

Paparella: Volume II: Otology and Neuro-Otology

Section 3: Diseases of the Ear

Part 4: Inner Ear

Chapter 44: Presbycusis

Paúl Hinojosa, Ralph F. Naunton

We are all familiar with the hearing impairment associated with advancing age. We are also familiar with the older person who "can hear everything he wants to hear" and who, while he cups his hand behind his ear when you speak quietly, starts back in discomfort when you raise your voice to help him. "Don't shout", he says, "I'm not deaf". This individual is probably a victim of presbycusis or presbyacusis (literally "the hearing of the old man") and, depending somewhat on the care with which we choose our ancestors, few of us will escape a similar fate should we live long enough.

Zwaardemaker, in 1891, was one of the first to recognize that, where pure-tone hearing is concerned, presbycusis is largely a matter of impairment of hearing for high-pitched sounds; with few exceptions, this impairment of hearing has been assumed to be sensorineural rather than middle ear in nature. Of the many conditions that can lead to sensorineural hearing impairment in adults, presbycusis is the most common and is the major example of hearing impairment in the United States. There may be multiple causes and attention should be given to a complete evaluation and differential diagnosis.

As is true in so many similar situations, recent additions to our understanding of presbycusis have taught us only that our explanations of the etiology and pathology of the condition remain inadequate.

Clinical Picture

Our methods of recognizing presbycusis are very often characterized as much by habit as by logic. We tend to identify a sensorineural hearing impairment as presbycusis not because it has any particularly distinctive characteristics but by recognizing concurrent signs of bodily aging. At the same time, our diagnosis is helped by a process of exclusion as we rule out other causes of sensorineural impairment.

When we encounter an otherwise unexplained sensorineural deafness of moderate degree in a man whose age is stated to be 70 years and who looks his age, we confidently diagnose presbycusis, but the same unexplained impairment in a 40-year-old patient would probably never be described as presbycusis but rather as "a sensorineural impairment of uncertain etiology". The changes seen in the aging auditory mechanism will be referred to in detail later; very clearly, however, we are dealing with a variety of changes that may occur singly or in groups of varying

size, begin early or later in life, and progress slowly or rapidly. Genetic factors play a role in determining the extent and character of the aging process in an individual and, although the chances are less, a 40-year-old may be as much a victim of an aging hearing mechanism as the 70-year-old.

Makishima (1967a) has claimed that there are seven kinds of pure-tone threshold audiograms to be seen in presbycusis: normal, flat, abrupt fall, slowly descending, convex upward, convex downward, and ascending. Traditionally, however, presbycusis is an otherwise unexplained high-tone sensorineural hearing impairment occurring in an elderly person. The hearing impairment tends to progress very slowly, occasionally taking large downward steps at times of physiologic stress (for example, during a severe illness or following administration of a general anesthetic).

The pure-tone impairment of presbycusis may be only moderate but accompanied by a severe speech discrimination difficulty, or the reverse may occur - a pure-tone impairment of a severe degree but with relatively little impairment of speech discrimination. Those persons whose speech discrimination is reduced out of proportion to their pure-tone threshold impairments were said by Gaeth (1948) to show phonemic regression.

In order to evaluate the effect of age changes on the retrocochlear pathways in the clinical situation, Kirikae (1969) studied the hearing for speech in a series of elderly persons with normal or nearly normal pure-tone threshold audiograms. Subjects were selected in this manner in order to limit the influence of inner ear lesions on the test results. In general, the results of these examinations indicated that elderly people with normal or nearly normal hearing for pure tones may show marked impairment of speech discrimination, reduction in discrimination of monaural interrupted speech, and considerable loss in the ability to recognize interaural time differences. Kirikae interpreted these results as confirmation that some of the auditory characteristics of presbycusis may be caused by lesions in the central auditory pathways.

Jerger (1973) evaluated the effects of aging on speech intelligibility by examining the relationship between PB scores and sensorineural hearing loss in 4095 ears of 2162 patients ranging in age from 6 to 89 years. The number of ears in each age decade ranging from a maximum of 740 in the sixth to a minimum of 109 in the eight. PBmax scores were determined for each ear of each patient. The results suggest that the elderly commonly show greater loss of speech understanding than can be accounted for by the loss in threshold sensitivity. When hearing loss was held constant across age, the progressive effect of aging on speech intelligibility loss was clearly documented. The phenomenon was not strongly related to the presence or absence of loudness recruitment, but was related to the difficulty of the listening task; in particular, the phenomenon can be dramatically enhanced by stress in the time domain. Available evidence suggests a central rather than peripheral mechanism for the phenomenon.

Similar conclusions were drawn by Welsh and colleagues (1985), who examined the hearing of a group of elderly listeners; their studies were interpreted as indicating a progressive loss of central auditory competence when measured by simultaneous binaural challenges and by

frequency and temporal distortion tests.

In a related study, Price and Simon (1984) demonstrated that age-related differences occur in the perception of temporal information in speech, thus accounting for the difficulty in understanding speech experienced by some older persons with normal pure-tone hearing.

Commonly, subjects with presbycusis perform poorly in audiometric tests requiring integration of or differentiation between binaurally applied sound stimuli (Matzker, 1954, 1958). It is often claimed that they are entirely unpredictable in presbycusis (Jerger et al, 1959). Auditory recruitment may be present or absent; when present, it is assumed that the lesion includes hair-cell damage; when absent, the lesion is assumed to be retrocochlear (Dix et al, 1948). In all probability, if the two types of lesions coexist, tests will indicate the presence of auditory recruitment rather than its absence (Shapiro, 1969).

Harris and Reitz (1985) measured the speech discrimination of "normally hearing" and hearing-impaired elderly subjects in noise and under reverberant conditions. The "normally hearing" performed well in the quiet, in noise, and with reverberation; but in noise with reverberation their performance was below normal. The hearing-impaired subjects performed poorly in all of the listening conditions.

The preceding discussion, while it may not provide an all-inclusive picture of presbycusis, may serve to remind us that normal auditory function requires far more than a normal peripheral sense organ. Efficient speech communication demands good peripheral function, but good function at all other levels within the auditory nervous system is equally necessary. Age changes or the other changes associated with presbycusis may produce peripheral damage or damage at other levels in the auditory system. The exact nature, locus, and combination of these changes, varying from person to person, will determine the audiometric test results and clinical outcome.

No description of the clinical picture of presbycusis can be complete without reference to the psychology or, better, the psychological problems of the hearing-impaired elderly subject. Understanding of speech can only follow a thought process in which the sounds entering the ears are interpreted. In this thought process, which occupies a brief but finite period of time, linguistic and other restraints are applied to aid in the interpretation and evaluation of what is heard. A knowledge of the topic under discussion provides one "restraint"; a knowledge of English grammar provides another set of "restraints". The young person can usually carry out this process of analysis rapidly, but the older person slows down and, while his peripheral ear may be functioning almost normally, his central auditory or interpretation functions may be reduced considerably below the normal level of efficiency. "Slow" is synonymous, in some quarters, with "mentally inadequate"; recognizing this, the older person will often prefer to remain silent and out of the conversation. He would like to join in the conversation but can do so only when the participants speak slowly so, rather than make a nuisance or fool of himself, he withdraws. And, as he withdraws, he becomes more and more "deaf", joining in only when the linguistic restraints are simple and the subject matter under discussion is very familiar - in other words, "he hears only what he wants to hear".

Incidence and Distribution

Hearing threshold levels for various segments of the population of the United States have been measured in a number of large-scale surveys and studies in recent years. One of the earliest of these studies was the clinical and audiometric investigation of approximately 9000 persons carried out in the course of the 1935-1936 National Health Survey (1938). At the 1954 Wisconsin State Fair, approximately 35,000 persons between the ages of 10 and 79 years were tested with standard pure-tone threshold audiometers (Glorig et al, 1957). Those tested mainly lived in Milwaukee and its surrounding areas, and there is some evidence that the subsample used for describing the threshold hearing levels characteristic of advancing age (for the most part male office workers) included an inappropriately large number of persons habitually exposed to industrial noise of an intensity likely to damage hearing (Nixon et al, 1962). In the 1959-1962 National Health Survey (National Center for Health Statistics, 1965) more than 6000 persons between the ages of 18 and 79 years were examined and tested. The results of these three different surveys are shown and, while there are significant differences among the findings of the various surveys, they all demonstrate a hearing impairment increasing with advanced age, at all test tone frequencies, greater for high frequencies than for low. The survey results also emphasize that at the higher frequencies there is a significant difference between hearing in men and women, with men showing greater losses than women.

In somewhat similar studies in 1959, Hinchcliffe (1959a, b) studied the pure-tone hearing threshold levels in a rural population in Great Britain, demonstrating that in all age groups there were significant differences between the hearing levels for men and women at 3, 4, and 6 kHz, and at 2 and 8 kHz in the older groups. He also demonstrated that the median hearing level of the population studied showed an increasing loss of sensitivity, or increased hearing impairment, with advancing age at all of the frequencies tested.

Rosen and colleagues (1962) carried out a very similar study on an entirely different population group. Rosen and his team visited an isolated tribal community living in a relatively noise-free environment in a remote area of the Sudan. The hearing of these people, the Mabaans, was strikingly different from the hearing of the population groups from the urban areas previously studied in that far less hearing impairment was seen at corresponding age levels of the Mabaans. Moreover, among the Mabaans, where men and women were equally exposed to the same environmental noises, the hearing of the average female was no different from the hearing of the average male.

Peculiar but impressive differences between the elderly Mabaan's hearing and the hearing of the average elderly citizen in any one of Western civilization's major cities may, in part, be attributed to the Mabaan's noise-free environment and to our own noisier existence. But the Mabaans do not suffer from hypertension, coronary artery disease, duodenal ulcer, allergies, or bronchial asthma; they have very little arteriosclerosis and live lives with far less stress and strain. It is likely that other as yet unidentified explanations exist and that the differences between some of the Western studies may be due to variations in testing conditions, population differences, or other factors; much of this remains in the realm of conjecture. While it is very

clear from the results of every study undertaken that the majority of the world's population suffers some degree of progressive hearing loss with advance of age beyond approximately 40 years, it is equally clear that some individuals reach a very advanced age and show near-normal or completely normal hearing in all respects. Many composite audiograms have been published by different authors, studying different populations, indicating the progressive loss of hearing sensitivity, particularly for high-pitched sounds, as age advances. It would be misleading to represent any one of these groups of findings as representative of the general population because of identified and unidentified population differences and because of differing measurement techniques. The only knowledge to be gained from these composite audiograms is that, on the average, hearing acuity for pure tones deteriorate with advancing age, the deterioration being greater at the higher frequencies.

Past "baby booms", advances in medical care, and probably other unidentified factors are determining that our population is aging. In a few decades from now the elderly will comprise a far larger proportion of our population than is the case at the present time. There seems little likelihood that the prevalence of hearing impairment in older persons will diminish; we must, therefore, expect that the number of hearing-impaired persons in our population will increase, and that the increase will be largely if not entirely due to the increase in the elderly (Naunton, 1986).

Pathology and Etiology

Early Reports of Histopathologic Changes

Crowe and associates (1934), in a classic study, examined histologically a number of human ears that in life had shown high-tone sensorineural losses. In some cases the prominent histologic lesion was atrophy of the organ of Corti in the basal turn; in other cases the most obvious histopathologic lesion was a partial atrophy of the cochlear nerve supply to the basal turn. In yet other cases the authors were unable to explain the clinical hearing losses on the basis of observed cochlear pathology. Also, they found no correlation between any vascular changes present in the vessels of the inner ear and degeneration of the membranous labyrinth; they therefore discounted arteriosclerosis as a cause of these changes.

Saxen (1937) described two types of pathologic change occurring in the auditory system, which he believed to be due to aging processes: the first was atrophy of the spiral ganglion, starting in the basal coil; the second was a progressive degeneration of the epithelial tissues of the cochlea, believed to be due to arteriosclerotic changes. Saxen also described a patient with marked hearing loss but in whose inner ear no cochlear nerve or labyrinthine changes could be found; the hearing impairment in this case was believed to be the result of degenerative changes in the central auditory nerve system. Crowe and co-workers and Saxen were therefore in agreement on the following points: (1) there are two types of histologic changes associated with presbycusis, one involving mainly the cochlear epithelial tissues, the other involving the spiral ganglion; (2) there are patients in whom hearing impairment cannot be explained on the basis of observable histopathologic changes in the inner ear.

Later Reports of Histopathologic Changes

In 1955 Schuknecht published the results of a quantitative study of the histopathology of the ears of several cats and humans with high-tone sensorineural hearing impairment. On the basis of these findings he concluded that there are two types of presbycusis. In the first, epithelial atrophy (later to be described as sensory presbycusis), degenerative changes begin at the basal end of the cochlear duct, including afferent and efferent nerve fibers. Schuknecht believed this variety of change to be the auditory manifestation of the aging process that affects all tissues of the body. Clinically it produces the familiar audiogram of presbycusis, starts at middle age, and progresses slowly.

Schuknecht's second type of presbycusis was described as neural atrophy (later to be described as neural presbycusis) and is characterized by a progressive degeneration of spiral ganglion cells (starting at the basal end), and of the neurons of the higher auditory pathways. Neural atrophy is the auditory manifestation of the well-recognized aging process that affects the central nervous system and results in loss of neurons; its clinical counterpart is a predominantly high-tone deafness with disproportionately severe impairment of speech discrimination. These two types of histopathologic changes and the clinical pictures associated with them can be superimposed upon each other in various proportions.

Schuknecht (1964) and Gacek and Schuknecht (1969) suggested that there are at least four varieties of presbycusis, including the two described earlier. "Sensory presbycusis" (previously described as epithelial atrophy) involves atrophy of the organ of Corti and the auditory nerve at the cochlear base, starting in the middle age and progressing slowly. It is manifest clinically by an abrupt high-tone hearing impairment but, even at an advanced age, the lesion is usually confined to the first few millimeters at the basal end and does not lead to impairment in hearing for the speech frequencies. The primary site of degeneration is probably in the supporting cells, the auditory nerve being only secondarily affected.

"Neural presbycusis" (previously described as neural atrophy) results from the loss of neurons in the auditory pathways and cochlea. Neuronal atrophy in the central nervous system starts early in life and continues remorselessly throughout life, the actual rate probably being under the control of genetic factors; it has no significant effect upon hearing until relatively late in life, when the neuronal population falls below a certain minimum. In cases of neural presbycusis, speech discrimination commonly falls significantly without a correspondingly severe pure-tone hearing impairment. This may be the clinical condition described by Gaeth (1948) and referred to as phonemic regression.

Schuknecht and Woellner (1955) showed that cats may preserve normal auditory thresholds even though they may lose as much as 86 per cent of the lower basal coil spiral ganglion cells. They found that, as a general rule, when more than 75 per cent of the spiral ganglion had degenerated in a particular region of the cochlea, an increased threshold occurred for frequencies having their locus of excitation in that region.

Schuknecht's third variety was "metabolic presbycusis". This is believed to be due to defects in the physical and chemical processes of energy production in the sense organs. The corresponding clinical manifestation is a flat audiogram, and the underlying pathologic change is atrophy of the stria vascularis. It is believed that the stria is responsible for the maintenance of the bioelectric and biochemical properties of the endolymph and that patients with atrophy of the stria vascularis commonly have flat audiograms.

The fourth variety was termed "mechanical presbycusis"; it is characterized clinically by a slowly progressive hearing loss for high tones without any abrupt descent. This is believed to be due to a disorder in the mechanics of the cochlear duct. In an electron microscopic study of temporal bones from individuals who had suffered from presbycusis, Nadol (1979) found thickening of the basilar membrane in the basal turn of one of the three bones examined; the thickening consisted of an increased number of fibrils and an accumulation of an amorphous osmiophilic material. This findings lends support to the reality of Schuknecht's earlier conjecture that there is a variety of presbycusis resulting from changes in the mechanics of the cochlear duct. Changes of this nature might, for example, affect the elasticity of the basilar membrane. The greatest threshold loss in this variety of presbycusis is for high frequencies, represented by the basal end of the cochlea where the basilar membrane is narrow; hearing for low frequencies is least affected, these being represented presumably by the apex of the cochlea where the membrane is widest.

Katsarkas and Ayukawa (1986) reviewed the pure-tone audiograms of 91 cases of what they defined as "pure" presbycusis. Only about 50 per cent of the audiograms could be visually fitted into the audiograms said to be associated with the histopathologic types of presbycusis described by Schuknecht. The other 50 per cent showed either pure-tone threshold curves that could partially fit any of the histopathologic types, or mild to moderate losses compatible with any type of presbycusis at an early stage.

Kirikae (1969) commented on the general recognition and agreement that presbycusis may be caused in part by disease of the inner ear, especially the organ of Corti and spiral ganglion. However, he also noted that there is considerable evidence to support the contention that histopathologic signs of degeneration due to age have been identified in the central auditory nerve system. Characteristic age changes occurring in the auditory pathways of the central nervous system and the cerebral cortex have been reported by several authors; for example, Kirikae and co-workers (1964) reported that changes of this nature were observed in the major nuclei of the auditory pathways (the ventral cochlear, superior olivary, lateral lemniscus, inferior collicular and medial geniculate nuclei, and in the cortex). The histologic changes observed included atrophy, nuclear pyknosis, and the appearance of ghostlike and indistinct ganglion cells. Hansen and Reske-Nielsen (1965) have also reported the observation of similar histologic changes in the auditory pathways and cortex. These changes clearly indicate that the functioning ganglion cells decrease in number as age advances. Arnesen (1982) studied the neuronal population of the cochlear nuclei of subjects with a prior diagnosis of presbycusis. In reaching the diagnosis, based finally upon clinical and audiometric criteria, subjects with a history of neurologic or systemic disorders or other variety of potential hearing disorder were excluded. Thus, the six subjects

whose ears were selected for examination had hearing losses attributable only to presbycusis. The neurons were counted in both right and left cochlear nuclei, following the technique described by Hall (1964). A reduction in the number of neurons was found in all 12 nuclei studied. The average loss of neurons was about 50 per cent relative to the number expected in normally hearing individuals (Hall, 1964). The author concluded that not only the cochlea but also the cochlear nuclei are involved in the degenerative processes associated with advancing age.

Makishima (1967b), in presenting the histologic findings in two unusual cases of presbycusis, commented that the most conspicuous histologic change observed was atrophy of the spiral ganglion; atrophy in the stria vascularis and depletion of hair-cells occurred, but these changes were slight. Degenerative changes in the central auditory pathways appeared to cause low-frequency as well as high-frequency hearing impairment. An additional finding was that arteriosclerotic narrowing of the internal auditory artery correlated positively with atrophy of the spiral ganglion and also with the degree of hearing impairment.

Suga and Lindsay (1976) reported the temporal bone histopathology of 17 elderly subjects who had shown spontaneous and gradually progressive bilateral sensorineural hearing losses. The most prominent histopathologic change observed was atrophy of the spiral ganglion. The audiogram and temporal bone sections shown are of one of their reported cases, showing bilaterally symmetric sensorineural pure-tone threshold impairment and speech discrimination, extensive bilaterally symmetric spiral ganglion cell loss, but limited hair-cell and stria atrophy. Diffuse senile atrophy was also often seen in the other structures of the inner ear, such as the organ of Corti and the stria vascularis. No consistent correlation was observed between the type of audiometric curve and the locus of lesions in the sensory, neural, or vascular elements of the cochlea, nor was any positive correlation found between the degree of arteriosclerosis and the degree of cochlear sensorineural degeneration. In 50 per cent of their cases, the spiral ganglion cells showed marked atrophy with no apparent histopathologic changes in the organ of Corti or the stria vascularis. In all six cases with the classical downward sloping audiogram of presbycusis, the spiral ganglion cells were degenerated in almost the entire length of the cochlea, degeneration being more marked in the lower than in the upper turns. Stria atrophy, on the other hand, was usually found in the middle or upper turns without corresponding atrophy of the spiral ganglion cells. Suga and Lindsay's observations suggest that audiometric configuration in presbycusis does not necessarily point to any specific locus of cochlear degeneration, findings that appear to be in sharp contrast with the earlier findings of Schuknecht (1955, 1964) and Gacek and Schuknecht (1969).

Etiology

In 1964 Hinchcliffe examined the hearing of a group of elderly men and women living in a rural area of Jamaica. A striking observation emerging from this study was that the women had greater hearing impairment at 2 kHz than an otherwise similar group of Scottish women studied under very similar circumstances (Hinchcliffe, 1959b). Possible explanations include exposure to noise, exaggerated aging changes, occurrence of a familial variety of sensorineural impairment, and the unexplained prevalence of sensorineural impairment in the area concerned.

Hinchcliffe believed the last explanation to be correct. The men in these two studies did not exhibit the same between-group differences as the women; this might be explained by the prevalence of noise-induced hearing loss in the Scottish men of similar magnitude to that of the unexplained sensorineural impairment occurring in the Jamaican men.

Hinchcliffe (1962) has also pointed out that many of our special and general senses exhibit an exponential decrement with advance of age. Among the senses specifically mentioned are hearing thresholds for random noise, visual sensitivity, gustatory sensitivity, corneal sensitivity, and hearing for sounds of frequencies 0.25 through 4.0 kHz. Hearing for sounds above 4 kHz behaves differently, showing a more rapid deterioration with advancing age. It is suggested that linear deterioration in brain cell count is the structural correlate of the observed exponential deterioration of various threshold sensitivities in the elderly. In the case of the ear it cannot be denied that histopathologic changes can also be seen in the peripheral sense organs, but, initially, these changes correlate poorly with function (Crowe et al, 1934), whereas later they affect hearing for those frequencies (4 kHz and above) at which Hinchcliffe (1962) showed a departure from the "exponential deterioration with age" dictum.

Tissue Aging in General

Human beings cannot claim exemption from the general rule of life that all living things age. Tortoises age slowly, some insects age rapidly and live for no more than a few hours, but we are all victims of the same aging process from the time of our birth. The process of aging is so universal that we commonly regard it as physiologic.

Animal tissues, organs, cells, subcellular structures, and enzymes have all been studied in the effort to explain this aging process; as a result, a number of different factors can now be implicated. These include the following:

1. A reduction of cell production.
2. Extracellular deposition of various materials (eg, cholesterol, atheroma, lipid in the cornea, and fluid in the retina).
3. Aging of extracellular substances such as collagen, elastic tissue, cartilage, and bone.
4. Overgrowth of cells and their products, most marked among fibroblasts of vessels and chondrocytes and osteoclasts of many joints.
4. Accumulation of substances in such cells as neurons, muscle cells, parenchymal cells of the liver, and adrenal cortex.
6. Intracellular aging changes of an uncertain nature.

It is likely that all these various types of aging processes occur in the auditory system, and it is equally likely that other processes associated with aging remain unidentified.

Summary

In summary, the core of our understanding of presbycusis is that an exponential "physiologic" aging process occurs, probably common to all of the special and general senses. The histopathologic correlates of this change, which occurs primarily in the central auditory nervous system, probably include a variety of changes at the cellular and subcellular levels. A characteristic hearing impairment commonly occurs at high frequencies. The histopathologic correlates of this impairment are the various changes seen in the peripheral organ, but the etiology remains uncertain; it seems that genetic factors may be involved, and Rosen and colleagues (1964) implicated such factors as metabolism, nutrition, and climate. A third factor, of surprisingly uncertain magnitude, is noise; we are very familiar with the hearing impairment shown by the punch press operator, but we are far less certain of the effects on the ear of household noise, airport and airplane engine noise, noisy cars, noisy trains, noisy music, and so forth.

Prevention and Alleviation

Little can be said about the prevention of presbycusis beyond avoidance of those factors, such as noise, which are believed to be involved in the general etiology of the problem. We are otherwise largely dependent upon the eventual positive identification of other controllable etiologic factors.

Hearing aids are commonly assumed to be of little value in presbycusis. The tendency of the elderly to leave their hearing aids in the bureau drawer is legendary and may be due in part to unrealistic expectations and in part to central auditory nervous system changes. Franks and Beckmann (1985) comment that there are estimated to be in excess of 6 million adults aged 65 years and older who have hearing problems that would justify consideration of hearing aid use, but that only about 20 per cent of this number actually use hearing aids. They found, in a study of 100 normally hearing and hearing-impaired individuals aged 65 years and older, that the foremost reasons for non-use of hearing aids included cost, calling attention to a handicap, hearing aid dealer practices, difficulty manipulating hearing aid controls, and not knowing where to obtain a hearing aid. Hearing aids have changed and are continuing to change; but perhaps even more dramatic are the improvements taking place in hearing aid fitting techniques. We are moving into an era in which we will be able, in a far more literal sense than has been possible in the past, to match the hearing aid to the hearing-impaired listener rather than the listener to the aid.

The introduction of the CROS hearing technique (Harford and Barry, 1965) and the use of vented earmolds also serve to improve the possibility that the elderly can use hearing aids effectively. The effects of the vent is to reduce the response of the hearing aid for low tones, tones for which the patient with presbycusis usually has little impairment. The vented earmold

also permits the entry of "unamplified" sounds to the ear wearing the hearing aid; this gives a more normal quality to the type of sound the listener hears. The CROS hearing technique is essential for widely vented earmolds of this type in order to prevent acoustic feedback.

Impressive progress has been made in recent years in the design, availability, and acceptability of assistive listening devices. These include telephone and doorbell alerts, volume-control telephone handsets, telephone amplifiers, and television and radio listening devices. Physicians must be prepared to refer hearing-impaired individuals not only to hearing aid providers, self-help groups, and counseling resources, but also to retailers and centers and other organizations able to provide information and counseling on the full and changing spectrum of available assistive listening devices.