Age-Related Hearing Loss

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Introduction

With advances in health care, life expectancy is increasing. Aging is associated with multiple related medical problems which have been referred to as geriatric syndromes. These syndromes typically interact with one another, have more than one cause, and broadly impact functional status of the geriatric patient. Sensory problems, such as impairments of vision, balance, and hearing, are well recognized geriatric syndromes, with hearing loss being the most common sensory problem among older adults.

In 2012, the U.S. Census Bureau projected that the U.S. population aged 65 and older will outnumber the population younger than 18 as of 2038. It is projected that, in 2015, 15% (47 million) and 2% (6 million) of the population will be 65 and 85 years old and older, respectively; but by 2060, the percent distribution will increase to 22% (92 million) and 4% (18 million), respectively. Studies based on the National Health and Nutrition Examination Survey (NHANES) show that an increasing proportion of the population suffers from hearing loss, reaching more than 80% of those older than 85 years. Age-related hearing loss (ARHL) is, by far the leading cause of hearing loss in developed countries.

By definition, ARHL is a progressive disorder. Another term commonly used for this disorder is presbycusis. New York otolgist St. John Roosa is credited with being the first to describe it as “a physiological . . . rather than a pathological, change in the ear . . . analogous to presbyopia, and . . . termed presbykousis.” The term is derived from the Greek presbus, (elder), and acoustes (to hear). Today, our understanding of this disorder has expanded to include pathological processes in the cochlea and the brain that were not suspected in the late 19th century. Whereas the peripheral pathology is universally accepted, our understanding of changes in the central auditory pathways and the global implications of these changes for management of patients with ARHL is continuing to evolve.

Presentation

Although onset is variable and dependent on several contributing factors, often the earliest symptoms of ARHL appear late in middle age. By this time, cochlear changes have progressed to affect hearing sensitivity in the frequency range that makes up our daily communication sounds. Patients commonly misunderstand similar-sounding words and tend to use context to compensate for this early deficit. In general, consonants are higher pitched than vowels and are spoken more softly than vowels. Age-related high-frequency hearing loss will result in difficulty hearing consonants and makes it easier for background noise to mask. Because consonants convey most of the information in a word, inability to hear them effectively will result in deterioration of speech intelligibility. Furthermore, consonants serve to separate syllables and words from one another. Therefore, with disruption of these breakpoints, words tend to run together and sound “mumbled.” Because voices of children and women tend to have a higher pitch, the initial complaints might be about the low volume and quality of a grandchild’s voice.

Over the years, as disruption of cochlear function extends toward the lower frequency regions, functional consequences become more substantial. With age-related cognitive changes ranging from slowed processing to frank impairment, the ability to use context efficiently to compensate for hearing deficits diminishes. Hearing in noisy or reverberant environments and accented or fast-paced speech become more challenging. A common complaint arising from these disruptions in speech intelligibility is “I can hear the words, but I can’t understand them.” Patients resort to varied strategies to cope. These strategies are influenced by several factors, including personality traits. When individuals have an external locus of control, complaints such as “my grandchildren mumble” are common. On the other hand, those with an internal locus of control display maladaptive strategies such as withdrawal from family conversations.
In general, evaluation and management of hearing loss require insights into the contributing pathophysiological conditions. However, management of age-related disorders is far more complex and challenging because these disorders, including hearing loss, rarely present in isolation. It is estimated that 50 to 75% of adults over age 65 have multiple chronic health conditions. Improving care of these individuals is a priority for the Department of Health and Human Services, and they are a focus of Healthy People 2020. Toward this goal, effort is being directed toward integrated care for older individuals with multimorbidity. This effort is compelling because persons with combined disabilities are at increased risk of cardiovascular and all-cause mortality.

The diagnosis of ARHL is based on patient history, physical examination, and a battery of audiological and other testing. Pathophysiology of ARHL is that of a progressive and insidious process, with affected individuals frequently less aware of their communication difficulties than the people around them. In longitudinal studies, deterioration of hearing is reported to be continuous and gradual for the majority of people, ranging from 1 to 6 dB/decade, although this rate may increase up to 9 dB/decade in older individuals. Because of the insidious nature of this disorder, patients often present reluctantly to the clinician at the insistence of family members.

Besides speech sounds, other important high-frequency warning sounds (alarms, ringing tones, turn signals, etc.) also become more difficult to hear. The common scenario in which a grandparent wearing a digital watch is unaware of the beeping high-pitched alarm is one typical example. Reduced ability to hear alarms raises concern about safety. For example, older individuals with hearing loss have been shown to be at increased risk of motor vehicle accidents while driving. There are also social ramifications to this attribute of age-related hearing loss. Difficulties hearing on the telephone, particularly cell phones in which quality of sound may fluctuate with the strength of the network signal, serve as a barrier to their effective use as an alternative to face-to-face communication. The use of high-pitched ring tones by adolescents to communicate via cell phones takes advantage of the inability of the older listeners to detect these sounds in structured settings such as classrooms. Older listeners are also typically unaware of unpleasant high-pitched noise emitted by some electronic devices and chargers, as well as antiloitering devices.

Besides difficulty in hearing communication sounds and alarms, other auditory functions are also impaired. For example, age and hearing loss also impact localization performance. A large body of evidence shows that the accuracy to localize sound sources declines with age, resulting in front–back confusions, especially for spectrally restricted sounds. Localization accuracy and acuity of acoustic stimuli decline with age and result in a blurred representation of sound sources. These deficits are attributed to age-related increase in the neural temporal jitter in the central auditory system that affects the accurate processing of timing information, which is crucial to acuity in representation of sound source position.

As hearing loss severity increases, overall functional status diminishes among older individuals. It has long been speculated that inability to communicate effectively, and potential decreased overall functional status, will lead to social isolation. For example, hearing impairment has been shown to be associated with poorer scores in social functioning assessments in older Australians. Similarly, poorer self-reported hearing scores predicted deterioration in social support among a large cohort of Dutch seniors. In a recent study by Mick et al, in which cross-sectional data for adults 60 to 84 years old were extracted from the 1999 to 2006 cycles of the U.S. NHANES, revealed that greater hearing loss is associated with increased odds of being socially isolated in women aged 60 to 69 years. This association was not affected by use of hearing aids. Based on their results, Mick et al conjectured that females may rely more heavily than males on verbal communication; therefore, hearing loss might impair their ability to receive emotional support to a greater degree than males. Social isolation has significant implications for the well-being of geriatric patients: lonely or isolated older adults are at greater risk for all-cause mortality and development and progression of cardiovascular disease, and lonely older individuals are more than twice as likely to develop Alzheimer disease (AD).

As noted earlier, hearing loss in older individuals was shown to be associated with increased all-cause mortality in the Australian Blue Mountains Hearing Study. Increased all-cause mortality is believed to be mediated by three variables: disability in walking, cognitive impairment, and self-rated health. Thus, besides the insidious nature of the disorder, the isolation associated with hearing loss may be another factor that leads to delayed presentation and diagnosis, primarily because there is little pressure to seek care for communication difficulties.

Inherent difficulties in communication that result in compounding psychosocial effects such as isolation may precipitate psychiatric disorders such as depression. Whether hearing loss can contribute to depression has been a subject of debate. “Limited” and “pervasive” degrees of depression were reported in 69% of community-dwelling elders with hearing impairment compared with 31% of non-hearing-impaired individuals. Davis and colleagues reported that hearing-impaired elders were 1.79 times more likely than non-hearing-impaired subjects to be depressed. Consistent with this view,
ARHL tends be associated with late-onset depression but not with early-onset depression. In a meta-analysis of published literature, the relationship between chronic diseases and risk for depression in old age was examined by calculating odds risk (OR) and relative risk (RR) for prevalence and incidence rates of depression, respectively. Loss of hearing was among a few chronic diseases, including stroke, loss of vision, cardiac disease, or chronic lung disease, that had both a significant OR and a significant RR for increased depression in old age.

On the other hand, the Nord-Trøndelag Hearing Loss Study with data from over 50,000 Norwegians aged 20 to 101 found substantial effects of hearing loss on symptoms of anxiety, depression, self-esteem, and subjective well-being in young and middle-aged persons, but not the older persons. Similarly, the Longitudinal Aging Study Amsterdam, in a 4-year follow-up study of older hearing-impaired subjects, found that, although hearing loss was associated with loneliness, it was not associated with depression.

In a cross-sectional study of Americans aged 50 and above, dual sensory loss, vision loss only, and hearing loss only were significantly associated with depression after age, gender, poverty, education, functional impairment, bed days, self-rated health, social support, and social activities were controlled for. In contrast, an English longitudinal study, controlling for health-related variables, including the number of medical conditions and functional disability renders insignificant the association between sensory loss in both vision and hearing with both the onset and the persistence of depression in older persons.

If depression is indeed a consequence of hearing loss and isolation, that may further reduce the likelihood of patients with ARHL seeking medical attention. Given its psychosocial implications, it has been recommended that clinicians maintain a low threshold for suspecting hearing loss in older patients, particularly when they present with comorbidities like anxiety, depression or apparent cognitive decline.

Another symptom that affects the well-being of patients with sensorineural hearing loss is tinnitus. About 85% of patients visiting an otologist have tinnitus. The incidence of tinnitus increases with age: tinnitus affects 15% of the general population and 33% of geriatric persons. In a longitudinal study of Swedish men and women in their 70s, 15% had continuous tinnitus, and 42% had occasional tinnitus, without any difference in the prevalence of tinnitus between men and women. It is not surprising that tinnitus commonly accompanies ARHL. This latter entity has been referred to as presbytinnitus. Presence of tinnitus by itself is not an independent risk factor for depression, but older individuals who perceive their tinnitus to be a problem or who have problems with tinnitus when going to bed often display depression symptoms. In patients who also have ARHL, tinnitus can be a source of emotional and sleep disorders, difficulties in concentration, and social problems. Tinnitus has been divided broadly into two groups based on age of onset: early- and late-onset. The two differ not only with regard to prevalence but also with regard to tinnitus-related distress, with late-onset sufferers being more distressed. Resting state electroencephalography source-localized activity and connectivity comparing the two groups revealed increased activity and functional connectivity in the late-onset group, supporting intrinsic differences in tinnitus-related neural activity, which may have implications for management.

In geriatric patients, the presence of tinnitus is associated with reduced systolic and diastolic blood pressure, reduced left ventricular ejection fraction, and increased brain natriuretic peptide (BNP) plasma levels. These findings suggest that tinnitus is associated with worse control of congestive heart failure in geriatric patients and may have important clinical implications for the early identification of patients who need more aggressive management of heart failure. These findings also lend support to the notion that hemodynamic imbalance can contribute to cochlear impairment in general and tinnitus in particular.

### Risk Factors

Several factors have been recognized as contributing to the development of ARHL. These might be broadly classified into two categories: intrinsic and extrinsic. Intrinsic factors are host factors and are primarily genetic (including sex and race), but also include health comorbidities (hypertension, diabetes, and stroke). Extrinsic factors in the environment include occupational and leisure noise exposure, smoking, ototoxic medications, socioeconomic status, and other factors. A more practical classification of risk factors is based on whether they can be modified to reduce their impact on ARHL. From this perspective, at the present time, genetic factors are not modifiable. In contrast, disease processes and environmental factors are believed to be modifiable such that their control could delay or minimize hearing loss.

### Genetic Factors

Individuals with ARHL often report a family history of hearing loss among parents, siblings, and close relatives. Therefore, it has been presumed that ARHL has a genetic component that influences the age of onset and severity of the loss. Challenges in separation of environmental from genetic factors...
have made it difficult to assess the contribution of genetics to ARHL. Several lines of evidence, including animal research, large population-based cohort studies, and gene studies using linkage and association analysis have led to estimations of heritability and identified several genetic foci that are thought to be contributory.

Similarities between the auditory systems of mice and humans have allowed researchers to use mice as a model for better understanding of ARHL. Specifically, mutation of the Ahi1 gene (age-related hearing loss gene 1), mapped to chromosome 10, is associated with elevated hearing thresholds at higher frequencies in middle- and older-aged inbred mice. Cadherin 23 is the gene associated with this locus and has been localized to stereocilia. Based on this finding, it is hypothesized that cadherin 23 plays a critical role in signal transduction in the inner ear. More recently, four other genes on the mouse chromosome 10 have been implicated. Using a genetically heterogeneous population of mice, several polymorphisms affecting ARHL and its modulation by noise have been defined, which included chromosome 10. The homologous genes regulating ARHL have yet to be identified in humans.

Large-population-based cohorts have proven useful in detecting the role of inheritance in ARHL. In the Framingham cohort, heritability of ARHL phenotypes was estimated to be 0.35 to 0.55. In that study, hearing levels in genetically unrelated and genetically related individuals with sensory and stria1 presbycusis were compared. The sensory presbycusis phenotype (described later in the chapter) showed a familial aggregation of hearing thresholds, which was greatest for mother–daughter pairs, sister pairs, and brother pairs. The correlations for the father–child pairs were not significant, which was suggestive of extrinsic factors playing a larger role in the father’s hearing loss patterns. The stria1 presbycusis phenotype (described later in the chapter) demonstrated a strong familial association in the sister–sister and mother–daughter pairs. Overall, the heritability estimates suggest that 35 to 55% of the variance of the sensory presbycusis phenotype and 25 to 42% of the stria1 presbycusis phenotype are attributable to genes. The results of this study demonstrated that, in a large group of biologically related people, hearing sensitivity is more similar than in a group in the same general environment but who are unrelated. A subsequent study in this population examined the genetic linkage between measures from audiometric examinations and markers from a genomewide scan. The scan identified multiple chromosomal locations with evidence of linkage to presbycusis, with some of these locations corresponding with genes implicated in congenital deafness. The analysis revealed three distinct regions on chromosome 11 (2, 79, 143 cM), as well as a region on chromosomes 10 (171 cM), 14 (126 cM), and 18 (116 cM) that showed evidence of linkage.

Heritability of audiometric shape parameters and the familial aggregation of different types of presbycusis were investigated in siblings. The authors found higher heredity for severe types of presbycusis compared with moderate or mild types, and low heredity for “concavity.”

The association between the magnitude of hearing loss and self-reported family history was explored in a study conducted in a population 50 years or older in Sydney, Australia. The prevalence of hearing loss was 33%, with 68.2% classified as mild and 31.8% classified as moderate to severe. Of the 2,669 subjects, 46.7% gave a family history of hearing loss. Participants who reported a family history of hearing loss were younger than those who reported no family history. Participants with increased severity of hearing loss were also more likely to report a family history of hearing loss among parents or siblings. After adjusting for known risk factors (age, sex, history of noise exposure, diabetes, smoking) a positive family history was shown to be strongly associated with hearing loss. This association was true regardless of whether the loss was reported in the mother, father, or siblings. The findings from this study support a strong association between family history and presbycusis, with the association seemingly stronger with more severe hearing loss. Strong associations were found between maternal family history and moderate to severe hearing loss in women and paternal family history and moderate to severe hearing loss in men.

A study of monozygotic and dizygotic twins explored the relative importance of genetic and environmental factors in self-reported reduced hearing among an older Danish population. That study showed that probandwise concordance rates (probability of disease for one twin given that the partner is affected), and odds ratios (the increased risk of reduced hearing for one twin given the presence vs. the absence of reduced hearing in the partner twin) were higher in the monozygotic twin pairs than in the dizygotic twin pairs, indicating a heritable effect. The heritability was estimated to be 40%.

Members of the National Academy of Sciences–National Research Council (NAS–NRC) twin panel underwent a linkage analysis for presbycusis. This study highlighted a region of chromosome 3 mapped to the DFNA18 locus and showed a heritability of 61% for presbycusis. Individuals carrying two mutations of gap junction gene, GJB2, are at increased risk of developing early presbycusis.

In another effort to identify specific ARHL genes, a genetic association study was performed with 2,418 samples from across nine European countries. One gene, the grainyhead-like 2 gene (GRHL2), was found to be associated with ARHL in this population.
contrast, no positive association was found between GRHL2 polymorphisms and ARHL in Han Chinese. Therefore, population differences might be a key factor in genetic expression.

Mitochondrial DNA mutations also have been implicated in the development of presbycusis. Mitochondrial function is essential for tissues with high metabolic activity such as the cochlea. Mutations in the mitochondrial genome accumulate with age; and once they reach a threshold level, oxidative phosphorylation and tissue function are compromised. Bai and colleagues reported that the Common Deletion (CD, a 4,977 base pair deletion most commonly associated with aging) was found in a higher frequency in temporal bones from individuals known to have been affected by presbycusis compared with those unaffected. Consistent with this finding, Markaryan and colleagues reported that, with increasing age, the quantity of the CD increased and that the amount of the CD directly and significantly correlated with the severity of hearing loss at 8,000 Hz. Mitochondrial mutation and deletion have been shown to contribute to the development of ARHL in a rodent model of presbycusis. On the other hand, ARHL in humans was not associated with mitochondrial mutations in a large sample of 200 patients with ARHL.

Oxidative stress is one possible mechanism for the aging process, and cochlear oxidative stress has been implicated in mouse models of ARHL. The dismutase 2 (SOD2) gene encodes a ubiquitous mitochondrial superoxide dismutase enzyme (manganese superoxide dismutase [MnSOD]) crucial for maintenance of reactive oxygen species homeostasis and has been implicated in the pathology of aging. SOD2 expression is reported to increase along a basal-to-apical gradient in cochlear spiral ganglion cells in a manner consistent with the known gradient of hair cell loss in ARHL. A genetic association between different polymorphisms in the SOD2 gene and noise-induced hearing loss has also been described. A role of common SOD2 promoter variation on SOD2 promoter regulation has been described, and SOD2 has been linked to ARHL risk in men, further implicating mitochondrial genes.

Antioxidant enzymes include those involved in glutathione metabolism, such as glutathione S-transferase (GST) and N-acetyltransferase (NAT), which are involved in the metabolism and detoxification of cytotoxic and carcinogenic compounds as well as reactive oxygen species (ROS). Individuals carrying polymorphisms of GSTM1, GSTT1 null genotype, and a NAT mutant allele are at increased risk of developing presbycusis. In fact, individuals with the GSTT1 null genotypes are almost three times more likely to develop presbycusis. The association of audiometric patterns and polymorphisms of antioxidant enzymes have also been explored in ARHL.

Mutant alleles for GSTT1 are more likely to have a high-frequency, steeply sloping audiogram, suggesting that the basal turn of the cochlea is susceptible to GSTT1-regulated oxidative stress.

On the threshold of the era of personalized medicine, identification of specific genetic factors may render gene therapy a possible treatment for presbycusis. For example, the introduction of the developmental gene Math1 has resulted in the recovery of hearing abilities of mature deaf mice. In the more proximate future, however, knowledge of genetic susceptibility may allow individuals with a family history of presbycusis to take preventive measures from a young age to help avoid or delay the development of hearing loss by addressing modifiable risk factors for ARHL. Steps that could particularly help these at-risk individuals include healthier diet, not smoking, minimization of noise exposure, and management of aggravating comorbidities such as diabetes and vascular disease.

### Modifiable Risk Factors

The influence of genetics is likely to be modulated by a set of nongenetic factors. Cross-sectional studies have identified several associations between chronic health conditions and hearing loss, although longitudinal analyses have failed to support consistently the association of some of these risk factors with incidence of ARHL. Cardiovascular disease and diabetes are well recognized as risk factors. Hypertension is also linked to hearing loss in some studies, but not consistently. Older persons with moderate-to-severe hearing loss have a significantly higher likelihood of reporting previous stroke; but unlike sudden hearing loss, age-related hearing loss is not predictive of increased risk of stroke, at least over a 5 year follow-up. Chronic kidney disease and systemic inflammation may contribute to progression of ARHL, although the latter may affect ARHL more actively in its early phases. A common thread among these disorders is vascular disease/arteriosclerosis. Along this line, it has been suggested that hearing loss precedes clinical manifestations of ischemic heart disease and may be an important “early marker” of a vascular or generalized arteriosclerotic process.

Although these conditions are risk factors for prevalence of hearing loss (cross-sectional studies), they are not always found to be predictive of incidence of hearing loss (longitudinal studies). Some studies may fail to implicate these risk factors because they are only weakly associated with hearing loss such that their effect is obscured by other factors.

Besides systemic disorders, otologic disease can also impact hearing in the aged. In one study chronic
Age-Related Hearing Loss

Aging-related worsening of hearing loss with age is likely mediated by vascular disease. Those with a history of noise exposure typically display more accelerated ARHL, although vulnerability to deleterious effects of noise exposure is not uniform across the population, and not all studies are in agreement on the effect of noise on ARHL. There is experimental evidence that early noise exposure can lead to accelerated age-related hearing loss in a mouse model. Among older adults, a history of exposure to workplace noise raises the risk of cardiovascular disease and angina, and severe exposure was associated with incident stroke (OR 3.44). Long-time smokers with occupational noise exposure tend to have a higher risk of permanent sensorineural hearing loss (SNHL).

Another recognized risk factor is history of exposure to ototoxic drugs. For example, chemotherapeutic agents such as cisplatin are used commonly in management of oncological disease, the incidence of which increases with age. Persons over 70 account for 45% of newly diagnosed malignancies. Not surprisingly, many cancer patients have ARHL before introduction of chemotherapeutic regimens. All markers of oxidant stress, lipid peroxidation, glutathionylation, and nitrosylation of proteins increase, whereas the measures of antioxidant defenses, mitochondrial apoptosis-inducing factor, and superoxide dismutase 2 (SOD2) decrease with age. Similarly, ototoxicity in general is believed to involve accumulation of ROS, leading to apoptosis. Therefore, at least theoretically, the dual demand on the antioxidant scavenger system might be expected to lead to poorer hearing outcomes. It is not known whether a synergistic interaction between ARHL and cisplatin ototoxicity is present. There is only one study that hints at possible outcomes. Older patients appear to show significantly greater incidence of audiometric

Fig. 6.1 Prevalence estimates for otosclerosis as a function of age and gender.
changes after cisplatin treatment. However, this study did not have adequate controls to be conclusive. The experimental finding that intratympanic dexamethasone has otoprotectant properties against cisplatin-induced ototoxicity might imply that the antioxidant scavenger system might have sufficient reserve to prevent a negative interaction between ARHL and ototoxins.

Because oxidative stress has been linked to ARHL, several studies have examined whether an antioxidant-rich diet can delay the progression of ARHL in animal models of presbycusis, with mixed results. Caloric restriction has been shown to suppress apoptosis in the cochlea and prevent presbycusis in a mouse model. Diet and nutrition certainly appear to influence human ARHL. Increased levels of dietary vitamin E and A (antioxidants) are associated with a reduced likelihood of prevalent hearing loss, but they do not affect risk of incident hearing loss in 5 year follow-up. There is an inverse association between higher dietary intake of long-chain omega-3 fatty acids and regular weekly fish consumption. Overall, healthy diets tend to be associated with better high-frequency thresholds in adults. High body mass index and central obesity, as measured by waist circumference, is an independent risk factor for age-related hearing loss in women older than 55. Moderate alcohol consumption is inversely correlated with hearing loss in the high, as well as in the low frequencies.

Chronic sun exposure, as measured by facial wrinkles, is positively associated with age-related hearing loss. Sunlight, a source of ultraviolet radiation, may be a source of systemic oxidative stress, which may be an underlying mechanism for presbycusis. Chronic sun exposure is more likely to produce hearing loss in those with low levels of antioxidants but without occupational noise exposure. Chronic low-level lead exposure may be an important risk factor for ARHL. On the other hand, higher educational attainment appears to be negatively associated with hearing impairment.

People born in more recent years are less likely to have hearing impairment at a given age than those born in earlier years. Over a typical generational span of 20 years, the prevalence of hearing impairment declined by 42% and 23% for men and women, respectively. This birth cohort effect likely is secondary to increased awareness of deleterious effects of noise and is consistent with the view that environmental and modifiable factors may be associated with the development of hearing impairment. Given concern about early exposure to noise resulting in accelerated age-related hearing loss, the decline in hearing impairment may be reversed with the popularity of personal listening devices among youth today. Indeed, the users of these devices have been demonstrated to exhibit elevated extended high-frequency (9–16 kHz) audiometric thresholds and reduced otoacoustic emission amplitudes, an early finding of noise-induced hearing loss.

### Relationship of Audiogram Characteristics to Cochlear Pathology

Audiometric evaluation for ARHL relies principally on the pure-tone threshold audiogram. It should be acknowledged that a standard audiometric evaluation is not a purely sensory test. Patient decisions about having heard a signal are governed by a set of self-generated rules that lie along a continuum from stringent to lenient. A patient who adopts a strict approach will respond only when absolutely certain that a signal was heard. In contrast, when lenient rules are adopted the patient will respond whenever presence of a signal is suspected. The decision variables reflect the central processes within listeners that mediate all stimulus-response tasks. The notion of a cognitive component for threshold testing (i.e., “did I hear the tone or not”) is supported by Gates and colleagues, who reported that pure-tone thresholds were worse in those with poorer executive function scores when they compared normal subjects to those with mild memory impairment but without other signs of dementia, and to those with an established diagnosis of AD.

For the purpose of the present discussion, cognitive implications of threshold testing will be set aside to focus on peripheral processes of ARHL. As highlighted in the preceding discussion, there are numerous factors that can affect hearing in old age and reflect history of genes, noise exposure, vascular health, diet, medications, and other factors. Therefore, it is not surprising that patients with ARHL do not present with a characteristic audiometric pattern or onset in a fixed age range. Whereas the conventional audiogram evaluates hearing thresholds up to 8 kHz, testing higher frequencies (i.e., extended high frequency) allows detection of elevated hearing thresholds at a younger age. This suggests that ARHL is a progressive sensorineural degenerative process whose onset may actually precede “old age.”

Given the diversity in presentation of ARHL, types of ARHL have been classified based on audiometric profile (Fig. 6.2). Perhaps the best-known scheme is that of Schuknecht, who correlated postmortem cochlear histopathology and pure-tone threshold audiogram findings to propose four main types of presbycusis: neural—associated with spiral ganglion loss; metabolic—associated with stria vascularis changes; sensory—associated primarily with hair cell loss; and conductive—not associated with a clear pathological correlate.
Sensory presbycusis appears to be from disruption or loss of outer hair cells, in the basal turn of the cochlea. Histologically, as hair cells and supporting cells undergo apoptosis, flattening and atrophy of the organ of Corti are seen. Microscopic examination of cochlear tissue reveals accumulation of lipofuscin intracellularly, a marker of senescence. The audiogram associated with sensory presbycusis typically shows a sharply sloping, high-frequency loss extending beyond the speech frequency range, with a slow, symmetric and bilateral progression of hearing loss over the years.

Neural presbycusis is associated with a loss of spiral ganglion cells and axons within the spiral osseous lamina, beginning in the basal turn of the cochlea. The organ of Corti in this type of presbycusis may show little sign of age-related degeneration. These changes disrupt transmission of the electrochemical signal from the cochlea into the auditory pathway via cranial nerve (CN) VIII, as reflected in increased thresholds of compound action potentials and dysynchronous neural activity, which may be related to synaptic abnormalities.131 Classically, audiograms of patients with neural presbycusis show a moderate downward slope into higher frequencies with gradual worsening over time. A severe loss in speech discrimination out of proportion to the threshold loss is often described, making amplification difficult due to poor comprehension.

Strial or metabolic presbycusis occurs with deterioration or atrophy of the stria vascularis. It is slowly progressive and often genetic within families. Functionally, the stria can be thought of as the “battery” of the cochlea that maintains endolymphatic potential. Audiograms classically associated with strial presbycusis show a flat loss with slow progression and good speech discrimination with no loudness recruitment. Although structurally intact, the function of the hair cells and consequently the spiral ganglion is disrupted by the inability to maintain endolymphatic potential. Significant improvement is possible with hearing aid amplification because speech discrimination is not usually affected. Strial loss usually occurs in small, focal lesions in the extreme ends of the apex and lower basal turns of the cochlea, but it can spread to involve larger segments or diffuse strial loss. Localized areas with only 20 to 30% loss may not result in much functional change, but > 50% loss leads to decreased endolymphatic potential and poor cochlear amplification with loss of gain (20 dB in the cochlear apex up to 60 dB in the base).
Mechanical (or conductive) presbycusis is a category of age-related hearing impairment that was interpreted as arising from stiffening of the basilar membrane and atrophy of the spiral ligament. Histologically, several structural deformities suggest disruption of cochlear mechanics. Spiral ligament atrophy occurs most commonly in the apical turn and least often in the basal turn. Severe deterioration and cystic degeneration may lead to full detachment of the organ of Corti from the lateral cochlear wall. Audiograms in mechanical presbycusis typically have an upward slope toward the high frequencies, with preserved speech discrimination.

Vascular and noise-induced presbycusis are among other proposed categories of presbycusis that reflect threshold elevations correlated to hypertension, cardiac disease, and stroke, or threshold elevations related to the intensity, duration, and frequency of noise exposure. In clinical practice, most cases of presbycusis do not separate into a specific type but have mixtures of these pathologies (mixed presbycusis), and ~25% of all cases of presbycusis show none of the foregoing characteristics. This latter group was classified as indeterminate presbycusis.130 Based on histological observations, Schucknecht and Gacek highlighted the importance of stria vascularis atrophy and neuronal losses over sensory cell losses.130

Other studies looking specifically at these classic categories have failed to establish a correlation between a pure-tone threshold pattern and structural abnormalities in the cochlea.132 For example, flat audiograms were associated with stria atrophy in Schucknecht’s scheme. However, Nelson and Hinojosa reported that this audiographic pattern was infrequently associated with stria atrophy, and more often occurred with outer hair cell loss alone or in combination with inner hair cell and spiral ganglion loss.132 In contrast, the classic downward-sloping audiogram has been associated with the extent of degeneration of the stria vascularis, inner and outer hair cells, and spiral ganglion cells.133 Ultrastructural features such as deformation of the cuticular plate in surviving hair cells134 or a peripheral neurite loss pattern135 may need to be considered in a full characterization of cochlear changes in presbycusis. Regardless of the debate on cochlear pathology–audiogram correlation, the notion that presbycusis arises from disruption of one or more of the key cochlear functional elements, including the inner and outer hair cells, spiral ganglion cells, and stria vascularis is widely accepted (Fig. 6.3).

Efforts at classification of audiograms of patients with age-related hearing loss can be aided by findings from animal models.136 The gerbil model of presbycusis has demonstrated that age-related hearing loss is not a sensory but a metabolic disorder. That is, hair cell losses are attributed to noise exposure; and in the absence of noise damage, age-related stria changes result in decreased endocochlear potential, which reduces cochlear sensitivity to a greater extent in the basal cochlea than in the apex. Using physiological findings in quiet-aged and furosemide-exposed gerbils as the conceptual framework, the main audiometric phenotypes (sensory, metabolic, and mixed sensory/metabolic) are believed to be consistent with predictions from animal findings associated with sensory and strial pathology.136

Central Presbycusis

Peripheral changes are expected to produce secondary central changes. This would be consistent with a model of “maladaptive” neural plasticity in which degeneration of spiral ganglion afferents132 induces slow secondary neural loss further up the auditory pathway. Well-described peripheral auditory declines in the cochlea have been shown in mouse models of presbycusis to have direct and indirect consequences on the loss of neurons in the central auditory nuclei and potential reorganization of tonotopic mapping in the primary auditory cortex and multiple associated cortices.137–141 In older human listeners, there is a linear relationship between hearing thresholds and gray matter volume in the primary auditory cortex, suggesting that even moderate declines in peripheral auditory acuity lead to a systematic downregulation of neural activity during the processing of higher-level aspects of speech, and may also contribute to loss of gray matter volume in the primary auditory cortex.142 In fact, individual differences in hearing sensitivity appear to predict the degree of language-driven neural recruitment.
during auditory sentence comprehension in the bilateral superior temporal gyri (including primary auditory cortex), thalamus, and brainstem for older listeners.\textsuperscript{142}

However, not all of the central changes are due to hearing loss. For example, by controlling for hearing loss, one study demonstrated that age alone substantially reduces spatial release from masking.\textsuperscript{143} The finding that aging can affect the ability to use spatial and spectrotemporal cues to separate competing speech streams suggests age-related changes in the cortical and/or subcortical structures essential for spatial hearing are independent of hearing loss and points to the importance of central processing.

Perception comes from the Latin word \textit{percipio} (to receive) and implies organization of sensory input. Perception leads to cognition, which comes from the Latin word \textit{cognoscere} (to learn or know), implying interpretation and assigning meaning. To move from signal detection to signal recognition and interpretation requires cognitive processing, which occurs through interaction of subcortical auditory pathways and multiple cortical regions. These interactions are collectively referred to as central auditory processing. Central auditory processing is used for successful completion of more challenging auditory tasks such as detection of a signal in background noise. Such tasks become more complex in understanding speech, during which the listeners must perceive and attend to relevant speech features, such as the pitch, timing, and timbre of the speaker’s voice. Background or competing noise increases complexity of the task, further taxing central mechanisms. Performance of these tasks would be expected to be influenced by age-related changes in central auditory processing, as well as in cognitive function.

Although cognitive skills such as processing speed, memory functioning, and ability to divide attention diminish with age, older adults with normal hearing can compensate successfully for degradations in speech perception.\textsuperscript{144} It is believed that this compensation arises from linguistic skills and a lifetime of accumulated vocabulary. In addition, slowed speech bestows additional restoration benefit because it provides older listeners more time to process noisy speech and to use available cues from the speech signal more effectively. It is believed that older people use context effectively\textsuperscript{145} toward this goal. On the other hand, older listeners with sensorineural hearing loss demonstrate deficits in the ability to compensate for degraded speech, and the severity of this deficit appears to be determined by the severity of the hearing loss.\textsuperscript{146} Saija and colleagues suggest that newly demonstrated top-down restoration skills of older individuals may lead to the development of new cognitive training methods to cope with complex listening environments of everyday life,\textsuperscript{144} as in perception of interrupted speech.\textsuperscript{147}

Functional magnetic resonance imaging (fMRI) has identified a core sentence-processing area located in the perisylvian region of the left cerebral hemisphere and an associated network of brain regions that support the working memory and other resources needed for comprehension of long or syntactically complex sentences in normal healthy older adults.\textsuperscript{148} This finding suggests that brain plasticity and compensatory neural recruitment contribute to maintenance of language comprehension with age. There are specific differences in activation of the auditory pathways observed through fMRI testing of speech listening in young and old listeners.\textsuperscript{149} Geriatric listeners show decreased activation of the auditory cortex compared with younger listeners, with even greater differences during speech listening in white noise compared with quiet listening. Specific sites of decreased activation included the anterior and posterior regions of the superior temporal gyrus bilaterally with particularly distinct differences within the posterior left superior temporal gyrus. Corpus callosum degeneration and resulting decreased interhemispheric neural transfer has also been implicated in asymmetric interaural responses during dichotic listening, with right-ear dominance frequently resulting.\textsuperscript{149}

Further support for compensatory age-related changes in auditory processing comes from a recent study correlating hearing in quiet and noise with cortical structures evaluated with magnetic resonance imaging (MRI).\textsuperscript{150} In older adults, a decline in the relative volume and cortical thickness of the prefrontal cortex was associated with a declining ability to perceive speech in a naturalistic environment. This finding is consistent with the decline–compensation hypothesis, which states that a decline in sensory processing caused by cognitive aging can be accompanied by an increase in the recruitment of more general cognitive areas as a means of compensation.\textsuperscript{150} These compensatory mechanisms were also investigated using fMRI to compare neural processing of degraded speech between young and older adults.\textsuperscript{151} Older adults adapted to degraded speech at the same rate as young listeners, although their overall comprehension of degraded speech was lower, driven by a reduced dynamic range. Neurally, both older and young adults relied on the left anterior insula for degraded more than clear speech. However, older adults relied on the middle frontal gyrus in addition to a core speech comprehension network. Once again, these findings lead to the conclusion that older adults recruit cognitive control networks as a compensatory mechanism.\textsuperscript{151} The foregoing studies suggest that older listeners appear to be able to compensate for the impact of aging per se on the brain, so long as age-related pathological processes such as significant peripheral threshold elevations and possibly cerebrovascular and cognitive disorders (yet to be discussed) are not present.
In one of the earliest studies of central presbycusis, the progressive loss in central auditory competence measured by simultaneous binaural challenges and frequency and temporal distortion tests correlated with age.\textsuperscript{152} It was suggested that central presbycusis, in addition to the peripheral form, further compounds hearing disorders in older patients and accounts for hearing disability in noise or speech competitive environments. Although some of the difficulties of hearing in noisy environments can be accounted for by loss of peripheral sensitivity, age-related deficits in interhemispheric information processing also contribute.\textsuperscript{153} In older individuals when speech-in-noise perception is poor, deficiencies in the subcortical spectrotemporal representation of speech, including low-frequency spectral magnitudes and the timing of transient response peaks, can be demonstrated.\textsuperscript{154}

A recent authoritative review emphasized that accumulating evidence supports the existence of central presbycusis as a multifactorial condition that involves age- and/or disease-related changes in the auditory system and in the brain.\textsuperscript{155} Dysfunction of central auditory processing is believed to contribute more significantly to the pathology of late presbycusis,\textsuperscript{156} making up a large component of presbycusis in people over 70 years of age.\textsuperscript{156}

Some patients with central auditory processing disorder perform better with a single hearing aid in the better ear than with binaural aids.\textsuperscript{157} In noise, 71% of geriatric patients perform better with one hearing aid, rather than two.\textsuperscript{158} This might be due to an imbalance or asynchrony in binaural signal or a cognitive processing deficit and serves to highlight the importance of dichotic tests when evaluating any older patient with hearing loss.

Given the preceding implications of central presbycusis, the standard approach of treating ARHL through compensation of peripheral functional deficits (i.e., hearing aids and cochlear implants) may not be optimal. Some have promoted a more comprehensive management strategy for ARHL consisting of diagnostic evaluation that goes beyond conventional audiometric testing and includes measures of central auditory function, such as dichotic tasks and speech-in-noise testing.\textsuperscript{159} A more comprehensive approach is expected to bestow substantial advantages on rehabilitation of the geriatric patient with ARHL.\textsuperscript{160}

Although a better understanding of central presbycusis is compelling because of its profound implications for auditory rehabilitation, the importance of this age-related process is further highlighted by the implications of the inherent relationship between central presbycusis and cognitive function. Gates and colleagues have shown that poor performance on the Synthetic Sentence Identification with an Ipsilateral Competing Message (SSI-ICM), one measure of central processing disorder, is common in people with AD.\textsuperscript{161} Using the SSI-ICM, in another study, a subset of older people from the Framingham Heart Study with normal cognitive screening test results on the Mini-Mental State Examination were identified to have very poor performance on the SSI-ICM (< 50% correct), yet normal word recognition in each ear. In this subset, the odds ratio for later diagnosis of dementia was over 12. This finding was interpreted as a common mechanism for AD and central processing disorder. Gates and colleagues reasoned that, because executive functioning is abnormal in people with AD and that many of the elements involved in central auditory processing, such as short-term memory, attention to task, and inhibition of unwanted signals, might involve executive functioning and undertook an examination of executive functioning and central auditory processing in another cohort. In that cohort, the significant relation of central auditory test results and the neuropsychiatric tests persisted even after adjustment for age, education, and pure-tone hearing thresholds, as well as, exclusion of AD cases.\textsuperscript{162} The prevalence of a poor central auditory test (including SSI-ICM) was 33% for the cognitively normal group, 80% for the memory-impaired group, and 90% for the AD group.

In summary, all “hearing” is a cognitive activity and difficult hearing environments (e.g., in the presence of background noise) can overload the cognitive aspects of understanding, especially when age and disease limit cognitive resources. Attention to central processes in evaluation of hearing loss has the potential to refine the current approach to rehabilitation and should include cognitive screening.

### Epidemiological Impact of Hearing Loss in Older Adults

As already noted, a growing number of epidemiological and clinical research studies have demonstrated strong associations between hearing impairment and measures of cognitive functioning and health outcomes. A conceptual model through which ARHL could be mechanistically associated with these downstream outcomes that are critical to aging and public health is depicted in Fig. 6.4. Investigating the potential mechanisms that underlie these associations begins with an understanding that age-related hearing loss reflects progressive damage to cochlear structures from aging and other factors (e.g., noise, vascular risk factors) that results in poorer encoding of sound by the cochlea. Earlier, we identified common factors that could underlie a simple correlation between hearing and aging outcomes, such as age, vascular risk factors (e.g., diabetes, smoking), and demographic or social factors (e.g., education). In contrast, mechanistic pathways through which hear-
Brain Structure and Function

Another pathway through which hearing impairment could contribute to impaired cognitive and physical functioning is through effects on brain structure. Neuroimaging studies have demonstrated that hearing loss is associated with reduced volumes in the primary auditory cortex and loss of integrity of central auditory white matter tracks. The basis of these associations remains unknown but may be related to alterations in the degree of neural activation provided by an impoverished auditory signal with subsequent changes in cortical reorganization and brain morphometry. In animal models, cochlear impairments are known to be associated with both tonotopic reorganization of the auditory cortex and morphological changes in central neuronal structures. Interestingly, degraded fidelity of peripheral encoding of sound likely results in recruitment and activation of broader neural networks needed for auditory processing, suggesting that peripheral hearing loss may carry cascading consequences for other regions of the brain and brain function. Under such a model, hearing impairment may constitute a "second hit" on the brain and thereby adversely affect cognitive performance and increase the risk of dementia in parallel to brain pathology caused by amyloid-β accumulation, neurofibrillary tangles, and microvascular disease. In support of this hypothesis, a recent neuroimaging study demonstrated that individuals with hearing impairment have accelerated rates of whole brain atrophy as well as specific volume declines in the right superior, middle, and inferior temporal gyri.

Mechanistic Pathways

Cognitive Load

Hearing impairment results in poor fidelity and distorted encoding of complex sounds (e.g., speech) in the cochlea. The effect of poor peripheral encoding of sound is demonstrated by studies in which a degraded auditory signal requires greater cognitive resources for auditory perceptual processing to the detriment of other cognitive processes, such as working memory. Neuroimaging studies have also demonstrated compensatory recruitment of regions in the prefrontal and temporoparietal cortex to maintain auditory speech processing in older adults. The increased auditory processing required for a distorted speech sound for an individual with hearing loss would affect the cognitive resources available for the performance of other tasks consistent with a resource capacity model. Importantly, such a cognitive load would always be present, given that hearing and cortical processing of sound are evolutionarily evolved processes that remain constantly active (e.g., monitoring of environmental sound cues). This cognitive “dual task” imposed by hearing impairment could, therefore, impact cognitive abilities as well as aspects of physical functioning that are dependent on attentional resources (e.g., gait, balance, driving).
over a mean 6.4 years of follow-up. These temporal regions are intriguing because they are important not only for spoken language processing, but also for semantic memory and sensory integration and are involved in the early stages of mild cognitive impairment or early AD.

**Social Engagement**

A final pathway through which hearing impairment could affect downstream outcomes is through effects on social engagement. Verbal communication is particularly susceptible to the effects of hearing loss given the inherent properties of spoken language. The components of spoken language consisting of the linguistic subsystems of phonology, semantics, and syntax are often encoded subtly in the auditory stream (e.g., *Sunday* and *someday* are phonetically similar but have markedly different meanings in conversation). Presbycusis leads to decrements in auditory sensitivity and loss of frequency resolution, which compromise an individual’s access to these fine auditory cues. These effects result in degraded verbal comprehension and impaired communication, particularly in situations with poor signal-to-noise ratio where effective communication is most critical (e.g., conversing with friends/family at dinner, participating in a meeting). Degraded communication can subsequently lead to impaired social functioning as demonstrated in several studies of older adults. Social relationships have powerful effects on physical and mental health that have been recognized since Durkheim first described the relationship between social integration and suicide in 1897. Subsequent prospective studies have consistently implicated a causal effect of poor social relationships on all-cause mortality, cognitive decline, dementia, heart disease, physical functioning, institutionalization, gene expression profiles, and depression. A conceptual framework developed by Berkman et al to explain these effects hypothesizes that an individual’s social network provides opportunities for social support, social influence, social engagement, person-to-person contact, and access to resources.

**Epidemiological Outcomes**

**Cognitive Functioning**

Most previous epidemiological studies of hearing and cognition have demonstrated positive associations between hearing and cognition, but some studies have shown no association. Heterogeneity in study results is likely attributable to differences in how hearing and cognition have been defined and measured in each study. Recent epidemiological studies have used objective audiometric assessments of hearing, both auditory and nonauditory tests of cognition, and have adjusted for multiple confounders (e.g., age, vascular risk factors). In two recent cross-sectional studies, the magnitude of the association of a 25 dB hearing loss (equivalent to shifting from normal to a mild hearing loss) with executive functioning was equivalent to ~ 7 years of aging. In subsequent longitudinal studies, greater hearing loss was also associated with accelerated rates of decline on both nonauditory and auditory tests such that individuals with hearing loss had a 30 to 40% faster rate of cognitive decline compared with those individuals with normal hearing over a 6 year period. Finally, hearing impairment has been found to be independently associated with a substantially increased risk of incident dementia. Compared with individuals with normal hearing, those individuals with a mild, moderate, and severe hearing impairment, respectively, had a two-, three-, and fivefold increased risk of incident dementia over > 10 years of follow-up.

**Physical Functioning**

Recent studies have demonstrated independent associations between hearing impairment and impaired daily functioning, mobility (e.g., gait speed and falls), and mortality. In contrast, other reports have indicated that there is no significant association between hearing and physical functioning and activity. Heterogeneity in study results is likely explained by subjective measurement or varying definitions of hearing. Similar mechanisms linking hearing and cognition could mediate these observed associations. Individuals with hearing impairment may perform less physical activity due to a greater likelihood of social isolation (and thus a lesser likelihood of exercise in a social setting) than individuals with normal hearing. Studies have also demonstrated that impaired hearing can contribute to cognitive load and can therefore affect attentional and cognitive resources that are important for maintaining posture and balance. Finally, impaired hearing could restrict an individual’s ability to monitor the auditory environment effectively (e.g., hearing footfalls and other auditory cues that provide orientation to the physical environment) and thereby affect an individual’s likelihood of performing physical activities.

**Role of Hearing Rehabilitative Therapies**

These epidemiological analyses of the association of hearing impairment with cognitive and physical functioning have adjusted for known confounders (e.g., age, education, diabetes). Although there is still likely
to be residual confounding from some unmeasured common pathological factor (e.g., inflammation), the consistency and robustness of these results across multiple studies and independent datasets strongly suggest that there are mechanistic pathways through which hearing loss directly or indirectly impacts cognitive and physical functioning in older adults. The most salient question, therefore, is whether hearing rehabilitative therapies could potentially mitigate these effects regardless of the mechanistic pathway. Results from observational epidemiological studies have generally demonstrated nonsignificant trends toward a protective effect with hearing aid use, but such results remain difficult to interpret. Individuals with hearing impairment who choose to use hearing aids and other technologies are likely to be healthier and of higher socioeconomic status (creating a positive bias of seeing a protective effect) but at the same time are also likely to have more severe hearing problems (leading to a negative bias) than individuals with hearing impairment who don’t use hearing aids. Answering this question, therefore, will require a definitive, randomized, controlled trial (RCT) of current best-practices hearing loss treatment (counseling/education, provision of hearing aid and other assistive devices) versus watchful waiting in a large cohort of older adults with untreated hearing impairment. Only one RCT of hearing aids focused on broader downstream outcomes has ever been performed and this moderately sized RCT of veterans performed over 20 years ago demonstrated positive effects of hearing aids on cognition and other functional domains at 4 months posttreatment.256 Trials incorporating more representative cohorts and technology (e.g., digital hearing aids and other assistive devices paired with counseling and education), following patients for several years, and providing observations of the effects of hearing rehabilitation on cognitive, physical, and social functioning have never been performed. A substantial advantage of such an RCT is that a well-designed and carefully planned trial can definitively answer the critical public health question at hand (does treating hearing loss reduce the risk of dementia?) while also providing the data to explore the various mechanistic pathways that underlie these associations.

Surgical Treatment of Conductive Hearing Loss in the Geriatric Patient

As already noted, ARHL can occur superimposed on other otologic disorders. This section is concerned with surgical treatments for conductive hearing loss in this subset of geriatric patients. Other treatments such as surgery to control cholesteatomas, infection, cochlear implants, or vestibular schwannomas are discussed elsewhere. Although the major issues are similar between older and younger otology patients, there are a few considerations that are of key importance in the geriatric patient.

First, there are some standard audiometric concepts that are of particular concern in older adults. For otologic surgery, the key considerations are hearing thresholds, word recognition (speech discrimination), and air–bone gap. The size of this air–bone gap assesses the physical contribution of the tympanic membrane and middle ear to hearing loss and represents the maximum potential improvement that surgery can achieve. Although variable, as sensorineural thresholds decline, word recognition also declines. Word recognition scores do not particularly deteriorate as the air–bone gap increases though, and this is of key interest for otologic surgery in the older adult. Some geriatric patients have word recognition scores that are disproportionately worse than one would expect from the bone thresholds. These patients may also have a large air–bone gap and have elevated bone thresholds. Surgery may reduce the air–bone gap, but this may be of little use to the patient if word recognition is poor. Surgery is not advisable for this subset of patients. Poor word recognition may be a major reason that many hearing-impaired people do not use hearing aids. Neither surgery nor hearing aids can substantially improve word recognition. People want to hear mostly to participate in conversation, but there is no reliable treatment for impaired word recognition. Word recognition scores < 60% are considered poor.257

Traditionally, stapedectomy, tympanoplasty, and ossiculoplasty of various types have made up the bulk of major otologic surgery for conductive hearing loss. Otosclerosis is usually diagnosed in middle age, therefore, most patients undergo stapedectomy earlier in life. For this reason revision stapedectomy is more likely in the older adult.258259 Proportionately more geriatric patients undergo stapedectomy/stapedotomy procedures for other indications, such as tympanosclerosis or other forms of stapes fixation. Bone may be soft in older patients so complications such as prosthesis erosion or fracture of the incus may be more common in the older adult. Tympanostomy tube insertion and even cerumen removal can be considered surgical procedures and should not be overlooked because these procedures may improve hearing in many people. There are no major differences in technique that apply only to older people, but some general considerations are important.

Bone-anchored hearing aids (BAHAs) are surgically implanted bone conduction hearing aids.
Results do not appear to differ between older or younger patients. Is age a factor in surgical treatment of hearing loss? It is difficult to find a paper that reports a significant difference in hearing outcome based on age alone. Sensorineural thresholds matter and they covary with age. Comorbidities matter, and they increase with age. For stapedectomy, Meyer and Lambert reported that results were similar in the older and younger adults. It would seem that, unless there are individual medical concerns, surgical treatment of hearing loss in the older patient is similar to treatment in younger patients. There are no randomized trials of surgical treatment for hearing loss specific to the geriatric population, possibly reflecting a lack of need to differentiate between older and younger patients. Age alone is not a valid reason to decline surgical treatment of conductive hearing loss. Obviously, many older patients have medical problems that must be considered and may be risk factors for surgery. These include factors, such as impaired vision, frailty, or preexisting increased fall risk, that could influence postoperative course, which may include dizziness and at least temporary hearing impairment. Individual preference and tolerance for surgery may be factors in patients’ decision making as well. In fact, most conductive hearing losses in geriatric patients are mixed losses with a significant sensorineural component that cannot be corrected with surgery.

Hearing aids are nonsurgical options for treatment of hearing loss. Many older adults decide against having any treatment; nevertheless, hearing aids can be worthwhile. Older patients may have high expectations for hearing aids, but many hearing-impaired candidates do not use them. Cited reasons for low usage rates include cost, difficulty using them, occlusion effect. These seem inadequate to explain fully the low usage rates. Many patients purchase hearing aids and then put them in a drawer; so cost does not always seem to drive usage. These people say that, “The hearing aid just doesn't work for me.” They must be right at least for their specific aid and level of training in use. For some reason, a hearing aid does not meet their needs. For some patients, an aid may have been fitted poorly; but audiometric reasons seem to offer the most likely explanation for low usage. Temporal processing abilities are disproportionately reduced in the geriatric patient (see Central Presbycusis earlier in the chapter). The most likely audiometric reason that hearing aids do not meet expectations may be poor word recognition. The lesson for surgeons is to be careful because many of the reasons that hearing aids are not effective for some people are the same reasons that surgery is less effective in those same people.

There are two main advantages of surgical options for the geriatric patient:

1. Otologic procedures are usually of short duration, are well tolerated, and can often be performed under local anesthesia.
2. Significant improvement in hearing quality can result if patient selection is appropriate.

Pitfalls and considerations include the following:

1. Late-age onset of conductive hearing loss is unusual. Otosclerosis typically begins in teen years, and chronic eustachian tube issues are typically lifelong problems that do not arise primarily in the older adult unless a tumor is present. If patients present with new, conductive hearing loss for the first time after the age of 60, we need to be suspicious that there may be an important etiology.
2. Conductive hearing loss occurs in the geriatric patient, but there is usually a significant sensorineural component as well. Surgery and amplification can both improve thresholds, but neither can improve speech discrimination appreciably. Failure to consider preoperative speech recognition (e.g., word recognition score < 60%) may be the most common error in surgical judgment in otologic surgery in the older patient.
3. Another audiometric consideration for surgery is the shape of the audiogram. The sloping, high-frequency loss that is typically seen in ARHL is more difficult for fitting a hearing aid than a flat loss. Surgery may also create a high-frequency loss. It is appropriate to ask whether a hearing aid will still be required if surgery is successful. If so, surgery may still be reasonable, but it is critically important that the patient and surgeon understand the goal of surgery in advance and that the patient is aware a hearing aid will be required postoperatively.
4. Consider comorbidities carefully. Although there are recognized risk criteria for anesthesia, surgically specific issues are important, as well.
   a. Bone strength and density might be of concern for some prostheses or procedures, particularly if crimping is involved, as in stapedectomy. In placing BAHAs, one should consider the possibility that the screw may not osseointegrate properly. It may be wise not to abbreviate the traditional 6 week wait time between surgery for BAHA placement and activation of the device because bone density may be inadequate in the older patient.
b. Healthy older patients do not have increased infection rates overall, but evidence of poor healing from other procedures or injuries in an individual can be a valid reason to decline surgery in some geriatric patients. Otologic surgery is not life-saving and should not involve significant risk to life, especially if other treatment options exist.

c. Many older patients have some cognitive decline, so the surgeon must be sure that the patient understands the risks and potential benefits, but most importantly, that he or she understands the rationale and does not have unrealistic expectations.

5. A hearing aid usage trial should be encouraged in elderly patients because it is easily reversible and may predict surgical success. Before embarking on surgical treatment for failed hearing aid use, the surgeon must be sure that the reasons for hearing aid failure are not also going to cause the surgery to fail.

6. Tinnitus treatment is not certain. Many older patients are troubled by tinnitus and hope that surgery will improve both the hearing and the tinnitus. Although this is a widely held belief, evidence that hearing correction improves tinnitus is not strong. Many patients who undergo surgery for tinnitus are disappointed.

7. Be gentle with geriatric patients. They have greater difficulty compensating for dizziness than young patients. They may require more recuperation time in the hospital. They need more explanation. They need more time.

8. Psychological concerns can be crucial in preoperative older patients. This is part of the true art of medicine. Patients who will never be happy or who play manipulative psychological games—and there are many of these—should be identified. They are generally to be avoided unless they can come to appreciate their problems preoperatively.

In summary, surgical treatment of conductive hearing loss in older patients without medical contraindications can be successful if performed for the right indications. Particular attention should be paid to speech discrimination scores. Geriatric patients may require more explanation and more recovery time.

■ Acknowledgment

The authors gratefully acknowledge George A. Gates, MD, for his leadership in age-related hearing loss research and review of this manuscript.

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Introduction

Hearing impairment is one of the most common maladies affecting older adults. Almost two thirds of individuals 70 years of age and older have some level of hearing loss. The prevalence of hearing loss increases over time and is generally associated with aging; however, it is often untreated. Hearing loss can cause difficulty communicating with others, localizing sound, and perceiving warnings, all contributing to a poorer quality of life. The National Institute on Deafness and Other Communication Disorders estimates there are nearly 36 million adults in the United States with some degree of hearing loss, most cases of which are caused by the loss of cochlear sensory hair cells. Cochlear hair cells are highly specialized mechanosensory receptors that are responsible for converting mechanical sound information into an electrical signal, amplifying it and transmitting it to the brain via auditory nerve fibers. In humans and other higher vertebrates, the inability of cochlear hair cells to regenerate after damage is the primary reason for the permanence of hearing loss. This chapter describes the multiple approaches being taken in pursuit of novel regenerative treatments for sensorineural hearing loss in addition to discussing the most critical challenges in the field.

Background

The organ of Corti is highly organized and consists of many cell types, including cochlear hair cells, supporting cells, and auditory nerve fibers (Fig. 7.1). At birth, the human cochlea contains ~15,000 sensory hair cells. There are two types of sensory hair
cells—inner hair cells and outer hair cells—both of which are important for hearing. Inner hair cells are responsible for converting sound information into an electrical signal, whereas outer hair cells are responsible for amplifying the signal. Auditory nerve fibers are responsible for sending sound information from cochlear hair cells to the brain for processing. Loss or damage of cochlear hair cells and auditory nerve fibers has been estimated to account for ~ 80% of cases of hearing loss.5

Currently, amplification devices and cochlear implants are the primary treatment options available for individuals with sensorineural hearing loss. Although these treatment options can return some hearing capability, the results vary between individuals. In addition, both hearing aids and cochlear implants require lifelong device usage, and they generally do not restore normal hearing qualities. As such, other approaches for restoring more normal cochlear function are actively being explored.


■ Hair Cell Regeneration

In mammals, it is known that skin and bone marrow cells are continually replenished throughout life.6,7 Furthermore, taste buds and olfactory bulb interneurons are constantly renewed in adult mammals8–11; however, such regeneration is not seen in the mammalian cochlea. It was previously thought that auditory hair cell regeneration did not occur in any context until 1988 when investigators found evidence of hair cell regeneration in the vestibular sensory epithelia of adult mammals.12–14 Although replacement of vestibular hair cells was observed, the newly regenerated cells occurred infrequently, and the amount of functional recovery that the regenerated cells may produce has been questioned.17

In the avian inner ear, hair cell regeneration begins after an auditory insult. Subsequent signaling after the insult begins a process whereby supporting cells divide and differentiate into immature hair cells and supporting cells.18–20 Alternatively, supporting cells may transform directly into immature hair cells,18,21,22 a process referred to as transdifferentiation. Using molecular, genetic, and environmental cues, these immature hair cells continue to become morphologically distinct as they mature over the course of several weeks.23,24 When compared with that of birds, fish, and amphibians, the cochlear sensory epithelium in mammals appears to have lost its ability to regenerate after hair cell loss. Exactly why this loss of regenerative ability has occurred with evolution remains unclear. Regardless, many investigators have focused attention on using what is known of mammalian hair cell development to guide efforts focused on regenerating hair cells after loss.

Treatments based on regenerating lost or damaged tissue are inevitably complex, and efforts to regenerate hair cells in the mammalian cochlea are subject to unique challenges beyond those seen in other organs. First, one needs to consider the complexity of an auditory hair cell. Hair cells are morphologically distinct with a round base and thinnest apex. They contain small hair-cell-like bundles called stereocilia, which extend from the apex of the cell and are embedded in the tectorial membrane. These hair cell bundles are mechanosensitive, responding to ionic flow, which transforms sound vibrations into electrical impulses. The electrical signal is then sent to the brain via auditory nerve fibers for further processing. Because of this, proper neural integration needs to be established to transmit sound information to the brain. Moreover, the cochlea has a distinct cytoarchitecture that is highly organized. This cytoarchitectural organization of the cochlea is critical to its proper function. Newly generated hair cells would need to integrate in the proper location within the cochlea and furthermore within the auditory sensory epithelium (the organ of Corti) to encode sound information accurately. Last, the cochlea is a delicate, membranous, and fluid-filled structure that is surrounded by dense otic capsular bone, making surgical access to the organ of Corti challenging. Efforts to outline meaningful regenerative therapies for hearing loss in humans need to address these unique challenges, and numerous laboratories worldwide are actively engaged in this exciting area of research.

■ Mechanisms for Avian Hair Cell Regeneration

Currently, there are two proposed mechanisms for cochlear hair cell regeneration in the avian inner ear: supporting cell proliferation and transdifferentiation. Cell proliferation is referred to as the growth and division of cells, during which a supporting cell reenters the cell cycle giving rise to two daughter cells that differentiate into one supporting cell and one hair cell. When cochlear hair cells are destroyed, they send a signal to neighboring supporting cells to activate proliferation.16,25 This signal prompts supporting cells to migrate through the sensory epithelium, reenter the cell cycle, and generate a daughter hair cell and a supporting cell.15 Alternatively, hair cells may be generated via supporting cell transdifferentiation, during which a differentiated cell is transformed into another cell type without cell cycle
reentry. In this approach, neighboring supporting cells are converted to hair cells via nonmitotic mechanisms with consequent depletion of the supporting cell population.

As applied to the mammalian inner ear, it is not clear how these mechanisms of regeneration might affect the organization, structure, and functional integrity of the organ of Corti. If supporting cells don’t replace themselves as they transdifferentiate into hair cells, the cytoarchitecture and function of the organ of Corti might be compromised. As such, a method to regenerate hair cells in the mammalian inner ear that does not lead to depletion of the cohort of endogenous supporting cells would seem logically preferable to one that relies on transdifferentiation alone. It is possible, however, that the mammalian inner ear has some tolerance to a level of depletion of the supporting cell population if it provides a healthy, functional cohort of hair cells. Ongoing research in this area through the approaches described here will likely provide insight into these unanswered questions in the years to come.

## Regenerative Approaches for the Treatment of Hearing Loss

There are several approaches being taken in the pursuit of novel, regenerative treatments for hearing loss, each having unique potential benefits and challenges. These approaches can generally be grouped into four categories:

1. Gene transfer
2. Pharmacotherapies
3. Exogenous delivery of stem cells
4. Promotion of endogenous stem cells

### Gene Transfer

Gene transfer has become an attractive avenue for regenerating hair cells by introducing a gene of interest to cells. To date, several studies expressing various genes of interest have produced promising results, which are discussed next.

**Atoh1**

The expression of the basic helix-loop-helix transcription factor *Atoh1* is one of the first indicators of hair cell differentiation in the cochlea. In developing mammals, *Atoh1* is expressed in prosensory patches that give rise to the auditory sensory epithelia and is both necessary and sufficient for hair cell development and formation. Mice without the *Atoh1* gene lack sensory hair cells in the auditory and vestibular portions of the inner ear. In contrast, when *Atoh1* is overexpressed in cultured cochlear explants, supernumerary hair cells are generated. Moreover, *Atoh1* has been found to be upregulated during hair cell fate specification in the adult chicken during hair cell regeneration. Collectively, these findings speak to the critical role that *Atoh1* plays in determining hair cell fate specification within the inner ear.

Investigators have introduced *Atoh1*-expressing viral vectors into the organs of Corti of a variety of different rodent species. Kawamoto and colleagues showed that delivery of *Atoh1*-expressing adenoviral vectors to the organ of Corti of mature, normal-hearing guinea pigs results in expression of the gene product in the organ of Corti and in some nonsensory locations (ectopic expression in cells outside of the organ of Corti) within the cochlea. Cells expressing the exogenously delivered *Atoh1* also expressed the hair cell marker myosin VIIa and displayed immature stereociliary bundles at the apex of the cell. In addition, these newly formed hair cells appeared to attract axons extended from the auditory nerve on some level. From this, the authors of this study concluded that cells in the normal-hearing adult mammalian inner ear are capable of generating new hair cells upon *Atoh1* misexpression.

Taking this a step further, Izumikawa and colleagues delivered an *Atoh1*-expressing adenovirus to the organs of Corti of deafened mature guinea pigs. Animals transfected with the virus showed new hair cell formation in the organ of Corti and in some ectopic locations in the cochlea. The authors also reported a significant improvement in auditory brainstem response thresholds in the ears of animals transfected with *Atoh1*. Cross-section analysis revealed that some of the cells displayed a mixed phenotype, having both hair cell and supporting cell features. As a whole, the source of the newly generated hair cells was unclear; however, it was hypothesized they arose from transdifferentiated and proliferated cells within the damaged regions that had been transfected with the *Atoh1*-expressing adenovirus.

In 2008, Gubbels and colleagues established a method to conduct gain-of-function studies in the developing inner ear using an in utero gene transfer technique. Plasmids encoding *Atoh1* and green fluorescent protein (GFP) were microinjected into the otic vesicle of mice on embryonic day 11.5 and examined on embryonic day 18.5 and later time points. Ears that received *Atoh1* demonstrated supernumerary hair cell formation throughout the cochlea. Cells that formed secondary to the delivery of exogenous *Atoh1* expressed myosin VIIa and displayed stereociliary bundles. Moreover, they attracted neuronal processes and expressed the ribbon synapse marker carboxy-terminal binding protein 2. Postnatal elec
trophophysiological analysis of the cells generated from in utero transfer of *Atoh1* revealed age-appropriate basolateral conductances and mechanoelectrical transduction properties. These results demonstrate that it is possible to generate cochlear hair cells by *Atoh1* misexpression after in utero gene transfer. Moreover, this study showed that the generated hair cells are functional on a cellular level and establish connections with the central auditory network.

Collectively, these studies suggest that a gene transfer approach using transcription factors known to be critical for hair cell development can generate hair cells in normal and deafened cochlea of both adult and developing rodents. Furthermore, these studies demonstrate that the newly generated hair cells are able to associate with the nearby auditory nerve and are functional on a cellular and possibly even an organ system level. It remains unclear if this type of approach leads to depletion of the supporting cell population and, if so, its implications. In addition, the long-term viability of hair cells generated through *Atoh1* gene transfer is similarly unclear. Regardless, *Atoh1* gene transfer–based approaches represent a promising and active area of investigation in the pursuit of novel, regenerative therapies for hearing loss.

**Cell Cycle Modulators**

Modulating genes that have a role in cell cycle regulation is another molecular approach being pursued to achieve hair cell regeneration. Although mammalian supporting cells are generally quiescent in vivo, several studies have reported that these cells have the capacity to reenter the cell cycle and generate hair cell–like cells in vitro.38–41 The concept of this approach is that, by altering the cell cycle of residual supporting cells following hair cell loss, proliferation may ensue, with subsequent differentiation of the progeny into hair and supporting cells.

It is well established that, during development, cell cycle exit continues progressively along the cochlear duct from the apex to the base, starting on embryonic day 12 and completing by embryonic day 14 in mice.29,44–46 While this is occurring, cochlear cells begin to express hair cell markers, including *Atoh1*, myosin VI, and myosin VIIa.29,31,32 At a similar developmental time, supporting cells begin to express the cyclin-dependent kinase inhibitor p27kip1.44,46 The expression of p27kip1 has been shown to coincide with the cell cycle exit of hair cell and supporting cell progenitors.28,44–46 P27kip1 is continually expressed in supporting cells, which may be responsible, to some degree, for maintaining the quiescent state of supporting cells.46 Alternatively, hair cells rapidly down-regulate p27kip1 during differentiation, expressing the cyclin-dependent kinase inhibitor p19Ink4d instead,46,48 which is thought to maintain them in a quiescent state (see later discussion).

Modulation of the expression of cell cycle inhibitors, such as p27kip1, may hold promise as a potential means for promoting some level of regeneration of hair cells in the mammalian inner ear. P27kip1 appears to play a significant role in the inability of mammalian hair cells to regenerate after damage.47,49 In the cochlea, P27kip1 is regulated at both transcriptional and posttranscriptional levels.49 Mice deficient for p27kip1 have supernumerary hair cells and supporting cells, most of which are located in the apical region of the cochlea.47 In addition, auditory brainstem response thresholds obtained from these mice were significantly elevated compared with controls, suggesting severe to profound hearing loss. The significant elevation in auditory brainstem response thresholds is thought to occur from excess hair cells and supporting cells disrupting the spatial organization and mechanical properties of the basilar membrane.47,49 In another study, White and colleagues examined the regenerative capacity of supporting cells isolated from the postnatal mouse.41 The authors found that postmitotic supporting cells are capable of transdifferentiating into new hair cells in vitro. In the first experiment, the ability of postmitotic supporting cells to reenter the cell cycle was examined by isolating supporting cells expressing green fluorescent protein under a p27kip1 promoter and culturing them in vitro. After 2 days, 60% of green fluorescent protein (p27kip1) positive cells downregulated expression of p27kip1, whereas 38% of these cells incorporated BrdU, signifying that these had reentered the cell cycle. Additional analysis determined that these cells were then able to differentiate into hair cells. A small number of these cells expressed the hair cell marker myosin VI. These results demonstrate that postnatal supporting cells from mice have the ability to divide and differentiate into hair cells via mitotic and nonmitotic means. Collectively, these studies suggest that modulation of p27kip1 in supporting cells may establish a method, or part of a method, to regenerate hair cells following their loss.

Although p27kip1 expression coincides with cell cycle exit, it is not essential for this occurrence.50 This suggests there are other genes that play a more central function in regulating cell cycle exit. Retinoblastoma (RB) 1 is another type of cell cycle regulator, which plays a role in holding inner ear hair cells in a state of quiescence and may represent a potential target for enabling regeneration to occur in the mammalian organ of Corti. The RB1 gene is a tumor suppressor involved in regulating cell cycle exit, differentiation, and survival. Although hair cells in the vestibular and auditory portions of the inner ear generally undergo similar processes during development and differentiation, RB1 appears to play different roles in these
regions. In 2005, Sage and colleagues found that targeted deletion of RB1 in the developing mouse utricle leads to proliferation of vestibular hair cells, suggesting a potential role for alteration of RB1 as a means to achieve regeneration of hair cells in the future. More recently, inactivation of RB protein in postmitotic supporting cells resulted in cell cycle reentry, with a subsequent increase in the number of supporting cells in the neonatal mouse cochlea. Moreover, some of the nuclei of proliferating supporting cells were intermittently observed in the hair cell layer above their normal position, similar to supporting cells undergoing regeneration in the avian auditory epithelium. There was no evidence of newly generated hair cells from supporting cells, suggesting that there may be a potential role involving other signaling factors to facilitate the continued differentiation of the newly generated cells into hair cells. One concern in the application of this type of strategy lies in the risk of tumor formation with manipulation of tumor suppressor genes such as RB1. As such, additional investigation will be needed to determine if efforts aimed at targeted alteration in the expression or function of genes, such as RB1, could be pursued safely as a potential therapeutic strategy for hair cell regeneration in the future.

Following hair cell formation, a mechanism to maintain the postmitotic status and continued viability of the cells is necessary to prevent their degeneration. The cyclin-dependent kinase inhibitor p19Ink4d is a factor that appears to sustain the postmitotic status of hair cells. During development, mice deficient for p19Ink4d develop in a normal manner; however, hair cell loss is observed beginning on postnatal day 17. In these mice, it appears that hair cells attempt to reenter the cell cycle, causing them to die by programmed cell death. As already discussed, hair cells rapidly downregulate p27kip1 during differentiation, suggesting that p19Ink4d alone is responsible for maintaining the postmitotic state of the hair cells. For the purpose of newly generated hair cells, continued maintenance is a constant process, and failure to regulate this accurately can have adverse effects on hearing. Accordingly, future efforts aimed at regenerating hair cells in the deafened cochlea will need to take into account the ongoing need for maintenance of the newly generated hair cells to ensure their permanence.

Collectively, studies evaluating gene transfer-based approaches for the treatment of hearing loss have produced promising results. Additional investigation is necessary to characterize key genes and cell cycle modulators that have been found to be critical for hair cell regeneration in animal and cell culture models. Furthermore, it is essential to determine if direct transdifferentiation of supporting cells into hair cells leads to a depletion of supporting cells, which might disrupt the cytoarchitecture of the organ of Corti. Moreover, the long-term viability of hair cells generated through gene transfer is similarly unclear. In addition, determining a safe and effective method to deliver genetic material into the inner ear remains a critical challenge. It is plausible that genes of interest could be introduced to the inner ear through transtympanic delivery to the middle ear with subsequent transport or diffusion through the round window membrane to the cochlea. Alternatively, direct injection of a gene of interest to the scala fluids of the inner ear might ultimately be required for meaningful delivery within the cochlea. The ability of the gene to penetrate all areas of the cochlea would need to be determined in addition to ensuring that no further damage results from these delivery approaches to the inner ear. Beyond the challenges of gene delivery on an organ basis, genetic material needs to be transported across the cell membrane for subsequent transcription to take place. Methods to accomplish this include packaging the gene in a viral vector that has tropism for the supporting cells of the organ of Corti, usage of electrical pulsations (electroporation) to drive the genetic material through the cell membrane or potentially packaging the genetic material in a lipid-based carrier that fuses with the cell membrane to release the gene of interest into the cytoplasm. Although these cellular delivery mechanisms are plausible approaches for gene delivery to the inner ear, a viral-based method for gene delivery appears to be the most logical candidate and the approach used in scientific studies on cochlear gene transfer to date. Although many questions remain to be answered, gene transfer–based approaches for the treatment of hearing loss remain a promising and attractive area of investigation.

**Pharmacotherapy**

Pharmacotherapeutics focuses on the use of drugs to modulate signaling pathways or gene expression in a cell. In regard to hearing loss, these drugs may target specific cellular pathways that signal supporting cells to divide, or target the regulation of specific genes such as Atoh1 in attempts to generate new hair cells. In concept, these synthetic molecules with biological activities could potentially be given systemically, transtympanically or by intracochlear administration to effect the generation of hair cells after loss; potentially avoiding some of the challenges associated with other approaches for hair cell regeneration. The Notch signaling pathway is one potential target for pharmacotherapeutically mediated efforts toward hair cell regeneration. This signaling pathway is responsible for establishing, at least in part, the hair cell–supporting cell mosaic of the organ of Corti during inner ear development.
upregulates Hes and Hey transcription factors, which inhibit Atoh1 expression.60–63 Thus Notch activation suppresses hair cell differentiation in supporting cells, thereby regulating the number of hair cells and supporting cells.60 In addition, Notch expression appears to increase during hair cell regeneration in the avian inner ear.64 Because of this, investigators have hypothesized that disrupting the Notch signaling pathway might promote hair cell generation. During embryonic development, the absence of the Notch ligand Jagged1 resulted in a severe reduction of hair cells in both the auditory and the vestibular portions of the inner ears of mice, indicating that it is required for the prosensory inductive function of Notch.65 Other studies inactivating the Notch ligands Delta1 and Jagged2 reported a greater number of hair cells relative to control cochleae, in addition to an increase in the number of supporting cells; suggesting Notch signaling may also play a role in regulating hair and supporting cell proliferation.65,66 Several other studies have also reported that, during development, disruption of Notch signaling can lead to a cellular conversion from a supporting cell fate to a hair cell fate.65–70 Collectively, these studies suggest that careful manipulation of Notch signaling in supporting cells may provide an avenue for regenerating hair cells following auditory insult.

One of the first reports of pharmacological inhibition of Notch signaling treated deafened guinea pigs using a g-secretase inhibitor.71 In this study, the authors reported that a small number of hair cells were generated following g-secretase administration, with newly generated cells expressing the hair cell marker myosin VIIa. More recently, pharmacological inhibition of Notch signaling using other types of small molecule g-secretase inhibitors has been shown to cause partial recovery of hearing thresholds in mice exposed to noise trauma.72 In this study, pharmacological inhibition of Notch signaling with LY411575 in deafened mice resulted in transdifferentiation of supporting cells into cochlear hair cells and partial restoration of hearing verified by auditory brainstem response testing. The supporting cells did not appear to reenter the cell cycle following g-secretase administration, indicating that hair cells were generated through transdifferentiation. Of note, both of these studies used direct delivery of g-secretase inhibitors to the scala tympani, which represents one potential advantage to a pharmacotherapeutic approach. In contrast, other approaches for hair cell regeneration such as gene transfer and cell transplantation (to be described) are more likely to require intracochlear delivery methods, which have a higher potential risk for hearing loss. In general, for this approach to become clinically relevant, the ability of the drug to penetrate all regions of the cochlea and prevent ectopic hair cell formation needs to be explored further. In addition, generating hair cells in proper locations within the organ of Corti while avoiding depletion of the supporting cell population will need to be accomplished. Although more investigation is needed, the foregoing studies demonstrate that pharmacotherapeutic targeting of Notch signaling may enable meaningful regeneration of hair cells in the deafened cochlea in the future.

Exogenous Stem Cells

One promising approach in regenerative therapies involves the use of stem cells as a substrate to generate mature cell types of interest with subsequent transplantation. There has been great progress with this approach for regeneration of tissue in other organ systems, with several ongoing clinical trials aimed at treating macular dystrophy and macular degeneration.73,74 With regard to the inner ear, several laboratories are attempting to generate cochlear hair cells in vitro using types of pluripotent stem cells such as embryonic stem cells (ESCs) and induced pluripotent stem cells (iPSCs). Pluripotent stem cells are defined as undifferentiated, self-renewing cells that have the ability to generate mature cell types from all three germ layers. Embryonic stem cells are a type of pluripotent cell derived from the inner cell mass of a blastocyst ~ 5 to 7 days after fertilization (Fig. 7.2). Induced pluripotent stem cells are also pluripotent; however, they are derived from fully differentiated adult cells (Fig. 7.2), typically fibroblasts from a skin punch biopsy that have been reprogrammed into a state of pluripotency by treatment with combinations of transcription factors. During development, pluripotent cells of the inner cell mass of a blastocyst undergo sequential differentiation, becoming more specialized and tissue-specific as the organism matures. The process of differentiation of pluripotent stem cells (ESCs or iPSCs) in culture to generate mature cell types recapitulates the process of development on some level. As applied to the goal of generating hair cells from pluripotent stem cells, the process of differentiating ESCs or iPSCs requires recapitulating inner ear, and subsequently hair cell, development in culture (Fig. 7.3). Several studies have reported successful differentiation of mouse pluripotent stem cells into otic progenitor cells and hair-cell-like cells, whereas reports of achieving the same goal using human stem cells are more limited.75,76

Differentiation of Pluripotent Stem Cells into Hair-Cell-Like Cells

In 2010, Oshima and colleagues published a stepwise guidance protocol using mouse ESCs and iPSCs to generate mechanosensitive hair-cell-like cells.77
Refine techniques so that the hair-cell-like cells generated from human stem cells have morphological refinement and functionality comparable with those seen in the mouse ESC and iPSC studies discussed earlier.

**Cell Transplantation to the Organ of Corti**

There have been several reports to date investigating the ability of stem cells to integrate into cochlear tissues upon inner ear transplantation. Multiple types of stem cells and methods for transplantation have been explored using both normal-hearing and acutely deafened cochleae. One of the first reports of stem cell delivery to the inner ear of mammals was published in 2001 by investigators in Japan. Neural stem cells were prepared from hippocampal tissues and injected into the scala tympani of newborn rat cochleae. Two to 4 weeks following transplantation, the authors reported grafted cell survival, with some resembling hair cells along the organ of Corti. Other studies transplanting stem cells into the damaged cochlea have described evidence of grafted cell survival and some integration into the sensory epithelia of the inner ear. In one study, investigators transplanted mouse fetal neural stem cells into the inner ears of mice following ototoxic injury. Grafted cells were identified in the cochlear, vestibular, and spi-
cells transplanted into the sound-damaged inner ear were able to migrate throughout the cochlea. The authors reported that the grafted neural stem cells in the organ of Corti expressed the hair cell markers myosin VIIa, oncomodulin, and calbindin. In addition, some of the grafted cells in the spiral ganglion exhibited a comparable phenotype to spiral ganglion neurons. In aggregate, the findings of these studies suggest that grafted stem cells can survive in the adult cochlea after transplantation for as long as 4 months in some cases. Despite the evidence of grafted cell survival, there have been a limited number of reports demonstrating sensory integration and/or differentiation of grafted cells into hair or supporting cells after transplantation in the adult mammalian cochlea. As such, a great deal of further investigation will be needed to identify stem cell or host-related factors that could be modified to allow for more successful integration of transplanted cells into the organ of Corti as hair cells.

Although stem cell transplantation into the adult mammalian cochlea has been met with limited success, several investigators have reported that mouse stem cells transplanted into the developing avian inner ear have the capacity to generate hair-cell-like cells. In one study, investigators generated otic progenitor-like cells in vitro from mouse ESCs, which were subsequently transplanted into the developing inner ear of chick embryos. The investigators reported that these otic progenitor cells were able to integrate into the avian auditory sensory epithelium and differentiate into cells expressing hair cell markers. More recently, another team of investigators used an alternative method to differentiate mouse ESC into hair-cell-like cells. Consistent with the previous report, when transplanted into the otic vesicle of developing chick embryos, these cells were found to incorporate in the correct location and function as host cells in the inner ear. Together, these studies demonstrate that mouse stem cells are capable of engraftment and differentiation under appropriate conditions when transplanted into the developing chick inner ear. This suggests that the microenvironment of the developing inner ear presents transplanted stem cells, even those from another species, with the signaling necessary to allow for engraftment and terminal differentiation as hair cells. Identification of host-related factors present in the developing inner ear that permit successful engraftment of transplanted stem cells may influence the future development of strategies, whereby deafened adult mammalian cochleae may be modified or primed in some way to allow for subsequent engraftment of transplanted stem cells. Clearly, a great deal of research will be needed to realize the potential of stem cell transplantation as a regenerative therapy for hearing loss in the future; however, steady progress continues to be made in this area.

Fig. 7.3 Cell fate decisions in differentiating pluripotent stem cells to an inner ear hair-cell-like cell. The highlighted lineage decisions recapitulate those made by inner ear hair cells during normal development. Discrete modifications of the cell culture environment during pluripotent stem cell differentiation act to guide the cells through these fate decisions to ultimately generate an enriched population of mature inner ear hair (or supporting) cells.
Cell Transplantation to the Auditory Nerve

In 2006, Corrales and colleagues transplanted neuronal progenitor cells derived from mouse ESCs into the cochlear nerve trunk of gerbils after experimentally induced damage to the auditory nerve. The transplanted cells were found to occupy substantial portions of the space previously occupied by spiral ganglion cells. Moreover, the cells were able to survive and extend processes throughout the cochlear nerve area, making contact with cochlear hair cells in the organ of Corti. From this study it appears that transplanted neuronal progenitor cells have the potential to survive, terminally differentiate, and morphologically specialize in an animal model of auditory neuronal degeneration. More recently, restoration of auditory brainstem response thresholds following lesioning of the rodent auditory nerve was reported using otic progenitor cells derived from human ESCs. In vitro, human ESCs were directed toward an otic progenitor fate using the signaling molecules fibroblast growth factor 3 and 10. Using these growth factors, human pluripotent cells were able to differentiate into hair-cell-like cells and functional auditory neurons in vitro. The authors transplanted the human ESC-derived neural progenitor-like cells into the spiral ganglion region of gerbils after deafening them by destroying the host auditory neurons. Analysis of the transplanted cells revealed that they were able to engraft, differentiate, and improve auditory brainstem response thresholds relative to deafened but untreated control animals. These studies demonstrate the ability of stem cell–derived neural progenitors to integrate successfully, terminally differentiate as auditory neurons, and ultimately improve auditory thresholds upon transplantation. Given these reports, the potential of stem cell transplantation for auditory nerve–related hearing loss may offer more immediate promise as a regenerative therapy when compared with stem cell transplantation into the cochlea.

Cell transplantation may be a viable treatment option in the future to replace damaged or lost cells in the inner ear; however, critical issues will need to be addressed to achieve this goal. First, an in vitro method to generate adequate and consistent numbers of hair cell progenitors from human pluripotent stem cells needs to be established to supply the cochlea with a sufficient number of cells to replace those that are damaged or have been lost. Because there are many types of pluripotent stem cells being used in this pursuit, the most effective cell type(s) would also need to be determined. Moreover, it is essential to identify the factors that are most critical for successful integration and terminal differentiation of grafted cells as hair cells to translate this approach to humans. Likewise, the transplanted cells also need to migrate to correct locations along the basilar membrane, avoiding the occurrence of ectopic or supernumerary hair cells to maintain the precise organization of the organ of Corti. In addition, grafted cells need to establish proper integration and neuronal circuitry for normal function. Furthermore, a safe and effective method to deliver cells to the organ of Corti must be established to ensure that it is not further damaged by the transplantation procedure itself. Last, transplanted cells face the possibility of immune responses that may ultimately lead to their rejection by the host, so immunosuppressive therapy may prove to be necessary. Although it remains to be determined if exogenous stem cells have the therapeutic potential to improve hearing ability, studies to date have provided promising results that may one day contribute to making this a viable treatment option.

Endogenous Stem Cells

The presence and potential of cells with stem cell–like properties in the mammalian inner ear is the topic of ongoing investigation in several laboratories. Mammalian organs with the capacity to regenerate generally contain a population of adult stem cells, which are responsible for preserving and repairing the tissue in which they are found. These cells differ from pluripotent stem cells in that they are multipotent, meaning they are only able to differentiate into tissue-specific cell types, not all cell types. When an adult stem cell divides, one of the resulting daughter cells replaces itself as an adult stem cell, whereas the other daughter cell becomes a tissue-specific progenitor cell. Thus adult stem cells act as a self-repair system, repopulating themselves as adult stem cells in addition to replacing the damaged or destroyed tissue.

Adult stem cells are found in a variety of organs and tissues, including the central nervous system, skin, bone marrow, and gut. Increasing evidence suggests that the inner ear may also possess a niche of stem cells. In one study, cells with a high proliferative potential and capacity to self-renew were isolated from the vestibular portion of the adult mouse inner ear. These inner ear–derived cells demonstrated the ability to form spheres, one feature of stem cells. When cultured in vitro, these cells showed the capacity to differentiate into cells expressing mature hair cell markers. In addition, when these sphere-derived cells were transplanted into the developing inner ear of chicken embryos, they were capable of differentiating into hair-cell–like cells.

In another study evaluating the presence of adult stem cells in the mouse inner ear, investigators isolated stem cells from cochlear and vestibular tissues in mice 1 to 4 months of age. Differences in the capacity for sphere formation were observed, with stem
cells from the vestibular sensory epithelia displaying a higher capacity for sphere formation compared with those isolated from the cochlear sensory epithelia. In addition, the sphere-forming ability of the cochlear sensory epithelia rapidly decreased from the second to the third postnatal week, whereas the sphere-forming ability of the vestibular sensory epithelia declined more slowly and into adulthood. As already stated, the mammalian vestibular organ has some capacity to replace lost hair cells, whereas the cochlea appears to lack this regenerative potential. Results from this study suggest that stem cell–like cells may be responsible for the persistence of some regenerative potential observed in the vestibular sensory epithelium. In addition, the inability of the cochlear sensory epithelium to regenerate might be due to a reduction in the number or potential of the tissue-specific stem cell population in the organ of Corti. Although there are reports of low/undetectable progenitor cells in the adult mammalian inner ear, other lines of investigation suggest that stem cell compartments may persist within the mature cochlea. The intermediate filament protein nestin is expressed in proliferating tissues and is widely regarded as a marker of mitotically active cells and neural stem cells. As such, nestin is commonly used to identify cells with stem cell characteristics in developing and adult tissues. Several studies have reported the existence of nestin-expressing cells in the inner ears of mice; however, their presence and location differ between reports. In the first report, nestin expression was observed in supporting cells below the inner and outer hair cells in the immature mouse cochlea, in addition to some mild expression in the outer hair cells; however, in the early adult mouse, nestin expression was downregulated and localized to only a few cells under the outer hair cell layer. In another study, nestin expression was observed in supporting cells near the inner hair cell layer, with some mild expression in a few inner and outer hair cells in the immature cochlea. In the mature inner ear, nestin expression was found to be limited to only the spiral ganglion. Most recently, nestin expression was found in supporting cells lateral to the outer hair cell region. This expression was observed throughout the whole cochlea in newborn mice; however, the number of nestin-expressing cells decreased as the cochlea matured. Of interest, the authors reported that there appeared to be an increase in the number of nestin-expressing cells following noise trauma. Collectively, the presence of nestin-expressing cells in the murine inner ear raises the possibility of the persistence of a population of stem-cell-like cells within the mammalian cochlea. Further work exploring their exact presence, localization, and overall function is essential to understand any therapeutic potential that they may possess.

More recently, Lgr5, a Wnt target gene, has emerged as another area of interest for hair cell regeneration. Lgr5 is a stem cell marker found in multiple proliferating adult tissues. During embryonic development, Lgr5 is expressed in nascent hair cells and supporting cells and is later downregulated as the cells mature, with expression limited to the third row of Deiters cells in the mature organ of Corti. Analysis of these cells by other investigators revealed that they give rise to hair cell lineages in vivo and in vitro. In this study, Lgr5-expressing cells were isolated and cultured in vitro and found to self-renew and differentiate into cells expressing the hair cell marker myosin VIIa. In vivo, these cells were able to give rise to hair cells. In agreement with the previous report, Lgr5 expression was downregulated to the third row of Deiters cells; however, inner pillar cells also appeared to retain this expression as well. Additional analysis revealed these Lgr5-expressing cells proliferate and generate hair cells under certain experimental conditions in neonatal mice. Evidence that these cells are able to reenter the cell cycle and proliferate offers the advantage that these cells could replenish both hair cells and supporting cells in the damaged mammalian cochlea. It remains unclear at this point how long the population of Lgr5-expressing cochlear stem cells persists into adulthood. As with nestin expression, further investigation of these cells is needed to determine their therapeutic potential as a means for regenerating cochlear hair cells.

In summary, the presence of endogenous stem cells in the mammalian inner ear presents the possibility of local therapy aimed at recruiting and directing these cells to repopulate the organ of Corti with functional cochlear hair cells after the native population of hair cells has been lost or damaged. Some studies on the topic have reported that, over time, the number and/or viability of multipotent stem cells in the cochlea decreases. Consequently, there exists the possibility that, by the time these cells are needed for most patients with acquired causes of hearing loss, they no longer exist. Accordingly, further research is warranted to determine the function, location, and persistence into adulthood of cochlear stem cells to better define their potential as the basis for a novel therapeutic approach for hearing loss.

Conclusion

The past 30 years of research have provided a better understanding of the mechanisms underlying inner ear development, avian cochlear hair cell regeneration, pluripotent stem cell differentiation, cell transplantation, the presence of endogenous
stem cells, and the potential therapeutic application of gene transfer and pharmacotherapies for hearing loss. Although much has been learned about the use of exogenous stem cells, inner ear stem cells, gene transfer, and pharmacotherapies for the replacement of damaged cells, these approaches for the treatment of hearing loss are still in experimental stages. There are many intricate components, and therefore challenges, involved in the process of auditory nerve and cochlear hair cell regeneration. As such, it is possible that a combination of the approaches described here or others not yet evident will yield novel, clinically meaningful treatments for hearing loss in the future. Although there are no clinical trials in this field to date for adults with hearing loss, several recent discoveries using cell culture and animal models give hope that otolaryngologists will have novel, regenerative therapeutic options for the management of sensorineural hearing loss in the future.

## Challenges of Regenerative Therapy

- Challenges facing all approaches
  - Identifying the safest and most efficient method to access the cochlea
  - Repopulating the cochlea with an adequate amount of cells
  - Avoidance of generating hair cells outside of the organ of Corti
  - Maintenance of newly generated cells
  - Reestablishment of neuronal circuitry
- Gene-based treatment and pharmacotherapies
  - Generation of replacement hair cells without significantly compromising the supporting cell population
  - Insuring that treatment does not compromise the cytoarchitecture of the organ of Corti
  - Unregulated cell proliferation carries the risk of tumor formation
- Exogenous stem cells
  - Determine the timing of transplantation relative to damage for optimal success
  - Proper migration, integration, and terminal differentiation of transplanted cells into the organ of Corti as hair cells
  - Immune rejection of transplanted cells
- Endogenous stem cells
  - Identifying if endogenous cochlear stem cells persist into adulthood and maintain potency to become hair cells
  - Determining the spatial and temporal organization of adult stem cells in the cochlea
  - Identifying a method for allowing cochlear stem cells, if present, to enter the cell cycle, proliferate, and replace lost hair cells

### Key Points

- Multiple approaches such as the following are being taken in pursuit of regenerative treatments for sensorineural hearing loss:
  - Gene transfer
  - Pharmacotherapies
  - Exogenous delivery of stem cells
  - Promotion of endogenous stem cells
- All approaches are still in experimental stages with no clinical trials to date in adults.
- Each approach has its own unique advantages and disadvantages.
- A combination of these approaches may be necessary for successful treatment of sensorineural hearing loss in the future.

## Acknowledgments

Samuel Gubbels receives support (KL2 award and Type I pilot) from the Clinical and Translational Science Award (CTSA) program, previously through the National Center for Research Resources (NCRR) grant 1UL1RR025011, and now by the National Center for Advancing Translational Sciences (NCATS), grant 9U54TR000021. The content is solely the responsibility of the authors and does not necessarily represent the official views of the NIH. SG also receives support from NIH/NIDCD 1 R03 DC012432–01, 1 R01 DC013912–01, as well as P30 HD003352.
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Hearing Aids: Considerations in the Geriatric Population

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Introduction

Hearing loss is the most common sensory deficit in the elderly.1 Hearing loss can impair communication, thus creating loneliness, isolation, dependence, frustration, and even communication disorders.1 When left untreated, hearing loss can substantially impair quality of life. Audiological rehabilitation, auditory rehabilitation, and aural rehabilitation are often used interchangeably to describe a patient’s management process designed for individuals who experience deficits in communication, as reported by Weinstein.2 Functional capabilities must be assessed on an individual basis due to wide variations in characterizing elderly adults. Chronological age is not a reliable predictor of physical, social, or mental status; therefore, it is important to understand how individuals view themselves. The impact of hearing loss varies by the degree of loss and the individual’s personality and activity level. The effects of hearing loss cannot be restricted to pathology alone because the mechanics of the ear cannot be isolated from the social aspects of hearing. The personality effects of hearing loss are largely dependent on an individual’s character, including mental, spiritual, societal, and economic resources. These components determine one’s reaction to hearing loss and the level of handicap it generates. Rehabilitation in the elderly must include a comprehensive approach to assessment and a multidimensional intervention. The purpose of rehabilitation with older adults, regardless of the severity or type of impairment, is to assist in recovering lost physical, psychological, and social skills.

The entire auditory system undergoes considerable change as the aging process progresses. Specific conditions may affect the type of aural rehabilitation to provide. For example, older adults develop changes of the outer ear and external auditory canal due to thinning of the epithelium, atrophy of subcutaneous tissue, and decline in secretory abilities of the glands. Hence certain types of hearing aids or audiological testing may not be appropriate due to changes in the ear structure. Although outer and middle ear pathologies are monitored under the care of a physician, the audiologist should be aware of any such issues. It is important to consider age-related changes in the brain when determining auditory rehabilitation in the elderly population. Aging is associated with progressive losses in function across multiple systems, including sensation, cognition, memory control, and affect. Age-related modifications in the central nervous system are associated with declines in the ability to perform selected cognitive and sensorimotor tasks; decreased functional capacity; and alterations in gait control, learning, and memory.3 These deficits may affect the patient’s ability to respond to aural rehabilitation strategies.

Hearing loss may be severe in older persons, whether from causes associated with aging or owing to other etiologies. More severe-to-profound losses may be associated with a change in personality and lifestyle due to the challenges that listening presents. Elderly individuals with hearing loss also find listening in the presence of multiple speakers or background noise especially difficult because their ability to detect signals in noise diminishes with age. All too often, the elderly person begins to believe that the inability to hear and understand a conversation is due to deterioration of the brain (intellectual impairment). Family, friends, and stereotypes of the elderly population may reinforce this belief. People may ignore the hearing-impaired person in group conversations and assume that the person does not know what is going on. Stereotypes of aging, such as physical and mental slowing, further undermine the elderly person’s weakened self-confidence and hasten his or her withdrawal from society. The isolation caused by hearing loss can contribute to delay in elderly individuals’ seeking medical attention to address their hearing handicap.

There is a significant relationship between hearing and speech. The ear is sensitive to a certain frequency range, and speech falls within that range. Speech can be divided into two types of sounds:
vowels and consonants. Roughly, vowels fall into the frequencies below 1,500 Hz, and consonants above 1,500 Hz. Vowels are relatively powerful sounds, whereas consonants are weaker sounds and are dropped often in everyday speech or not pronounced clearly. In essence, low-frequency speech sounds provide the listener with a sense of volume, whereas higher-frequency speech sounds provide meaning and clarity. Most commonly, older adults experience a hearing loss configuration that reflects comparatively good low-frequency hearing and poorer high-frequency hearing. High-frequency sensorineural hearing loss often causes deterioration of a person’s ability to understand speech. Speech recognition ability can be correlated with the aging process. In some cases, diminished speech understanding is due to peripheral hearing loss. This type of loss typically presents with the ability to hear speech but not understand it. A second cause of trouble understanding speech is central auditory processing issues, such that age-related changes or other changes in the auditory pathways of the brainstem or portions of the auditory cortex degrade the speech signal. Individuals with hearing loss may ask people to speak louder in an attempt to achieve better speech clarity. Unfortunately, “louder” is not always the answer. Loudness may actually reduce discrimination ability due to distortion of the speech signal. Distortion occurs more frequently in people with high-frequency hearing losses because overall loudness also amplifies the low-frequency sounds, such as vowels, which they usually hear at a normal or close to normal volume. Speaking in a louder voice creates overpowering vowels with relatively weaker consonants and does not improve the clarity of speech. These factors must be assessed and are critical to proper hearing aid selection.

Before a hearing aid is recommended, it is necessary to determine whether the patient will be helped by it enough to justify purchasing one. Hearing aids are generally not covered by Medicare; however, Medicaid or private insurance plans may cover the cost of hearing aids in whole or in part. Regardless of insurance type, patients must be offered the same services, the cost of the services must be equitable, and national procedure codes must be used for requesting reimbursement. It is important to assist the elderly patient in figuring out what his or her specific insurance will cover, if anything, before proceeding with purchasing hearing aids. For those patients that have served in the military, it is advisable to obtain amplification through the Veteran’s Administration because hearing aids and other assistive devices are fully covered through veteran’s benefits. Both economic factors and individual hearing loss should be taken into account. This is particularly important in a sensorineural impairment in which the problem is more one of discrimination than of amplification. Typically, high-frequency sensorineural hearing loss is attributed to presbycusis—age-related hearing loss that is associated with the cochlear degenerative process of aging. Humes et al reported that the hearing loss of older adults is greatest in the frequency region (³ 2,000 Hz) for which the amplitude of speech is the lowest.

Beyond presbycusis, there are also medical causes of hearing loss, including infection, autoimmune disease, medication effects, and many other conditions. These causes should always be evaluated by a physician, and medical causes of hearing impairment generally should be treated in conjunction with auditory rehabilitation and amplification for every patient. A patient being considered for a hearing aid should undergo otologic evaluation first.

## Hearing Aid Considerations

Hearing aids, assistive listening devices, and implantable devices may be helpful for older adults with hearing loss and communication issues. A hearing aid is a portable personal amplifying system used to compensate for a loss of hearing. Almost all hearing-impaired patients are candidates for a hearing aid, although some will receive greater benefits from their aids than others. Any patient who is motivated to use a hearing aid deserves a thorough evaluation and a trial with an appropriate instrument. Assistive listening devices include amplified telephones, television amplifiers, and other such tools that can increase signal intensity for the listener. Implantable devices can include bone-anchored hearing aids, cochlear implants, and the auditory brainstem implant. Hearing aids are appropriate for the vast majority of patients, and this chapter does not address cochlear implants or auditory brainstem implants.

According to Kochkin, the average age of new hearing aid users is 71.1 years. Of the 34 million people with hearing loss in the United States, only 25% use hearing instruments, which suggests that over 25 million people are living with untreated/unaided hearing loss. Despite recent advances in hearing aid technology and miniaturization of hearing aids, negative attitudes persist. Before fitting a hearing aid in an older adult, various factors must be considered, such as communication, physical, psychological, and sociological factors. Some of the issues older adults face when considering use of hearing aids include the following:

1. Experience with hearing aids
2. Financial considerations
3. Attitudes toward hearing aids
4. Degree of hearing loss
5. Lack of need
6. Visual/manual dexterity issues
7. Recommendations from professionals
8. Recommendations from family and friends
9. Stigma
10. Trust
11. Lack of knowledge.

The audiologist is responsible for informing patients fully about the entire process and providing realistic expectations, counseling, and support before fitting a hearing aid.

■ Prefitting Assessment

The audiologist must administer tests to determine whether a hearing loss is present, and if so, the nature of the hearing loss. Threshold measures used in conjunction with otoscopy and immittance testing can help determine the need for medical or surgical remediation. If a condition requires immediate medical attention, the patient should be referred to the appropriate medical professional. Regardless of any degree or nature of impairment, medical clearance for hearing aids from a physician should always be obtained. If no medically treatable condition is present, the severity of hearing impairment, symmetry, configuration, type of hearing loss, and speech perception should be documented. Once medical contraindications are ruled out and the patient is determined to be a candidate for amplification, the audiologist must discuss thoroughly the nature of the hearing loss, its consequences, and realistic expectations, and must evaluate personal factors and the patient's motivational level to use amplification.

The patient should be given a clear explanation of the hearing problem and why he or she has trouble hearing or understanding speech. Patients should understand the difference between hearing difficulty and understanding difficulty, and how amplification affects both. The problems that might easily lead the patient to develop frustration and behavioral changes should be explained clearly so that these problems can be met forthrightly and intelligently. The goal of aural rehabilitation is to prevent or mitigate psychosocial changes and quality-of-life impairments that may result from hearing loss.

Psychological adjustment for each patient involves giving the patient more penetrating insight into the "personality problems" that are already in evidence or likely to develop as a result of hearing loss. Therapy should not use a predetermined technique but must be designed to meet the needs of the specific hearing-impaired individual. Frequently, it is advisable to implement aural rehabilitation not only with the patient but also with the patient's spouse or family because it is impossible to separate a person's individual problems from family problems. The patient must be encouraged to associate with friends and not become isolated because of difficulties in communication. It must be impressed on individuals that using residual hearing effectively allows them to enjoy life and interact as usual with only minor modifications. Use of questionnaires can be very helpful during the prefitting assessment. The Hearing Handicap Inventory for the Elderly Screening Version described by Weinstein (HHIE-S) is a good tool for determining patients' perception of their hearing loss. The HHIE-S is a 10-item questionnaire developed to assess how an individual perceives the social and emotional effects of hearing loss. A higher HHIE-S score suggests a greater handicapping effect of a hearing impairment. The information obtained from this questionnaire can help the audiologist tailor counseling and intervention strategies.

As discussed previously, speech discrimination problems experienced by older adults often have a central auditory or cognitive basis. Because this issue has been identified, part of amplification candidate selection should include a test battery that at least screens for central auditory processing disorder (CAPD). A relationship between sensorineural hearing loss and cognitive impairment has also been identified. It can be difficult to separate cognitive and central auditory effects from peripheral effects in the elderly. Recent data suggest that central auditory dysfunction may be an early manifestation of more general cognitive impairment and therefore may be a contributing factor to poor performance of older adults.

■ Physical Factors

In the elderly, vision status, manual dexterity, ear/ear canal variables, and overall health status should affect the hearing aid decision process. Vision problems may dictate the choice of hearing aid design, style, and type of signal processing. Otologic issues such as excessive wax buildup, active infections, stenosis of the external auditory canal, or unusual growths (such as exostoses) can inhibit the insertion of a hearing aid or limit the effectiveness of a specific style of hearing aid. Patients with a tendency to accumulate excess earwax should be acquainted with options for controlling earwax so that it doesn't affect hearing aid performance. Wax buildup inside of a hearing aid can impair the overall sound quality. Acute middle ear problems such as active infection or effusion can also contraindicate hearing aid use until the problem is resolved. Acute otitis externa will prevent use of a hearing aid until the infection is cleared as determined by their physician. In all cases of suspected medical pathology, patients should be urged to seek medical intervention.
Today's digital hearing aids are considerably smaller than the older technology. Smaller hearing aids can pose a challenge for many older adults who may suffer from poor vision, reduced manual dexterity, diminished tactile sensitivity, or reduced fine motor coordination. Most state-of-the-art hearing aids are digital and adapt their settings automatically for best listening in various environments. This feature can be useful, especially for patients with dexterity issues, because it bypasses the need for manual hearing aid control. Additionally, the adaptive feature can make hearing aids more serviceable for patients with cognitive or memory issues. When selecting a hearing aid, assessment of these factors may be helpful. For example, those elderly patients that may experience reduced mobility, tactile sensitivity, and tremors may have exceptional difficulty changing the small hearing aid batteries; batteries generally need to be changed on a weekly basis. If batteries are left unchanged, the hearing aid itself is no longer of any use and will likely sit in the ear as an earplug rather than an assistive device. Additionally, visual issues that occur in the elderly population can further create a barrier in proper care for amplification devices. It is crucial to give a family member or caregiver the responsibility of caring for the device in terms of changing the batteries as well as general cleaning. Manual dexterity can be measured using the Nine-Hole Pegboard Test, which is designed to evaluate fine-motor coordination and finger dexterity. Visual-motor coordination and touch recognition may affect successful use of amplification and should also be evaluated.

### Acclimatization

The concept of acclimatization must be discussed and addressed with the patient as part of the counseling. It is critical to allow ample time for auditory and cognitive acclimatization to hearing aids, especially in the case of overall auditory deprivation, or switching from a monaural to binaural array. Arlinger et al reported that acclimatization to hearing aids is associated with improvement in auditory performance over time, and acclimatization usually results in a 3 to 5% improvement in speech recognition ability. Factors impacting acclimatization include time course, age of patient, degree/configuration of hearing loss, previous experience, training effects, and the amount of audibility that it restores. Specifically, results have shown a significant acclimatization effect in a group of elderly individuals that were fitted monaurally with a linear algorithm. Although linear processing is less common in today's hearing aid fittings, this further brings up the question of whether a monaural fitting is more appropriate in the elderly population.

### Hearing Aid Arrangement: Monaural versus Binaural

In the case of bilateral hearing loss, choosing to invest in one versus two hearing aids is a decision that must be considered thoroughly. In general, binaural amplification is associated with increased speech understanding, improved directional hearing, improved spatial organization, and signal redundancy. Although many audiologists would consider binaural amplification to be the preferable option in the case of bilateral hearing loss, there is variable evidence regarding success in monaural versus binaural hearing aid fittings.

It is accepted generally that a binaural fitting will provide the greatest localization and speech perception in both quiet and noise. Most studies indicate that binaural fittings help improve binaural squelch, head shadow effects, and binaural redundancy. Binaural amplification can also prevent auditory deprivation, a phenomenon described as a decrease over time in auditory performance associated with the reduced availability of acoustic information.

In the case of elderly patients, however, this approach may not always be optimal. Contrary to these findings, other studies have suggested that binaural fittings can actually be more detrimental to the elderly user. In some cases, auditory processing disorders can present with binaural interference, making binaural speech perception worse than binaural hearing aids. Additionally, it has been found that elderly individuals often exhibit reduced speech intelligibility when aided binaurally as opposed to monaurally. This is referred to as the Binaural Interference Effect. Unless it is determined during the evaluation that the patient exhibits binaural interference, a binaural fitting should still be considered because the majority of patients will benefit from binaural amplification. Subjective reports or dichotic listening tasks are more helpful in determining this than typical audiometric speech tests, which have limited diagnostic value. It may take 6–12 weeks for an older adult to acclimate to binaural amplification, especially if one ear has been unaired for a lengthy period of time.

Cox et al found that specific hearing loss parameters (severity, configuration) were not predictors of a monaural versus binaural preference. The investigators found that patients who preferred a monaural fit attributed this to comfort and quality. Patients who preferred a binaural fit attributed this to restoration of balance, clarity of sounds, and comfort. Nearly all hearing aid manufacturers and audiologists implement a trial period for hearing aids, allowing flexibility for the patient and audiologist to determine the fitting arrangement that provides the greatest benefit. Audiologists and otologists should remain alert for
binaural interference in older patients and should not hesitate to recommend removal of one hearing aid if binaural amplification does not provide the expected result.

# Hearing Aid Technology

Old hearing aid technology did very little to improve a person's ability to understand but improved the ability to hear by making sounds louder. Recent hearing aid technology targets sensorineural losses with poorer discrimination, improving the amount of benefit individuals can receive from a hearing aid, although not necessarily truly improving discrimination ability. In addition to advances in technology, there is also a lot of flexibility regarding discretion. Many companies offer multiple color options so that the patient can choose if they want their hearing aid to match the tone of their skin or hair. Modern technology and color options have made hearing aids more appealing.

One of the most important things that a hearing aid does for people with hearing loss is it permits the individual to hear sounds with greater ease, reducing the strain of listening. Although the individual may not necessarily be able to understand more with an aid than without one, the device may relieve tension, fatigue, and some of the complications of a hearing impairment.

## Over-the-Counter Hearing Aids

Amplification systems are often available over the counter at a considerably reduced price in comparison to those dispensed by a licensed professional. Although these may seem appealing due to their reduced cost and accessibility, these devices should be used with caution. This option may be beneficial for a select number of elderly patients that present with a relatively flat hearing loss and require only some additional gain for speech clarity. In this case, an over-the-counter amplification system may provide the needed benefit at a lower cost. Although a basic amplifier may work for a small percentage of elderly patients, they often do not take into account individual frequency and gain requirements and will often produce more distortion and discomfort rather than offering any noticeable benefit. Patients in these cases may end up wearing a device that is inappropriate for their audiological needs. For some, the poor performance experienced from these devices may deter them from trying other amplification altogether. Another unfortunate outcome may be that the patient often spends unnecessary costs before deciding to purchase devices from a licensed professional.

## Body-Worn Hearing Aid

Given today's microtechnology, the body aid is no longer dispensed. The body aid is a large, high-powered instrument worn on the body and connected to the ear via an earmold. Body aids offer a wide range of amplification and are often used by patients with severe to profound hearing impairment. The microphone, amplifier, and battery are located in the case, which is worn on the body or carried in a pocket. The receiver is connected to the amplifiers by a long wire and is attached directly to the earmold—a custom earpiece designed to collect sound into the ear. This separation of receiver and microphone helps eliminate acoustical feedback in high-amplification instruments. Body aids can be fit to losses of 40 to 110 dBHL. Given that newer technology can also fit a wide range of hearing losses, body aids are now obsolete. Additionally, newer technology contains numerous digital feedback suppression algorithms, eliminating the issue of feedback for patients with significant amplification needs.

## Behind-the-Ear (BTE)

Behind-the-ear (BTE) hearing aids are currently the best choice for severe to profound hearing losses. All of the necessary components of the amplifying system, including the battery, are held in a single case that sits behind the ear. The amplified sound is then fed to the ear via a plastic tube attached to a custom ear mold. This design provides adequate separation of microphone and receiver to reduce acoustical feedback, which can be common in severe losses. These hearing aids can be adapted for mild to profound losses, making them very flexible.

## In-the-Ear (ITE)

Previously, in-the-ear (ITE) hearing aids (Fig. 8.1) were the most widely dispensed. In these types of instruments, the entire hearing aid system is actually housed inside the earmold shell. The aids can help in various cases of hearing loss, typically anywhere in the range from 25 to 80 dBHL. Additionally, various modifications can be made to accommodate different degrees and configurations of hearing loss. There are several styles of ITE instruments available: full-shell (Fig. 8.2a); half-shell (Fig. 8.2b); in-the-canal (ITC) (Fig. 8.2c); and completely-in-the-canal (CIC) (Fig. 8.2b), listed from largest to smallest, respectively. One drawback of the smaller ITE styles is that they cannot adequately provide as much amplification as the larger shells, making them inappropriate for more severe hearing loss. Generally, the larger the device, the larger the fitting range. For those with dexterity and cognitive issues, a larger ITE style
is often preferable for ease of insertion and manipulation. Also, the batteries tend to be larger in larger devices, therefore maintenance of batteries is easier, and battery life may be longer.

**Receiver-in-Canal (RIC) Hearing Aids**

Receiver-in-canal (RIC) hearing aids (Fig. 8.2d) are a newer style that looks very similar to open fit or slim tube BTE hearing aids. The difference is that the hearing aid’s speaker is housed inside the ear canal. The hearing aid speaker's proximity to the tympanic membrane offers a sharper sound quality. Additionally, the small device that is seated behind the ear makes the device more appealing cosmetically as opposed to other styles. RIC hearing aids are popular due to their small size, discrete appearance, and ability to minimize occlusion. Minimizing occlusion creates a more natural sound quality for the patient. The devices also fit hearing losses from mild to severe. The popularity of these open-fit devices is attributable largely to feedback suppression systems. Given the small size of these instruments, elderly patients with dexterity issues may have difficulty manipulating these devices, particularly with inserting and removing the aids.

**Lyric**

The Lyric (Phonak, Zurich, Switzerland) (Fig. 8.4) is a continuous-wear hearing aid designed for moderate to severe hearing loss. A specially trained audiologist or otologist inserts the device completely in the ear canal only 3 or 4 mm from the eardrum. The patient rarely touches the device, and it can remain in the ear for up to 4 months at a time because it is not affected by contact with water. Users can turn the device on and off and raise the volume using an external magnet. One advantage of this device is lack of handling and changing of batteries, which can be particularly helpful for those with both cognitive and dexterity issues. However, the device is more suited for those with mild hearing losses and is susceptible to damage due to wax or moisture given its deep insertion in the ear canal.

**Hearing Aid Orientation**

After the device has been fitted, the audiologist should educate the patient as to the hearing aid parts, and if possible, the patient's frequent com-
munication partners should be oriented, as well. Patients require an understanding of the hearing aid features, overall use (including, but not limited to insertion, removal, program button, volume control), and general maintenance and care. Cognition, memory, and physical factors should be kept in mind when orienting an elderly person to a hearing aid. The older patient may require more and different reinforcement. The clinician should speak at a slow rate to ensure adequate speech understanding; however, it is advised that the clinician not adopt an exaggerated tone because this can be misconstrued as condescending.

Smith and West discussed the importance of positive reinforcement when working with elderly hearing aid patients. They also stressed emphasizing self-efficacy, even if it means presenting information in a very simple manner. The audiologist should be prepared with vision-enhancing aids such as a magnifying glass to ensure that the patient can see small components of the hearing aid. It can also be helpful to point out tactile landmarks such as the catch on the battery door. Sufficient time should be spent ensuring that the patient can insert/remove the hearing aids, change the batteries, and perform basic maintenance. Realistic expectations and a wearing schedule should be reviewed with the patient at this appointment as well.

Fig. 8.3 Examples of various earmolds used to couple with behind-the-ear (BTE) or receiver-in-the-canal (RIC) hearing instruments. Size and style vary depending on individual factors such as degree of hearing loss and ear canal anatomy. (Republished from Siemens Hearing USA; with permission.)

Postfitting Process

Following the initial fitting, the patient should return for a postfitting appointment to ensure that the hearing aids have been used correctly. It is possible that the clinician may have to review the same procedures that were discussed in the fitting appointment. Postfitting questionnaires can help to determine benefit from the patient's perspective. If the patient is unsatisfied with the hearing aids at this point, the audiologist should determine why. There are many adjustments that can be made to the hearing aid circuitry. The audiologist should be prepared to counsel the patient and family as needed; modify the aids, change the number of aids worn, repeat counseling, and apply any intervention strategy needed to help elderly patients use hearing aids successfully.

Summary

Selecting amplification for any patient requires careful individualized consideration of numerous objective factors. These considerations become especially crucial when fitting the elderly population. Sensory deficits as well as issues with cognition, memory, and motor factors require extra care on both the part of the audiologist as well as the family or caregivers throughout the entire fitting process and thereafter. Counseling in particular becomes especially important because there are various psychological effects due to hearing loss involved in addition to the hearing deficit itself. An individualized aural rehabilitation plan must be created to ensure that all needs of each individual are met.
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Cochlear Implantation in the Elderly

Daniel H. Coelho and Brian J. McKinnon

Introduction

Cochlear implantation (CI) has become a well-established means of addressing severe to profound sensorineural hearing loss in children and adults who cannot benefit from conventional amplification. It is understood to be both clinically effective and cost-effective,\(^1-3\) and it is estimated that over 324,200 patients have received this medical device.\(^4\) Nevertheless, although there is evidence supporting the benefits of CI in the general population, concern remains that there is insufficient evidence to support geriatric CI as appropriate, safe, effective, and cost-effective.\(^5\) This chapter provides a broad description of the clinical challenges associated with CI and the potential benefits to recipients.

Epidemiology

Hearing loss is one of the most common disabilities in the elderly, the fastest-growing segment of our population. The U.S. population aged 65 and older will grow from 40.2 million in 2010 (13.5% of the population) to 88.5 million in 2050 (20.5%). One fifth of this 20% will be 85 and older. Studies based on the National Health and Nutrition Examination Survey (NHANES) show that an increasing proportion of the population has age-related hearing loss, reaching over 80% of those over 85 years of age.\(^6\) For up to 10% of older patients with hearing loss, the impairment is so severe that conventional amplification devices fail to provide significant benefit.\(^7\) Beyond speech perception, inability to communicate significantly impacts quality of life and overall well-being and is associated with cognitive impairment, dementia, personality changes, depression, and reduced functional status.\(^8,9\)

Fortunately, as with younger patients, CI has proven to be an extremely effective intervention for older patients and is widely gaining in popularity. Several factors have contributed to the rapid increase in the number of elderly CI recipients. Chief among them is the growth in the overall population over the age of 65, increasing the absolute number of eligible recipients. In addition, not only does the incidence of hearing loss increases with age, but those with hearing loss will experience worsening of their hearing over time. The prevalence of hearing loss nearly doubles with every decade of age.\(^6\) These factors, combined with a small but significant increase in awareness of this technology, have led to more patients opting for CI than ever before. Of all patients receiving CI, the over-65 age cohort is the fastest-growing segment, with the biggest growth in the over-80 subgroup.\(^6\)

Despite the many elderly patients who have benefited from this life-changing technology, the rate of CI use in older adults who meet candidacy criteria is less than 5%.\(^6\) Numerous myths and barriers exist—beliefs widely held by both the public and the medical communities. Among them are the perception that CI is exclusively for congenital hearing loss or children, CI is an untested/experimental technology, outcomes are poor for older adults, CI is not covered by insurance, the surgery poses an unacceptably high risk, or the device is too complicated to use, among others. Referral patterns likewise contribute to the bottleneck of access to CI. Few primary care providers screen for hearing loss at a new patient visit, with even fewer addressing hearing at follow-up visits.\(^10\) In addition, some audiologists and otolaryngologists may have well intentioned though misplaced faith in conventional hearing aids, irrespective of a potential financial disincentive to refer.

Preoperative Considerations

Once an elderly patient has been identified audiologicaly as a candidate for CI, the process does not differ greatly from that for younger candidates. A thorough history must be obtained, with particular
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attention given to duration of hearing loss, especially severe-to-profound hearing loss. As with younger patients, speech performance outcomes are closely related to deafness duration and are critical in counseling patients and their family on reasonable expectations. Cognitive evaluations, although not common practice, can help guide assessment and counseling when appropriate. Likewise, insofar as varying etiologies may have a higher risk of labyrinthine ossification (postmeningitic, posttraumatic, ototoxic, etc.), this too may influence both preoperative imaging choice and expected performance outcomes. Speech performance and expectations must be managed accordingly if traumatic or incomplete implantation is a possibility.

Careful attention must be paid to medical comorbidities. Although age itself is not a known risk factor for perioperative complications, the likelihood of coexisting cardiopulmonary pathology does increase with age. Medical optimization and clearance by the patient’s primary care provider, cardiologist, pulmonologist, and care providers can be extremely helpful in assuring a successful procedure and recovery. Many older patients may be on aspirin, clopidogrel, warfarin, or other anticoagulative therapy. Collaborating with the prescribing physician can help to bridge the perioperative period, leading to a timely transition back to therapeutic anticoagulation.

### Intraoperative Considerations

There is a pervasive, but erroneous, perception among both health care professionals and consumers that age is a significant risk factor for general anesthesia and for anesthetic and surgical complications. Current literature suggests that comorbidities and the American Society of Anesthesia (ASA) rating of physical status are more important than age as prognostic factors for adverse anesthetic outcome. Coexisting conditions of advanced age that potentially have an impact on the risk of anesthesia include cardiopulmonary insufficiency, arthritis, hepatorenal disease, endocrine dysfunction, nutritional status, and pharmacokinetic issues.

Coelho et al reviewed 70 patients over the age of 70 undergoing CI and found that general anesthesia is well tolerated without significant risk in the majority of patients. In their review of 50 patients 80 years or older, Carlson and colleagues found no higher rates of surgical complications when compared with younger CI recipients, though they did find a small but statistically significant higher risk of cardiovascular complications and hospital admission rate for this population. Nonetheless, of great import to patients and physicians, there was no mortality associated with this elective procedure. This is not surprising: unlike other, nonelective surgeries that elderly patients frequently must undergo, CI usually requires only 1 to 2 hours to perform with ~1.5 to 2 hours of general anesthesia. Further, the small incision and minimal blood loss do not result in significant hematologic or fluid imbalance.

Other studies have agreed that age alone is not an independent risk factor in geriatric populations, particularly in the nonemergent or outpatient procedures in geriatric populations. Lau and Brooks demonstrated that age itself is not a reliable predictor of unanticipated hospital admission after laparoscopic cholecystectomy. In comparing age and ASA status, Trus and colleagues and Matin and colleagues independently found no increased risk contribution from age in patients older than 65 years who underwent laparoscopic reflux and urological surgery, respectively.

Although “best anesthetic” technique has yet to be defined in patients with cardiovascular disease, hemodynamic stability and speed of recovery are impacted by choice of anesthesia. Kirkbride et al showed improved intraoperative blood pressure maintenance in older outpatients induced with the high-dose inhalation agent sevoflurane compared with those randomly assigned for intravenous propofol induction. In addition to myocardial depressant effects of anesthetic agents, atelectasis associated with mechanical ventilation, and volume loading due to intravenous fluid administration contribute to cardiac and pulmonary complications, including congestive heart failure, hemodynamic instability, and pulmonary insufficiency. Cardiopulmonary complications can be reduced by minimizing intraoperative fluid administration and the duration and amount of anesthesia used. Elderly patients require up to 30% less minimum alveolar concentration of inhalational anesthetic compared with young adults. The use of bispectral index (BIS) monitors may aid in the titration of anesthetic and improve early recovery.

### Postoperative Considerations

Surgical complications associated with CI are not increased in the elderly. Specifically, the older patient does not have a higher incidence of flap necrosis, improper electrode placement, infection, facial nerve stimulation or injury, or cerebrospinal fluid (CSF) leakage. In addition, no perioperative deaths have been reported. Significant postoperative pain or nausea is rarely encountered, and patients frequently return to their normal routine within days. In fact, nausea and vomiting are less common in older than in younger adults.

Postoperative urinary retention has been reported in patients with and without benign prostatic hyper-
trophic (BPH). Conversely, many CI patients with a history of BPH did not experience postoperative urinary retention. The type of anesthesia administered (including analgesia and sedation) may play an important role in the development of postoperative urinary retention. Derangements of the sympathetic/parasympathetic balance result in increased bladder capacity and decreased rate of bladder contraction. Similarly, opioids relax the detrusor muscle and increase bladder capacity. Other indirect effects of anesthesia contribute to retention, including excessive intravenous volume administration, sedation leading to decreased awareness of bladder filling, and postoperative positioning or situations. Because postoperative urinary retention is a rare complication of general anesthesia, the authors do not routinely place a Foley catheter.

In reviewing the published literature on surgical outcomes in elderly CI recipients, no studies reported a higher rate of device failure (hard or soft), infection, or extrusion. However, certain long-term situations unique to the geriatric population must be considered. For those implants not secured in a bony well or with bony tie-down sutures, the temporalis pocket serves as an important barrier to extrusion or anterior migration. Great care must be taken to avoid tearing the temporalis periosteum or creating an overly large subperiosteal pocket—both of which are more likely in the elderly population—and can result in postoperative migration of the receiver-stimulator. In addition, as patients age, and certainly during periods of poor health, lean body mass decreases. Therefore, the external coil magnet may become too strong, resulting in inadvertent pressure necrosis. Close inspection at each programming session can help to identify this problem and prevent further injury.

### Postoperative Audiological and Quality of Life Outcomes

With respect to postoperative rehabilitation, geriatric and younger adult cochlear implant users share similarities in terms of time frames between surgery and activation, the number of initial and follow-up sessions, and program strategies used. There appear to be no apparent differences in the factors used to guide geriatric and younger adult cochlear implant rehabilitation, with speech perception testing being the most important. Reported barriers for geriatric cochlear implant users receiving rehabilitation include reimbursement for services, limited time for rehabilitation services, and transportation.

An excellent review summarizes and details current postoperative audiological and quality of life (QOL) findings. There are several measures and instruments used to assess the audiological and QOL outcomes achieved by geriatric cochlear implant recipients, the complexity of which are beyond the scope of this chapter. The various measures and instruments make comparison of different studies difficult, and on occasion, impractical. Younger adults may have less restrictive candidate criteria, and cochlear implant candidate criteria can vary from country to country; many audiological and QOL outcome tests are specific to language, health system, or country. Although most audiological outcome testing is standardized, not all QOL outcomes are, compounding the challenge. Lastly, there have been substantial advances in available technology over time, with concurrent improvement with CI outcomes, further complicating assessment of long-term performance, as well as comparison of current cochlear implant user outcomes with past cochlear implant user outcomes.

However, there are consistent trends being identified. Geriatric cochlear implant users enjoy improved speech perception and have outcomes for speech perception in quiet that are comparable to younger cochlear implant users. Younger postlingual cochlear implant users do have better speech perception in noise than geriatric cochlear implant users, which may reflect a longer duration of hearing loss and poorer preoperative speech perception in the latter group. Geriatric patients tend to have a somewhat slower rate of speech perception gain. However, there is evidence of a strong correlation between length of daily cochlear implant use and speech perception performance. When preoperative speech perception was taken into account, age was not predictive of postoperative speech perception outcome.

Interestingly, unilateral geriatric cochlear implant users report a speech perception benefit similar to younger unilateral cochlear implant users, but less speech perception benefit was reported by bilateral geriatric cochlear implant users than by unilateral geriatric cochlear implant users or younger bilateral cochlear implant users. Although many geriatric cochlear implant users reported continued challenges with telephone conversation, and conversation in noise and groups, larger speech perception gains have been seen in those who report increased social activity. Speech perception achievements appear stable over the long term, and speech perception may continue to improve.

In aggregate, QOL outcomes of geriatric cochlear implant users, like speech perception, mirror those of younger cochlear implant users, though validated instruments were not used in many studies. Greater QOL outcomes were seen with greater speech perception scores. Geriatric cochlear implant users show greater confidence and participation in social settings than they did preoperatively. Physical health
and social support QOL scores were, in general, not improved. Overall, geriatric cochlear implant users and their families reported high levels of satisfaction and hearing benefits.

Although there is benefit to taking the data in aggregate, as been done here, it is worth taking a closer look at a more detailed level before leaving this topic. The studies that show subtle but significant differences are worthy of closer examination because they may provide insight into the physiology of normal aging. First, some studies show that performance is in fact slightly worse (but still excellent) in older individuals. Second, all geriatric implantees are not the same, and even when controlling for duration of deafness, the cohort over age 70 may not perform as well as those under age 70. Third, the learning curve for older individuals may be different, taking them years to achieve speech recognition levels reached after only 1 year by younger matched adults. Fourth, some studies show that performance is in fact slightly worse (but still excellent) in older individuals—limitation that, unlike hearing in quiet, does not improve with time. Fifth, similar to some studies in children but unlike matched adults, side may play a role in outcomes, with right side implantation resulting in improved speech perception.

Economic assessment of efficacy further finds that geriatric CI compares favorably with pediatric and adult CI, despite shorter life expectancy, and that rates of long-term use and nonuse compare favorably with children and adult recipients. Nonetheless, funding and reimbursement issues are particularly relevant in this patient population. A RAND Corporation–funded study reviewed payments received for cochlear implants by providers and facilities in the United States and found substantial shortfalls in reimbursement. With Medicare alone, the study determined that on average a hospital faced a $5,000 to $10,000 loss on every Medicare patient implanted, making the provision of CI to geriatric candidates economically tenuous. Additionally, Medicare uses candidate criteria that are significantly more restrictive than those put forward by the Food and Drug Administration. Such selection biases likely skew the outcomes data, under-representing the true benefit for older patients with moderate-to-severe hearing loss. Taken together, the lack of adequate reimbursement and the restrictive candidate criteria risk reduced access to CI for many geriatric patients who could benefit. Attempts to improve levels of reimbursement for cochlear implants are being impeded by the trend to control health care costs in the context of the current economic and political era, and the implementation of health care reform legislation, the Patient Protection and Affordable Care Act.

■ Conclusion

An important question to consider asks not only what the geriatric patient can teach us about CI but also what CI can teach us about the geriatric patient. The answers may lie within what is already known about CI. Cochlear implants have been studied sufficiently in the geriatric population to conclude that geriatric patients with severe-to-profound hearing loss achieve substantial and incontrovertible benefit from CI compared with no intervention or conventional hearing aids. In aggregate, many studies show that the benefit for older individuals (variably defined but generally older than 65) is comparable to that for younger matched adult controls.

Although geriatric CI candidates and recipients face many hurdles, there is growing evidence to support geriatric CI as appropriate, safe, effective, and cost-effective. Despite the evidence, access to CI and cochlear implant services will remain constrained due to restrictive candidate criteria and inadequate reimbursement. Efforts should be made to resolve these impediments so that CI can be made available to any geriatric patient likely to benefit from this remarkable technology.
References


Subjective Idiopathic Tinnitus in the Geriatric Population

Paul F. Shea and Brian J. McKinnon

Introduction

Tinnitus is the perception of sound in the absence of an external auditory stimulus; tinnitus can be distressing and annoying, contributing to disruption of sleep, anxiety, and depression. It has been reported that 16% of the general population have experienced tinnitus to some extent. About 25 to 30% of those who report tinnitus seek medical help; ~ 2 to 4% of all those with tinnitus report being unable to lead a normal life because of their tinnitus. Nearly a quarter of geriatric patients report tinnitus, a finding that has been stable for some time; the rate of distress and negative impact is higher than in younger patients, and the perceived severity increases with later onset of tinnitus.

Tinnitus can be separated into two broad categories, objective and subjective. Objective tinnitus can be heard by both the patient and the examiner. It is less common than subjective tinnitus and can be caused by somatosounds. Subjective tinnitus is perceived only by the patient and may be idiopathic or secondary to another disorder. Tinnitus is commonly associated with hearing loss, traumatic brain injury, ototoxicity, and other conditions. When a diagnosis can be identified to which the tinnitus may be secondary, such as Ménière disease, otitis media, or cerumen impaction, treatment of the condition may provide improvement in the tinnitus. A significant percentage of tinnitus patients with normal hearing have abnormalities of outer hair cell function that can be measured by otoacoustic emissions, as well as abnormal central activity measured by auditory brainstem responses, and these cases are therefore not strictly idiopathic. Thus an effort should be made to identify any underlying conditions before making the diagnosis of idiopathic tinnitus. This review discusses subjective idiopathic tinnitus that is bothersome and that persists for longer than 6 months in the geriatric patient.

Evaluation

The evaluation of the geriatric patient with tinnitus does not differ significantly from that of other patients with hearing loss or tinnitus. A complete otolaryngological history is taken, and a physical examination is performed. In the geriatric patient, attention should be paid to family medical history (particularly to hearing loss history) past and present noise exposure (both recreational and work related), use of hearing protection, past and current medications, previous surgery, and past hearing aid use. It should not be assumed that geriatric patients are inactive and free of ongoing noise exposure. A history of arthritis, head injury, or smoking is associated with an increased risk of developing tinnitus. The concurrent complaint of anxiety and insomnia is also common in this population.

A challenge in the evaluation of the geriatric tinnitus patient is the lack of a generally accepted instrument to document or describe the nature, severity, or quality of tinnitus. Visual analog scale scores can be used to assess loudness, pitch, and disturbance of the tinnitus. Questionnaires such as the Tinnitus Handicap Inventory and the Tinnitus Reaction can help with grading the tinnitus severity. The Tinnitus Functional Index has the advantage of being able to grade tinnitus severity and to measure effectiveness of tinnitus interventions.

Physical examination should include meticulous otoscopy (ideally with magnification), and audiological testing. Auscultation should be performed in complaints of pulsatile tinnitus. Audiological testing should not be limited to routine audiometry and tympanometry; otoacoustic emission testing and auditory brainstem response can help identify possible causes, even in those with normal routine audiometry. Laboratory tests such as autoimmune studies, tests for infectious causes (e.g., Lyme disease, syphilis), thyroid studies, hematocrit, blood
chemistry, lipid profile, and others should be considered based on the level of suspicion created by the history and physical exam.\textsuperscript{8,16}

Imaging is not performed routinely in elderly patients with symmetric hearing loss or tinnitus, nor in those with nonbothersome symmetric tinnitus without hearing loss. Imaging should be considered in patients with asymmetric hearing loss and tinnitus, asymmetric tinnitus without hearing loss, and pulsatile tinnitus.\textsuperscript{6,10} An excellent algorithm for the evaluation of pulsatile tinnitus is described by Mattox and Hudgins.\textsuperscript{17}

### Management

Despite the immense amount of literature on the management of tinnitus, there is a dearth of studies of sufficient quality to permit specific recommendations regarding treatment.\textsuperscript{1} Very little of the literature is specific to the management of tinnitus in the geriatric patient, and the studies that do exist are of insufficient quality to guide age-specific recommendations.

Currently there is no Food and Drug Administration (FDA)-approved pharmaceutical agent for tinnitus, and evidence-based pharmacological approaches are limited to the treatment of comorbidities such as depression, anxiety, and insomnia.\textsuperscript{18} Many medications recommended to assist in the management of tinnitus (antidepressants, anticonvulsants, anxiolytics, and herbal preparations) may be inappropriate or unsafe in the geriatric patient, and some may exacerbate tinnitus.\textsuperscript{1,19} Intratympanic medications studied in randomized, controlled trials have not shown benefit; in the case of lidocaine, no randomized, controlled trials have been performed,\textsuperscript{1,18} and no conclusion can be drawn toward efficacy or safety. Novel therapies, such as acupuncture,\textsuperscript{20,21} hold promise in the adult tinnitus population and await study in the geriatric tinnitus population, although benefit has not been established for hypnosis.\textsuperscript{22-24}

Because cochlear implantation is not currently approved by the FDA for the management of tinnitus, this chapter does not review cochlear implantation for tinnitus management in the geriatric patient. However, it should be noted that tinnitus can improve in geriatric patients who undergo unilateral and bilateral cochlear implantation for severe to profound hearing loss.\textsuperscript{25,26}

With the dearth of evidence supporting the safety or efficacy of pharmacological management of tinnitus, alternative therapies should be considered. In 2009, the UK Department of Health issued the Provision of Services for Adults with Tinnitus.\textsuperscript{27} The recommendations included providing tinnitus patients with information/education, hearing aids, psychological support, relaxation therapy, cognitive behavioral therapy (CBT), sleep management, sound enrichment therapy, and habituation therapies.\textsuperscript{27,28} These guidelines were not specific to the geriatric population and did not provide specific recommendations for assessing therapeutic benefit.\textsuperscript{28} However, the guidelines do suggest a systematic and orderly approach to the management of the tinnitus patient. Of those therapies recommended by the guidelines, educational counseling and CBT appear to hold significant promise of benefit.

Tinnitus retraining therapy (TRT) and sound therapy are commonly recommended in tinnitus patients,\textsuperscript{1} though research specific to geriatric tinnitus patients is lacking. TRT (which the Provision of Services for Adults with Tinnitus guideline refers to as habituation therapy or simplified tinnitus retraining therapy) and sound therapy (also known as masking) have both been subjects of a Cochrane review.\textsuperscript{31,32} Like many of the therapies mentioned previously, the Cochrane review could not make a determination on the efficacy of sound therapy due to the lack of good evidence in the literature.\textsuperscript{31} A Cochrane review of TRT did identify a single, low-quality randomized, controlled trial suggesting TRT is more effective than masking;\textsuperscript{32} other work has found CBT combined with TRT is beneficial.\textsuperscript{33}

Tinnitus often occurs with hearing loss. Hearing aids have been the standard treatment of hearing loss for decades, particularly in geriatric patients, but are increasingly recognized for their role in the treatment of tinnitus as well. This is believed to be
accomplished by several mechanisms. Amplification of speech diverts attention away from tinnitus, and amplification of other ambient sounds serves to partially mask tinnitus.24 Hearing aids have also been developed that can deliver continuous masking noise, and others attempt to transpose the tinnitus with sound of a different frequency. A recent study demonstrated long-term benefit in tinnitus patients with linear octave frequency transposition (LOFT) hearing aids measured with a visual analog scale.35 More studies are needed to determine which hearing aid programming strategies are most effective for the general and geriatric patients with hearing loss and tinnitus because there has been a lack of evidence supporting the use of hearing aids in the past.26

■ Conclusion

It is well established that geriatric patients find tinnitus disruptive, and with advancing time and age, tinnitus can become more intrusive and burdensome. The concern should be taken seriously, and the evaluation should be meticulous and thorough. The care of the geriatric tinnitus patient requires the recognition that there is little evidence to support aggressive medical or surgical therapy. Interventions making use of educational counseling, hearing aids, CBT, and sound therapy currently appear to hold the best chance of providing relief.

■ References


Introduction

Dizziness and imbalance are common conditions affecting the elderly population. They can be challenging to manage given that the symptoms can be nonspecific and may represent multiple underlying diagnoses. This chapter begins by defining dizziness and imbalance in the elderly and describing age-related vestibular loss (ARVL), which is of particular interest to the geriatric otolaryngologist. The chapter then reviews the epidemiology of dizziness, imbalance, and ARVL and discusses the physiological and pathological evidence for ARVL. Finally, the chapter reviews the evaluation and management of an older patient with dizziness and imbalance and closes with a discussion of falls risk assessment.

Definitions of Dizziness and Imbalance in the Geriatric Population

Dizziness connotes a subjective perception of disorientation or involuntary motion, which can occur during movement or at rest. Dizziness can be subdivided further into the subtypes of vertigo and presyncopal lightheadedness. Vertigo is the false sensation that either the body or the environment is moving (usually spinning) and may be a symptom of vestibular, visual, or neurological impairment, psychological factors, or the use of multiple medications (polypharmacy). Vertigo that occurs in the elderly has been termed presbyvertigo. Presyncopal lightheadedness is the sensation of impending faint associated with transient diffuse cerebral hypoperfusion. Causal factors for lightheadedness include cardiovascular disease and orthostatic hypotension (e.g., resulting from excessive medication use or autonomic instability). Imbalance can be equated with disequilibrium, and it connotes a sense of postural instability generally associated with the trunk and legs without a sensation in the head. Imbalance is usually described either while standing or walking and typically does not occur at rest. Imbalance results from neuromuscular impairment related to muscle weakness, loss of peripheral sensation or proprioception, and/or arthritis. The imbalance or disequilibrium that occurs with aging has been termed presbystasis or presbyequilibrium.

The typology of dizziness and imbalance is conceptualized in Fig. 11.1 as a set of overlapping conditions. Age-related vestibular loss (ARVL), which is in the realm of geriatric otolaryngology, is also depicted. The vestibular system plays an integral role in maintaining the vestibulo-ocular and vestibulospinal reflexes (VOR and VSR). The VOR is important for stabilization of gaze during head movement, and VOR impairment manifests as dizziness (i.e., abnormal sensation of motion). The VSR is important for trunk and limb stabilization during head movement. VSR dysfunction manifests as postural instability. The VOR and VSR are depicted in Fig. 11.1 as the overlap between ARVL and presbyvertigo and presbystasis, respectively. Interestingly, there is increasing recognition of the physiological importance of vestibuloautonomic projections. Vestibuloautonomic impairment has been associated with orthostatic hypotension. Thus ARVL may also be a causal factor for the symptom of presyncopal lightheadedness. Emerging evidence is suggesting that a certain amount of ARVL is present in older individuals but may not be manifesting symptomatically as dizziness or imbalance. This may be because the level of vestibular impairment has not crossed a critical threshold, or because an individual is able to compensate for the ARVL. ARVL is thus depicted in Fig. 11.1 as asymptomatic or “subclinical” and symptomatic or “clinical.”

As is evident in Fig. 11.1, multiple causative factors have been associated with dizziness and imbalance in the geriatric population. It is well known among researchers who study aging that geriatric conditions often result from numerous coexisting
Epidemiology of Dizziness, Imbalance, and Age-Related Vestibular Loss in the Geriatric Population

Estimates of the prevalence of dizziness and imbalance in the geriatric population depend largely on the definitions of dizziness and imbalance used, and on the populations surveyed. Definitions vary as noted previously. The populations surveyed can vary with respect to their age ranges, whether they are population-based or clinic-based, and what types of clinics are being studied (e.g., primary vs. specialty care). Several large population-based studies have found a 20 to 30% prevalence of dizziness and imbalance in the elderly population (age ≥ 65 years).\(^7\)-\(^8\) The prevalence of dizziness and imbalance rises steeply with age, with levels over 50% in the community-dwelling population over age 80.\(^10\)

A study in institutionalized nursing home residents observed a prevalence of dizziness and vertigo of 68%.\(^11\) Among patients presenting to a primary care clinic, 24% reported dizziness and 17% identified dizziness as their major presenting complaint.\(^12\) Within the otolaryngology clinic, one study of 131,000 consecutive patients found that 6% of patients over age 65 presented with vertigo or a presumed vestibular diagnosis.\(^13\) Interestingly, this large-scale survey of otolaryngological practice found that visits from geriatric patients increased from 14.3% in 2004 to 17.9% in 2010. Moreover, this study noted that the five most common geriatric diagnoses were otologic (including hearing loss, external ear disorders, tinnitus, otitis media/eustachian tube disorders, and vertigo).

A landmark series of studies based in Germany estimated the population prevalence and incidence more specifically of vestibular vertigo (i.e., vertigo resulting from vestibular impairment). Community-dwelling participants in a national telephone survey were queried about symptoms of dizziness and vertigo. Those who reported moderate symptoms were administered a detailed neurotologic interview, from which vestibular vertigo was diagnosed based on symptoms of rotational vertigo, positional vertigo, or recurrent dizziness with nausea and oscillopsia or
imbalance. Of note, the neurotologic interview was found to have good validity based on a gold standard of neurotology clinic–based diagnosis in establishing a vestibular diagnosis. The lifetime prevalence, 1 year prevalence, and incidence of vestibular vertigo were observed to be 7.8%, 4.9%, and 1.5%, respectively.\(^1\) The 1 year prevalence of vestibular vertigo increased with age to 7.2% in those aged 60 to 69 and 8.8% in individuals over age 80. This study was among the first to estimate the population prevalence of ARVL.

A more recent study estimated the prevalence of vestibular impairment in the U.S. population using an objective, rather than subjective (self-report based), test. Data were drawn from the 2001–2004 National Health and Nutrition Examination Survey (NHANES). Vestibular function was assessed in NHANES using the modified Romberg test, whereby vestibular impairment was inferred from an inability to stand on a foam pad with eyes closed. Thirty-five percent of U.S. adults age 40 years and older had evidence of balance dysfunction based on this postural metric.\(^4\) The odds of balance dysfunction increased significantly with age, such that 85% of individuals age 80 and above had evidence of balance dysfunction. These estimates are considerably higher than the prevalences of vestibular vertigo mentioned earlier from the German population. It is possible that the symptom of vestibular vertigo represents a component of clinical ARVL, whereas vestibular impairment based on the modified Romberg test represents subclinical ARVL.

Epidemiological analyses of dizziness, imbalance, and ARVL have also investigated risk factors for these conditions. Most studies have observed an increased prevalence of dizziness and imbalance in women.\(^1\) Vestibular vertigo was also more prevalent in women.\(^15\) However, the prevalence of vestibular impairment based on objective modified Romberg testing did not differ by gender.\(^4\) Findings from a review of the most frequently reported causes of dizziness in primary care practice are presented in Table 11.1.\(^1\) The review found that peripheral vestibular disease was the most common cause of dizziness, observed in 20 to 50% of cases. Peripheral vestibular diseases included benign paroxysmal positional vertigo (BPPV), labyrinthitis, and vestibular neuritis. Other common causes of dizziness were cardiovascular disease, systemic infection (leading to orthostatic hypotension), psychiatric disorders, metabolic disturbances, and use of multiple medications. A more recent epidemiological survey of the elderly population in England found that dizziness was associated with abnormal heart rhythm, hearing loss, vision loss, and low grip strength, whereas imbalance was associated with diabetes, arthritis, low grip strength, and vision loss.\(^8\) With respect to vestibular vertigo, independent risk factors were depression, tinnitus, and cardiovascular risk factors, including hypertension and dyslipidemia.\(^14\) Finally, independent risk factors for vestibular impairment as measured by the modified Romberg test included low socioeconomic status and diabetes mellitus.\(^4,16\)

Epidemiological studies also have examined the impact of dizziness, imbalance, and ARVL on diverse outcomes, including falls, quality of life, health care utilization, and other economic outcomes. Dizziness has been associated with a two- to threefold increased risk of falling.\(^5,10\) Specifically with respect to ARVL, the study from NHANES found that individuals with objective vestibular impairment who were also clinically symptomatic (i.e., reported dizziness) had a 12-fold increase in the odds of falling. In a small pilot study, older fallers were found to have significantly higher rates of peripheral vestibular dysfunction than older nonfallers.\(^17\) A prospective study reported that elderly patients with vestibular asymmetry were significantly more likely to experience an incident fall.\(^18\) Moreover, several studies have

### Table 11.1 Most common causes of dizziness in primary care practice

<table>
<thead>
<tr>
<th>Category</th>
<th>Percent</th>
<th>Examples</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peripheral vestibular disease</td>
<td>20–50</td>
<td>Benign paroxysmal positional vertigo (BPPV), labyrinthitis, vestibular neuritis</td>
</tr>
<tr>
<td>Cardiovascular disease</td>
<td>10–30</td>
<td>Arrhythmia, congestive heart failure, vasovagal conditions (e.g., carotid sinus hypersensitivity)</td>
</tr>
<tr>
<td>Systemic infection</td>
<td>10–20</td>
<td>Systemic viral and bacterial infection</td>
</tr>
<tr>
<td>Psychiatric conditions</td>
<td>5–15</td>
<td>Depression, anxiety, hyperventilation</td>
</tr>
<tr>
<td>Metabolic disturbances</td>
<td>5–10</td>
<td>Hypoglycemia, hyperglycemia, electrolyte disturbances, thyrotoxicosis, anemia</td>
</tr>
<tr>
<td>Medications</td>
<td>5–10</td>
<td>Antihypertensives, psychotropic medications</td>
</tr>
</tbody>
</table>

observed an association between vestibular asymmetry and fall-related hip and wrist fracture risk.19-21

Quality of life measures assess general quality of life (e.g., the Short-Form Health Survey [SF-36]) as well as health-related quality of life (i.e., related to a specific health condition). Dizziness and vestibular vertigo have been associated with significantly poorer quality of life, in both the physical and the mental domains. One population-based study in Sweden found that dizziness was one of the most influential symptoms affecting general quality of life in older individuals.22 The most widely used measures of dizziness- and imbalance-related quality of life are the Dizziness Handicap Inventory (DHI),23 the Activities Balance Confidence (ABC) scale,24 and the Falls Efficacy scale (which measures fear of falling).25 Two studies that administered the DHI in patients presenting with dizziness to a primary care clinic and a specialized dizziness clinic found that over 60% of patients reported moderate to severe handicap associated with their dizziness in both clinical contexts.26,27 With respect to health care utilization and economic outcomes, the German population-based study found that vestibular vertigo was more likely than nonvestibular vertigo to be associated with a medical consultation, sick leave, interruption of daily activities, and avoidance of leaving the house.28 Similarly, a population-based study in the United States observed that 50% of older individuals with dizziness and balance problems saw at least one medical provider, and 35% saw three or more providers.29 A single provocative longitudinal study found that patients with disequilibrium at baseline were at significantly increased risk only for new-onset cognitive decline compared with controls.30

The epidemiological data reviewed thus far suggest that dizziness, imbalance, and ARVL are prevalent in the population and have significant clinical, functional, and economic implications. Several final points deserve mention at the conclusion of this section. First, as has been highlighted by numerous authors, the goal of research on dizziness and imbalance in the geriatric population should be the development of evidence-based clinical practice guidelines for the effective diagnosis and management of these conditions.1 To this end, the use of a common nomenclature is an important first step. Second, although dizziness and imbalance are prevalent in the geriatric population they are not universal. As one study specifically points out, not all individuals over age 90 had dizziness.10 As such, dizziness and imbalance in the geriatric population may be considered “age-concomitant” rather than “age-dependent” conditions.10 As a corollary, these conditions should be viewed as pathological, and efforts should be made to treat them. Finally, it follows that the potential scope of managing dizziness and imbalance in the geriatric population is enormous, likely far exceeding the capacity of geriatricians and otologists.11 Thus it might be necessary to train other types of health care professionals such as nurses and physical therapists to assist more in managing these conditions.

### Physiological and Pathological Evidence for Age-Related Vestibular Loss

As already described, there is epidemiological evidence that ARVL is prevalent in the geriatric population. The vestibular system consists of five organs: three semicircular canals (anterior or superior, posterior and horizontal, or lateral), and two otolith organs—the saccule and the utricle (Fig. 11.2). The semicircular canals detect angular head rotations along the planes of the canals, whereas the otoliths detect linear translations of the head, as well as head orientation with respect to gravity. The saccular neuroepithelium is oriented in a vertical direction and preferentially detects vertical linear head movements, whereas the utricular neuroepithelium is horizontally oriented and preferentially detects horizontal head movements.

In recent years, numerous vestibular physiological tests have been developed that allow for localization of dysfunction within the five organs of the vestibular system. The most widely used vestibular tests are caloric and rotational chair testing, which evaluate the function of the horizontal semicircular canal.31 Recently, video-oculography techniques have made possible quantitative angular vestibulo-ocular reflex (AVOR) testing during head impulses to assess the function of each of the six semicircular canals.32,33 The vestibular-evoked myogenic potential (VEMP) tests are gaining increasing popularity as measures of otolith function. The sound-evoked cervical VEMP (cVEMP) is a product of the sacculocollic reflex and is thought to specifically reflect saccular function.34 The vibration-evoked ocular VEMP (oVEMP) has been suggested to selectively measure utricular function.35 Several classic studies have reported a decline in horizontal semicircular canal function with aging. Peterka and colleagues tested over 200 healthy subjects across a wide age range (7–81 years) and observed increased postural sway and decreased VOR gain to sinusoidal rotation with age.36 Caloric responses, however, were not observed to change with age. Paige similarly observed declining VOR responses with age to high-amplitude and high-velocity sinusoidal rotations in 81 patients age 18 to 89.17 The author concluded that “aging entails a progressive bilateral peripheral vestibular loss.” Baloh and colleagues completed one of the only longitudinal studies of vestibular function in 57 normal older
individuals who were followed annually for 5 years. They observed a significant decrease over the 5 years in VOR gain to sinusoidal stimuli, again only at higher velocities. Interestingly, none of the older individuals reported symptoms of dizziness or imbalance. It is possible that a threshold of ARVL must be crossed for the disease to move from being subclinical to clinical.

In addition to age-related declines in semicircular canal function, studies also suggest that otolith function decreases with age. Welgampola and Colebatch performed cVEMP testing in 70 adults age 25 to 85. They observed decreasing click-evoked response amplitudes with age, notably a 25 to 30% decline in amplitude per decade from the sixth decade. Brantberg and colleagues measured tone-burst evoked cVEMPs in 1,000 consecutive patients seen in their clinic and observed a steady decline with age starting as early as age 40. A pilot study of 50 healthy older individuals age 70 and over evaluated the five vestibular end-organs simultaneously to assess which if any organ was disproportionately affected. The study observed that 80 to 90% of subjects had semicircular canal dysfunction, whereas only 50% of participants had abnormal saccular function and 20% had utricular impairment.

Histopathological analyses of human temporal bones also demonstrate the adverse effects of age, demonstrating declines in vestibular hair cell populations and progressive otoconial degeneration associated with aging. Interestingly, findings from temporal bone specimens corroborate the foregoing physiological data, demonstrating a greater loss of vestibular hair cells in the cristae ampullares of the semicircular canals relative to the otolithic maculae.

### Evaluation and Management of an Older Patient with Dizziness and Imbalance

It is critical to take a systematic approach in the evaluation of an older patient with dizziness or imbalance. Some older individuals may report primarily vertigo, lightheadedness, or disequilibrium, which can suggest the predominance of certain underlying etiologies. However, it should be noted that older patients often have multiple concomitant impairments, such as low vision (e.g., from presbyopia or macular degeneration), cardiovascular disease (e.g., hypertension), muscle weakness, and arthritis, and the use of multiple medications. Although a particular impairment may predominate, it is possible that this impairment in combination with another deficit creates the clinical problem (i.e., the clinical problem is multifactorial). As such, it is critical that the most
common contributors to dizziness and imbalance in the elderly be addressed systematically every time in every patient.

The first step in the otolaryngologist’s evaluation of an elderly patient with dizziness and imbalance is to obtain a history. This includes asking when the symptoms started, whether the symptoms are progressive, how the patient would further characterize the symptoms (vertigo, lightheadedness, and/or imbalance), whether the symptoms are constant versus episodic (if episodic whether the duration is seconds, minutes, hours, or days), and whether the episodes occur at rest or only during head movement, standing, or walking. Of the major vestibular diagnoses, BPPV is particularly common in older adults and bears special mention. Increased BPPV in the elderly may reflect age-related degeneration of the otoconial membrane, leading to abnormal seeding of otoconia in the endolymph.46 A study of the German population observed a prevalence of 3.4% in individuals over age 60, and a cumulative lifetime incidence of almost 10% by age 80.47 BPPV accounted for 39% of cases of vertigo in older patients presenting to neurotology clinics.48 However, older patients do not always experience the classic presentation of BPPV—short episodes of rotatory vertigo associated with changes in head position. A study of 100 older patients presenting to general geriatric practices for chronic medical conditions found that 9% had unrecognized BPPV.49 Moreover, patients with BPPV had significantly increased fall risk. Another study found that older patients with BPPV were more likely to experience postural instability.50 Of note, this instability could be improved through canalith repositioning maneuvers.

The next step is to elicit a medical history to understand the multiple factors that might be contributing to the patient’s dizziness. Specifically, the following conditions should be noted: (1) eye disorders (e.g., cataracts, macular degeneration, or glaucoma), (2) cardiovascular disease (e.g., hypertension, arrhythmias, syncope), (3) musculoskeletal disorders (e.g., arthritis, weakness), (4) peripheral sensory loss (e.g., neuropathy), (5) psychiatric disease (e.g., depression, anxiety), (6) cognitive impairment, and (7) systemic conditions (e.g., diabetes). If any unrecognized nonvestibular impairment is identified, an appropriate referral should be made. The otolaryngologist should also ask about a history of hearing loss. Emerging evidence is suggesting a link between hearing loss, imbalance, and elevated fall risk.51

The otolaryngologist should then review the patient’s medications. Polypharmacy (i.e., the use of four or more medications) contributing to dizziness is a particular concern among older individuals. A study in the ambulatory adult population age 65 or older found that 44% of men and 57% of women take five or more medications.52 Moreover, older adults metabolize drugs differently than younger adults and may be exposed to higher drug levels at the usual doses. An increase in the number of medications used in the elderly has been associated with an increased risk of impaired balance.53 Certain classes of medications, including antihypertensive, psychotropic, and narcotic pain medications, have been associated with a particularly increased risk of dizziness and falls and have been termed fall-risk-increasing drugs (FRIDs).54–56 Several clinical tools have been developed to evaluate potentially inappropriate medication use in older patients. The Beers criteria is the most commonly used tool.57 It contains one list of medications that should be avoided independent of diagnosis, and a second list of medications that should be avoided considering the diagnosis. Further tools have been developed, including the Screening Tool of Older People’s potentially inappropriate Prescriptions) (STOPP) criteria, which enumerate medications to be avoided by organ system, and the Screening Tool to Alert doctors to Right Treatments (START) criteria, which list medications that should be recommended in older patients according to condition in the absence of any contraindications.57

With respect to the physical examination, we recommend screening for orthostatic hypotension (a systolic blood pressure decrease of at least 20 mm Hg or a diastolic blood pressure decrease of at least 10 mm Hg within 3 minutes of standing). Evidence of orthostasis could indicate excessive dose of antihypertensive medications (among other conditions) and should prompt referral to the primary care provider or cardiologist. Eye movements should be assessed, particularly the cardinal movements of smooth pursuit, saccade, and vergence. Deficits may indicate brainstem or cerebellar pathology and should prompt a referral to a neurologist. Cerebellar function should also be assessed with maneuvers such as the finger-nose-finger test, the rapid alternating hand movement test and heel-to-shin test. Deficits in any of these tests may indicate cerebellar dysfunction and should prompt referral to a neurologist.

Clinical vestibular testing should include assessment for spontaneous nystagmus (which would indicate vestibular asymmetry), postheadshaking nystagmus (which would indicate a latent vestibular asymmetry), and the horizontal head impulse test (HIT) (which evaluates horizontal semicircular canal function). One study showed that 50% of older adults age 70 and older had an abnormal HIT.58 Clinical testing should also include the Dix-Hallpike test to evaluate for BPPV, which, as mentioned previously, is very common in older individuals and is treatable. The Romberg test should be performed with eyes closed on foam to evaluate standing balance in the absence of vision and proprioceptive information (such that the patient is relying only on vestibular information).
The patient’s gait should also be observed, to assess stability and the potential need for physical therapy and/or an assistive device. The timed up and go (TUG) test is an efficient and reproducible measure of fall risk. The patient is asked to stand from a seated position, walk 3 m, turn around, then return to sitting. Older adults who take longer than 14 seconds to complete the TUG are at significantly increased risk of falling.59

At present, the mainstays of management of the older patient with dizziness or imbalance seen in the otolaryngology clinic are management of polypharmacy, identification of nonvestibular contributors and appropriate referrals, management of hearing loss, home safety modification (including installing night lights and grab bars, removing throw rugs, and creating clear passageways within the home), use of assistive devices (cane and walker), and exercise programs (e.g., tai chi, or even light walking). For older patients with evidence of vestibular impairment, vestibular rehabilitation is the primary treatment. Vestibular rehabilitation is a program whereby patients learn to compensate for their vestibular loss by using visual or proprioceptive cues under the direction of a therapist.60,61 Studies have shown that vestibular rehabilitation is as effective in older patients as in younger patients.62 One randomized, controlled trial administered vestibular rehabilitation to a group of older patients with chronic dizziness seen in primary care clinics.63 The study found that vestibular rehabilitation significantly reduced dizziness symptoms and improved postural stability and dizziness-related quality of life. This trial is among the first to administer vestibular rehabilitation in primary care patients who did not have a specific vestibular diagnosis (except perhaps ARVL). Further research is needed to establish the appropriate timing for vestibular rehabilitation. Evidence that vestibular function starts to decline in middle age suggests the potential benefit of vestibular exercises prior to the onset of significant vestibular loss.64 This phenomenon has been termed prerehabilitation, or “prehab.”65 More recent studies have investigated the benefit of biofeedback prostheses in the treatment of vestibular impairment. The prostheses consist of body-worn devices that deliver sensory feedback (e.g., vibrotactile, auditory) to patients to orient the trunk during movement.66 Early reports are promising that the prostheses effectively improve dizziness and imbalance. Additionally, the multichannel implantable vestibular prosthesis represents a potential new technology for the treatment of ARVL that is awaiting human trials.67

Finally, pharmacological therapies for dizziness should be used judiciously and sparingly in older individuals. The most commonly-used medications to treat dizziness are vestibular suppressants, which include antihistamines (e.g., meclizine), anticholinergics (e.g., scopolamine), and benzodiazepines (e.g., lorazepam).68 Vestibular suppressants can be effective in reducing symptoms of vertigo and motion sickness. However, they have been shown to blunt the error signal that drives vestibular compensation.69 As such, vestibular suppressants are not indicated in the setting of chronic, progressive vestibular impairment (e.g., ARVL) where compensation is critical. Moreover, vestibular suppressants have sedating effects and are metabolized and cleared more slowly in older individuals.70 Thus they are not recommended in the elderly; indeed, antihistamines, anticholinergics, and benzodiazepines are listed in the Beers criteria. Interventions that challenge the vestibular system and foster compensation—such as vestibular therapy—are preferable to treat dizziness and imbalance in the elderly.

### Falls Risk Assessment

The American Geriatrics Society (AGS) recommends that all patients older than age 65 with a history of falls or a balance and gait disorder should undergo multifactorial falls risk evaluation.71 If the opportunity exists, otolaryngologists should consider joining or developing multidisciplinary teams that provide multifactorial falls risk evaluation to older patients. Such a multidisciplinary falls prevention clinic has been established at the first author’s (YA’s) institution. All patients seen in the clinic are given a standardized questionnaire (Appendix). The questionnaire was developed based on AGS guidelines and with input from a multidisciplinary group of providers at the institution, including geriatricians, neurologists, otolaryngologists, ophthalmologists, orthopedists, cardiologists, physiatrists, psychiatrists, and physical and occupational therapists. Notably, the questionnaire quantifies fall history with the use of a falls severity grading scale developed in the clinic.72 Additionally, standard batteries such as the Geriatric Depression Scale, the Activities Balance Confidence Scale, the Lawton Instrumental Activities of Daily Living Scale, and the Falls Efficacy Scale are administered to measure the impact of imbalance and falls risk on the patient’s functional status and quality of life.74,25,75,76

A standardized physical examination was also developed for patients seen in the falls prevention clinic (Table 11.2), using measures in wide clinical and research use such as the Balance Evaluations Systems Test (the Mini-BEST), the Scale for the Assessment and Rating of Ataxia (SARA), and the Montreal Cognitive Assessment (MOCA).77–79 Vestibular testing is performed in all patients, including assessment of spontaneous nystagmus, gaze-evoked nystagmus, postheadshake nystagmus, visual VOR suppression,
Dix–Hallpike test, and HIT using video-oculography. We also evaluate the other key contributors to fall risk, including vision loss (specifically loss of contrast sensitivity), peripheral sensory loss (particularly loss of joint proprioception), muscle weakness (particularly of the lower limbs), and neurocognitive decline (including loss of cerebellar and cognitive function). All patients receive personalized counseling on home modification and the need for assistive devices and are prescribed a program of physical and/or occupational therapy as needed. The screening questionnaire is used to direct any specialty referrals (e.g., to neurology, otolaryngology, or ophthalmology).

### Conclusion

As the population ages, increasingly otolaryngologists will be called on to manage the common geriatric problem of dizziness. This chapter reviews a nomenclature for dizziness and imbalance conditions in the elderly and emphasizes the need for a systematic approach to this multifactorial problem. Otolaryngologists should recognize the high prevalence of dizziness in the elderly, its potentially profound impact on quality of life and even length of life (shortened by fall injuries), and the availability of treatment to improve symptoms. Most older patients with dizziness can be helped, but first we must recognize them and treat them with enthusiasm and knowledge.

### References


FALLS PREVENTION CLINIC QUESTIONNAIRE

The Johns Hopkins Hospital
600 North Wolfe Street, Meyer 1130  Baltimore, MD 212877142
Appointments: (410) 6143234/ Fax: (410) 6140503

Thank you for arranging to visit our clinic.

Please complete this questionnaire before coming for your visit. It is confidential and will be part of your record. It asks for information about your current problems and your past medical history. This form will give us a better understanding of your problem, and will allow us to spend more time evaluating your problem and discussing treatment plans.

When you come for your first visit, please bring this completed form along with other medical information you or your doctor think is necessary. Should you have any questions, please do not hesitate to contact us at the number at the top of this page.

Thank you very much. We look forward to seeing you.

Johns Hopkins Falls Prevention Team

Name __________________________________________________   Date ____________________
Birth date ____________________   Age __________   Gender:  □ Male  □ Female
Race:   □ Black or African American   □ American Indian or Alaska native   □ Asian
   □ Native Hawaiian or Pacific Islander   □ White
Are you Hispanic or Latino:   □ Yes   □ No

Contact Information:
Home Phone: _________________________   Work Phone: _________________________
Email: _____________________________________________
Address: __________________________________________________________________________
I live with:  □ Alone   □ Spouse   □ Family
 □ Other, please describe: ____________________________________________________________
Who referred you to us (name, address, phone)? ______________________________________
__________________________________________________________________________________

Who is your primary care physician? ___________________________________________________

Are there any other physicians involved in your care? Please list them below:

<table>
<thead>
<tr>
<th>Name</th>
<th>Specialty</th>
<th>Phone Number</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
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</tr>
</tbody>
</table>
1. Past Medical History

*Have you ever been told you have or had any of the following (check all that apply):*

**Cardiovascular**
- □ High blood pressure
- □ Chest pain/angina
- □ Heart attack
- □ Rheumatic fever
- □ Blood clots/phlebitis
- □ High cholesterol

**Respiratory/Lung**
- □ Feeding tube (PEG)
- □ Emphysema
- □ Asthma
- □ Chronic bronchitis
- □ Allergies/hay fever
- □ Tracheostomy

**Neurological**
- □ Stroke
- □ Hyperactivity
- □ Learning disability
- □ Attention problems
- □ Head injury
- □ Depression/anxiety

**Gastrointestinal**
- □ Ulcers
- □ Reflux
- □ Hepatitis

**Renal**
- □ Kidney failure
- □ Hemodialysis
- □ Kidney stones

**Endocrine**
- □ Diabetes
- □ Thyroid disease
- □ Osteoporosis
- □ Rheumatologic disease
- □ Hearing difficulties
- □ Vision problems
- □ Injuries
- □ Anemia
- □ Cancer

*Please describe other health problems that you have:*
___________________________________________________________________________________
___________________________________________________________________________________
___________________________________________________________________________________
___________________________________________________________________________________

2. Medications

**Allergies:**
__________________________________________________________________________

**Current Medications:** Please list all medications you are currently taking (including injections and skin patches), and when possible please provide the dosage:
___________________________________________________________________________________
___________________________________________________________________________________

3. Have you had any x-rays, MRI or CT scans of your brain, neck or spine?

□ No □ xray □ MRI □ CT scan □ Neck □ Back □ Brain

**List and/or explain:**  
___________________________________________________________________________________
___________________________________________________________________________________
4. About your life

What is the highest degree you have earned?
□ High school  □ Technical certificate  □ Associate’s  □ Bachelor’s  □ Master’s
□ Doctoral

What is your marital status?   □ Married   □ Single   □ Divorced   □ Widowed   □ Separated
□ Other

If you have children, how many and how old?
___________________________________________________________________________________

Do you currently live in a:   □ House   □ Apartment   □ Nursing facility
□ Other ________________________________

Are there any steps to get in the house/apartment?:   □ Yes   □ No
If yes, how many? _______________

Are there any steps inside the house/apartment?:   □ Yes   □ No
If yes, how many? _______________

Is there elevator access to other levels of your house/or to your apartment?   □ Yes   □ No

Is your house or apartment wheelchair accessible?   □ Yes   □ No

Do you smoke? (please include cigarettes, cigars or pipes)   □ Yes   □ No
If yes, how many packs per day? __________   For how many years? __________

If no, did you smoke formerly?   □ Yes   □ No
Packs per day? __________   For how many years? __________

Are you able to walk:   □ Independently   □ I use an assistive device  Please choose below:
□ Cane  □ Walker or Rollator  □ Wheelchair
□ Other device, describe ____________________________________________________________
5. About how you are feeling

Choose the best answer for how you have felt over the past week:

1. Are you basically satisfied with your life? □ Yes □ No
2. Have you dropped many of your activities and interests? □ Yes □ No
3. Do you feel that your life is empty? □ Yes □ No
4. Do you often get bored? □ Yes □ No
5. Are you in good spirits most of the time? □ Yes □ No
6. Are you afraid that something bad is going to happen to you? □ Yes □ No
7. Do you feel happy most of the time? □ Yes □ No
8. Do you often feel helpless? □ Yes □ No
9. Do you prefer to stay at home, rather than going out and doing new things? □ Yes □ No
10. Do you feel you have more problems with memory than most? □ Yes □ No
11. Do you think it is wonderful to be alive now? □ Yes □ No
12. Do you feel pretty worthless the way you are now? □ Yes □ No
13. Do you feel full of energy? □ Yes □ No
14. Do you feel that your situation is hopeless? □ Yes □ No
15. Do you think that most people are better off than you are? □ Yes □ No
6. About your falls

Using the picture above let us know how many falls you had:

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description</th>
<th>Number last year</th>
<th>Number in last 5 years</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Near Fall: Slip, trip or loss of balance, no fall to the ground</td>
<td></td>
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<tr>
<td>2</td>
<td>Fall to the ground or a lower level (e.g., chair), did not receive medical</td>
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</tr>
<tr>
<td>3</td>
<td>Fall to the ground or a lower level (e.g., chair), received medical attention, not admitted to the hospital</td>
<td></td>
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<tr>
<td>4</td>
<td>Fall to the ground or a lower level (e.g., chair), admitted to the hospital</td>
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</tbody>
</table>

If you experienced a fall in the last 5 years, please answer the following questions, otherwise skip to section 4.
Did any of your falls result in:

1. **Minor injury**—Treated at home resulted in pain, bruise or scrape. Used ice, wound cleaned at home: □ Yes □ No

2. **Moderate injury**—Required a doctor’s evaluation resulting in stitches or other closure, splinting. A pulled muscle or tendon was diagnosed: □ Yes □ No

3. **Major injury**—Required a prolonged visit to the hospital and resulted in surgery, casting, head injury (skull fracture, brain bleed) or internal injury: □ Yes □ No

Please think of the worst fall you had. After falling:

1. Did you require medical assistance? □ Yes □ No

2. Did you call your physician? □ Yes □ No

3. Did you go to your physician's office? □ Yes □ No

4. Did you go to the emergency department? □ Yes □ No

5. Did you have surgery? □ Yes □ No

6. Did you have a broken bone requiring surgery? □ Yes □ No

7. Were you admitted to the hospital? □ Yes □ No

8. Were you in the hospital's Intensive care unit? □ Yes □ No

9. Did you have any other treatment that you think is important? □ Yes □ No

Please describe the problem: ____________________________________________________________
___________________________________________________________________________________
The following questions are about the circumstances during which the fall occurred. During the fall were you/did you:

1. Changing positions (laying down to sitting, or sitting to standing) □ Yes □ No
2. Moving from the bed to a chair, or otherwise □ Yes □ No
3. Moving from one room to another inside the house □ Yes □ No
4. Walking up or down the stairs □ Yes □ No
5. Going from one room to the other in the dark □ Yes □ No
6. Taking a shower □ Yes □ No
7. Moving outside the house □ Yes □ No
8. Using alcohol □ Yes □ No
9. Using bifocal or multifocal eyeglasses □ Yes □ No
10. Having symptoms (shortness of breath, lightheaded, foot pain, etc.) □ Yes □ No
11. On a slippery surface (raining, snowing, liquid on the floor, etc.) □ Yes □ No
12. Trip over an object □ Yes □ No
13. Fall during day time □ Yes □ No
14. Fall during night time □ Yes □ No
15. Alone □ Yes □ No
16. With someone else in the same room □ Yes □ No
17. Being helped by someone (supporting or assisting in any way) □ Yes □ No
18. Have any other important circumstances at the time of the fall □ Yes □ No

Please describe the circumstances: ____________________________________________
_____________________________________________________________________________
7. **SCREENING QUESTIONNAIRE**

<table>
<thead>
<tr>
<th>Question</th>
<th>No</th>
<th>Yes</th>
<th>If yes, please explain</th>
</tr>
</thead>
<tbody>
<tr>
<td>7a. Do you have dizziness (includes imbalance, vertigo, room-spinning, light-headedness)? If yes answer the following questions:</td>
<td></td>
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<tr>
<td>7b. Does your dizziness include imbalance?</td>
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<tr>
<td>7c. Does your dizziness include room-spinning or vertigo?</td>
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<tr>
<td>7d. Has your dizziness been getting worse?</td>
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<td>7e. Does rolling over in bed or getting in or out of bed make you dizzy?</td>
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<td>7f. Does your dizziness last seconds?</td>
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<tr>
<td>7g. Does your dizziness last minutes to hours?</td>
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<td>7h. Does your vision jump or blur while walking?</td>
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<tr>
<td>7i. Do you experience sudden dizziness with loud sounds?</td>
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<tr>
<td>7j. Do you experience dizziness with sneezing, coughing, straining?</td>
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<td>7k. Do you get dizzy/light-headed when you stand up quickly?</td>
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<td>7l. Is your dizziness triggered by certain foods/beverages?</td>
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<tr>
<td>7m. Do you have a family history of dizziness or vertigo?</td>
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<tr>
<td>7n. Have you lost consciousness when you were dizzy?</td>
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<tr>
<td>7o. Do you have hearing loss or ringing in your ears?</td>
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<tr>
<td>7p.</td>
<td>Do you have headaches, head pressure, sinus pressure, visual changes, light sensitivity, sound sensitivity, or nausea?</td>
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<tr>
<td>7q.</td>
<td>Do you have difficulties with memory, attention, decision-making, i.e., cognitive changes?</td>
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<tr>
<td>7r.</td>
<td>Do emotional changes, sudden falling asleep cause you to fall?</td>
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<tr>
<td>7s.</td>
<td>Do you have slurred speech or difficulty swallowing?</td>
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<tr>
<td>7t.</td>
<td>Do you have shaking in your hands or recent change in handwriting?</td>
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<tr>
<td>7u.</td>
<td>Do you have bowel or bladder problems or erectile dysfunction?</td>
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<tr>
<td>7v.</td>
<td>Do you have weakness, numbness, tingling or pain running down your legs? Is it recent or chronic?</td>
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<tr>
<td>7w.</td>
<td>Do you have heart palpitations or an abnormal heart rhythm?</td>
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<tr>
<td>7x.</td>
<td>Do you have shortness of breath with walking?</td>
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<tr>
<td>7y.</td>
<td>Have you had a recent change in your vision?</td>
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<tr>
<td>7z.</td>
<td>Have you had a recent change in your glasses?</td>
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<tr>
<td>7aa.</td>
<td>Do you see double?</td>
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<tr>
<td>7bb.</td>
<td>Do you have low back pain?</td>
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<tr>
<td>7cc.</td>
<td>Do you have hip pain?</td>
<td></td>
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<tr>
<td>7dd.</td>
<td>Do you have knee pain?</td>
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</tbody>
</table>
8. FEAR OF FALLING (Falls Efficacy Scale International)

For each activity below, please mark from 1–4 to show how concerned you are that you might fall if you did this activity. Please reply thinking about how you usually do the activity. If you currently don’t do the activity (example: if someone does your shopping for you), please answer to show whether you think you would be concerned about falling IF you did the activity.

<table>
<thead>
<tr>
<th>Activity</th>
<th>Do you perform this activity?</th>
<th>Not at all concerned 1</th>
<th>Somewhat concerned 2</th>
<th>Fairly concerned 3</th>
<th>Very concerned 4</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Cleaning the house (e.g. sweep, vacuum, dust)</td>
<td>□ Yes □ No</td>
<td></td>
<td></td>
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<tr>
<td>2. Getting dressed or undressed</td>
<td>□ Yes □ No</td>
<td></td>
<td></td>
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<tr>
<td>3. Preparing simple meals</td>
<td>□ Yes □ No</td>
<td></td>
<td></td>
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<tr>
<td>4. Taking a bath or shower</td>
<td>□ Yes □ No</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>5. Going to the shop</td>
<td>□ Yes □ No</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>6. Getting in or out of a chair</td>
<td>□ Yes □ No</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>7. Going up or down stairs</td>
<td>□ Yes □ No</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>8. Walking around in the neighborhood</td>
<td>□ Yes □ No</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>9. Reaching for something above your head or on the ground</td>
<td>□ Yes □ No</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>10. Going to answer the telephone before it stops ringing</td>
<td>□ Yes □ No</td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>11. Walking on a slippery surface (e.g., wet or icy)</td>
<td>□ Yes □ No</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>12. Visiting a friend or relative</td>
<td>□ Yes □ No</td>
<td></td>
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<td>13. Walking in a place with crowds</td>
<td>□ Yes □ No</td>
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<td>14. Walking on an uneven surface (e.g., rocky ground, poorly maintained pavement)</td>
<td>□ Yes □ No</td>
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<td>15. Walking up or down a slope</td>
<td>□ Yes □ No</td>
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<td>16. Going out to a social event (e.g., religious service, family gathering, or club meeting)</td>
<td>□ Yes □ No</td>
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9. LAWTON SCALE—INSTRUMENTAL ACTIVITIES OF DAILY LIVING

Are you able to perform the following activities? For each activity, circle the item description that most closely resembles your functional level (either 0 or 1).

9a. Telephone use

9ai. Operates telephone on own initiative; looks up and dials numbers: □ 1
9a(ii). Dials a few well-known numbers: □ 1
9a(iii). Answers telephone, but does not dial: □ 1
9a(iv). Does not use telephone at all: □ 0

9b. Shopping

9b(i). Takes care of all shopping needs independently: □ 1
9b(ii). Shops independently for small purchases: □ 0
9b(iii). Needs to be accompanied on any shopping trip: □ 0
9b(iv). Completely unable to shop: □ 0

9c. Food preparation

9c(i). Plans, prepares and serves adequate meals independently: □ 1
9c(ii). Prepares adequate meals if supplied with ingredients: □ 0
9c(iii). Heats, serves and prepares meals or prepares meals but does not maintain adequate diet: □ 0
9c(iv). Needs to have meals prepared and served: □ 0

9d. Housekeeping

9d(i). Maintains house alone or with occasional assistance (e.g., “heavy work domestic help”): □ 1
9d(ii). Performs light daily tasks such as dish-washing, bed making: □ 1
9d(iii). Performs light daily tasks but cannot maintain acceptable level of cleanliness: □ 1
9d(iv). Needs help with all home maintenance tasks: □ 1
9d(v). Does not participate in any housekeeping tasks: □ 0
9e. **Laundry**

9ei. Does personal laundry completely: □ 1

9eii. Launders small items; rinses stockings, etc.: □ 1

9eiii. All laundry must be done by others: □ 0

9f. **Mode of transportation**

9fi. Travels independently on public transportation or drives own car: □ 1

9fii. Arranges own travel via taxi, but does not otherwise use public transportation: □ 1

9fiii. Travels on public transportation when accompanied by another: □ 1

9fiv. Travel limited to taxi or automobile with assistance of another: □ 0

9fv. Does not travel at all: □ 0

9g. **Responsibility for own medication**

9gi. Is responsible for taking medication in correct dosages at correct time: □ 1

9gii. Takes responsibility if medication is prepared in advance in separate dosage: □ 0

9giii. Is not capable of dispensing own medication: □ 0

9h. **Ability to handle finances**

9hi. Manages financial matters independently (budgets, writes checks, pays rent and bills, goes to bank), collects and keeps track of income: □ 1

9hii. Manages day-to-day purchases, but needs help with banking, major purchases, etc.: □ 1

9hiii. Incapable of handling money: □ 0
10. The Activities-Specific Balance Confidence (ABC) Scale

Instructions to Participants:

For each of the following, please indicate your level of confidence in doing the activity without losing your balance or becoming unsteady from choosing one of the percentage points on the scale from 0% to 100%. If you do not currently do the activity in question, try and imagine how confident you would be if you had to do the activity. If you normally use a walking aid to do the activity or hold onto someone, rate your confidence as it you were using these supports. If you have any questions about answering any of these items, please ask the administrator.

For each of the following activities, please indicate your level of self-confidence by choosing a corresponding number from the following rating scale:

0% 10 20 30 40 50 60 70 80 90 100%

no confidence completely confident

How confident are you that you will not lose your balance or become unsteady when you...

1. ...walk around the house?  ____%
2. ...walk up or down stairs?  ____%
3. ...bend over and pick up a slipper from the front of a closet floor  ____%
4. ...reach for a small can off a shelf at eye level?  ____%
5. ...stand on your tiptoes and reach for something above your head?  ____%
6. ...stand on a chair and reach for something?  ____%
7. ...sweep the floor?  ____%
8. ...walk outside the house to a car parked in the driveway?  ____%
9. ...get into or out of a car?  ____%
10. ...walk across a parking lot to the mall?  ____%
11. ...walk up or down a ramp?  ____%
12. ...walk in a crowded mall where people rapidly walk past you?  ____%
13. ...are bumped into by people as you walk through the mall?  ____%
14. ...step onto or off an escalator while you are holding onto a railing?  ____%
15. ...step onto or off an escalator while holding onto parcels such that you cannot hold onto the railing?  ____%
16. ...walk outside on icy sidewalks?  ____%