



Consensus Statement

NIH Consensus Development Conference
January 22-24, 1990

Volume 8, Number 1



Consensus Statement

NIH Consensus Development Conference
January 22-24, 1990

Volume 8, Number 1

NIH Consensus Development Conferences are convened to evaluate available scientific information and resolve safety and efficacy issues related to a biomedical technology. The resultant NIH Consensus Statements are intended to advance understanding of the technology or issue in question and to be useful to health professionals and the public.

NIH Consensus Statements are prepared by a nonadvocate, non-federal panel of experts, based on: (1) presentations by investigators working in areas relevant to the consensus question during a 1-1/2 day public session; (2) questions and statements from conference attendees during open discussion periods that are part of the public session; and (3) closed deliberations by the panel during the remainder of the second day and morning of the third. This statement is an independent report of the panel and is not a policy statement of the NIH or the Federal Government.

Copies of this statement and bibliographies prepared by the National Library of Medicine are available from the Office of Medical Applications of Research, National Institutes of Health, Building 1, Room 260, Bethesda, MD 20892.

For making bibliographic reference to the consensus statement from this conference, it is suggested that the following format be used, with or without source abbreviations, but without authorship attribution:

Noise and Hearing Loss. NIH Consens Dev Conf Consens Statement 1990 Jan 22-24; 8(1).

ABSTRACT

The National Institutes of Health Consensus Development Conference on Noise and Hearing Loss brought together biomedical and behavioral scientists, health care providers, and the public to address the characteristics of noise-induced hearing loss, acoustic parameters of hazardous noise exposure, individual and age-specific susceptibility, and prevention strategies. Following a day and a half of presentations by experts and discussion by the audience, a consensus panel weighed the evidence and prepared a consensus statement.

Among their findings, the panel concluded that sounds of sufficient intensity and duration will damage the ear and result in temporary or permanent hearing loss at any age. Sound levels of less than 75 dB(A) are unlikely to cause permanent hearing loss, while sound levels above 85 dB(A) with exposures of 8 hours per day will produce permanent hearing loss after many years. Current scientific knowledge is inadequate to predict that any particular individual will be safe when exposed to a hazardous noise. Strategies to prevent damage from sound exposure should include the use of individual hearing protection devices, education programs beginning with school-age children, consumer guidance, increased product noise labeling, and hearing conservation programs for occupational settings.

The full text of the consensus panel's statement follows.

INTRODUCTION

Hearing loss afflicts approximately 28 million people in the United States. Approximately 10 million of these impairments are at least partially attributable to damage from exposure to loud sounds. Sounds that are sufficiently loud to damage sensitive inner ear structures can produce hearing loss that is not reversible by any presently available medical or surgical treatment. Hearing impairment associated with noise exposure can occur at any age, including early infancy, and is often characterized by difficulty in understanding speech and the potentially troublesome symptom, tinnitus (i.e., ringing in the ears). Very loud sounds of short duration, such as an explosion or gunfire, can produce immediate, severe, and permanent loss of hearing. Longer exposure to less intense but still hazardous sounds, commonly encountered in the workplace

or in certain leisure time activities, exacts a gradual toll on hearing sensitivity, initially without the victim's awareness. More than 20 million Americans are exposed on a regular basis to hazardous noise levels that could result in hearing loss. Occupational noise exposure, the most common cause of noise-induced hearing loss (NIHL), threatens the hearing of firefighters, police officers, military personnel, construction and factory workers, musicians, farmers, and truck drivers, to name a few. Live or recorded high-volume music, recreational vehicles, airplanes, lawn-care equipment, woodworking tools, some household appliances, and chain saws are examples of nonoccupational sources of potentially hazardous noise. One important feature of NIHL is that it is preventable in all but certain cases of accidental exposure. Legislation and regulations have been enacted that spell out guidelines for protecting workers from hazardous noise levels in the workplace and consumers from hazardous noise during leisure time pursuits. Inconsistent compliance and spotty enforcement of existing governmental regulations have been the underlying cause for their relative ineffectiveness in achieving prevention of NIHL. A particularly unfortunate occurrence was the elimination of the Office of Noise Abatement and Control within the Environmental Protection Agency in 1982.

On January 22-24, 1990, the National Institute on Deafness and Other Communication Disorders, together with the Office of Medical Applications of Research of the National Institutes of Health convened a Consensus Development Conference on Noise and Hearing Loss. Cosponsors of the conference were the National Institute of Child Health and Human Development, the National Institute on Aging, and the National Institute for Occupational Safety and Health of the Centers for Disease Control. The effects of environmental sounds on human listeners may include:

- Interference with speech communication and other auditory signals.
- Annoyance and aversion.
- Noise-induced hearing loss.
- Changes in various body systems.
- Interference with sleep.

This conference was entirely centered on NIHL. The panel focused on five questions related to noise and hearing loss:

- What is noise-induced hearing loss?
- What sounds can damage hearing?
- What factors, including age, determine an individual's susceptibility to noise-induced hearing loss?
- What can be done to prevent noise-induced hearing loss?
- What are the directions for future research?

Following a day and a half of presentations by experts in the relevant fields and discussion from the audience, a consensus panel comprising specialists and generalists from the medical and other related scientific disciplines, together with public representatives, considered the evidence and formulated a consensus statement in response to the five previously stated questions.

WHAT IS NOISE-INDUCED HEARING LOSS?

Sounds of sufficient intensity and duration will damage the ear and result in temporary or permanent hearing loss. The hearing loss may range from mild to profound and may also result in tinnitus. The effect of repeated sound overstimulation is cumulative over a lifetime and is not currently treatable. Hearing impairment has a major impact on one's communication ability and even mild impairment may adversely affect the quality of life. Unfortunately, although NIHL is preventable, our increasingly noisy environment places more and more people at risk.

Studies of NIHL

Most studies of the association between sound exposure and hearing loss in humans are retrospective measurements of the hearing sensitivities of numerous individuals correlated with their noise exposures. The variability within these studies is usually large; thus, it is difficult to predict the precise magnitude of hearing loss that will result from a specific sound exposure. Prospective studies of selected workers' hearing levels over a long time while their sound exposures are carefully monitored are costly and time-consuming and, due to attrition, require a large number of subjects. When significant hearing loss is found, for ethical reasons, exposures must be reduced, interfering with the relationships under study. Although studies of NIHL in humans are difficult, they provide valuable information not available from animal studies and should be continued.

In prospective animal studies, sound exposures can be carefully controlled, and the anatomic and physiologic correlates of NIHL can be precisely defined. Although there may be interspecies differences with respect to the absolute sound exposure that will injure the ear, the basic mechanisms that lead to damage appear to be similar in all mammalian ears.

Anatomic and Physiologic Correlates of NIHL

Two types of injury are recognized: acoustic trauma and NIHL. Short-duration sound of sufficient intensity (e.g., a gunshot or explosion) may result in an immediate, severe, and permanent hearing loss, which is termed acoustic trauma. Virtually all of the structures of the ear can be damaged, in particular the organ of Corti, the delicate sensory structure of the auditory portion of the inner ear (cochlea), which may be torn apart.

Moderate exposure may initially cause temporary hearing loss, termed temporary threshold shift (TTS). Structural changes associated with TTS have not been fully established but may include subtle intracellular changes in the sensory cells (hair cells) and swelling of the auditory nerve endings. Other potentially reversible effects include vascular changes, metabolic exhaustion, and chemical changes within the hair cells. There is also evidence of a regional decrease in the stiffness of the stereocilia (the hair bundles at the top of the hair cells), which may recover. This decrease in stereocilia stiffness may lead to a decrease in the coupling of sound energy to the hair cells, which thereby alters hearing sensitivity.

Repeated exposure to sounds that cause TTS may gradually cause permanent NIHL in experimental animals. In this type of injury, cochlear blood flow may be impaired, and a few scattered hair cells are damaged with each exposure. With continued exposure, the number of damaged hair cells increases. Although most structures in the inner ear can be harmed by excessive sound exposure, the sensory cells are the most vulnerable. Damage to the stereocilia is often the first change, specifically, alteration of the rootlet structures that normally anchor the stereocilia into the top of the hair cell. Once destroyed, the sensory cells are not replaced. During the recovery period between some sound exposures, damaged regions of the organ of Corti heal by scar formation. This process is very important because it reestablishes the barrier between the two fluids of the inner ear (perilymph and endolymph). If this barrier is not reestablished, degeneration of hair cells may continue. Further, once a sufficient number of hair cells are lost, the nerve fibers to that region also degenerate. With degeneration of the cochlear nerve fibers, there is corresponding degeneration within the central nervous system. The extent to which these neural changes contribute to NIHL is not clear.

With moderate periods of exposure to potentially hazardous high frequency sound, the damage is usually confined to a restricted area in the high-frequency region of the cochlea. With a comparable exposure to low-frequency noise, hair cell damage is not confined to the low-frequency region but may also affect the high-frequency regions. The predominance of damage in different cochlear regions with different frequency exposures reflects factors such as the resonance of the ear

canal, the middle ear transfer characteristics, and the mechanical characteristics of the organ of Corti and basilar membrane.

Assessment of NIHL

Hearing loss is measured by determining auditory thresholds (sensitivity) at various frequencies (pure-tone audiometry). Complete assessment should also include measures of speech understanding and middle-ear status (immittance audiometry). Pure-tone audiometry is also used in industrial hearing conservation programs to determine whether adequate protection against hazardous sound levels is provided.

The first audiometric sign of NIHL resulting from broadband noise is usually a loss of sensitivity in the higher frequencies from 3,000 through 6,000 Hertz (Hz) (i.e., cycles per second), resulting in a characteristic audiometric "notch." With additional hearing loss from noise or aging, the threshold at 8,000 Hz may worsen and eliminate this characteristic audiometric pattern. Thus, the presence or absence of NIHL cannot be established on the basis of audiometric shape, *per se*. The hearing loss is usually bilateral, but some degree of asymmetry is not unusual, especially with lateralized noise sources such as rifles. After moderate sound exposure, TTS may occur, and, during a period of relative quiet, thresholds will return to normal levels. If the exposure continues on a regular basis, permanent threshold shifts (PTS) will result, increasing in magnitude and extending to lower and higher frequencies. If the exposures continue, NIHL increases, more rapidly in the early years. After many years of exposure, NIHL levels off in the high frequencies, but continues to worsen in the low frequencies. Although TTS and PTS are correlated, the relation is not strong enough to use TTS to predict the magnitude of permanent hearing loss.

An important consequence of the sensitivity loss associated with NIHL is difficulty in understanding speech. Whereas a large proportion of the *energy* in speech is contained within the low frequency range, much of the *information* required to differentiate one speech sound from another is contained within the higher frequencies. With significant hearing loss in the high frequencies, important speech information is often inaudible or unusable. Other interfering sounds such as background noise, competing voices, or room reverberation

may reduce even further the hearing-impaired listener's receptive communication ability. The presence of tinnitus may be an additional debilitating condition.

NIHL may interfere with daily life, especially those social activities that occur in noisy settings. Increased effort is required for understanding speech in these situations, which leads to fatigue, anxiety, and stress. Decreased participation in these activities often results, affecting not only hearing-impaired individuals but also friends and family members. Hearing loss is associated with depression in the elderly and may be related to dementia and cognitive dysfunction. Systematic study of the effects of hearing loss on the quality of life have only lately focused specifically on individuals with NIHL; therefore, continued studies of this kind are desirable.

The impairment in hearing ability resulting from NIHL may vary from mild to severe. An individual's ability to communicate and function in daily life varies with the degree of loss and the individual's communication needs although these relationships are complex. The magnitude of the effect on communication ability may be estimated by a variety of scales, which are often used in disability determinations. These scales, which vary substantially in the frequencies used, the upper and lower limits of impairment, age correction, and adjustment for asymmetric hearing loss, attempt to predict the degree of communication impairment (understanding of speech) on the basis of pure-tone thresholds. There is no consensus about the validity or utility of the scales, which scale should be used, whether measures of speech understanding should be included, or whether self-assessment ratings should be incorporated into either impairment rating scales or disability determinations.

WHAT SOUNDS CAN DAMAGE HEARING?

Some sounds are so weak physically that they are not heard. Some sounds are audible but do not have any temporary or permanent after-effects. Some sounds are strong enough to produce a temporary hearing loss from which there may appear to be complete recovery. Damaging sounds are those that are sufficiently strong, sufficiently long-lasting, and involve appropriate frequencies so that permanent hearing loss will ensue.

Most of the sounds in the environment that produce such permanent effects occur over a very long time (for example, about 8 hours per workday over a period of 10 or more years). On the other hand, there are some particularly abrupt or explosive sounds that can cause damage even with a single exposure.

The line between these categories of sounds cannot be stated simply because not all persons respond to sound in the same manner. Thus, if a sound of given frequency bandwidth, level, and duration is considered hazardous, one must specify for what proportion of the population it will be hazardous and, within that proportion, by what criterion of damage (whether anatomical, audiometric, speech understanding) it is hazardous.

The most widely used measure of a sound's strength or amplitude is called "sound level," measured by a sound-level meter in units called "decibels" (dB). For example, the sound level of speech at typical conversational distances is between 65 and 70 dB. There are weaker sounds, still audible, and of course much stronger sounds. Those above 85 dB are potentially hazardous.

Sounds must also be specified in terms of frequency or bandwidth, roughly like the span of keys on a piano. The range of audible frequencies extends from about 20 Hz, below the lowest notes on a piano, to at least 16,000 or 20,000 Hz, well above the highest notes on a piccolo. Most environmental noises include a wide band of frequencies and, by convention, are measured through the "A" filter in the sound-level meter and thus are designated in dB(A) units. It is not clear what effect, if any, sound outside the frequency range covered in dB(A) measurements may have on hearing. At this time, it is not known whether ultrasonic vibration will damage hearing.

To define what sounds can damage hearing, sound level, whether across all frequency bands or taken band by band, is not enough. The duration of exposure—typical for a day and accumulated over many years—is critical. Sound levels associated with particular sources such as snowmobiles, rock music, and chain saws, are often cited, but predicting the likelihood of NIHL from such sources also requires knowledge of typical durations and the number of exposures.

There appears to be reasonable agreement that sound levels below 75 dB(A) will not engender a permanent hearing loss, even at 4000 Hz. At higher levels, the amount of hearing loss is directly related to sound level for comparable durations.

According to some existing rules and regulations, a noise level of 85 dB(A) for an 8-hour daily exposure is potentially damaging. If total sound energy were the important predictor, an equivalent exposure could be as high as 88 dB(A) if restricted to 4 hours. (A 3-dB increase is equivalent to doubling the sound intensity.) This relation, enshrined in some standards and regulations, is a theory based on a dose or exposure defined by total energy.

In spite of the physical simplicity of a total-energy concept, other principles have been invoked to define equivalent exposures of different sound levels and durations. Early research suggested that NIHL after 10 years could be predicted from temporary threshold shifts (TTS) measured 2 minutes after a comparable single-day exposure. Those results, however, were taken to indicate that a halving of duration could be offset by a 5-dB change in sound level rather than a 3-dB change. This 5-dB rule is implemented in the Walsh-Healey Act of 1969 and subsequent Occupational Safety and Health Administration regulations for the purpose of requiring preventive efforts for noise-exposed workers. The 3-dB trading rule is agreed to in International Standards Organization (ISO) Standard 1999.2 (1989) for the purpose of predicting the amount of noise-induced hearing loss resulting from different exposures. There is no consensus concerning a single rule to be used for all purposes in the United States.

Generally, for sound levels below about 140 dB, different temporal forms of sound, whether impulse (gunshot), impact (drop forge) or steady state (turbine), when specified with respect to their level and duration, produce the same hearing

loss. This does not appear to follow at levels above 140 dB, where impulse noise creates more damage than would be predicted. This may imply that impulse noise above a certain critical level results in acoustic trauma from which the ear cannot recover.

Although sound exposures that are potentially hazardous to hearing are usually defined in terms of sound level, frequency bandwidths, and duration, there are several simple approximations that indicate that a sound exposure may be suspected as hazardous. These include the following: If the sound is appreciably louder than conversational level, it is potentially harmful, provided that the sound is present for a sufficient period of time. Hazardous noise may also be suspected if the listener experiences: (a) difficulty in communication while in the sound, (b) ringing in the ear (tinnitus) after exposure to the sound, and/or (c) the experience that sounds seem muffled after leaving the sound-exposure area.

In the consideration of sounds that can damage hearing, one point is clear: it is the acoustic energy of the sound reaching the ear, not its source, which is important. That is, it does not matter if the hazardous sound is generated by a machine in the workplace, by an amplifier/loudspeaker at a rock concert, or by a snowmobile ridden by the listener. Significant amounts of acoustic energy reaching the ear will create damage—at work, at school, at home, or during leisure activities. Although there has been a tendency to concentrate on the more significant occupational and transportation noise, the same rules apply to all potential noise hazards.

WHAT FACTORS, INCLUDING AGE, DETERMINE AN INDIVIDUAL'S SUSCEPTIBILITY TO NOISE-INDUCED HEARING LOSS?

One thoroughly established characteristic of NIHL is that, on the average, more intense and longer-duration noise exposures cause more severe hearing loss. A second is that there is a remarkably broad range of individual differences in sensitivity to any given noise exposure. Several factors have been proposed to explain differences in NIHL among individuals; others may be associated with differences over time within the same individual. It is important to distinguish those factors whose roles in determining susceptibility are supported by a consistent body of theory and empirical evidence from other factors whose roles have been proposed but for which theory, data, or both are less conclusive.

Differences Among Individuals

Both temporary threshold shift (TTS) and permanent threshold shift (PTS) in response to a given intense noise may differ as much as 30 to 50 dB among individuals. Both animal research and retrospective studies of humans exposed to industrial noise have demonstrated this remarkable variation in susceptibility. The biological bases for these differences are unknown. A number of extrinsic factors (e.g., characteristics of the ear canal and middle ear, drugs, and prior exposure to noise) may influence an individual's susceptibility to NIHL. However, animal studies that have controlled these variables suggest that individual differences in inner ear anatomy and physiology also may be significant. Additional research is necessary to determine whether vascular, neural feedback (efferent system), or other mechanisms can account for and predict such individual variation.

One factor that may be associated with decreased susceptibility to NIHL is conductive hearing loss; the cochlear structures may be protected by any form of acoustic attenuation. For similar reasons, middle ear muscles, which normally serve a protective function by contracting in response to intense sound, when inoperative, can result in increased susceptibility. Among the other factors that are *theoretically* associated with differences in susceptibility are (a) unusually efficient acoustic transfer through the external and middle ear, as a determinant of the amount of energy coupled to the inner ear structures,

and (b) preexisting hearing loss, which could imply that less additional loss would occur if the sensitive structures have already been damaged. Support for these hypotheses has been modest, in the case of the transfer function, because little empirical work has been done to test that hypothesis, and, in the case of reduced sensitivity, because several studies disagree. In general, when there is a difference in average loss to a given noise exposure, those ears with previous PTS or TTS have shown somewhat less additional loss than those not previously exposed.

Findings have sometimes implicated degree of pigmentation, both of the receptor structures (melanization) and of the eye and skin, as related to susceptibility. However, these results, too, are equivocal.

Gender. There is little difference in hearing thresholds between young male and female children. Between ages 10 and 20, males begin to show reduced high-frequency auditory sensitivity relative to females. Women continue to demonstrate better hearing than men into advanced age. These gender differences are probably due to greater exposure of males to noise rather than to their inherent susceptibility to its effects.

Differences Within Individuals

Ototoxic drugs. Among the causes of differences of susceptibility to noise exposure *within* individuals are ototoxic drugs and other chemicals. In animal research, certain antibiotics (aminoglycosides) appear to exacerbate the damaging effects of noise exposure. Clinical evidence of corresponding effects in human patients has not been established, but precautions should be taken with regard to noise exposures of individual patients treated with these medications. Although high doses of aspirin are widely known to cause TTS and tinnitus, aspirin has *not* been shown to increase susceptibility to NIHL.

Age. In certain animal models there is evidence of heightened susceptibility to noise exposure shortly after birth—a "critical period" (possibly following the time when fluids fill the middle ear but before complete development of the cochlear structures). However, it is *not* clear that data from such animal models can be generalized to full-term normal human infants. Premature infants in noisy environments (e.g. neonatal intensive care units), however, may be at risk.

At the other extreme, increasing age has been hypothesized to be associated with decreasing susceptibility. This contention is based on the existence of presbycusis, hearing loss that increases with age and that is *not* known to be attributable to excessive noise exposure or other known etiology. The typical levels of presbycusis at various ages have recently been incorporated as Annex A in International Standards Organization Standard 1999.2 (1989). That standard may be used to estimate the portion of overall hearing loss that is attributable to exposure to excessive noise.

In summary, scientific knowledge is currently inadequate to predict that any individual will be safe in noise that exceeds established damage-risk criteria, nor that specific individuals will show greater-than-average loss following a given exposure. Among the many proposed explanations, the hypothesis that the resonant and transmission properties of the external and middle ear affect individual susceptibility deserves further attention. Empirical support for this hypothesis should not be difficult to obtain, but very few data have been collected on this question, both for TTS (experimentally) and PTS (retrospectively). Differences in susceptibility of the cochlear structures to NIHL may exist, but no practical approach to predicting them is yet available. Identification of susceptible humans will almost certainly be delayed until a successful animal model is available.

WHAT CAN BE DONE TO PREVENT NOISE-INDUCED HEARING LOSS?

Noise-induced hearing loss occurs every day—in both occupational and nonoccupational settings. The crucial questions for prevention are as follows: (1) What can individuals do to protect themselves from NIHL? (2) What role should others, such as educators, employers, or the Government, play in preventing NIHL? (3) What general strategies should be employed to prevent NIHL? Answers to these questions have long been known, but solutions have not been effectively implemented in many cases. As a result, many people have needlessly suffered hearing loss.

Individual Protection Strategies

Hearing conservation must begin by providing each individual with basic information. NIHL is insidious, permanent, and irreparable, causing communication interference that can substantially affect the quality of life. Ringing in the ears and muffling of sounds after sound exposure are indicators of potential hazard. Dangerous sound exposures can cause significant damage without pain, and hearing aids do not restore normal hearing. Individuals should become aware of loud noise situations and avoid them if possible or properly use hearing protection. It is important to recognize that both the level of the noise and its duration (i.e., exposure) contribute to the overall risk. Certain noises, such as explosions, may cause immediate permanent damage.

Many sources, such as guns, power tools, chain saws, small airplanes, farm vehicles, firecrackers, some types of toys, and some medical and dental instruments may produce dangerous exposures. Music concerts, car and motorcycle races, and other spectator events often produce sound levels that warrant hearing protection. Similarly, some stereo headphones and loudspeakers are capable of producing hazardous exposures. Parents should exercise special care in supervising the use of personal headset listening devices, and adults and children alike should learn to operate them at safe volume settings.

Nonoccupational Strategies

Hearing loss from nonoccupational noise is common, but public awareness of the hazard is low. Educational programs should be targeted toward children, parents, hobby groups,

public role models, and professionals in influential positions such as teachers, physicians, audiologists and other health care professionals, engineers, architects, and legislators. In particular, primary health care physicians and educators who deal with young people should be targeted through their professional organizations. Consumers need guidance and product noise labeling to assist them in purchasing quieter devices and in implementing exposure reduction strategies. The public should be made aware of the availability of affordable, effective hearing protectors (ear plugs, ear muffs, and canal caps). Hearing protection manufacturers should supply comprehensive instructions concerning proper protector use and also be encouraged to increase device availability to the public sector. Newborn nurseries, including neonatal intensive care units, should be made quieter. Medical and dental personnel should be trained to educate their patients about NIHL.

Individuals with significant noise exposure need counseling. Basic audiometric evaluations should be widely available. The goal is to detect early noise-induced damage and interrupt its progression *before* hearing thresholds exceed the normal range.

Occupational Strategies

Hearing conservation programs for occupational settings must include the following interactive components: sound surveys to assess the degree of hazardous noise exposure, engineering and administrative noise controls to reduce exposures, education to inform at-risk individuals why and how to prevent hearing loss, hearing protection devices (earplugs, earmuffs, and canal caps) to reduce the sound reaching the ear, and audiometric evaluations to detect hearing changes. Governmental regulations that currently apply to most noisy industries should be revised to encompass *all* industries and all employees, strengthened in certain requirements, and strictly enforced with more inspections and more severe penalties for violations.

Many existing hearing conservation programs remain ineffective due to poor organization and inadequately trained program staff. Senior management must use available noise controls, purchase quieter equipment, and incorporate noise reduction in planning new facilities. Noise exposures must be measured accurately and the degree of hazard communicated

to employees. Hearing protection devices must be available that are comfortable, practical for the demands of work tasks, and provide adequate attenuation. Labeled ratings of hearing protector attenuation must be more realistic so that the degree of protection achieved in the workplace can be properly estimated. Each employee must be individually fitted with protectors and trained in their correct use and care. Employees need feedback about their audiometric monitoring results annually.

Employers need to monitor program effectiveness by using appropriate techniques for analysis of group audiometric data. By detecting problem areas, managers can prioritize resource allocations and modify company policies to achieve effectiveness. Potential benefits include reduced costs for worker's compensation, enhanced worker morale, reduced absenteeism, fewer accidents, and greater productivity.

Enactment of uniform regulations for awarding worker's compensation for occupational hearing loss would stimulate employers' interest in achieving effective hearing conservation programs. Equitable criteria for compensability should be developed based on scientific investigations of the difficulties in communication and other aspects of auditory function encountered in everyday life by persons with differing degrees of NIHL.

General Strategies

Both nonoccupational and occupational NIHL could be reduced by implementing broader preventive efforts. Labeling of consumer product noise emission levels should be enforced according to existing regulations. Incentives for manufacturers to design quieter industrial equipment and consumer goods are needed along with regulations governing the maximum emission levels of certain consumer products, such as power tools. Reestablishment of a Federal agency coordinating committee with central responsibility for practical solutions to noise issues is essential. Model community ordinances could promote local planning to control environmental noise and, where feasible, noise levels at certain spectator events. High-visibility media campaigns are needed to develop public awareness of the effects of noise on hearing and the means for self-protection. Prevention of NIHL should be part of the health curricula in elementary through high schools. Self-education materials for adults should be readily available.

WHAT ARE THE DIRECTIONS FOR FUTURE RESEARCH?

The panel recommends that research be undertaken in two broad categories: (1) Studies that use existing knowledge to prevent NIHL in the immediate future, and; (2) research on basic mechanisms to prevent NIHL in the long-term future.

- Development of rationale and collection of empirical data to evaluate systems for combining sound level and duration to predict NIHL.
- Longitudinal studies to further delineate responses of the ear to noise over time in different groups of people with varying levels of exposure.
- Continued investigation of engineering noise measurement and control techniques, such as acoustic intensity measurement, active noise-cancellation systems, and cost-benefit analyses of noise reduction.
- Development and investigation of hearing protector designs that provide improved wearer comfort, usability, and more natural audition.
- Development of repeatable laboratory procedures that incorporate behavioral tests to yield realistic estimates of hearing protector attenuation performance that are accepted for device labeling purposes.
- Empirical evaluation of the efficacy of hearing conservation programs and the field performance of hearing protection devices in industry.
- Development and validation of evaluation techniques for detection of the following:
 - (a) subtle changes in hearing resulting from noise exposure and
 - (b) early indicators of NIHL.
- Determination of the pathophysiological correlates of TTS and PTS.
- Investigation of the anatomic and physiologic bases of presbycusis and interactive effects with NIHL.
- Investigation of genetic bases for susceptibility to NIHL, using contemporary techniques, including molecular biology.
- Further studies of drugs (e.g., vasodilating agents) and other pre-exposure conditions (e.g., activation of efferent systems or exposure to "conditioning" noise) that have been suggested in preliminary reports to protect the inner ear from NIHL and elucidation of the underlying mechanisms.
- Investigation into the physiologic mechanisms underlying the synergistic effects of certain drugs and noise exposure in animal models.

CONCLUSIONS AND RECOMMENDATIONS

- Sounds of sufficient intensity and duration will damage the ear and result in temporary or permanent hearing loss at any age.
- NIHL is characterized by specific anatomic and physiologic changes in the inner ear.
- Sounds with levels less than 75 dB(A), even after long exposures, are unlikely to cause permanent hearing loss.
- Sounds with levels above 85 dB(A) with exposures of 8 hours per day will produce permanent hearing loss after many years.
- There is a broad range of individual differences among people in the amount of hearing loss each suffers as a result of identical exposures.
- Current scientific knowledge is inadequate to predict that any particular individual will be safe when exposed to a hazardous noise.
- Because sources of potentially hazardous sound are present in both occupational and nonoccupational settings, personal hearing protection should be used when hazardous exposures are unavoidable.
- Vigorous enforcement of existing regulations, particularly for the workplace and consumer product labeling, would significantly reduce the risk of workplace NIHL. Regulations should be broadened to encompass all employees with hazardous noise exposures.
- Application of existing technologies for source noise control, especially in the manufacture of new equipment and construction of new facilities, would significantly reduce sound levels at the ear.
- In addition to existing hearing conservation programs, a comprehensive program of education regarding the causes and prevention of NIHL should be developed and disseminated, with specific attention directed toward educating school-age children.

Consensus Development Panel

Patrick E. Brookhouser, M.D.

Conference and Panel
Chairperson
Director
Boys Town National Research
Hospital
Professor and Chair
Department of Otolaryngology
Creighton University
Omaha, Nebraska

John Gordon Casali, Ph.D.

Director
Auditory Systems Laboratory
Department of Industrial
Engineering and Operations
Research
Virginia Polytechnic Institute and
State University
Blacksburg, Virginia

Francis I. Catlin, M.D., Sc.D.

Professor
Department of Otolaryngology
and Communicative Sciences
Baylor College of Medicine
Houston, Texas

Marilyn E. Demorest, Ph.D.

Professor
Department of Psychology
University of Maryland Baltimore
County
Catonsville, Maryland

Judy R. Dubno, Ph.D.

Associate Professor of Surgery
Division of Head and Neck
Surgery
University of California at Los
Angeles School of Medicine
Los Angeles, California

George A. Gates, M.D.

Professor and Vice Chairman
Department of Otolaryngology
Washington University
St. Louis, Missouri

Ira J. Hirsh, Ph.D.

Professor
Department of Psychology
Washington University

Director of Research Emeritus
Central Institute for the Deaf
St. Louis, Missouri

Patricia A. Leake, Ph.D.

*Associate Professor in
Residence*
Department of Otolaryngology
Coleman-Epstein Laboratories
University of California
San Francisco, California

Kevin John Murphy, M.D., F.A.A.P.

*Assistant Professor of Clinical
Pediatrics*
Washington University Medical
School/Northwest Pediatrics
Florissant, Missouri

Julia Doswell Royster, Ph.D.

President
Environmental Noise Consult-
ants, Inc.
Cary, North Carolina

Evelyn D. Talbott, Dr.P.H.

*Assistant Professor of Epidemi-
ology*
Department of Epidemiology
University of Pittsburgh Gradu-
ate School of Public Health
Pittsburgh, Pennsylvania

Bonnie Tucker, J.D.

Attorney/Professor of Law
Arizona State University College
of Law
Tempe, Arizona

Charles S. Watson, Ph.D.

Professor and Chair
Department of Speech and
Hearing Sciences
Speech and Hearing Center
Indiana University
Bloomington, Indiana

Laura Ann Wilber, Ph.D.

Professor
Department of Audiology and
Hearing Impairment
Northwestern University
Evanston, Illinois

Speakers

Peter W. Alberti, Ph.D., M.B.
"Clinical Criteria: Noise or Not?"

Alf Axelsson, M.D., Ph.D.
"Noise Exposure in Adolescents and Young Adults"

Elliott H. Berger, M.S.
"Hearing Protection—the State of the Art (Circa 1990) and Research Priorities for the Coming Decade"

Barbara A. Bohno, Ph.D.
"Patterns of Cellular Degeneration in the Inner Ear Following Excessive Exposure to Noise"

William W. Clark, Ph.D.
"Noise Exposure and Hearing Loss From Leisure Activities"

Robert A. Dobie, M.D.
"Effects of Noise-Induced Hearing Loss on Quality of Life"

Mary Florentine, Ph.D.
"Prevention Strategies: Education"

Kenneth J. Gerhardt, Ph.D.
"Prenatal and Perinatal Risks"

Donald Henderson, Ph.D.
"Acoustic Parameters of Hazardous Noise Exposures"

Larry E. Humes, Ph.D.
"Individual Susceptibility—Nonauditory Factors"

M. Charles Liberman, Ph.D.
"Biological Bases of Acoustic Injury"

John H. Mills, Ph.D.
"Noise and the Aging Process"

Anna K. Nabelek, Ph.D.
"Interactions Between Hearing Loss and the Environment"

William Noble, Ph.D.
"Evaluation of Disability and Handicap"

Gerald R. Popelka, Ph.D.
"The Effects of Certain Auditory Factors on Individual Susceptibility to Noise"

Branda Ryals, Ph.D.
"Critical Periods and Acoustic Trauma"

Richard Salvi, Ph.D.
"Interaction Between Noise and Other Agents"

Edgar A.G. Shaw, Ph.D.
"The Measurement of Noise Exposure and the Assessment of Risk"

Norat D. Stewart, Ph.D.
"Noise Reduction to Prevent Hearing Damage—State of the Art, Implementation Problems, and Future Directions"

Henning E. Von Gierke, Dr.Eng.
"The Noise-Induced Hearing Loss Problem"

W. Dixon Ward, Ph.D.
"Impulse/Impact Vs. Continuous Noise"

Planning Committee

Ralph F. Naunton, M.D.
Planning Committee Chairperson
Director
Extramural Program
Division of Communication Sciences and Disorders
National Institute on Deafness and Other Communication Disorders
National Institutes of Health
Bethesda, Maryland

Patrick E. Brookhouser, M.D.
Conference and Panel Chairperson
Director
Boys Town National Research Hospital
Professor and Chair
Department of Otolaryngology

Creighton University
Omaha, Nebraska

William W. Clark, Ph.D.

Director
Graduate Program in Communi-
cation Sciences
Central Institute for the Deaf
St. Louis, Missouri

Jerry M. Elliott

Program Analyst
Office of Medical Applications of
Research
National Institutes of Health
Bethesda, Maryland

John H. Ferguson, M.D.

Director
Office of Medical Applications of
Research
National Institutes of Health
Bethesda, Maryland

William H. Hall

Director of Communications
Office of Medical Applications of
Research
National Institutes of Health
Bethesda, Maryland

Donald Henderson, Ph.D.

Professor and Chairman
Communicative Disorders and
Sciences
State University of New York at
Buffalo
Buffalo, New York

Karen Jackson

Information Coordinator
National Institute on Deafness
and Other Communication
Disorders
National Institutes of Health
Bethesda, Maryland

James F. Kavanagh, Ph.D.

Associate Director
Center for Research for Mothers
and Children
National Institute of Child Health
and Human Development
National Institutes of Health
Bethesda, Maryland

William Melnick, Ph.D.

Professor
Department of Otolaryngology
University Hospital Clinics
Ohio State University
Columbus, Ohio

Andrew Monjan, Ph.D., M.P.H.

Chief
Neurobiology and Neuropsy-
chology Units
Neuroscience and Neuropsy-
chology of Aging Program
National Institute on Aging
National Institutes of Health
Bethesda, Maryland

Lt. Col. Michael J. Moul, Ph.D.

Assistant Director
Army Audiology and Speech
Center
Walter Reed Army Medical
Center
Washington, D.C.

J. Buckminster Ranney, Ph.D.

Deputy Director
Division of Communication
Sciences and Disorders
National Institute on Deafness
and Other Communication
Disorders
National Institutes of Health
Bethesda, Maryland

Dina Rice

Conference Coordinator
Prospect Associates
Rockville, Maryland

Alice H. Suter, Ph.D.

Visiting Scientist
National Institute on Occupa-
tional Safety and Health
Centers for Disease Control
Cincinnati, Ohio

Susan Wallace, M.F.A.

Conference Coordinator
Prospect Associates
Rockville, Maryland

Conference Sponsors

**National Institute on Deafness
and Other Communication
Disorders**

Jay Moskowitz, Ph.D.
Acting Director

**Office of Medical Applications of
Research**

John H. Ferguson, M.D.
Director

**National Institute of Child Health
and Human Development**

Duano F. Alexander, M.D.
Director

National Institute on Aging

T. Franklin Williams, M.D.
Director

**National Institute for
Occupational Safety and Health
of the Centers for Disease Control**

J. Donald Millar, M.D.
Director



U.S. DEPARTMENT OF HEALTH AND
HUMAN SERVICES
Public Health Service
Office of Medical Applications of Research
Building 1, Room 260
National Institutes of Health
Bethesda, MD 20892

Official Business
Penalty for private use \$300

BULK RATE
Postage & Fees
PAID
PHS/NIH/OD
Permit No. G291

Ken Mittelhotz
Office of Noise Abatement
Federal Activities
Environmental Protection Agency
401 M Street, S.W., A-104
Washington, D.C. 20460