scale, which enables the unmodified signal to be analyzed.

Another instrument used to measure noise exposure in the workplace is the commercial audiodosimeter, which provides a readout of the dose or the percent exposure. The logging dosimeter integrates a function of sound pressure over time and calculates the daily (8-hour) dose with respect to the current permissible noise level for a continuous noise of 90 dBA SPL lasting 8 hours. The response characteristics of standard sound meters are not adequate to describe the level of impulsive noise. Consequently, specially designed impulse meters or oscillographic instruments having high temporal-resolution capabilities are used to make these measurements.

Nature of the Loss

For many years it has been known that intense levels of noise cause irreversible damage to the hearing mechanism. The primary site(s) of damage is at the level of the sensory receptors of the inner ear's cochlea, that is, the inner and outer hair cells of the organ of Corti. In some instances, supporting-cell elements can also be affected. Depending on the physical attributes of the exposure stimulus (that is, the intensity, frequency or spectral content, duration or schedule, and time-varying characteristics), noise can cause damage to hair cells ranging from total destruction, to effects evident only in the ultrastructure of specialized subcellular regions (for example, the fusing or bending of the stereocilia). Whenever degenerative process or structural modifications to the end organ reach a significant level, an associated reduction in hearing capability can be detected.

Traditionally, hearing loss caused by excessive noise has been separated into two distinct categories. One type, called *acoustic trauma,* is caused by a single, short-lasting exposure to a very intense sound (for example, an explosive blast) and results in a sudden, usually painful, loss of hearing. The other type of hearing loss is commonly referred to as noise-induced hearing loss and results from chronic exposure to more moderate levels of sound. A great deal more is known about the anatomic processes underlying both the symptoms of and recovery from acoustic trauma than is known about NIHL. It is well established that a single exposure to a severe sound can result in direct mechanical damage to the delicate tissues of the peripheral auditory apparatus, including components of the middle ear (tympanic membrane, ossicles) and inner ear (organ of Corti). In contrast, regular exposure to less intense, but still noisy sounds, involves the insidious destruction of inner-ear components that eventually and unavoidably leads to an elevation in hearing levels. Such permanent threshold shifts are often accompanied by a number of other common symptoms of hearing dysfunction including tinnitus, loudness recruitment, and frequency distortion or diplacusis.

A patient with NIHL commonly consults a physician because of difficulties in hearing and understanding ordinary speech, especially in the presence of background noise. Many variations may be found in the detailed configuration of the audiogram of a noise-damaged ear, depending on the temporal and spectral distribution of the noise stimulus, as well as on the stage of hearing loss. The pattern of hearing loss most commonly associated with the onset stages of NIHL is illustrated in Fig. 161-1. The initial region of impairment involves the sensitive midfrequency range, between 3 and 6 kilohertz (kHz), and the corresponding impairment is classically described as the "4-kHz notch". This particular pattern of maximal hearing loss, with little or no low below 1 kHz, typically appears regardless of the noise-exposure environment. The audiogram in Fig. 161-1 also demonstrates the sensorineural

aspect of NIHL, in that thresholds for bone-conducted stimuli are essentially identical to those of air conduction. Note also that the profile of noise-induced threshold hearing is usually symmetric for both ears, for individuals who have been working in noisy industrial settings. It is not uncommon, however, for other forms of noxious sound, such as the gunfire associated with sport shooting, to cause an asymmetric pattern of hearing loss. In this case, the ear pointed toward the source of noise (gun barrel), for example, the left ear of a right-handed shooter, would have worse hearing, by 15 to 30 dB or more, particularly at high frequencies, than the one directed away from the source, due to the protective head-shadow effect.

The development of a hearing loss caused by habitual exposure to moderately intense levels of noise typically consists of two stages. The first phase involves a decreased hearing acuity (that is, an increase in the threshold of sound detection) immediately after exposure that lasts for minutes to hours thereafter, and is referred to as a temporary threshold shift (TTS). After exposure, if the threshold shift does not recover, a second stage of cochlear damage has occurred, which is called a permanent threshold shift (PTS). In PTS, the elevation in hearing threshold is irreversible. Although the precise relationship between TTS and PTS remains unsettled, the concept of "equal-temporary effects", or the "principle of equinocivity" has been proposed, in which the magnitude of the initial reversible threshold shifts produced by a given exposure sets the upper limit for growth of PTS under the continual influence of the TTS sound. However, that PTS eventually develops from such repeated exposures suggests that the early, so-called TTS episodes are most likely accompanied by microscopic alterations to hair cells that go undetected by current assessment techniques.

One common observation in cases of NIHL has been that as the length of time of exposure to loud noise increases, hearing loss becomes greater and begins to affect adjacent higher and lower frequencies. For example, in a cross-sectional study of occupational NIHL, Taylor et al (1984) demonstrated the gradual loss of hearing sensitivity in men caused by an habitual exposure to the intense sounds of drop-forging machines. Fig. 161-2 illustrates the progressive effects of exposure tothe wide-band noise (Fig. 161-2, A) of two types of forging devices, hammers or presses, on the magnitude of hearing loss as the length of exposure increased. For the operators of both the press and hammer equipment (Figs. 161-2, B and C, respectively), the approximate 10- to 20-dB shifts, typically observed at the higher frequencies during the first 1 to 2 years of exposure, grew to be a 20-dB or greater loss, from 3 to 6 kHz, after a 3-year exposure, and a 40-dB or greater threshold shift, after an 8-year exposure. Detailed examination of the NIHL-growth curves of Figs. 161-2, B, and C, reveals that with continuing exposure, hearing loss worsened at the higher frequencies and spread to the lower frequencies. In addition, for average exposure times of < 10 years, hearing levels for the press and hammer operators, who were exposed to mean levels of 108 and 99 dB SPL, respectively, deteriorated similarly. However, for long-term exposures of 10 years or more, the results of Taylor et al (1984) indicated that hearing losses resulting from the hammer/impact noise were greater than those resulting from the more continous noise of the press equipment. Finally, a characteristic feature of NIHL, which is clearly documented in Figs. 161-2, B and C, is that it rarely exceeds 70 to 80 dB.

Johnsson and Hawkins (1976) were among the first investigators to describe the typical patterns of cochlear lesions in the inner ears of humans exposed chronically to different categories of loud sound. The photomicrograph of Fig. 161-3, illustrating cochlea, prepared as a soft-surface preparation, from a patient with a lengthy history of noise exposure, depicts some of the common histopathologic correlates of NIHL. The authors reported that the 50-year-old patient had worked intermittently over a 5- to 6-year period in an automobile-stamping plant, and that he had a long history of the recreational use of firearms. Note the sharp zone of transition in the basal end (right) from the normal-looking organ of Corti (a darkish stripe corresponding to the region of inner and outer hair cells), with its dense network of nerve fibers, to the complete absence of hair cells and their corresponding nerve fibers (the much lighter adjacent area). The lower portion of Fig. 161-3 graphically reconstructs the histopathologic features of this cochlea as a cytocochleogram by depicting the number of remaining hair cells, in the form of percentages, averaged over 1-mm sections. A typical finding in individuals exposed to the occupational noise exemplified in this case is the almost symmetric pattern of degeneration observed for the two ears. The inset at right, showing the patient's audiogram obtained about 1 year before his death, demonstrates the severity of the anatomic damage in functional terms by revealing an abrupt hearing loss for test frequencies above 2 kHz.

Examination of human temporal-bone specimens by a number of researchers (Bredberg, 1968; Igarashi et al, 1964; Johnsson and Hawkins, 1976; McGill and Schuknecht, 1976) has yielded documentation of the progressive stages of noise damage as depicted by conventional demographic data like those of Fig. 161-2. First, in the predictable sequence of events, a small region of hair-cell and nerve-fiber degeneration appears at a cochlear region corresponding to the 4-kHz notch. The discrete bilateral lesions gradually grow to involve a greater portion of the organ of Corti. Finally, as exposure to noise continues over years, the remaining sensory and neural elements in the basal end of the cochlea are destroyed, resulting in an abrupt loss of high-frequency hearing.

Mechanisms of Noise-Induced Hearing Impairment

Scientific interest in the damaging effects of excessive sound on hearing has a long history for a number of reasons. First, the experimental strategy of exposing animals to noise and examining their ears for the sites of the resulting acoustic injury has been used in the past as the independent variable in establishing some of our basic knowledge of hearing. With the use of noise as the damaging agent, frequency information relating to physical distance along the basilar membrane to the "best" frequency of the damaged region has provided a basis for understanding the tonotopicity of the cochlea, and the central projection of frequency-related information (for example, Eldredge et al, 1981). More recently, noise-damage strategies have been used to contribute to our understanding of the function of inner and outer hair cells by permitting differences in their central terminations in the ventral and dorsal cochlear nuclei, respectively, to be distinguished (Morest and Bohne, 1983). The major impetus behind an experimental interest in the effects of noise on hearing, however, stems from a desire to understand the fundamental processes by which exposure to loud sound leads to acoustic injury. By achieving an understanding of the mechanisms underlying NIHL, it is hoped that some means of preventing or predicting an individual's susceptibility to PTS can be developed.

The research literature on noise is voluminous. Initial experiments 50 years ago (for example, Lurie, 1942) were straightforward histologic studies based on the strategy of exposing animals to intense noises, followed by a general description of the resulting histopathology. More recent noise studies (eg, Lonsbury-Martin et al, 1987) have attempted to establish a structure/function relationship between anatomic damage and the inability to detect auditory signals. However, in this extensive literature, a great disparity in the experimental findings relating the effects of missing hair cells to the corresponding hearing sensitivity is sometimes apparent. For example, in the results of different studies it is not unusual to find normal hearing despite extensive hair cell losses (for example, Ades et al, 1974; Eldredge et al, 1973; Ward and Duvall, 1971), or severe losses of auditory sensitivity in the absence of any measurable effect on the cochlea (for example, Hunter-Duvar and Elliott, 1973; Moody et al, 1978). These disparate results and the confusion they cause are related to a number of factors including poor analysis of the problem, experimental error, and a failure to understand the limitations of the techniques used. Also, such experiments usually exposed the animals to a single noise at levels well above 100 dB SPL in an attempt to mimic damage patterns that develop in humans from intermittent exposure to much less intense noises over many years. Consequently, although early noise studies clearly demonstrated that the longer an animal was exposed to extreme levels of sound, the greater was the resulting cochlear injury, they contributed little to our knowledge about how NIHL develops in real-life situations.

Most recent work has made use of more realistic research paradigms, with intermittent stimuli of intensities and durations designed to approximate the effects of a working lifetime of exposure to occupational noise. These studies have been developed sequentially so that a thorough understanding of a particular effect has been achieved. Bohne and Clark (Bohne and Clark, 1982; Clark and Bohne, 1978) have exemplified such a systematic approach by establishing an animal model of noise exposure in the chinchilla, an experimental subject with humanlike hearing capability, using a standard-exposure stimulus representing a low-frequency, steady-state noise common to many industrial settings. The chinchillas were exposed to a 95-dB SPL octave band of noise, centered at 500 Hz, with intensities not exceeding 65 dB SPL below about 300 Hx, or above 1 kHz. In some experiments, animals were exposed continuously for varying periods of time, whereas, in others, exposures were interrupted by regularly spaced rest periods. Detailed studies of hearing acuity coupled with careful histologic examination of the entire cochlea using high-resolution, light microscopic assessment of plastic-embedded flat preparations have revealed a number of findings relevant to the patterns of damage observed in human ears exposed regularly to noisy sounds.

One of the most important observations of Bohne and Clark (1982) with respect to humans concerns their findings that exposure to a low-frequency octave-band noise routinely damaged both the low- and high-frequency regions of the cochlea. Low-frequency lesions in the apical half of the cochlea consisted of a scattered loss of outer hair cells, and high-frequency lesions developing in the basal portion, by definition, included discrete lesions of about 0.04 mm consisting of a loss of inner or outer hair cells that equaled or exceeded 50%. In Fig. 161-4, the relationship of the two types of low- and high-frequency damage to the number of exposure days is shown. It is clear from these details that as the duraction of exposure was lengthened, more apical outer hair cells degenerated, and both the size and incidence (the latter not shown) of high-frequency lesions increased.

With moderate high-frequency exposure (for example, continuous exposre for 9 days to an 86-dB SPL octave band of noise with a center frequency of 4 kHz), as shown in the cytocohleogram of Fig. 161-5, A, damage is confined predominantly to a region of the organ of Corti that is basal to the frequency place of the noise (Bohne et al, 1987). With a similar exposure to a low-frequency noise (for example, a continuous exposure for 9 days to a 95-dB SPL, 0.5-kHz octave band of noise), damage consisted mainly of a scattered degeneration of outer hair cells over a reasonably broad portion of the low-frequency organ of Corti, along with one or more regions of punctate damage in the basal turn (Fig. 161-5, B) (Bohne and Clark, 1982). When the same low-frequency noise was presented on an intermittent (18 hours of rest between 36 successive 6-hour exposures), rahter than on a continuous schedule, chichillas sustained significantly less loss of outer hair cells in the low-frequency region compared to those receiving a continuous exposure of equal energy (95 dB SPL, for 9 days) (Bohne et al, 1985). However, the 18-hour rest period did not protect the basal cochlea from the development of high-frequency lesions (Fig. 161-5, C).

Bohne's anatomic work raises the possibility that low- and high-frequency lesions result from distinctly different damage mechanisms. It is not unexpected that the low-frequency exposure would induce damage in the apical region because such a sound, although involving a great portion of the cochlear partition, maximally displaces the apical part of the basilar membrane. But, why does a loss of hair cells in the basal turn occur, despite the low levels of acoustic energy measured at higher frequencies? The current understanding of the cochlea's mechanical analysis in the form of traveling-wave theory does not provide an obvious explanation for these observations. Alternative interpretations include the possibilities that the traveling wave peak-displacement pattern is level-dependent, that lower levels of prolonged activation of the basal region can eventually lead to injury, or that prolonged overstimulation alters the macromechanical displacement pattern.

One important research area that has not received a great deal of attention is the development of more sensitive measures that can detect small acoustic injuries to the inner ear (that is, the beginning stages of NIHL) so that individuals vulnerable to the long-term damage caused by noise can be identified. In recent years evidence has been accumulating that the audiometric testing of thresholds to pure tones at octave intervals does not meet this need, because, by the time such a loss is identified, permanent cochlear damage has occurred. The sensitivity of a number of threshold and suprathreshold psychoacoustic tests to detect subtle deteriorations in hearing acuity, including psychophysical-tuning curves and frequency-discrimination tasks, have either not proved to be of general usefulness, or have proved too cumbersome methodologically to implement in a clinical or workplace setting.

One new diagnostic technique that is currently under development promises to be sensitive to localized regions of structural damage. This noninvasive, objective procedure is based on the systematic measurement of a relatively newly discovered class of cochlear response referred to as otoacoustic emissions (Kemp, 1978). Because evidence suggests that emissions are primarily generated by the outer hair cells (for example, Mountain, 1980; Siegel and Kim, 1982), they provide an ideal opportunity to assess the normality of cochlear processing in ears suspected of being overstimulated by agents such as loud sound, which are known to initially damage this particular sensory cell type. Indeed, the functional status of outer hair cells in established instances of NIHL has been well described in a number of recent studies on the practical applicability of otoacoustic-emissions testings in the

otolaryngology clinic (for example, Lonsbury-Martin and Martin, 1990; Martin et al, 1990; Ohlms et al, 1991; Probst et al, 1987).

In Fig. 161-6, the ability of the two major types of evoked emissions, the distortion-product, in the "audiogram" form (top right), and the transiently evoked (bottom) otoacoustic emissions, to describe the configuration of a developing NIHL is illustrated for a 25-year-old woman, who worked in the military for 3 years in a missile assembly plant. In this example, it is clear that the magnitude and frequency extents of both emission types reflected the generally poorer hearing of the right compared with the left ear. An example of the adequacy of another type of distortion-product emissions, in the form of response/growth or input/out functions, to detect the early stages of a noise-induced cochlear injury is provided in Fig. 161-7. For this 23-year-old woman, who was a ground-support member of an Air Force crew, an elevation from the normal range in the detection threshold of emissions of these objective measures to the 20-dB hearing level measured in the left ear (solid symbols). These examples of the ability of evoked otoacoustic emissions to accurately track the frequency pattern described by behavioral-threshold shifts caused by noise exposure attest to the potential usefulness of emission procedures in identifying the site of a sensorineural loss and in monitoring the development of potential hearing impairments in hearing-conservation programs.

Experimental studies have led to an increased understanding of other major issues in the field of NIHL. For example, the results of previous investigations suggest that the origin of the 4-kHz notch in the audiogram is related to the resonator function of the external auditory ear canal (Caiazzo and Tonndorf, 1977), rather than to indeterminable innate properties of the inner ear, including a reduced vascular supply to this region of the basilar membrane (Schuknecht, 1974). The focal point of research interest, however, remains the fundamental mechanism by which the sensory cell degenerates or is damaged after exposure. A number of mechanisms (Bohne, 1976) have been proposed, including mechanical injury caused by severe motion of the basilar membrane, metabolic exhaustion of activated cells, vascular narrowing that causes ischemia, and ionic poisoning from interruption of the chemical gradients of the inner ear. Although the many years of experimental research have not produced a major breakthrough in our understanding of damage mechanisms, the current, most convincing morphologic evidence supports a combination of the mechanochemical theories. First, it is likely that alterations in the stereocilia in the form of shortened or broken rootlets are the initial pathologic processes underlying TTS and PTS, respectively (Liberman, 1987; Liberman and Dodds, 1987). As exposure continues, a discrete but direct mechanical disruption probably results in a toxic mixing of endolymph and perilymph through microbreaks in the structural framework of the cochlear duct (Bohne and Rabitt, 1983), which leads to secondary effects, including loss of hair cells and their corresponding nerve fibers.

In one relatively new area of investigation, the results of a number of studies have established that avian hair cells can regenerate after exposure to damaging levels of sound (Corwin and Cotanche, 1988; Cotanche, 1987; Ryals and Rubel, 1988). Whereas it is clear that the sensory cells of higher vertebrates do not regenerate, the outcomes of other experiments indicate that the mammalian cochlea may be capable of actively adapting to certain high-level sounds by undergoing functional recovery during on-going exposure. For example, the chinchilla studies of Bohne and Clark and colleagues (Clark et al, 1987) demonstrated that during regular exposure to an intermittent, low-frequency octave band of

noise, a recovery of up to 30 dB in behaviorally measured threshold shifts can occur during the recurring episodes of overstimulation. Further, by comparing the sensitivity and tuning characteristics of cochlear nerve-fiber activity in animals exposed for 40 rather than for 4 days, these same investigators observed significant recovery of the physiologic responses (Sinex et al, 1987).

The notion that the cochlea can become resistant over time to the effects of excessive sound is also supported by the "conditioning" effects noted by Canlon and colleagues (Canlon, 1988; Canlon et al, 1988) in the guinea pig and Franklin et al (1991) in the rabbit. In the guinea pig, continuous exposure to a 1-kHz, 81-dB SPL tone for 24 days resulted in a 20-dB reduction in the threshold shifts of auditory brainstem responses caused by a subsequent exposure to the same sound at 105 dB SPL, for 72 hours. For rabbits, a systematic schedule of overstimulation consisting of regular exposure to a 95-dB SPL octave band noise centered at 1 kHz, followed by a 3-week recovery period, altered gthe susceptibility of distortion-product otoacoustic emissions to the effects of the repeated exposure sessions. For these animals, the primary result illustrated in Fig. 161-8 was that the number of days of overstimulation required to reach a criterion loss in emission amplitudes increased for each successive exposure session. Together, the chinchilla, guinea pig, and rabbit findings suggest that the mammalian cochlea may, under some conditions, be capable of dynamically adapting to excessive sound.

For humans, the practical implications of the capacity to develop a "resistance" to loud sounds are obvious. However, human studies of the relationship between noise exposure and hearing loss are difficult to perform for a number of reasons. Experimental studies are clearly hindered by the ethical issues involved in the deliberate exposure of test subjects to noises, even though they are thought to be reversibly damaging. The complexity of measurements demanded by the alternate cross-sectional study that describes the hearing of humans exposed occupationally is considerable. These difficulties include differences inherent in the population (factors such as race, gender, presence of ear disease); problems involved in controlling exposure to non-occupational noise or past exposure history; and technical problems with the descriptive techniques themselves, ranging from variability in audiometric measures to difficulties in performing valid measurements of the noise environment itself. Given the complicated experimental designs demanded by all these controls, it is no wonder there have been few faultless clinical, epidemiological, or experimental studies of the effects of excessive noise on hearinhg in humans. The importance of performing longitudinal field studies of communities or particular population segments, such as the elderly, children, and the chronically ill, who are habitually exposed to noises produced by road traffic or aircraft, is obvious to determine effective environmental-health criteria for noise control.

Although the findings of recent studies in particular have resulted in advancing scientific knowledge of the sound-damage process, a number of major empiric issues remain to be satisfied. These include development of low-cost technical methods for controlling noise at the source, protection of individuals from excessive exposure, identification of individuals who are in the early stages of NIHL, assessment of the degree of risk from potentially hazardous noises, and determination of whether particular individuals or ears already damaged by noise are more susceptible to injury.

Susceptibility

A long-standing observation in the field of NIHL has been that some ears are more easily damaged by noise than others. Individually varying susceptibilities to noise-induced hearing impairment have been found in both humans and research animals. The widespread interest in NIHL and its prevention makes important the development of valid and reliable indices to predict human susceptibility to various noise levels. It is commonly assumed that such variability in susceptibility is a manifestation of biologic factors unique to each subject. For example, a genetically based imperfection in the physical characteristics of the cochlea (for example, stiffness of the cochlear partition), or variability in cochlear structure (for example, density of hair cells) has been proposed as contributing to susceptibility (Ward, 1979).

Identifying individual differences among a variety of factors has always been a focus of interest. A number of potentially important variables that have been investigated in the past and continue to be examined include age (Bhattacharyya and Dayal, 1986), gender (Ward, 1966), race (Jerger et al, 1986), previous damage to the inner ear (Cantrell, 1979), efficiency of the acoustic reflex (Borg et al, 1983), smoking habits (Barone et al, 1987), and the influence of certain disease states such as hypercholesterolemia (Axelsson and Lindgren, 1985), diabetes (Hodgson et al, 1987), or cardiovascular disease (Iki et al, 1985). Although a number of pertinent factors have been demonstrated, the majority of data are inconclusive (Humes, 1984). In addition, whereas a few factors such as pigmentation (Garber et al, 1982) appear to have some relationship to potentiating noise damage, others such as age simply produce additive effects (Davis and Silverman, 1978). Possibly, as McFadden and Wightman (1983) suggested in their review of the contribution of the psychoacoustic method toward understanding the symptomatology of clinical hearing disorders, the orthogonally based research approach, which assumes a causal relationship between factors, will not uncover meaningful relationships. Consequently, perhaps the uncovering of interrelationships among a myriad of individual differences by application of a multivariate test "battery" will be more successful in identifying the basic factors that predict susceptibility to NIHL.

One of the most exciting areas in current animal research aimed at determining potential factors underlying an individual's susceptibility to NIHL has focused on the role of the cochlea's neural-feedback network, the olivocochlear efferent system. Most of the findings concluding that efferent-related activity protects the ear from the damaging effects of high-level sound have been used primarily on the artificial activation of relevant descending pathways in the guinea pig by strong electrical stimulation (eg, Rajan and Johnstone, 1988, 1989). Additional research is needed, however, to resolve the significant disparities between these results and those of others (eg, Liberman, 1991) who have deliberately attempted to achieve greater control over the potentially contaminating influence of concomitant stimulation of the middle-ear muscle reflex or cochlear blood flow. In this manner systematic study will determine whether inherent efferent processes can account for, and predict, the remarkable individual variation in susceptibility to NIHL.

Interactive Effects

It is well established that noise in combination with certain chemical agents produce stronger reactions than each stimulus applies singly. The three major categories of ototoxic drugs are the aminoglycoside antibiotics, the "loop" diuretics, and the salicylates. The latter two classes of drug appear to cause reversible effects, whereas the aminoglycosides cause permanent damage to the inner ear and to hearing. A number of laboratories have established that either kanamycin or neomycin applied in combination with different types of noise produces a marked potentiating interaction (for example, Brown et al, 1978). Other studies of the temporal aspects of interactive effects indicate that the degree of potentiating interaction is the same whether the drug is given concurrently with the noise exposure or several months later (Ryan and Bone, 1982). Although earlier studies (Woodford et al, 1978) concluded that salicylates appear to be an ototoxic drug in which no interaction with noise seems to occur, more recent evidence from controlled laboratory research indicates that additional loss may occur when humans are treated with aspirin and exposed to noise concomitantly (Carson et al, 1989; McFadden and Plattsmier, 1983). Finally, in a recent experimental study in chinchillas, the heavy-metal, antineoplastic agent cis-diamine-dichloroplatinum, commonly known as cisplatin, has been shown to significantly increase the amount of hearing and sensory-cell losses from exposure to noise (Boettcher et al, 1987).

In recent years the interactive effects of noise with chemical agents common to industry and the environment have been reported. For example, in an experimental study in rats, Fechter et al (1988) discovered that the simultaneous exposure to noise and the environmental pollutant, carbon monoxide, produced more permanent hearing loss at the high frequencies than the sum of the losses produced by each agent administered alone. A variety of other chemicals present in the environment as commercial products or chemical intermediaries and contaminants, such as the organic solvents toluene and hexane, the pollutants methyl mercury and lead acetate, and the organic metal trimethyltin chloride used in the manufacture of plastics and polyurethane foam, have been identified as potent ototoxic agents that potentially interact synergistically with noise sounds. It is important to note that whereas many of these environmental toxicants have been associated with direct injury to peripheral structures, possible additional anatomic damage to more central auditory pathways is also likely.

Other Adverse Effects Caused by Noise

Damage to the vestibular system is a potential problem with noise because balance receptors are coupled physically with the auditory receptors; that is, they share the membranous labyrinth. Theoretically, the limiting membrane, which separates the utricle and semicircular canals from the rest of the vestibule, protects the majority of vestibular sensory cells from intensive stapes vibration. In the past, some effects of a transient nature caused by noise stimulating the vestibular apparatus have been observed (Parker, 1976), but the sound levels involved were extremely severe (above 125 dB SPL). For example, it is not uncommon for commercial divers to complain about dizziness after operating excessively noisy equipment under water (Molvaer and Gjestland, 1981). Presumably, their vestibular function has been affected more directly through bone conduction. Again, loss of equilibrium sense is typically transient, and consequently, a direct correlation between hearing loss and vestibular upset is not clearly established.

Recently, there has been considerable interest in the interaction of vibration and noise, which are common cofactors in the workplace. Although the vast majority of research shows that vibration alone does not affect hearing, the results of both epidemiological studies in humans and controlled laboratory studies in animals indicate a synergistic interaction of concomitant vibration, either whole-body or segmental, and noise resulting in an increase in the degree of NIHL (see review in Boettcher et al, 1987).

Exposure to both infra- and ultra-frequency sounds outside of the human-hearing range has also been studied. Infrasonic or vibratory stimuli are defined as sounds in the range of 0.1 to 20 Hz (Johnson, 1982). Although infrasound exposure of humans can cause TTS for the normal audiometric frequencies of 0.25 to 8 kHz (for example, Nixon and Johnson, 1973), there are no known instances in which infrasound clearly caused permanent damage to the human inner ear. On the other hand, some reports have demonstrated detrimental effects of microwaves on hearing (Chou et al, 1982); however, the thermal properties of ultra-high frequency sound waves in the gigahertz range, rather than mechanical energy, appear to have produced these adverse experimental effects. Thus, it is probably safe to conclude that the auditory system's built-in, middle-ear filter somewhat limits the frequencies of sound that are hazardous to the inner ear (Durrant, 1978).

Other nonauditory problems concern the general bothersome or fatiguing effects of noise, which may lead to nonspecific health disorders because of interference with the restorative processes associated with sleep. In conditions of chronic exposure, noise is considered to act as a biologic stressor that may lead to prolonged activation of the autonomic nervous system and the pituitary-adrenal complex, resulting in health impairment. Noise also has been related to circulatory problems, such as hypertension (Peterson et al, 1981), and to disorders involving gastrointestinal motility, such as peptic ulcers (Doring et al, 1980). Certain types of noise may also be annoying and lead to emotional unrest (Mason, 1969). Finally, noise can have a deleterious effect on task performance, especially if speech is involved. In general, data relevant to the nonauditory effects of noise tend to be inconclusive because the variables are difficult to isolate for objective study.

Legal Issues

One practical issue that the lawmakers of industrialized societies have had to consider concerns the balancing of two conflicting goals; that is, how can laborers be protected against the hazards of the workplace, without placing an enormous financial burden on society either by satisfying compensatory obligations, or by preventing such work-related effects through costly engineering modifications of the industrial process? Over the years, concerns about protecting the public and work force from environmental insults such as noise have been written into a combination of law and governmental regulations. Federal, state, and community legislative and regulatory actions are continually being reviewed and altered. Although the purpose of this review is not to detail such legal controls, either with respect to their historical development or their current status, a brief discussion is appropriate. For a lucid review of governmental statutes and regulations pertaining to noise control, see Suter and von Gierke (1987).

The major difficulty encountered in composing regulatory control has been in defining in practical terms what constitutes a hazardous noise. The equal-energy principle (Ward and Nelson, 191 embodies one popular approach toward expressing the danger of a particular noise by a simple number. The equal-energy principle assumes that permanent damage to hearing is related to total sound energy, which is a product of the noise level in dBA and the duration of exposure. One tenet is that an equal amount of noise energy causes an equal amount of hearing loss. Burns and Robinson (1970), who investigated the hearing losses of industrial workers, concluded that the equal-energy principle could be applied to determine daily exposure doses because hearing loss caused by industrial noise exposure appeared to be a simple function of noise energy. Atherley and Martin (1971) extended the concept to impulse noise by application of the equivalent continuous sound level (Leq) principle. The Leq is defined as the A-weighted level of a continuous, steady sound that produces, in a specified interval, an exposure that has the same total acoustic energy as that of an actual time-varying sound over the identical interval (Goldstein, 1978). In other words, if one sound contains twice as much energy as a second sound, but lasts half as long, both sounds would be characterized by the same equivalent sounds level. The Leq concept holds that these two exposures produce equivalent damage to the ear.

Although notions such as the equal-energy principle have proven useful in the practical definition of noise-control variables, their validity has been more difficult to establish. For example, over the years, experimental evidence has been conflicting in that it has tended to be either decidedly for or against the equal-energy rule. In general, it appears that the principle cannot be generalized indiscriminately to stimuli throughout the range of noise parameters because exposure to impulse noises (Spoendlin and Brun, 1973) and intermittent noises (Bohne et al, 1985) may lead to more or less degeneration, respectively, in the organ of Corti than would be expected according to the equal-energy principle. However, some aspect of the equal-energy concept has been adopted by most industrialized nations, including the USA, as a means of measuring the hazard of a particular noise exposure. The present US regulations use a 5-dB trading relationship, along with a 90-dBA time-weighted average (TWA), the maximal permissible limit for exposure to noise, over an 8-hour day. Consequently, exposure to 90 dBA for 8 hours is equivalent in sound-energy terms to an exposure of 95 dBA, for half that period, that is, for 4 hours.

Currently industries that employ laborers who work where noise is above 85 dBA must implement a hearing-conservation program consisting of several components (OSHA, 1983). First, preemployment audiometric assessment and annual audiometric monitoring are required so that noise-induced cochlear impairment can be detected before becoming too severe. Further, when a hearing loss is identified, the worker must be notified of the disorder and counseled about the use of personal hearing protectors. The second portion of the conservation program requires workers in areas of high-noise level (above 85 dBA) to wear ear protectors and to participate in a noise-education program, which informs the employee of the hazardous effects and the correct fitting of personal protectors. An important part of hearing conservation is the otologic referral (discussed later), which is essential when in-plant audiometry has detected a substantial hearing loss.

Role of the Otolaryngologist

Patient with NIHL make up a significant portion of the otolaryngologist's patient population. With the rise in numbers of individuals over the age of 65, the psychologic, economic, and social impact of NIHL is still growing. Consequently, the physician will continue to be conspicuously involved with the problem. It is certain that a known prevention exists, but it is unlikely for fiscal and technical reasons that the general sound levels of our environment will be reduced. Although the inhalation of hyperoxygenated air as a prophylaxis (Joglekar et al, 1977) has been proposed, no proven treatment or cure for noise damage exists. Consequently, detecting the early stages of NIHL is important to prevent further injury to the organ of Corti.

The role of the otolaryngologist is first to identify the cause and extent of a reported hearing loss through systematic medical and audiometric evaluation. As part of the course of otologic management, the patient should be educated about the hazards of noise and about preventive measures to preserve remaining hearing. The physician should then make basic decisions about the appropriate aural rehabilitative course of action. Finally, the physician should be sensitive to any emotional problems the patient may have in accepting a hearing impairment, which may be dealt with by counseling or special provisions.

One of the otolaryngologist's most important contributions is in teaching the patient to guard against further losses. When recommending the use of personal hearing protectors or reinforcing compliance with ongoing hearing-protection measures, the physician should be aware that the commonly used personal protectors vary considerably in effectiveness and produce an attenuation that is highly frequency dependent. In Fig. 161-9, the frequency-specific attenuation properties of an insert earplug and earmuff worn separately, or together, are compared. This comparison shows that when sealed correctly into the ear canal, earplugs reduce the noise reaching the middle ear by 15 to 30 dB. Earmuffs, on the other hand, are more effective protectors, especially for frequencies between 500 Hz and 1 kHz. In areas with extremely high noise levels, earplugs do not afford sufficient protection and individuals should be counseled to wear both earplugs and earmuffs. It is also important to appreciate that sound energy associated with high noise levels may reach the inner ear by passing through vibrating bone and tissues adjacent to the ear. Therefore, bone- and tissue-conduction thresholds set a practical limit to the possible attenuation provided by hearing-protection devices. Fig. 161-9 describes these limits with respect to the approximate attenuation limits for earplugs and earmuffs.

A final point concerning hearing protectors should be impressed on the patient. Fig. 161-10 illustrates the relationship of maximal protection in dBA to the percentage of time that the devices are worn. Essentially, hearing protectors should be worn all the time, because if they are removed for even a few minutes, their effective cumulative attenuation capability is severely reduced (Martin, 1976). For example, Fig. 161-10 illustrates that the efficiency of even an ideal (infinite) protector, that is, one that attenuates 30 dB, is reduced to about a maximum of 20-dB efficacy level, if worn for only 99% of the time.

In many states occupational NIHL is compensable. Thus physicians are required at times to serve as expert witnesses as to the probable cause of a claimant's hearing loss. Such medicolegal testimony requires that the patient undergo careful otologic and audiometric

evaluation so that hearing losses caused by unrelated ear disorders, such as cerumen impaction, middle-ear effusion, aging, genetic abnormalities, otosclerosis, and an array of other ear diseases, can be excluded. To permit the reversible (TTS) effects to recover, most states require recovery time (ranging from 14 "quiet" hours to 24 weeks) away from the occupational-noise source before audiometric assessment. By ruling out organic disease, by giving careful attention to the history of both job- and recreation-related noise exposure, and by documenting the extent and degree of involvement of both ears, an informed decision concerning any causal relationship between the noted disability and the work environment can be made.

Hearing impairment is the medical expression used to refer to the hearing level at which individuals begin to experience difficulties in everyday life. Hearing impairment expresses itself in practical terms, such as a difficulty in understanding speech. By the time an individual becomes aware of decreased speech intelligibility, considerable damage to the organ of Corti can have occurred because speech reception is not altered greatly until a hearing loss is more than 40 dB. The amount of loss at frequencies most important to speech (that is, at 2, 3, and 4 kHz) is in fact used by OSHA (1983) as a basis for calculating amounts of compensation because NIHL occurs initially at 2 kHz and above. The measure of hearing impairment is called the *hearing handicap,* which is always based on the functional state of both ears. An official guide published by the American Academy of Otolaryngology - Head and Neck Surgery (1979) provides a detailed explanation of how to evaluate and compute an NIHL handicap.

Because of an increasingly noisy environment and the probability that many individuals will unwittingly suffer hearing losses because of exposure to loud sounds at home, on the job, and during recreational activity, otolaryngologists, along with other hearing health-care professionals, should educate the public about hearing conservation. Such education is needed especially for children who are regularly exposed to the amplified music associated with personal cassette players, stereos and radios, and discos and concerts. The physician can be particularly effective in teaching individuals how to recognize the danger signals of potential NIHL, including the need to shout to be heard and the development of muffled sound, pain, and ringing in the ears (Florentine, 1990). The influence of otolaryngologists as hearing experts in the areas of education and motivation can be a major force in preventing noise-induced hearing loss.

Summary

We know more than ever before about the workings of the human ear: how it responds initially to excessive sound, how it eventually fails to reverse the progressive damage, and how much more we need to learn about basic ear processes before such damage can be corrected. With an understanding of the fundamental mechanisms involved in the process of NIHL, medical intervention to minimize permanent injury may be possible. In the meantime, we know what needs to be done to prevent or check the damage process. In light of the economic impracticability of noise control at the engineering or administrative level, especially in industry, it appears that education of the public about the potential hazards of excessive sound and the beneficial use of protective devices will be the major weapons against NIHL. Consequently, the educational role of the otolaryngologist is paramount for the conservation of hearing that is threatened by habitual noise exposure.