Association Between Age-Related Hearing Loss and Stroke in an Older Population

Bamini Gopinath, PhD; Julie Schneider, PhD; Elena Rochtchina, MSc; Stephen R. Leeder, PhD, MD; Paul Mitchell, PhD, MD

Background and Purpose—Very few studies have investigated the association between hearing loss and stroke. A recent article in Stroke reported an increased incidence of stroke among patients with sudden hearing loss over a 5-year follow-up period. Our study aimed to explore this association among subjects with age-related hearing loss from a representative population. Further, we looked at the association between severity of hearing loss and risk of stroke in older persons, acknowledged as a limitation by the authors of the Stroke report.

Methods—The Blue Mountains Hearing Study is a population-based survey of age-related hearing loss conducted during 1997 to 1999 and 2002 to 2004, among participants of the Blue Mountains Eye Study. Pure-tone air conduction hearing thresholds from 0.25 to 8.0 kHz were measured by audiologists. Hearing loss was defined as the pure-tone average of frequencies 0.5, 1.0, 2.0, and 4.0 kHz >25 dB HL in the better ear.

Results—Persons with moderate to severe hearing loss had a significantly higher likelihood of reporting previous stroke (OR, 2.04; 95% CI, 1.20–3.49) after multivariable adjustment. However, moderate to severe hearing loss did not predict incident stroke after 5-year follow-up (OR, 1.14; 95% CI, 0.59–2.23).

Conclusions—We observed a strong cross-sectional association between stroke and moderate to severe hearing loss. However, age-related hearing loss did not increase risk of incident stroke in our cohort. Insufficient study power or differing underlying pathologies of sudden sensorineural hearing loss and typical age-related hearing loss may account for the discrepant findings between these studies. (Stroke. 2009;40:1496-1498.)

Key Words: Blue Mountains Eye Study ■ hearing loss ■ stroke incidence

Recent reports indicate that sudden sensorineural hearing loss may be an early sign of stroke. An article in Stroke by Lin et al investigated the incidence of stroke in persons experiencing sudden sensorineural hearing loss. After adjusting for other factors, the hazard risk for stroke over 5 years was 1.64-fold (95% CI, 1.31–2.07; P<0.001) greater in patients with sudden hearing loss than in controls. This study, however, did not assess the association between stroke and hearing loss severity, and did not adjust for smoking, a documented risk factor for both conditions.

We explored prevalent and incident stroke among older persons with hearing loss (both gradual and sudden) at baseline and 5-year follow-up, among participants of Blue Mountains Hearing Study (BMHS), a community-based cohort. We also investigated severity of sensorineural hearing loss and also included smoking in the multi-variable analyses.

Materials and Methods

The BMHS is a population-based survey of hearing loss conducted during 1997 to 1999 and 2002 to 2004, among participants of the Blue Mountains Eye Study. Methods to ascertain this population are described. During 1992 to 1994, 3654 participants aged 49 years or older were examined (82.4% participation). Surviving participants were invited to follow-up examinations after 5 (1997–1999) and 10 (2002–2004) years, at which 2334 (75.1% of survivors) and 1952 (75.6% of survivors) were reexamined, respectively.

Pure-tone audiometry at both visits was performed by audiologists in sound-treated booths, using TDH-39 earphones and Madsen OB822 audiometers (Madsen Electronics). Hearing impairment was determined as the pure-tone average of audiometric hearing thresholds at 500, 1000, 2000, and 4000 Hz (PTA0.5 to 4KHz), defining any hearing loss as PTA0.5 to 4KHz ≥25 dB HL, and moderate to severe hearing loss as PTA0.5 to 4KHz >40 dB HL in the better ear. Subjects were asked whether their hearing loss had a gradual or sudden onset.

Persons reporting a stroke during this period had medical records cross-checked. Stroke diagnoses used MONICA criteria. Most had either CT or MRI performed. Incident or prevalent stroke was defined if typical clinical symptoms were reported and mostly confirmed by neuroimaging.

SAS statistical software (SAS Institute) was used, including t tests, χ² tests, and logistic regression. Multivariable logistic regression analysis calculated adjusted OR and 95% CI. P<0.05 indicated statistical significance.
Results
Of the 2956 BMHS-1 (first hearing survey) participants, 62 were excluded as they had incomplete audiological or stroke data, and 92 were excluded on the basis that they had conditions such as conductive or middle ear hearing loss, resulting in 2802 BMHS-1 subjects with complete audiological and stroke data. Of these, 921 had hearing loss and 119 reported a history of stroke. Participants who did not report a history of stroke at BMHS-1 and had complete audiological data (n=1394) were included for incident stroke analysis at BMHS-2 (Figure). Of these, 474 had hearing loss and 43 reported incident stroke. Table 1 shows the baseline characteristics of participants involved in incidence analysis.

Table 1. Demographic and Clinical Characteristics of BMHS-1 and BMHS-2 Participants With Complete Audiological and Stroke Data Included in the 5-Year Incidence Analyses

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Hearing Loss (n=474)</th>
<th>No Hearing Loss (n=920)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex, n (%)</td>
<td></td>
<td></td>
<td>0.01</td>
</tr>
<tr>
<td>Female</td>
<td>257 (54.2)</td>
<td>564 (61.3)</td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>217 (45.8)</td>
<td>356 (38.7)</td>
<td></td>
</tr>
<tr>
<td>Age, mean±SD, yr</td>
<td>72.7±7.1</td>
<td>66.1±6.9</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Current smoker, n (%)</td>
<td>31 (6.6)</td>
<td>73 (7.9)</td>
<td>0.35</td>
</tr>
<tr>
<td>BMI, mean±SD, kg/m²</td>
<td>27.2±4.4</td>
<td>27.6±4.5</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Hypertension, grade 2, n (%)</td>
<td>271 (57.3)</td>
<td>465 (50.8)</td>
<td>0.02</td>
</tr>
<tr>
<td>Type 2 diabetes, n (%)</td>
<td>58 (12.2)</td>
<td>78 (8.5)</td>
<td>0.03</td>
</tr>
<tr>
<td>No tertiary qualifications, n (%)</td>
<td>192 (41.7)</td>
<td>304 (34.8)</td>
<td>0.01</td>
</tr>
</tbody>
</table>

Figure. Distribution of BMHS-1 and BMHS-2 participants included for incident stroke analysis. *Participants who completed questionnaires at both hearing studies but had incomplete stroke or audiological data. †Participants excluded because they had conductive hearing loss, middle ear hearing loss, childhood hearing loss, or a history of diagnosed otosclerosis.

The likelihood of reporting stroke was 1.6-fold higher among subjects with than without sensorineural hearing loss (OR, 1.55; 95% CI, 1.01–2.38), but was marginally nonsignificant after multivariable adjustment (Table 2). The odds of reporting stroke increased 2-fold with increasing hearing loss severity, and persisted after multivariable adjustment (OR, 2.04; 95% CI, 1.20–3.49). Compared to subjects without sensorineural hearing loss, those with any hearing loss had no increased stroke risk during follow-up after multivariable adjustment. We also assessed the association between prevalent stroke in persons with any hearing loss who reported gradual decline in hearing function (n=43) compared to persons who reported sudden hearing loss at BMHS-1 (n=6). Participants with sudden hearing loss had 2-fold greater nonsignificant odds of reporting stroke (OR, 2.23; 95% CI, 0.91–5.63) after adjusting for age and sex. At BMHS-2, 13 subjects with stroke history reported gradual loss of hearing, whereas 3 reported sudden hearing loss. The increased odds of incident stroke after follow-up in those reporting sudden or gradual hearing loss at baseline was not significant (OR, 2.44; 95% CI, 0.67–8.94; and OR, 1.01; 95% CI, 0.48–2.12, respectively).

Discussion
We observed a significant association between moderate to severe sensorineural hearing loss and prevalent stroke. However, unlike the study by Lin et al,2 we could not demonstrate that either gradual or sudden hearing loss predicted incident stroke after 5 years in our cohort. In persons reporting sudden hearing loss at baseline, the odds of reporting incident stroke increased 2-fold, but this was not significant.

The disagreement between our findings and those of Lin et al2 may relate to differences in underlying pathologies for
In sudden hearing loss, strong evidence exists for vascular involvement, infection, or autoimmune disease, among other factors contributing to its pathogenesis. Age-related hearing loss is caused by deficits in hair cells, cochlear neurons, stria vascularis, a combination, or aging itself. Age-related hearing loss is also influenced by genetic risk factors, exposure to occupational noise, and toxins. Vascular occlusion has not been clearly established as an underlying cause of age-related hearing loss, in contrast to sudden hearing loss. This may explain why sensorineural hearing loss among older subjects in our study did not predict development of stroke at follow-up. Second, our study power may have been insufficient to detect an association between hearing loss and stroke attributable to the smaller number of surviving participants. Third, the differing age–gender distributions between the 2 studies may account for the discrepant findings. Finally, we acknowledge that 281 (14%) subjects died between BMHS-1 and BMHS-2; this could have introduced survival bias into the analysis and we may have underestimated incident stroke in our population.

In conclusion, we observed a significant cross-sectional association between moderate to severe hearing loss and stroke. Sudden or gradual hearing loss at baseline did not increase risk of incident stroke after 5 years of follow-up, in contrast to findings by Lin et al., possibly reflecting differing underlying pathologies of these 2 types of sensorineural hearing loss, or a different onset of hearing loss compared with physician-diagnosed sudden hearing loss. Our study highlights the need for awareness of these 2 types and to clearly distinguish between them before requesting detailed clinical examinations to identify patients at potential risk of stroke, as suggested by Lin et al.

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Disclosures
B.G. interpreted the data and drafted the article. J.S. revised data critically for important intellectual content. E.R. performed analysis of the data. S.R.L. revised data critically for important intellectual content. P.M. designed the study and directed its implementation and revised it critically for important intellectual content.

References
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