Presbycusis

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Presbycusis—literally, elderly hearing—is used in this report as a generic term to encompass all aspects of age-related decline in auditory performance. As such, this term implies a complex multifactorial etiology with a variety of co-factors, both known and unknown. Basic etiologic issues remain unresolved. For example, it is not clear that hearing loss is an inevitable concomitant of aging. Indeed, some believe the vast majority of age-related hearing loss is due to the effects of wear and tear and a lifetime of noise exposure, to both hazardous and normal environmental sounds. Others believe that genetically programmed biologic degeneration of the auditory system is an inevitable sequel of aging. The extent to which these two chief factors play a role in presbycusis is debated. Even the prevalence of this disorder is based on estimates that vary widely. There is little agreement on the best method of assessment and uncertainty exists concerning age norms and the definitions of impairment and handicap.

The sections of this report provide capsule summaries of our current knowledge about presbycusis in the areas of basic science, prevalence, interaction with noise, and etiopathogenesis. The report concludes with a listing of specific research topics that the working group consider of high priority.

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ANATOMY AND PHYSIOLOGY OF THE COCHLEA AND CENTRAL AUDITORY PATHWAY

Presbycusis is a complex state that may reflect pathologic influences along the entire auditory neuraxis. Gacek and Schuknecht[1] defined four types of presbycusis: (1) mechanical or conductive; (2) metabolic, which is characterized by strial atrophy; (3) sensory, which is characterized by hair- and supporting-cell loss from the organ of Corti; and (4) neural presbycusis, which could be described as loss of neurons or neuronal function from the spinal ganglia up the auditory neuraxis to the temporal cortex.

Morphologic studies in human beings and in several animal models have described age-related loss of inner and outer hair cells and supporting cells, primarily from basal turns of the cochlea. Outer hair cell numbers appear to decrease to a greater extent than do numbers of inner hair cells. Unfortunately, previous studies have not systematically looked at age-related morphologic changes and correlated them to auditory function. Studies in human beings have begun to correlate audiologic and brainstem evoked response data with quantitative descriptions of cochlear morphology.

In certain mouse models of presbycusis, there is good correlation between structural losses in the cochlea and objective measures of threshold, but this correlation is lacking in other strains. Other morphologic age-related changes in the cochlea include strial changes, which may be reflected as reductions in the endocochlear potential. A recent study in aged gerbils described endocochlear potential losses as high as 30 mV in the apical coil, associated with only a 14 dB loss in action potential threshold.[2] Age-related losses of spiral ganglion cells have been described in human beings and animals. Age-related loss of eighth nerve fibers has been reported to be as high as 20 percent in very old rats.[3] Results from studies of age-related neuronal losses in the human cochlear nuclei have been controversial, but when these data are compared to the literature on rodents, a significant age-related loss of cochlear nucleus neurons probably occurs. Excellent light and electron microscopic studies in rodents have described age-related morphologic changes at the level of the superior olivary complex.[4] If similar changes appear in human material, they could provide an explanation for the psychophysically observed loss of the ability to lateralize sounds.[5] These kinds of changes have been associated with poor performance by elderly individuals during synthetic speech tests.[6] Age-related loss of neurons displaying GABA immunoreactivity in the inferior colliculus suggests a loss of inhibitory function in aged

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rats. Differential loss of inhibitory function in key auditory circuits could explain the loss of speech intelligibility and loss of the ability to detect signal in noise seen in the elderly.[7] Age-related changes in brainstem evoked response measures have been reported in human beings as well as rodents. Results from rodent studies show age-related latency changes of the N_0, while the wave IV-N_0 conduction time was prolonged.[8] These findings suggest that changes are occurring at the level of the superior olivary complex, lateral lemniscus, or inferior colliculus. Similar changes have been reported in certain human brainstem evoked response studies,[9] but not in others.[10] Changes in the use of glucose with senescence have been observed in the colliculus of rats,[11] further suggesting sensitivity of this structure to age-related changes in the rat. This brief review of some of the literature on aging in the auditory system is an attempt to point out certain areas in which work has been done. While a considerable number of studies have examined hair-cell loss with age, few have attempted to correlate objective measures of function or functional changes with age in animal models at any level of the auditory system. There is a clear need for additional work in this area.

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INTERACTION OF AGING AND NOISE

Although the issue of interaction of noise exposure and aging is of considerable theoretical and practical interest, it has received surprisingly little attention in the literature. The regulations of the Occupational Safety and Health Administration, as well as most state compensation laws, assume that noise and aging interact in an additive manner: that is, a factor is calculated as an "age correction," which is then subtracted from the worker's hearing level in order to determine the amount of hearing loss due to noise alone. Aside from the issue of the determination of compensable hearing loss, the interaction of noise and presbycusis permeates virtually all large-scale retrospective studies of noise-induced hearing loss in man.

Studies that support the concept of additivity include Macrae's[1] longitudinal study of hearing levels at 1 and 4 kHz in 200 military veterans, ages 30 to 75 years no longer exposed to noise, and Wellenschik and Raber's[2] cross-sectional study of 25,744 industrial employees. The subjects in the latter study were ages 20 to 60 years, exposed to noise levels ranging from <85 dBA to more than 97 dBA/hr, from 0 to more than 25 years. They were grouped into categories of age (<25, 26-35, 36-45, 46-55, and >55) and years of exposure (0, 1-5, 6-15, 16-25, and >26 years). Additivity was observed in all groups except for the oldest group at durations shorter than 15 years.

Two studies by Novotny[3,4] address the issue of additivity. A study of TTS produced by exposure to a broadband noise at 85 dB for 3 min in four groups of human listeners with mild high-frequency hearing losses, ages 30 to 64 years, failed to show any difference in TTS with age. However, a cross-sectional study of 80 industrial employees, ages 30 to 59 years who had been exposed for 5 or 10 years indicated that, for equal exposures, both age groups showed similar hearing levels; that is, no interaction was observed.

Perhaps more important than the interaction issue per se is the more general question, "What is the role of environmental noise exposure in presbycusis?" A recent study by Goycoolea et al.[5] has revived interest in this. Goycoolea's study of hearing in natives of Easter Island who had (1) always lived on the island, (2) lived in civilization for 3 to 5 years, or (3) lived in industrialized society for more than 5 years indicated a progressive deterioration of hearing levels as a function of number

of years in civilization. The median hearing thresholds of natives always living on the island (men and women combined) was found to be similar to those female citizens of the United States; there was no significant difference in hearing thresholds between men and women among these natives. The relation between aging and hearing level in natives always living in the quiet environment was interpreted as true or intrinsic presbycusis; the results also suggested that there are no significant inherent racial differences and no significant inherent differences between males and males in sensitivity of hearing.

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PATHOGENESIS

Progressive bilateral hearing loss in the elderly, primarily affecting high-frequency sounds, is the most common cause of auditory deficiency in adults. In the United States, 23 percent of the population between 65 and 75 years of age and 40 percent of the population above 75 years is affected. Despite the importance of presbycusis as a health problem, little is known of its pathogenesis. Although the most common measure of impairment has been the pure-tone audiogram, this measure does not necessarily give a good indication of the degree of difficulty experienced by the impaired person in everyday life. The most common complaint is difficulty in understanding speech in noise. It now seems clear that hearing loss in the elderly should not be regarded just as an attenuation of sound. Rather, the hearing loss is characterized by a reduced ability to discriminate sounds, which lies at the heart of the difficulty in understanding speech in noise.

Statistically, there are approximately 11,000,000 adults in the United States, ages 18 to 79 years, considered hearing impaired. Corso[1] suggests that the two major factors having deleterious effect on hearing are noise exposure and aging. It is reasonably clear that some important auditory functions do change with age. In general, the losses are greater at high frequencies, and the rate of loss at high frequencies increases with age. Temporal integration—the summation of acoustic energy over time—appears to be reduced by presbycusis.[2] Frequency selectivity, the ability to resolve the frequency components of a complex sound, has been found to worsen progressively with increasing age, although individual variability is much greater in elderly listeners than in young listeners, and not all elderly listeners show reduced selectivity.[3-5] Von Wedel[6] found that temporal resolution was reduced in cases of presbycusis and suggested that this might be important for speech perception. Price and Simon[7] support this idea.

Finally, studies of impaired hearing (including elderly listeners, but not specifically concerned with the effects of aging) suggest that several other auditory functions may be adversely affected by the impairment. Individual differences may be related to differences in the underlying pathology. Schuknecht[8] described four types of presbycusis, classified according to the location of atrophy in the cochlea and eighth nerve. However, his evidence was based on case studies rather than a statistical analysis of a large number of subjects. Other workers have questioned his classification system, since the atrophy is often not restricted to just one or two sites.[9,10] Katsarkas and Ayukawa[11] described that only 50 percent fit into these categories and another 50 percent of the subjects' presbycusis was caused by more than one degenerative process. In 86 percent of the subjects, speech discrimination was excellent or good in quiet; this test therefore may not reflect the difficulties persons with presbycusis have in understanding speech under adverse auditory conditions. Tone decay and recruitment, in most cases, showed that hearing loss was caused by cochlear pathology. However, these data did not exclude the possibility of central auditory involvement. Soucek et al.[12] investigated hearing by evoked audiometry in 90 elderly subjects and correlated data with histologic changes in the cochlea. They observed that the pattern of the waveforms suggested hearing loss of peripheral origin. The histologic portion of the study demonstrated degeneration of the outer hair cells in all three turns of complete loss of the structures in the basal end.

It seems clear that presbycusis can involve both cochlear and retrocochlear degeneration, but it is not easy to determine the relative importance of the two in a given individual on the basis of psychophysical or speech perceptual measures.[13] Measures of tone decay

(adaptation), "rollover" in PB word scores, and measures of binaural processing may be useful for this. In a task involving the use of interaural differences to improve the detection of a tone in noise (the binaural masking level difference), Novak and Anderson[14] found that subjects assumed to have neural presbycusis did not improve as much as other presbycusis subjects. Many recent studies suggest that the proportion of presbycusis caused by retrocochlear involvement may be rather small. For example, in a survey of recent studies, Marshall[13] found that only 4 percent showed abnormal rollover. The relationship between these tests of auditory function and speech intelligibility in noise as a function of age have not been systematically examined.

It is generally accepted that hearing loss occurs among the elderly as a result of aging; however, it has been shown that in some primitive societies, hearing loss is not inevitable. The lack of presbycusis in these societies has been attributed to diet and environmental factors. Human and experimental studies have shown that not only does noise exposure damage hearing, but high levels of lipids in the blood, elevation of blood pressure, and vascular disease also contribute to hearing loss.[15-17] Few animal models have been developed. Keithley and Feldman[18] described degenerative changes associated with aging in rats; Henry,[19] in mice; Schmiedt et al.,[20] in gerbils; and Bennett et al.[21] in primates. Prazma et al.[22] demonstrated smaller blood flow in the lateral portion of the stria vascularis in aged gerbils when compared with young ones.

With an increasing elderly population in the United States, presbycusis will become a more significant health problem. This pathologic process and its relationship to the environment need further investigation.

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THE PREVALENCE OF AGE-RELATED AUDITORY DYSFUNCTION: A SUMMARY

Prevalence

Since 1971, a generally consistent growth has occurred in the estimated number and proportion of people in the United States reporting hearing problems in one or both ears. In 1986, the estimated number of hearing impaired people was 20,732,000, or 8.8 percent of all Americans.[1] The actual number may be larger because audiologic data indicate that people tend to underreport their hearing loss. This increase in hearing impairment

may be largely attributable to the rapid growth of the elderly population in America. For example, 2.0 percent of persons under the age of 18 years were reported to have some degree of hearing impairment in 1986. However, the respective percentages of persons ages 18 to 44, 45 to 64, 65 to 74, and 75 years and over were 5.2, 13.6, 24.4, and 37.8, respectively.[1] Audiologic data show that hearing sensitivity begins to deteriorate more rapidly in the fourth decade of life for both men and women. In interpreting age-related hearing losses, it is important to keep in mind that most people with hearing problems are reported to have less than severe losses.

Data Quality

Major sources of data about the prevalence of hearing impairment are (1) the National Health Interview Survey (HIS), conducted annually by the National Center for Health Statistics (NCHS), and (2) the first Health and Nutrition Examination Survey (HANES), conducted between 1971 and 1975 by NCHS and the 1984 Survey of Income and Program Participation (SIPP), conducted by the Census Bureau. In terms of sampling and interviewing technique, the data are quite good. Because HIS and SIPP survey data, they do not contain accurate measures of hearing impairment. Only broad generalizations can be obtained from these data. HANES contains detailed audiologic data, but the sample size is smaller, limiting study of presbycusis. The HANES data are also quite old. However, NCHS will be undertaking a new HANES in the near future.

Gap Areas

Reliable, current data involving the prevalence of the various degrees of self-reported hearing loss do not exist. Only the overall prevalence of hearing impairment is dependably updated on an annual basis in the National Health Interview Survey. Because of the focus on persons with the more severe hearing losses, we do not have good data on the prevalence of different kinds of mild and moderate hearing losses—both unilateral and bilateral hearing losses. Because of a lack of longitudinal audiologic data, we do not have a complete understanding of how the function of the prevalence of age-related hearing impairment changes as a population grows older. Most demographic studies assume period data by age represents longitudinal change. Moreover, in terms of prevalence, there are not as yet agreed-upon statistical techniques to factor out presbycusis from hearing loss that is caused by elements that exist in environment.

Suggestions

1. NCHS should conduct hearing impairment supplements to the HIS similar to those in 1971 and 1977 on a regular basis. These would update information on various types of hearing losses.
2. The World Health Organization's International Classification of Impairments, Disabilities, and Handicaps should be tested as a framework from which to view these data. Such a system offers the potential to explore the prevalence of different kinds of less than severe losses, as well as speech discrimination impairment as opposed to auditory sensitivity impairment, measured by pure-tone air conduction tests.
3. A longitudinal audiologic data study is needed to determine the extent to which the pattern of hearing dysfunction of a cohort as it ages is similar to that of the age pattern of an entire population at one point in time. Such a study would allow us to measure the impact of cohort effects. The HANES followup offers some potential to study cohort effects on reported hearing loss over time.
4. Feasibility of determining some positive markers of presbycusis loss for survey analysis needs to be explored more. If impossible, statistical indicators of presbycusis need development.

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MULTIDISCIPLINARY INVESTIGATION OF AGE-RELATED AUDITORY DYSFUNCTION

At the basic science level, what used to be considered "multidisciplinary" is increasingly commonplace, often in the hands of a single investigator. Animal studies correlating structural and functional changes (e.g., stria vascularis atrophy vs. endolymphatic potential) in aging animals would be an example. Other approaches include psychoacoustic, micromechanical, and biochemical characterization of the aging auditory system. This eclecticism has become an important feature of modern hearing science which, while certainly welcome, hardly needs further encouragement, except as might be provided by targeted funding for aging research.

There is a great lack of multidisciplinary research in human presbycusis. Among areas open for fruitful clinical collaboration are epidemiology, genetics, internal medicine (regarding interaction of presbycusis with endocrine, nutritional, and cardiovascular disorders), and psychiatry (regarding psychosocial impact of presbycusis and the effects of intervention). These different clinical research disciplines are less likely to be learned by single investigators, so "multidisciplinary" work will be needed. Many very desirable studies in human presbycusis would necessarily be complex and time-consuming, and specialists in biostatistics and clinical trials management would be needed.

The otolaryngologist's role in presbycusis, until now, has primarily been to exclude the uncommon cases of hearing loss due to other etiologies in older hearing-impaired patients. He or she should be an integral part—logically, the coordinator—of multidisciplinary research in presbycusis. If and when medical treatment of presbycusis and/or its putative precursors prove worthwhile, otolaryngologists would naturally assume the responsibility for such treatment or for the identification of patients requiring treatment for other medical specialists. Audiologists will also be crucial members of both research and clinical teams attacking presbycusis. Age-related programs of aural rehabilitation might be developed; demonstrating their efficacy convincingly would probably require help from colleagues in psychology and health care delivery research.

In summary, "multidisciplinary" research is becoming commonplace in basic science, but is rare in clinical studies of presbycusis, which will require large teams and extended study periods in order to attack many problems of interest.

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RESEARCH PRIORITIES

1. To determine the most appropriate statistical models for the interaction of age and hearing loss and of the influence of demographic and economic factors, such as sex, race, religion, marital status, education, and occupation, on this interaction. The models will be evaluated by how well they account for the risk of hearing loss, its age at onset, and the rate of hearing decline.
2. To discover the degree to which socioeconomic status influences the risk of hearing loss, its age at onset, and the rate of hearing decline.
3. To identify whether the process of age-related hearing loss exists independently of identifiable diseases.
4. To explore the health-related consequences of age-related hearing loss, particularly those involving mortality.
5. To measure the applicability of these results for other impairments, other countries, and a wide variety of data sets.
6. To determine the contribution of diet, diabetes, arteriosclerosis, and noise exposure to the development of sensorineural degeneration in the cochlea.
7. To assess differences in regulation of cochlear blood flow in aged and young animals.
8. To quantify the effects of aging on auditory function and speech discrimination and develop valid measures to define impairment and social handicap.
9. Assess the effect of environmental noise by careful study of auditory function in noise-free societies and correlate these with biologic markers of aging, such as aspartic acid polymerization, visual acuity, and pulmonary function.