

# Risk Factors Related to Age-Associated Hearing Loss in the Speech Frequencies

Larry J. Brant\*  
Sandra Gordon-Salant†  
Jay D. Pearson\*  
Lisa L. Klein\*  
Christopher H. Morrell\*  
E. Jeffrey Metter\*  
James L. Fozard\*

## Abstract

This paper examines the relationship between several risk factors and the development of age-associated hearing loss in the speech frequencies. Hearing loss is defined as an average threshold level of 30 dB HL or greater at the frequencies of 0.5, 1, 2, and 3 kHz. Hearing thresholds from 0.5 to 8 kHz using a pulse-tone tracking procedure were collected on participants of the Baltimore Longitudinal study of Aging since 1965. A proportional hazards regression model was used to study the relationship between several risk factors that have previously been found to be associated with numerous health-related outcomes and the length of follow-up time until the occurrence of unilateral or bilateral hearing loss in a screened group of 531 men. Risk factors considered are age, blood pressure, and alcohol and cigarette consumption. After controlling for age, only systolic blood pressure showed a significant relationship with hearing loss in the speech frequencies ( $p < .05$ ). Since blood pressure is a modifiable risk factor, these results suggest that preventing hypertension might contribute to an effective program for the prevention of apparent age-associated hearing loss.

**Key Words:** Aging, follow-up study, hearing loss, presbycusis, pure-tone thresholds, risk factors

Recent estimates of hearing loss or impairment in the United States suggest that approximately 28 million people are afflicted (NIH, 1990). Results from the 1987 National Health Interview Survey indicate that the rate of self-reported hearing impairment ranges from 5.4 percent in 18- to 44-year-old individuals to 29.6 percent in those 65 years and older (Schoenborn and Marano, 1988). Even though hearing loss is an important public health problem, risk factors associated with age-associated hearing loss are not well understood. A better understanding of the factors relating to

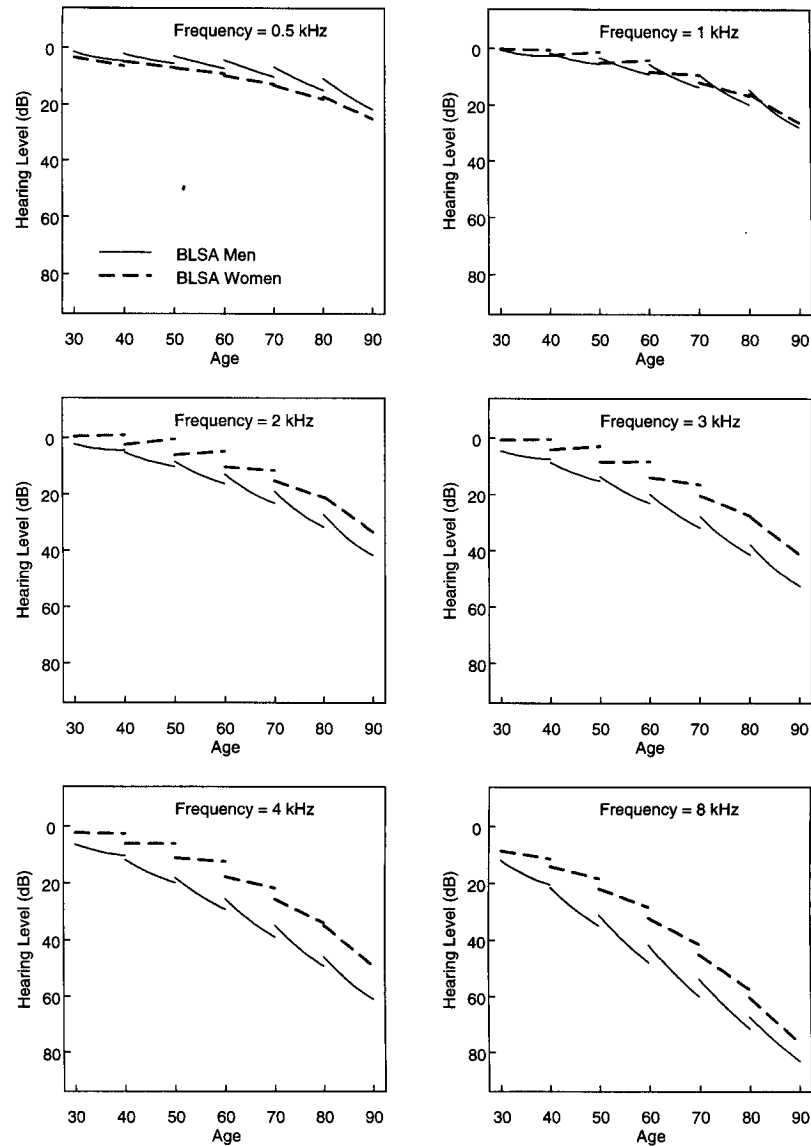
hearing loss may lead to methods of prevention for a health problem concerning a large proportion of the general population.

Using a definition of hearing loss as threshold levels greater than 20 dB above audiometric zero for at least one frequency from 0.5 to 4 kHz in the better ear, Moscicki et al (1985) estimated the prevalence of hearing loss in 57- to 89-year-old participants in the Framingham Heart Study to be 83 percent. In a later paper from the same study, Gates et al (1993) found low-frequency (0.25-1.0 kHz) hearing loss to be related to cardiovascular disease events, which included coronary heart disease, heart attack, and intermittent claudication. While these cross-sectional findings do not prove that cardiovascular disease and hearing loss have a cause and effect relationship, they suggest the need for a longitudinally based risk factor study of hearing loss.

Studies of age-associated hearing loss are complicated by the fact that hearing sensitivity decreases at different rates with age for each of

\*Gerontology Research Center, National Institute on Aging, Baltimore, Maryland; †Department of Hearing and Speech Sciences, University of Maryland, College Park, Maryland

Reprint requests: Larry J. Brant, Gerontology Research Center, National Institute on Aging, 4940 Eastern Ave., Baltimore, MD 21224



**Figure 1** Age-specific patterns of longitudinal hearing loss for males (—) and females (---) from the Baltimore Longitudinal Study of Aging. Each line segment represents the 10-year changes in hearing level for successive age cohorts. The figure is based on data reported by Pearson et al (1995).

the frequencies tested between 0.5 and 8 kHz (Brant and Fozard, 1990; Pearson et al, 1995). Using a mixed-effects regression model with linear and quadratic terms for longitudinal age changes and linear, quadratic, and cubic terms for cross-sectional age changes along with higher order interaction terms involving these age changes, Pearson et al (1995) reported predicted 10-year longitudinal patterns for various age decades. Figure 1 summarizes the average of the age-specific patterns of longitudinally determined hearing loss for females and males from the Baltimore Longitudinal Study of Aging

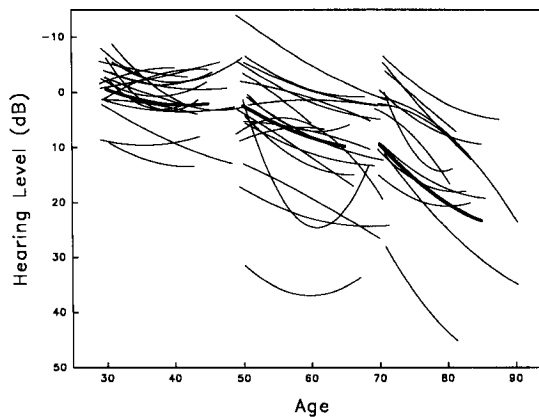
(BLSA) beginning the study at 30, 40, 50, 60, 70, and 80 years of age. The rates of decline (as seen from the slopes of the individual line segments) in hearing levels are different between men and women in the different age groups at the six different frequencies reported. At 0.5 kHz, women have slightly greater hearing levels than men, but men appear to have greater rates of decline (as evidenced by the steeper slopes of the predicted curves) at ages 50 years and older. On the other hand, at 8 kHz, Figure 1 shows that men have greater hearing levels than women and that the men's rates of decline

are greater than the women's as well, except in possibly the 80-year-old group, where both the men's and women's predicted lines appear almost parallel.

Besides age and gender differences in hearing levels at different frequencies, there are large individual differences in the longitudinal patterns of hearing loss. The mixed-effects regression model used to study longitudinal patterns of hearing loss allows for the estimation of both average and individual patterns or trends in the context of a combined or unified analysis. Figure 2 demonstrates the heterogeneity in hearing loss by presenting the predicted patterns of hearing loss at 1 kHz for 19 30-year-old, 19 50-year-old, and 13 70-year-old BLSA male participants with at least three visits and 10 or more years of follow-up. Since each curved line represents the predicted hearing level for an individual, the figure dramatically portrays the considerable differences in the individual patterns of hearing levels. While most individuals display an increase in hearing level with age, a few individuals actually show a decrease in hearing level with age following an initial increase. In addition to age accounting for differences in the individual patterns in hearing level, these data suggest that there may be other underlying variables or risk factors that might be related to hearing loss. Thus, any study of hearing loss should give careful attention to the effect of age and should examine factors that are associated with or help to explain individual differences in hearing loss.

Much of the hearing loss that is associated with age may not be caused by aging in the pure sense but may represent the accumulated effects of various risk factors. In 1962, Rosen et al reported pure-tone hearing thresholds of male and female adults from a nonindustrialized society, the Mabaan tribe of Africa. These thresholds were considerably better than those reported for people of comparable ages from an industrialized society who were free of recognized specific noise exposure and otologic disease (e.g., Robinson and Sutton, 1978) that would impair hearing. This suggests that there may be risk factors for hearing loss associated with daily life in industrialized societies beyond noise exposure that are not inherent in nonindustrialized societies.

Three possible disease-related risk factors that are usually more prevalent in industrialized societies than in nonindustrialized societies are hypertension, cigarette smoking, and alcohol consumption. As a person ages, systolic blood



**Figure 2** Individual patterns of predicted hearing loss at 1 kHz for 30-year-old, 50-year-old, and 70-year-old males having at least three visits and 10 or more years of follow-up in the Baltimore Longitudinal Study of Aging. The thick lines represent the average patterns of hearing loss for each of the three age groups and are the same as in the corresponding panel of Figure 1.

pressure increases due to a progressive vascular stiffening (Kannel and Vokonas, 1986). High blood pressure including clinical hypertension is thus possibly related to age-associated hearing loss because of subsequent vasoconstriction. Vasoconstriction of the inner ear blood vessels adversely affects the supply of blood and oxygen to the inner ear, and because the inner ear relies on oxidative metabolism, oxygen deprivation to the inner ear is thought to produce deficits in auditory sensitivity (Ito, 1991; Ohlsen et al, 1992; Miller et al, 1995). A history of cigarette smoking has often been noted in cases of presbycusis. Smoking produces increased levels of carbon dioxide and nicotine, which can constrict blood vessels or cause vasospasm and thrombotic occlusions (Zelman, 1973). Alcohol consumption may also affect the auditory nervous system. Because the principal effect of alcohol absorption by the blood is the depression of the central nervous system, the direct action of alcohol as well as related nutritional deficiencies may cause peripheral nerve degeneration and brain alterations, particularly in those individuals who chronically consume alcohol. Auditory brainstem potentials of alcoholics, obtained during periods of abstinence, have demonstrated a significant increase in interpeak latencies compared to nonalcoholics (Begleiter et al, 1981). However, it is unknown whether these electrophysiologic changes in the auditory system associated with alcohol consumption are accompanied by changes in behavioral measurements such as auditory thresholds.

The purpose of this paper is to examine the relationship between hearing loss in the speech frequencies and chronic disease-related risk factors such as systolic blood pressure, cigarette smoking, and alcohol consumption, while controlling for age. This paper uses information collected from a longitudinal study and reduces the bias due to underlying hearing disorders by only including participants in the analysis who have been screened for otologic disorders and evidence of noise-induced hearing loss prior to the start of the study.

## METHOD

### Subjects

Participants in the Baltimore Longitudinal Study of Aging (BLSA) served as subjects in this study. The BLSA is an ongoing multidisciplinary study of normal human aging (Shock et al, 1984). The study is carried out at the Gerontology Research Center in Baltimore, Maryland and is part of the intramural research program of the National Institute on Aging (NIA). The BLSA originated with the study of male participants in 1958 and was expanded to include female participants in 1978. Hearing testing was added to the BLSA protocol in 1968. The study population represents a predominantly white upper-middle class group of community-dwelling male and female volunteers who live primarily in the Baltimore-Washington metropolitan area.

A sample of 1247 men and 588 women was evaluated in the hearing protocol. Study participants were tested on at least two occasions and the longitudinal measurements were taken at approximately 2-year intervals. Thus, longitudinal hearing data are available for a maximum follow-up of 22.8 years in men and for a maximum follow-up of 13.0 years in women.

The medical records of the BLSA participants were reviewed at every examination at which an audiogram was available to exclude participants with any of the following diagnoses of otologic disorders: chronic otitis media, Meniere's disease, cholesteatoma, perforation of the tympanic membrane, congenital hearing loss, otosclerosis, ototoxicity, stroke-induced hearing loss (all stroke patients were excluded because of the possibility of a central or peripheral hearing loss caused by the stroke or alteration in the ability to accurately complete the test), middle ear effusion, and impacted cerumen. Also, participants with a unilateral hearing loss

(i.e., the average hearing level at 0.5, 1, 2, and 4 kHz differed between ears by more than 10 dB at one or more visits), which presumably reflects an unidentified pathology, were excluded from the study. In addition, participants with any evidence of noise-induced hearing loss from the audiometric record were also excluded from the study. Noise-induced hearing loss was assumed to be present if a participant's hearing threshold at 3, 4, or 6 kHz exceeded the thresholds at both 2 and 8 kHz by 15 dB or more (Ward, 1980; Kryter, 1985). Finally, for an individual to be included in the study, their first examination average hearing threshold at 0.5, 1, 2, and 3 kHz had to be no greater than 20 dB for either ear. The number of subjects who satisfied the inclusion criteria of normal otologic history, had no evidence of noise exposure, and had normal hearing in the speech frequencies at entrance to the study were 531 men and 310 women.

### Measurements

**Audiometric Thresholds.** Pure-tone thresholds by air conduction were obtained from the subjects using a Bekesy audiometer (GSI 1701). The stimulus was a tone swept from 0.1 to 10 kHz and was presented in the pulsed mode with a 50 percent duty cycle (200 msec on, 200 msec off). The stimuli were delivered using TDH-39 circumaural earphones with MX 41/AR cushions. A method of adjustment was used to track the subject's auditory threshold. Each ear of the subject was tested individually. All testing was conducted in a sound-proof booth that conforms to the standard criteria for background noise in audiometric rooms for 500 to 8000 Hz (ANSI, 1977).

**Blood Pressure.** During each examination, blood pressure measurements were obtained using standard procedures. Blood pressure was measured by auscultation of the first and fifth Korotkoff sounds using a manual sphygmomanometer appropriately sized to the participant's arm. Each participant's blood pressure was taken in both arms while in a seated position.

**Case History.** In advance of visiting the Gerontology Research Center, the participant was sent a medical-history questionnaire to complete and bring to the Center. During the interview, the medical examiner (physician, clinical nurse practitioner, or physical's assistant) reviewed the information with the participant and, if necessary, supplemented it. The case

history questionnaire asks information about alcohol consumption, including whether or not the participant drinks alcoholic beverages, the types of alcoholic beverages consumed, and the amount of consumption. Questions pertaining to smoking history include whether or not the participant smokes, the type of tobacco smoked, and the frequency of tobacco smoking.

**Procedures.** Participants were followed longitudinally and were classified as having obtained an "event" of hearing loss if the average pure-tone threshold at 0.5, 1, 2, and 3 kHz reached or exceeded 30 dB HL in either ear. These frequencies were selected to represent average hearing sensitivity in the speech frequencies. The elimination of participants whose pure-tone average was between 20 or 30 dB HL at baseline together with the careful screening for hearing-related illnesses reduces the bias due to underlying disease and reduced the chance of artifactually high hearing loss due to events that occur at the beginning of the longitudinal follow-up period.

The length of follow-up and information on the event of "hearing loss" were used in a survival analysis of hearing loss. A survival analysis evaluates the length of time until the development of an event, which is hearing loss in this study. A proportional hazards regression model (Cox, 1972) was the specific type of survival analysis used, which permitted assessment of the association between risk factors and the time until the occurrence of the event. In particular, this model controls for age by

using four age strata: < 50, 50–59.9, 60–69.9, and ≥70 years (Kalbfleisch and Prentice, 1980; Hopkins, 1990). The inclusion of strata in the model helps adjust for previously observed differences in rates of hearing loss by age by allowing for the estimation of different survival patterns for each age strata. Covariates or risk factors considered in the model as having a possible effect on hearing loss include alcohol use (drinks per week), cigarette smoking status (packs per day), and systolic and diastolic blood pressure (mm Hg). Relative risks of developing hearing loss with regard to each risk factor were calculated using the estimated coefficients from the regression analysis. Tests of significance and 95 percent confidence intervals (CIs) for each covariate from univariate and multivariate analyses were obtained using the usual asymptotic properties of these estimates.

## RESULTS

Using the definition of an event for hearing loss as the average pure-tone threshold 30 dB HL or greater in either ear at 0.5, 1, 2, and 3 kHz, 46 of the 531 men and 7 of the 310 women in the study population attained an event of hearing loss during the follow-up period. The small number of events in the population of women provides insufficient information for the subsequent analysis and, hence, only data for the men are analyzed and reported in this paper.

Table 1 gives the sample characteristics of the male study population for each of the four age groups: <50, 50–59.9, 60–69.9, and ≥70

**Table 1 Sample Characteristics of BLSA Male Participants by Age at First Examination for Hearing Risk Factor Study**

Characteristic	Age at First Examination			
	<50 (n = 303)	50–59.9 (n = 77)	60–69.9 (n = 82)	≥70 (n = 69)
	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD
Age at entry	33.4 ± 8.0	55.4 ± 2.9	64.2 ± 3.0	75.1 ± 3.9
Length of follow-up (yr)	10.9 ± 7.1	11.2 ± 7.6	7.8 ± 6.2	5.1 ± 4.2
Systolic blood pressure (mm Hg)	121.0 ± 14.8	131.9 ± 17.8	137.5 ± 20.9	139.4 ± 20.9
Cigarette smoking (%) <sup>*</sup>				
None	39.6	37.7	41.4	52.2
Moderate	35.6	41.5	35.4	39.1
High	24.8	20.8	23.2	8.7
Alcohol consumption (%) <sup>†</sup>				
None	11.6	11.7	13.4	21.7
Moderate	55.1	55.8	39.0	50.7
High	33.3	32.5	47.6	27.6

<sup>\*</sup>Moderate is one pack or less/day, high is more than one pack/day; <sup>†</sup>moderate is six drinks or less/week, high is more than six drinks/week.

years. The average length of follow-up was greatest in the 50-year-old group (11.2 [± 7.6] years) and was smallest in the 70 years and older group (5.1 [± 4.2] years). The table also gives the mean and standard deviation for the age at entry and systolic blood pressure for each of the four age groups. As expected, the mean systolic pressure increases with age, as does the standard deviation. In addition, the table shows the percentage of men with no (none), moderate, and high cigarette smoking and alcohol consumption. Cigarette smoking is less prevalent in older groups, while alcohol use remains relatively constant.

Figure 3 shows the average hearing thresholds (dB HL) for the frequencies (500, 1000, 2000, 3000 Hz) used in the determination of hearing acuity (only right ear data are shown) for the 531 men tested at the first examination and included in one of the four age categories. As stated in the subject inclusion criteria for the study, the overall mean hearing level at the four frequencies is no greater than 20 dB at baseline examination. In fact, the greatest single frequency mean is 20.2 dB in the ≥70-year-old group at 3000 Hz. During the succeeding follow-up period of approximately 20 years (maximum follow-up was 22.8 years), 9 events of hearing impairment were observed in the <50 age group, while 14, 11, and 12 events occurred in the 50- to 59.9-, 60- to 69.9-, and ≥70-year-old age groups, respectively.

Results from the age-adjusted analyses using an age-stratified proportional hazards model for the risk factors systolic blood pressure,

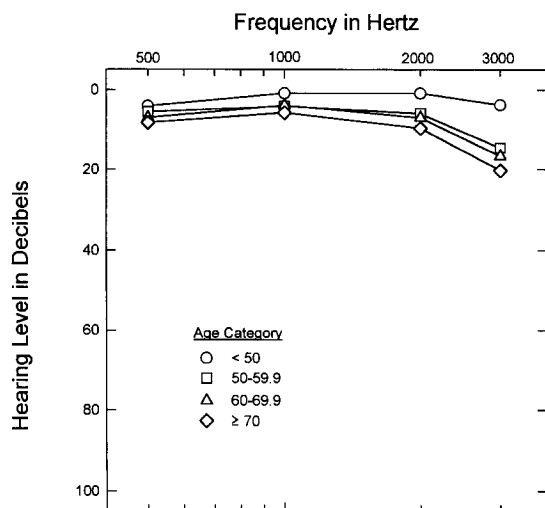


Figure 3 Mean audiograms for the <50-, 50 to 59.9-, 60 to 69.9-, and ≥70 year-old age groups.

cigarette smoking, and alcohol consumption are given in Table 2. Univariate analyses were done for each risk factor separately while controlling for age using the four age strata in the proportional hazards model. In addition, since systolic blood pressure, cigarette smoking, and alcohol consumption are often interrelated or cigarette smoking and alcohol consumption may serve as intermediate mechanisms between systolic blood pressure and hearing loss, a multivariate proportional hazards regression model was carried out for the three risk factors simultaneously while also controlling for age with a similar stratified analysis. The parameter estimates of the models are related to the relative risks or the ratios of the chances of an individual incurring a hearing loss during the follow-up period compared to a reference group. A statistically significant relative risk is larger or smaller than 1 (the value of 1 represents no evidence of a difference in risk between the different levels of the risk factor). When  $p < .05$ , the 95 percent CI of the relative risk does not overlap the value of 1. After adjustment for age and the simultaneous effect of the potentially confounding risk factors of cigarette smoking and alcohol consumption, systolic blood pressure remained at about the same level of statistical significance ( $p = .05$ ) as the age-adjusted univariate result ( $p = .04$ ). Systolic blood pressure showed a significant

Table 2 Relative Risks and 95% Confidence Intervals (CIs) of Hearing Loss for Several Risk Factors in BLSA Hearing Study\*

Risk Factor	Relative Risk (95% CI)	p value
<b>Univariate</b>		
Systolic blood pressure†	1.32 (1.01–1.74)	.04
Cigarette smoking		
Moderate vs none	1.38 (0.71–2.70)	.35
High vs none	1.23 (0.56–2.70)	.61
Alcohol consumption		
Moderate vs none	0.81 (0.37–1.79)	.61
High vs none	0.83 (0.34–1.98)	.67
<b>Multivariate</b>		
Systolic blood pressure†	1.32 (1.00–1.76)	.05
Cigarette smoking		
Moderate vs none	1.37 (0.67–2.79)	.39
High vs none	1.30 (0.56–3.04)	.54
Alcohol consumption		
Moderate vs none	0.78 (0.34–1.80)	.56
High vs none	0.65 (0.25–1.68)	.38

\*Relative risks computed from an age-stratified proportional hazards model; †relative risk per 20 mm Hg difference.

association with hearing impairment having a relative risk of 1.32 for a 20 mm Hg rise (95% CI 1.01 to 1.74) from the age-adjusted univariate analysis. For example, this result indicates that men who are borderline hypertensive with a systolic pressure of 140 have a 32 percent greater risk of developing a hearing loss than normotensive men with a systolic pressure of 120. Similarly, men who are definite hypertensive with a systolic pressure of 160 have a 74 percent (1.32<sup>2</sup>) greater risk of hearing loss than those with a 120 mm Hg reading. The other risk factors examined, cigarette smoking and alcohol consumption, show no significant association with hearing loss.

The age-stratified proportional hazards model gives one overall estimate of the relative risk for each risk factor level that pertains to all four age strata. Separate independent proportional hazards model analyses were performed for each of the four age groups individually to check for the appropriateness of a single measure of the relative risk for systolic blood pressure from the unified analysis. The results of these analyses (not shown) suggest that the single age-stratified value representing all ages adequately summarizes the relative risk of systolic blood

pressure in the entire male study population, since the relative risks calculated separately for the different age strata have similar values and do not show any trend with age.

Figure 4 shows the probabilities (%) of normal hearing for three levels of systolic blood pressure (120 mm Hg [normotensive], 140 mm Hg [borderline hypertensive], and 160 mm Hg [definite hypertensive]) for the four age groups (<50, 50–59.9, 60–69.9, and ≥70 years). An examination of the survival curves for normal hearing indicates that the largest difference between the curves for the different blood pressure levels during the follow-up period occur in the men aged 50–59.9 and 60–69.9 years. There is also a precipitous drop in the survival curves for the men under age 50 at the end of the follow-up period and a somewhat lesser drop in the ends of the survival curves for the 50–59.9 age group. These declines result because events of hearing loss are occurring more frequently in the smaller interval at the end of the follow-up period and, subsequently, estimates of the percentage with normal hearing are computed at each occurrence of these events. As might be expected, the oldest age group (≥70 years) had the fastest decline of those with normal hearing level, with

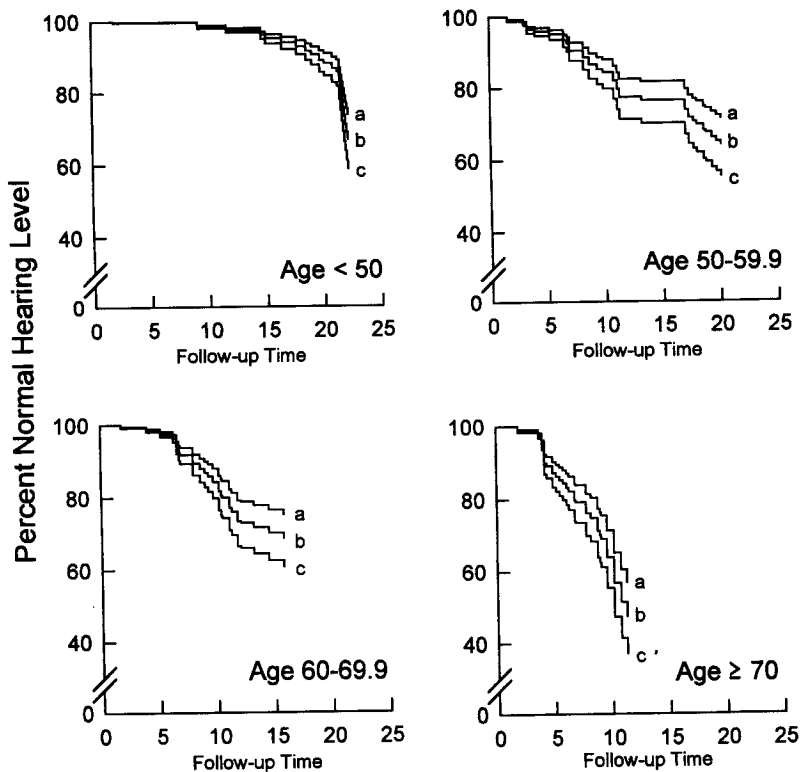


Figure 4 Probabilities (%) of maintaining normal hearing at systolic blood pressure levels of 120 (a), 140 (b), and 160 (c) mm Hg.

only 50 percent continuing to have normal hearing after about 10 years of follow-up. Thus, there is evidence that high systolic blood pressure is associated with age-related hearing loss in the speech frequencies, while there is no evidence from this study that either cigarette smoking or alcohol consumption have an association.

## DISCUSSION

The findings from the longitudinal follow-up study reported in this paper provide evidence to suggest that blood pressure may have an effect on auditory thresholds in the speech frequencies. Age-adjusted analyses of a population of men with no evidence of noise-induced hearing loss or other hearing-related disorders showed a significant association between systolic blood pressure and hearing loss with a 32 percent greater risk of developing hearing loss for a 20 mm Hg increase in systolic pressure. Age-stratified survival curves for hearing loss show different time trends for the different age groups in the study. Figure 4 shows that, although the onset of hearing loss occurs at different points of follow-up for each of the four age groups, blood pressure has a significant association with the onset of hearing loss at all ages.

Other studies that have examined the relationship between cardiovascular-related factors and hearing loss have not come to any general consensus. Rubinstein et al (1977), Makishima (1978), and Gates et al (1993) have reported an association between cardiovascular disorders and hearing loss. Rubinstein et al (1977) found hearing loss to be greater in elderly individuals (aged 65–85 years) suffering from cardiovascular disease than in those who were apparently healthy, while Makishima (1978) found a relationship between arteriolar sclerosis and presbycusis. Gates et al (1993) reported that pure-tone hearing levels were significantly related to cardiovascular disease in an older cohort of men (with mean and standard deviation  $72.7 \pm 6.2$  yrs) and women ( $73.0 \pm 6.0$  yrs) participating in the Framingham Heart Study. On the other hand, studies reported by Hansen (1968) and Pykko et al (1988) present somewhat different findings. On the basis of comparing averaged audiograms of subjects 45 years and older with normal blood pressure to those with elevated blood pressure, Hansen concluded that neither increased systolic nor diastolic blood pressure affected hearing ability. The method of data analysis may, however, have obscured individual variations in the data. Pykko et al (1988)

also found that diastolic and systolic blood pressure did not contribute significantly to the development of sensorineural hearing loss in a large sample of forest workers. The primary factors identified in this study of workers in a noisy environment were age and noise exposure.

In the present study, no evidence was found to suggest an association between cigarette smoking or alcohol consumption and age-related hearing loss in the speech frequencies. These findings agree with those reported by Pykko et al (1988) and Cunningham et al (1983), even though the latter study noted a trend toward a relationship between cigarette smoking and hearing thresholds. In addition, Zelman (1973) found that smokers exhibited poorer high-frequency pure-tone thresholds than nonsmokers of comparable age. The choice of frequencies for defining an event in the present study may have obscured an observation of an effect of smoking on hearing loss since the events were not based on high frequencies. Thus, this study suggests that smoking is not a strong risk factor for hearing loss in the speech frequencies, but we cannot rule out the possibility that there is an association between high-frequency hearing loss and smoking behavior.

Alcohol consumption also did not appear to be associated with age-related hearing loss in the speech frequencies in this risk factor follow-up study. Previous studies reported effects of chronic alcoholism on the latencies of auditory evoked potentials (Begleiter et al, 1981) and of ethyl alcohol ingestion on acoustic reflex thresholds measured immediately after ingestion (Cohill and Greenberg, 1977). The case histories of our subjects indicate that most of them (approximately 85%) consumed alcohol at some time. However, their alcohol consumption could not be characterized as chronic alcoholism, nor did they consume alcohol immediately before the audiologic assessment. Thus, a casual history of drinking alcohol does not appear to be a major causative factor for apparent age-related hearing loss in the speech frequencies.

In summary, the present results for men indicate that casual alcohol consumption and cigarette smoking are not strong risk factors for age-related hearing loss in the speech frequencies. However, the results do suggest that elevated blood pressure does present a significant risk for shifts in auditory thresholds independent of the effects of age. Thus, these findings represent the first longitudinal report of a significant modifiable risk factor, other than noise exposure, for hearing loss in men and suggest that preventing



hypertension might contribute to a program for the prevention and treatment of hearing impairment. Finally, we are continuing to collect data on women, so that as our current study sample gets older and there are more events of hearing loss and hypertension, we will be able to analyze this causative relationship between blood pressure levels and hearing loss in women as well.

## REFERENCES

- American National Standards Institute. (1977). *Criteria for Permissible Maximum Ambient Noise During Audiometric Testing* (ANSI S3.1-1977). New York: ANSI.
- Begleiter H, Porjesz B, Chow CL. (1981). Auditory brainstem potentials in chronic alcoholics. *Science* 211:1064-1066.
- Brant LJ, Fozard JL. (1990). Age changes in pure-tone hearing thresholds in a longitudinal study of normal human aging. *J Acoust Soc Am* 88:813-820.
- Cohill EN, Greenberg HJ. (1977). Effects of ethyl alcohol on the acoustic reflex threshold. *J Am Audiol Soc* 2:121-123.
- Cox DR. (1972). Regression models and life-tables. *J Royal Stat Soc B* 34:187-202.
- Cunningham DR, Vise LK, Jones LA. (1983). Influence of cigarette smoking on extra-high-frequency auditory thresholds. *Ear Hear* 4:162-165.
- Gates GA, Cobb JL, D'Agostino RB, Wolf PA. (1993). The relation of hearing in the elderly to the presence of cardiovascular disease and cardiovascular risk factors. *Arch Otolaryngol* 119:156-161.
- Hansen CC. (1968). Perceptive hearing loss and arterial hypertension. *Arch Otolaryngol* 87:119-122.
- Hopkins A. (1990). Survival analysis with covariates. In: Dixon WJ, Brown MB, Engelman L, Jennrich RI, eds. *BMDP Statistical Software Manual, Volume 2*. Berkeley, CA: University of California Press, 769-806.
- Ito H. (1991). Effects of circulatory disturbance on the cochlea. *J Otol Rhinol Laryngol* 53:265-269.
- Kalbfleisch JD, Prentice RL. (1980). *The Statistical Analysis of Failure Time Data*. New York: John Wiley & Sons.
- Kannel WB, Vokonas PS. (1986). Primary risk factors for coronary heart disease in the elderly: the Framingham Study. In: Wenger NK, Furberg CD, Pitt E, eds. *Coronary Heart Disease in the Elderly*. New York: Elsevier, 60-92.
- Kryter KD. (1985). *The Effects of Noise on Man*. 2nd ed. New York: Academic Press.
- Makishima K. (1978). Arteriolar sclerosis as a cause of presbycusis. *Otolaryngology* 86:322-326.
- Miller JM, Ren TY, Nuttall AL. (1995). Studies of inner ear blood flow in animals and human beings. *Otolaryngol Head Neck Surg* 112:101-113.
- Moscicki EK, Elkins EF, Baum HM, McNamara PM. (1985). Hearing loss in the elderly: an epidemiologic study of the Framingham Heart Study cohort. *Ear Hear* 6:184-190.
- National Institutes of Health. (1990). Noise and hearing loss. *NIH Consensus Development Conference Consensus Statement*. Vol. 8, No. 1. Bethesda, MD: Office of Medical Applications of Research.
- Ohlsen KA, Didier A, Baldwin D, Miller JM, Nuttall AL, Hultcrantz E. (1992). Cochlear blood flow in response to dilating agents. *Hear Res* 58:19-25.
- Pearson JD, Morrell CH, Gordon-Salant S, Brant LJ, Metter EJ, Klein LL, Fozard JL. (1995). Gender differences in a longitudinal study of age-associated hearing loss. *J Acoust Soc Am* 97:1-10.
- Pykko I, Koskimies K, Starck J, Pekkarinen J, Inaba R. (1988). Evaluation of factors affecting sensory neural hearing loss. *Acta Otolaryngol (Stockh)* 449:155-158.
- Robinson DW, Sutton GJ. (1978). *A Comparative Analysis of Data on the Relation of Pure-Tone Audiometric Thresholds to Age*. Teddington, Great Britain: National Physical Laboratory, AC 84.
- Rosen S, Bergman M, Plester D, El-Mofty A, Satti MH. (1962). Presbycusis study of a relatively noise-free population in the Sudan. *Ann Otol* 71:727-743.
- Rubinstein M, Hildesheimer M, Zohar S, Chilarovitz T. (1977). Chronic cardiovascular pathology and hearing loss in the aged. *Gerontology* 23:4-9.
- Schoenborn CA, Marano M. (1988). Current estimates from the National Health Interview Survey: United States 1987. *Vital and Health Statistics, Series 10*, No. 166. Washington, DC: Government Printing Office.
- Shock NW, Greulich RC, Andres R, Arenberg D, Costa PT, Lakatta EG, Tobin JD. (1984). *Normal Human Aging: The Baltimore Longitudinal Study of Aging*. Publ. No. 84-2450. Washington, DC: Government Printing Office.
- Ward WD. (1980). Noise-induced hearing damage. In: Paparella M, Shumrick D, eds. *Otolaryngology*. Vol III. Philadelphia: WB Saunders, 1788-1803.
- Zelman S. (1973). Correlation of smoking history with hearing loss. *JAMA* 223:920.