Associations Between Hearing Impairment and Mortality Risk in Older Persons: The Blue Mountains Hearing Study

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PURPOSE: To assess whether hearing loss predicts an increased risk of mortality.

METHODS: The Blue Mountains Hearing Study examined 2956 persons (49+ years) during 1997 to 2000. The Australian National Death Index was used to identify deaths until 2005. Hearing loss was defined as the pure-tone average (0.5–4 kHz) of air-conduction hearing thresholds greater than 25 dB HL. Associations between hearing loss and mortality risk were estimated using Cox regression and structural equation modeling (SEM).

RESULTS: When we used Cox regression, we discovered that hearing loss was associated with increased risk of cardiovascular (hazard ratio [HR] 1.36, 95% confidence interval [CI] 1.08–1.84) and all-cause (AC) mortality (HR 1.39, 95% CI 1.11–1.79) after adjustment for age and sex but not after multivariable adjustment. SEM pathway analysis, however, revealed a greater AC mortality risk (HR 2.58, 95% CI 1.64–4.05) in persons with hearing loss, which was mediated: cognitive impairment (HR 1.45, 95% CI 1.08–1.94) and walking disability (HR 1.63, 95% CI 1.24–2.15). These variables increased mortality both directly and indirectly through effects on self-rated health.

CONCLUSIONS: Hearing loss was associated with increased AC mortality via three mediating variables: disability in walking, cognitive impairment, and self-rated health. It is important to recognize that persons with combined disabilities are at increased risk of cardiovascular and AC mortality.

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INTRODUCTION

Hearing impairment increases with age and was the third most frequent chronic condition reported by elderly persons in the 2002 U.S. National Health Interview Survey (1), affecting between 35% and 45% of persons aged 50+ years (2–5). This common disability is independently associated with many mortality risk markers, including stroke (6), ischemic heart disease (7), diabetes (8, 9), and smoking (10). Hearing impairment is also associated with increased functional, physical and psychosocial impairment (11–13), poorer health-related quality of life (12, 14), increased risk of institutionalization (15), increased risk of falls (16), cognitive impairment (17), increased risk of car collisions (18), and a poorer understanding of one's health and its treatment (19). Despite these associations, there are no reports in which the authors assessed potential pathways between hearing impairment and mortality. Better understanding this association may help guide mortality lowering interventions in persons with hearing loss.

Prospective studies suggest that hearing impairment increases the mortality risk associated with visual impairment (20–23). However, the association with mortality independent of visual impairment is lost after adjustment for co-morbidities and self-reported health status (20, 23–25). The association with mortality has been attributed to the effect of hearing loss on contextual variables such as self-rated health, mood, functional status, and social relationships (20, 23–25). This was determined on the basis of the finding that the association was lost after adjustment for these variables. To our knowledge there have been no reports in which the authors use structural equation...
modeling (SEM) to identify mediating variables between hearing impairment and mortality.

SEM is a modern statistical method that permits modeling of complex relationships that are difficult to estimate with the use of traditional regression techniques (26). SEM facilitates the examination and quantification of direct pathways, plus indirect pathways via intermediate or mediating variables. Estimates for such variables can be summed to determine the total indirect effect of the variable of interest on the outcome. Adding the indirect and direct effects then provides an estimate of the total effect of the variable of interest on the outcome. We aimed, by the use of SEM in an older Australian population-based cohort, to confirm first that no direct link existed between hearing impairment and mortality, and second, to determine whether indirect associations existed through mediating variables associated with increased mortality risk.

METHODS

Study Population

The Blue Mountains Hearing Study (BMHS) is a population-based survey of age-related hearing loss in a representative older Australian community. The BMHS invited participants who attended the second cross-sectional survey of the Blue Mountains Eye Study (BMES II). Persons who moved into the study area or study age group (50 years or older), identified from a repeat door-to-door census in 1992 to 1994 (BMES I, 82.4% participation rate). Of the 3654 residents aged 49 years were conducted during 1997-9 (BMES II). After also including persons identified in the 1999 door-to-door census, 2956 of 3914 invited participants attended the BMHS, giving an overall participation rate of 75.5%.

Medical, alcohol, and smoking histories were determined by interviewer-administered questionnaire at baseline. A history of angina, myocardial infarction, diabetes, hypertension, stroke, or cancer was determined by responses to questions phrased “Has a doctor advised you that you have…?” History of smoking was defined as never, past, or current smoking. Current smokers included those who had stopped smoking within the past year. Weight in kilograms, height in meters, and systolic and diastolic blood pressures were also recorded at baseline. Self-reported hearing loss was determined by an audiologist-administered questionnaire and was defined as a positive response to the question: “Do you feel you have hearing loss?”

The hearing examination included pure-tone air- and bone-conduction audiometry conducted in a sound-treated room by an audiologist with the use of a Madsen OB822 audiometer (Madsen Electronics, Taastrup, Denmark). Hearing thresholds at octave and inter-octave frequencies from 0.25 to 8 kHz (0.25, 0.5, 1, 2, 3, 4, 6, 8 kHz) were measured. Audiometer calibration was conducted regularly and complied with the International Standards Organization protocol 389 (1991).

To identify and confirm persons who died after the baseline examination, demographic information, including surname, first and second names, sex, and date of birth of the 2965 participants were cross-matched with Australian National Death Index data for deaths, to the end of 2005. A probabilistic record linkage package was used via the adoption of a multiple-pass procedure in which both data sets were grouped on the basis of different characteristics (eg, date of birth, name, sex) each time. Matches were divided into exact and nonexact. All nonexact matched records were examined manually and accepted if there was only one nonexact-matched characteristic that was not critical. Information provided by family members during follow-up was also included if the participant was reported to have died on or before December 2005. The International Classification of Diseases, Ninth Revision (28) and International Statistical Classification of Diseases and Related Health Problems, 10th Revision (29) cause of death codes were also obtained. The primary cause of death was used in statistical modeling.

Definitions

Participants were classified as having hypertension if systolic blood pressure was greater than 160 mmHg or if diastolic blood pressure was greater than 100 mmHg or they were
currently using blood pressure-lowering medication. Body mass index (BMI) was calculated as weight in kilograms divided by height in meters squared, with less than 20 defined as low. Disability in walking at baseline was assessed as present if the participant was observed by a trained examiner to have walking difficulties, or used walking aids or a wheelchair. Cognitive impairment was defined as mini-mental state examination score of 24 or less. Alcohol was modeled as no alcohol versus any alcohol. High serum urate was defined as serum urate greater than 0.5 mmol/L. Any hearing impairment was defined as the pure-tone average of air-conduction hearing thresholds greater than 25 decibels hearing level (dB HL) for the pure tone average of four frequencies (0.5, 1, 2, and 4 kHz) in the better ear. Mild hearing loss was defined as less than 25 to 45 dB HL or greater, and moderate-to-severe hearing loss as greater than 45 dB HL for the average of four frequencies (0.5, 1, 2, and 4 kHz) in the better ear.

Statistical Analysis
Statistical analyses were performed by the use of SAS software v9.13 (SAS Institute, Cary, NC) and MPlus (Mutén and Mutén 1998-2008). Simple statistics included student t tests for comparing means and chi-square tests for comparing proportions. Cox regression models were used to estimate hazard ratios (HRs) and 95% confidence intervals (95% CIs). Multivariable-adjusted models included variables found significantly associated with mortality after age adjustment. These were previous history of acute myocardial infarction (AMI), stroke, angina and hypertension, current smoking, BMI, cancer, diabetes, walking disability, high serum urate, alcohol consumption of more than 1 unit per day, cognitive impairment, depression, and self-rated health. A p value of less than .05 was considered statistically significant.

SEM pathway analysis (26) was used to model the relationship between hearing impairment, mortality, and covariables found to be significantly associated with mortality by Cox regression. The model was fit by the use of MPlus (30) with maximum likelihood and Monte Carlo integration methods. Standard errors were calculated by the use of the delta method and HRs obtained from the coefficients by exponentiation. Hearing loss was defined as pure tone average less than 25 dB in the better ear. Covariables used in the model were previous history of AMI, stroke, angina and hypertension, current smoking, BMI, cancer, diabetes, walking disability, high serum urate, alcohol consumption of more than 1 unit per day, cognitive impairment, depression, and self-rated health.

For this model, the cardiovascular (CV) risk factors AMI, stroke, angina, and hypertension were modeled as latent variables. Each mediating variable was adjusted for age, sex, and hearing loss. The multiple potential pathways to mortality are shown in Figure 1. The indirect effect of hearing loss was then calculated for each covariable by multiplying the effects of that covariable on mortality and hearing loss on the covariable. The total indirect effect of hearing loss was then calculated by summing the coefficients of the estimated indirect effects of each mediating variable and then converting to HRs. The total estimated effect of hearing loss on mortality was calculated by summing the coefficients of the indirect and direct effects and then converting to HRs. Models were simplified by removing indirect pathways for individual covariables that were not significant at a p value level of .1.

RESULTS
Ninety-two persons were excluded from analysis as the result of hearing loss from birth (n = 15), otosclerosis (n = 19),
and conductive hearing loss (n = 60). (Two persons had more than one reason for exclusion.) As of December 31, 2005, 403 BMHS participants with detailed cause of death data available. Table 1 shows the distribution of significant mortality risk markers in persons with and without hearing loss. Compared with those with normal hearing, participants with hearing loss at baseline were more likely to be male, older, cognitively impaired, diabetic, or underweight. They were more likely to have a self-reported history of angina, myocardial infarction, stroke, low self-rated health, an observed difficulty in walking or use of walking aids, and reported lower alcohol consumption. There were no significant differences in the proportions of current smokers or persons with a history of cancer between the groups with and without hearing loss.

Table 2 shows age and sex adjusted as well as multivariable adjusted CV and all-cause (AC) mortality rates, stratified by severity of hearing loss, expressed as HRs with 95% CIs. After age and sex adjustment, CV and AC mortality was greater in persons with any hearing loss compared with those with normal hearing (for CV: HR, 1.36; 95% CI, 1.00–1.84 and for AC: HR, 1.39; 95% CI, 1.11–1.75). There was no difference in the association with mortality between any, mild, or moderate-to-severe hearing loss.

### Table 1. Prevalence of mortality risk markers in participants of the Blue Mountains Hearing Study by hearing impairment

<table>
<thead>
<tr>
<th>Mortality risk marker</th>
<th>All subjects, n (%)</th>
<th>Hearing impairment, n (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(n = 2815)</td>
<td>No, a n (%)</td>
</tr>
<tr>
<td>Male</td>
<td>1218 (43.3)</td>
<td>750 (39.8)</td>
</tr>
<tr>
<td>Age, mean (SD)</td>
<td>66.6 (9.3)</td>
<td>63.5 (8.0)</td>
</tr>
<tr>
<td>Current smoker</td>
<td>267 (9.6)</td>
<td>188 (10.0)</td>
</tr>
<tr>
<td>Body mass index &lt; 20</td>
<td>74 (2.6)</td>
<td>36 (1.9)</td>
</tr>
<tr>
<td>Body mass index 20 to 30</td>
<td>2023 (72.4)</td>
<td>1343 (71.6)</td>
</tr>
<tr>
<td>Body mass index &gt; 30</td>
<td>696 (24.9)</td>
<td>497 (26.5)</td>
</tr>
<tr>
<td>Alcohol &gt;1 unit/day</td>
<td>2160 (76.7)</td>
<td>1480 (78.3)</td>
</tr>
<tr>
<td>Diabetes</td>
<td>289 (11.0)</td>
<td>163 (9.3)</td>
</tr>
<tr>
<td>Stroke</td>
<td>119 (4.2)</td>
<td>55 (2.9)</td>
</tr>
<tr>
<td>Angina</td>
<td>291 (10.3)</td>
<td>144 (7.6)</td>
</tr>
<tr>
<td>Previous myocardial infarction</td>
<td>210 (7.5)</td>
<td>114 (6.0)</td>
</tr>
<tr>
<td>History of cancer</td>
<td>314 (11.2)</td>
<td>208 (11.0)</td>
</tr>
<tr>
<td>Disability in walking</td>
<td>194 (6.9)</td>
<td>58 (3.1)</td>
</tr>
<tr>
<td>Cognitive impairment</td>
<td>79 (2.9)</td>
<td>21 (1.2)</td>
</tr>
<tr>
<td>Self-rated health</td>
<td>536 (19.1)</td>
<td>319 (17.0)</td>
</tr>
</tbody>
</table>

aExcludes subjects with hearing loss from birth, otosclerosis or conductive hearing loss.
b<25 dB HL.
cO>25 dB HL.
dFor comparison between normal hearing and hearing impaired.

### Table 2. Association between the severity of hearing loss and mortality by cause after covariable adjustment in the Blue Mountains Hearing Study using Cox regression, expressed as hazard ratio (HR) with 95% confidence intervals (95% CI); the reference group is persons without hearing loss (n = 1886)

<table>
<thead>
<tr>
<th>Cause of mortality</th>
<th>Any hearing loss (n = 929)</th>
<th>Mild hearing loss (n = 635)</th>
<th>Moderate-to-severe hearing loss (n = 294)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Deaths</td>
<td>HR (95% CI)</td>
<td>p</td>
</tr>
<tr>
<td>Age and sex</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>adjusted</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cardiovascular</td>
<td>144</td>
<td>1.36 (1.00–1.84)</td>
<td>0.048</td>
</tr>
<tr>
<td>All-cause</td>
<td>245</td>
<td>1.39 (1.11–1.75)</td>
<td>0.004</td>
</tr>
<tr>
<td>Multivariable</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>adjusted</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cardiovascular</td>
<td>122</td>
<td>1.06 (0.76–1.48)</td>
<td>0.728</td>
</tr>
<tr>
<td>All-cause</td>
<td>208</td>
<td>1.12 (0.88–1.44)</td>
<td>0.363</td>
</tr>
</tbody>
</table>

aExcludes subjects with hearing loss from birth, or with otosclerosis or other causes of conductive hearing loss.
b<25 dB HL.
Adjusted for age, sex, smoking, alcohol, hypertension, home ownership, low body mass index (<20 kg/m²), previous history of diagnosed angina, acute myocardial infarction, walking disability, cognitive impairment (mini mental state examination <24), fair or poor self-reported health, high serum urate.
TABLE 3. The association of hearing loss and all-cause mortality assessed using structural equation modeling and expressed as hazard ratios with 95% confidence intervals (95% CIs) stratified by pathway: reference group is persons with normal hearing.

<table>
<thead>
<tr>
<th>Pathway</th>
<th>Hazard Ratio (95% CI)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total</td>
<td>2.58 (1.64–4.05)</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td>Direct</td>
<td>1.09 (0.84–1.41)</td>
<td>.5083</td>
</tr>
<tr>
<td>Indirect</td>
<td>2.37 (1.58–3.54)</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td>Covariates of indirect pathway</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Disability in walking</td>
<td>1.63 (1.24–2.15)</td>
<td>.005</td>
</tr>
<tr>
<td>Cognitive impairment</td>
<td>1.45 (1.08–1.94)</td>
<td>.0136</td>
</tr>
<tr>
<td>Direct through covariate to survival</td>
<td>1.37 (1.11–1.68)</td>
<td>.0028</td>
</tr>
<tr>
<td>Cognitive impairment</td>
<td>1.25 (1.00–1.57)</td>
<td>.0535</td>
</tr>
<tr>
<td>Through Covariate via Poor Health to Survival</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Disability in walking</td>
<td>1.19 (1.05–1.37)</td>
<td>.0089</td>
</tr>
<tr>
<td>Cognitive impairment</td>
<td>1.16 (1.01–1.32)</td>
<td>.0343</td>
</tr>
</tbody>
</table>

*Excludes subjects with hearing loss from birth, otosclerosis or conductive hearing loss.

However, there was no statistically significant association between hearing loss and CV or AC mortality after multivariable adjustment when Cox regression was used (Table 2). There was no significant associations of hearing impairment with other causes of death (including cancer, cerebrovascular, respiratory, renal, liver, gastrointestinal, injury, neurological, diabetes, and other; data not shown).

Table 3 shows the association between hearing loss and mortality when SEM pathway analysis was used. In contrast to the Cox multivariable adjusted model, SEM identified a significant association between hearing impairment and mortality after confounders were adjusted for. This occurred only via indirect links to mortality through the mediating variables of disability in walking and cognitive impairment. The pathways from disability in walking and cognitive impairment to mortality occurred both directly and indirectly, via a third mediating variable, self-rated health. Figure 2 shows the path model with HR and 95% CIs. However, there was no significant link between mortality and self-rated health that was independent of disability in walking or cognitive impairment. Other covariates, including smoking, alcohol intake, hypertension, home ownership, low BMI (<20 kg/m²), previous history of diagnosed angina or AMI, hypertension, or high serum urate were found not to be significant mediating variables between hearing loss and mortality.

Comparing the results of Table 2 (using Cox regression models) for AC mortality after multivariable adjustment (HR, 1.12; 95% CI, 0.88–1.44, p = .363) and Table 3 (using SEM) the direct association between hearing loss and mortality (HR, 1.09; 95% CI, 0.84–1.41, p = .5083) demonstrates similar risk estimates obtained from the SEM and the Cox regression models for the direct association of hearing loss with AC mortality after multivariable adjustment.

DISCUSSION

Consistent with previous reports using Cox regression, we observed that hearing loss predicted mortality after adjustment for age and sex, but the association became nonsignificant after adjusting for other covariables associated with mortality in the study population (mortality risk markers) (20, 23–25). Using SEM pathway analysis, we found no direct pathway between hearing loss and mortality but identified disability in walking and cognitive impairment as mediating variables for the increased mortality risk associated with hearing loss. These variables acted both directly on mortality and indirectly via a third mediating variable, self-rated health. It is important to point out that the arrows in the path models (Figs 1 and 2) are determined based on biological hypotheses, and that the SEM cannot provide information to determine either the association direction or causality. However, SEM provides estimated HRs incorporating the direct and indirect contribution of the study factor that shared variances with other factors. Our findings from the SEM suggest that hearing impairment is associated with mortality via indirect pathways.

Although the authors of previous studies (20, 23–25) concluded that the association between hearing loss and mortality was likely to be mediated by contextual variables such as self-rated health and functional status, these conclusions were determined on the basis of findings that the association was lost after adjustment for these variables. In a further advancement in this field, our study using SEM has now documented that disability in walking and self-rated health are two mediating factors likely to account for the link between hearing impairment and mortality. To our knowledge, this is the first report to suggest that cognitive impairment may be a mediating variable between hearing loss and mortality.

Our proposed model is supported by previous work. Studies (31–33) suggest that both functional and physical decline as well as cognitive impairment are associated with low self-rated health. Functional and physical impairment, cognitive impairment, and low self-rated health are each independently associated with increased mortality (25, 34–36). In keeping with these findings, our study showed that disability in walking and cognitive impairment are associated with an increased mortality risk, both directly and indirectly via self-rated health. Associations between hearing loss and measures of functional and physical decline have also been reported previously (16, 37–43).
Mechanisms that could explain the association of hearing loss with disability in walking include increased fear of falling, infirmity caused by declining physical and social activities associated with hearing loss—reflecting a decreased ability to seek professional help for hearing impairment (19)—and impaired balance from accompanying decreased vestibular function (16).

Associations between hearing loss and cognitive impairment have also been reported (37, 44–47). These may be explained by sensory underload (lack of intellectual stimulation reducing cognitive ability) (44, 48), attentional demands of sensory measurement (measurement of hearing loss is sensitive to negative age differences in cognitive processes such as sustained attention and discrimination) (44), or some common cause (hearing loss and cognitive function are both measures of the physiological architecture of the brain) (46,49). These reports support our finding that associations exist between hearing loss and cognitive impairment. Our study adds a new dimension to these associations and extends the hearing loss-cognitive impairment association to mortality risk by identifying pathways from hearing loss to mortality through cognitive impairment and disability in walking.

Our finding that hearing loss increased the odds of CV death but not other causes may be explained by the pathways through disability in walking and cognitive impairment. Affected persons are more socially isolated and may be less likely to see their doctor regularly or to have prescriptions for preventive medications filled. They may also have a poorer understanding of their own health issues and its treatments (19). They may have relatively poorer diets and be less able to seek urgent help when needed. They may also be less likely or unable to exercise regularly, leading to lower cardiorespiratory reserve and greater risk of CV death. Other causes of death may either be less susceptible to these associations, or because of fewer events, we may have had insufficient statistical power to detect associations.

Our results also suggest that severity of hearing impairment may not be so important in predicting mortality risk. This is significant as persons with mild hearing loss may not report it or the loss may go unnoticed by treating clinicians, particularly if they have cognitive or functional impairment. This makes identification of at risk groups more difficult. It also raises the possibility of some unidentified common mechanism leading to an increased risk of hearing loss, cognitive and functional impairments and mortality.

The strengths of our study include its large population-based dataset, with high participation and long follow-up period, standardized hearing assessment, use of Australian National Death Index mortality and causes of death data, and detailed data on the health and functional status of participants. Limitations include the possibility that not all potential pathways were included in the model, such as the variables, exercise, diet, and nutrition variables.

This study supports the contention that hearing loss is associated with an increased risk of mortality through mediating variables, including disability in walking and self rated health and identifies cognitive impairment as a further mediating variable between hearing loss and mortality risk. We could not, however, document a gradient effect from the severity levels of hearing loss on mortality risk. It is important for clinicians to recognize that persons with this combination of disabilities are at increased risk of CV and AC mortality so they can implement strategies that may reduce mortality risk.
REFERENCES