

Relationship of Hearing Impairment to Dementia and Cognitive Dysfunction in Older Adults

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We conducted a case-control study in 100 cases who had Alzheimer's-type dementia and 100 age-, sex-, and education-matched, nondemented controls to evaluate the hypothesis that hearing impairment contributes to cognitive dysfunction in older adults. The prevalence of a hearing loss of 30 dB or greater was significantly higher in cases than in controls (odds ratio, 2.0; 95% confidence interval, 1.2 to 3.4), even when adjusted for potentially confounding variables. In addition, we observed a dose-response relationship in which greater hearing loss was associated with a higher adjusted relative odds of having dementia. Hearing loss was also significantly and independently correlated with the severity of cognitive dysfunction, as measured by the Mini-Mental State Examination, in nondemented as well as demented patients. These results demonstrate an association between hearing impairment and dementia and lend support to the hypothesis that hearing impairment contributes to cognitive dysfunction in older adults.

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HEARING impairment has been hypothesized to contribute to cognitive dysfunction in older adults. The public health ramifications of such a relationship could be significant because the treatment of dementia is usually palliative, and hearing impairment is highly prevalent,^{1,2} frequently undetected,³ and usually treatable.

If hearing impairment does contribute to cognitive dysfunction, one would expect a higher prevalence of hearing impairment in demented persons than in nondemented persons. In addition,

one might expect a dose-response relationship in which progressively greater amounts of hearing loss would be associated with a progressively higher risk of dementia and cognitive dysfunction.⁴ Although previous studies of hearing and cognition in older adults have consistently demonstrated these conditions to be associated, the associations have not always been statistically significant.⁵⁻¹¹ These studies, however, have been criticized for low statistical power, imprecise diagnostic criteria and instrumentation, and failure to control for potentially confounding variables such as age and depression.⁵

This article presents the results of a case-control study that examined the relationship of hearing impairment to dementia and cognitive dysfunction in older adults. The study was designed to have adequate statistical power, diagnostic criteria, and instrumentation to control for known potentially confounding variables.

METHODS

Subjects

The study was conducted from 1985 to 1987. Subjects included 100 cases with dementia and 100 age-, sex-, and education-matched nondemented controls who were outpatients at the Adult Medicine Clinics at Harborview Medical Center and the University Hospital in Seattle, Wash. In addition to primary care, these clinics provide specialized dementia evaluations for which patients are referred from throughout the Pacific Northwest.¹² The sample size was chosen to afford a statistical power of 80% for detecting a twofold increase in the risk of dementia, assuming a 40% prevalence of hearing impairment in controls.^{1,2} Study-eligibility criteria for both cases and controls were as follows: 65 years of age or older; English speaking; education of eighth grade or beyond; pure tone air conduction threshold audiometry test/retest agreement within ± 5 dB at 1000 Hz; and speech reception threshold-pure tone average agreement within ± 10 dB. Potential cases and controls who fulfilled age, sex, and diagnostic criteria were identified from systematic manual and computerized searches of clinic registries, with supplementary telephone calls, if necessary, to determine educational attainment. This pool of potential cases and controls also fulfilled separate eligibility criteria for cognitive performance, as described later herein, and provided informed consent.

Cases who fulfilled eligibility criteria and consented to participate were selected consecutively. Cases met criteria from the National Institute of Neurological Disorders and Stroke/Alz-

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heimer's Disease and Related Disorders Association for the clinical diagnosis of Alzheimer's disease ("probable" classification).¹³ We chose to study Alzheimer's disease because it is the most common dementia and its auditory neuropathology is relatively well described. All cases also had Mini-Mental State Examination (MMSE) scores of 14 or greater.¹⁴ More severely demented cases (MMSE score <14) were excluded, since a high proportion of these were not expected to fulfill the audiometric reliability criteria described previously. Two otherwise eligible demented patients were excluded because they did not fulfill audiometric reliability criteria.

Controls were matched with cases by age (± 2.5 years), sex, and educational attainment (whether or not they had graduated from high school)—the last to control for socioeconomic status and the effect of education on MMSE performance.¹⁵ When more than one patient was identified as a potential control for a given case, the patient to be approached was randomly selected from this pool. To screen for clinically unrecognized dementia, controls were selected for scores of 24 or greater on the MMSE, since this score has high discriminant validity for dementia or delirium.¹⁴ One potential control was excluded from the study on this basis.

Informed consent was provided directly by controls and, for cases, by legal guardians, if available, or "patient advocates" (usually spouses or other family members). Participation rates were 73% (100/137) for potentially eligible cases and 67% (100/150) for controls. The age, sex, and educational levels of participants and nonparticipants were nearly identical (P , not significant) for both cases and controls. For cases, participants and nonparticipants had nearly identical MMSE scores as well (P , not significant). Mini-Mental State Examination scores were usually not available for nonparticipating potential controls.

Data Collection and Instruments

Subjects received complete medical evaluations, including history, physical examination, and laboratory evaluations, by their general internists in the Adult Medicine Clinic. Current diagnostic, medication, and most demographic data were obtained from medical records.

Controls and cases' "advocates" (in nearly all instances, a spouse or other family member) completed a questionnaire regarding other possible risk factors for dementia (family history, head trauma), hearing-aid use, and additional demographic data, including educa-

Table 1.—Characteristics of Demented and Nondemented Patients

Variable	Patients (N = 100 Pairs)	
	Demented	Nondemented
Mean age, y (\pm SD)*	77.1 (6.3)	77.0 (6.3)
% Female*	58	58
High school graduate, %*	71	71
Medical diagnoses, %		
Hypertension	51	40
Arthritis	44	23
Coronary artery disease	31	17
Chronic lung disease	22	13
Mini-Mental State Examination, mean score (\pm SD)	18.6 (5.1)	27.0 (2.3)

*Matching variable.

Table 2.—Prevalence of Potential Risk Factors for Cognitive Dysfunction in Demented and Nondemented Patients

Variable	Patients, % (N = 100 Pairs)		Odds Ratio (95% Confidence Interval)
	Demented	Nondemented	
Family history of dementia	53	23	3.3 (1.7-6.4)
Diagnosis of depression	29	12	2.9 (1.4-6.2)
History of head injury	17	14	1.1 (0.6-2.3)
≥ 3 Prescription medications*	37	64	0.4 (0.2-0.7)
Hearing-aid use	17	13	1.4 (0.6-3.0)

*The mean number (\pm SD) of prescription medications in cases and controls was 2.5 ± 2.1 and 3.7 ± 2.5 , respectively ($P < .001$).

tion and source of primary care. Subjects were administered the MMSE and the Hamilton Rating Scale for depression¹⁶ by study personnel. Participants with hearing aids wore them during administration of the MMSE and the Hamilton Rating Scale. The MMSE score was used as an indicator of the severity of dementia.¹⁷ For study purposes, the Hamilton Rating Scale was used only to quantify depressive symptomatology; diagnoses of depression were based on findings of the patients' regular physicians.

Two certified audiologists performed audiometric evaluations in a sound-isolated audiometric testing suite with a clinical audiometer calibrated to specifications of the American National Standards Institute. Audiologists were not informed of the subject's status as a case or a control. The audiological test protocol included pure tone air conduction, bone conduction, speech reception threshold measurements, and speech discrimination assessment for each ear. In addition, speech reception thresholds and speech discrimination scores were obtained with the patient seated 1 m from a loudspeaker at 0° azimuth, both unaided and with hearing aids, when indicated.

Data Analysis

Mean values (\pm SD) are noted. The degree of hearing loss was defined, ac-

cording to guidelines of the American Academy of Otolaryngology, as a single, weighted, binaural average of monaural air-conducted, pure tone average thresholds at 500, 1000, 2000, and 3000 Hz.¹⁸ (For this, hearing in the better ear is weighted five times greater than hearing in the worse ear.) The crude relative odds (odds ratio) was computed as the ratio of discordant pairs, and McNemar's Test for correlated proportions was used to evaluate the statistical significance of the association in the unadjusted analysis. Univariate case-control comparisons for continuous variables were performed with paired t tests.

Adjusted odds ratios were calculated with conditional logistic regression.¹⁹ A test for trend was performed by testing the statistical significance of a single hearing-impairment variable, scored 1 for 20- to 29-dB losses, 2 for 30- to 39-dB losses, and 3 for losses of 40 dB or greater, using a likelihood ratio test.

For cross-sectional analyses (ie, correlation of auditory acuity with cognitive function within cases or controls), both cognitive functioning and hearing loss were expressed as continuous variables on the basis of MMSE and pure tone average threshold values, respectively. Multivariate analyses within these cohorts were conducted with multiple linear regression.

Table 3.—Prevalence of 30-dB Hearing Loss in Demented and Nondemented Patients (N = 100 Pairs)*

	Demented		Total
	≥30 dB	<30 dB	
Nondemented	29	15	44
	30	26	56
	59	41	100

*Odds ratio, 30/15 = 2.0; 95% confidence interval, 1.2 to 3.4.

RESULTS

Characteristics of the Study Population

The patients' sociodemographic and medical diagnostic characteristics are given in Table 1. Among the potential risk factors for Alzheimer's disease or cognitive dysfunction examined, family history of dementia and diagnosis of depression were significantly more common among cases than controls, and the number of prescription medications was significantly lower among cases than among controls (Table 2). The use of hearing aids was uncommon and did not affect the relative odds for having dementia. In addition, significantly fewer cases (28%) than controls (74%) received their primary care from study clinics (odds ratio, 0.2; 95% confidence interval, 0.1 to 0.3).

Prevalence of Hearing Impairment in Cases and Controls

The mean hearing loss was significantly higher in cases than in controls (31.8 ± 13.2 and 28.5 ± 13.3 dB HL [hearing level], respectively; $P = .03$). The prevalence of hearing impairment at the median level (>30 dB) was significantly higher in cases than in controls (odds ratio, 2.0; 95% confidence interval, 1.2 to 3.4) (Table 3).

We next adjusted for potentially confounding variables on which cases and controls had not been matched, including source of primary care, to control for otherwise unrecognized differences in health status or other selection biases at the clinic level. Hearing impairment remained statistically significantly associated with dementia when controlled both individually and collectively for family history of dementia, diagnosis of depression, number of prescription medications, and source of primary care (Table 4). Based on these data, the proportion of observed cases *potentially* due to hearing impairment (ie, the adjusted "etiologic fraction") would be approximately 32%.²⁰

Dose-Response Relationship of Hearing Loss to Dementia

To determine whether a dose-response relationship between hearing

Table 4.—Relative Odds for Dementia of 30-dB Hearing Loss, Adjusted for Potentially Confounding Variables (N = 100 Pairs)

Variables in Model	Model				
	1	2	3	4	5
Hearing loss ≥30 dB					
Adjusted odds ratio	1.9	1.7	1.9	2.5	2.3
95% Confidence interval	(1.1-3.3)	(1.0-3.3)	(1.1-3.3)	(1.3-4.9)	(1.1-5.2)
Family history of dementia	X*	X*
Diagnosis of depression	...	X*	X*
≥3 Prescription drugs	X*	...	X*
Source of primary care	X*	X*

*X indicates variable included in model.

Table 5.—Risk of Dementia at Various Levels of Hearing Loss

Hearing Loss, dB	Adjusted Odds Ratio*	95% Confidence Interval
Mild (20-29)	1.5	0.4-5.4
Moderate (30-39)	2.2	0.6-7.8
Moderate/severe (≥40)	4.1	1.1-15.8

*Odds ratio was adjusted for family history of dementia, depression diagnosis, number of prescription medications, and source of primary care. Reference odds ratio for normal hearing (<20-dB loss) is 1.0. Trend of increasing risk of dementia for increasing level of hearing loss is statistically significant ($P < .05$).

loss and dementia existed, we divided the study population into four strata of hearing loss (<20, 20 to 29, 30 to 39, and >40 dB HL), which closely approximated the quartiles of hearing loss within the population. After adjusting for potentially confounding variables, we found that the risk of dementia significantly increased as a function of hearing loss (Table 5). Although the risk of dementia was increased for mild (20 to 29 dB) and moderate (30 to 39 dB) hearing loss, it was statistically significant only for moderate/severe (>40 dB) hearing loss.

Association of Amount of Hearing Loss to Severity of Cognitive Dysfunction Among Cases and Controls

The amount of hearing loss was significantly correlated with the severity of cognitive dysfunction in both demented and nondemented patients in univariate analyses ($r = .26$ [$P < .01$] for demented patients; $r = .29$ [$P < .01$] for nondemented patients). These associations remained statistically significant when controlled for potential confounders including age, education, sex, medication use, and depression ($P < .05$ in both demented and nondemented patients).

COMMENT

This study was designed to test the hypothesis that hearing impairment contributes to cognitive dysfunction in older adults. In our study population, we found that hearing impairment was more prevalent in demented than nondemented patients and that the risk of

dementia increased with progressively greater amounts of hearing loss. In addition, hearing loss was correlated with poorer cognitive functioning in nondemented as well as demented patients. These associations were statistically significant even after controlling for potential confounders. Thus, the findings of this study lend support to the hypothesis that hearing impairment contributes to cognitive dysfunction in older adults.

If hearing impairment does contribute to cognitive dysfunction, we do not wish to imply that it might "cause" Alzheimer's disease in a pathophysiological sense; rather, hearing impairment would probably expose or exacerbate the symptoms of dementia, thereby promoting its diagnosis²¹ or resulting in so-called excess disability.²² For example, hearing loss may reduce the input of environmental stimuli and information and, as a result, directly decrease orientation. This effect could be compounded in individuals whose hearing impairment causes social isolation.²³ In addition, hearing impairment is thought to predispose an individual to depression,^{7,9} which itself may impair cognitive functioning.²⁴

Several other mechanisms might explain the relationships we observed. First, hearing impairment could lower measured performance as an artifact of administering the cognitive test verbally,²⁵ resulting in false diagnoses of dementia and underestimation of cognitive functioning. Since the diagnoses of Alzheimer's disease in our study patients were clinically and not pathologi-

cally derived, it is impossible to determine if some cases were falsely diagnosed. However, given the accuracy of current clinical diagnostic criteria for Alzheimer's disease,^{26,28} it seems unlikely that a large proportion of patients would be falsely diagnosed in this manner. In our study, the MMSE was administered by seasoned clinicians who were sensitized to potential testing bias due to hearing impairment. If such a bias occurred under these circumstances, auditory mental status artifact must be insidious and pervasive in clinical practice and research. Moreover, we have previously shown in a subset of these patients that hearing-impaired patients do not achieve higher scores on a written MMSE than they do on the standard, verbally administered MMSE.²⁸ Thus, confounding of mental status testing by hearing impairment is a very unlikely explanation for the findings of this study.

Second, a selection bias could have contributed to these findings if hearing-impaired cases were proportionately more likely to participate than hearing-impaired controls. We cannot directly rule out a bias of this nature since the auditory status of nonparticipants is unknown; however, participants and nonparticipants were very similar demographically. Most important, a bias of this nature would not explain the correlation of hearing loss and cognitive dysfunction that was observed separately within case and control cohorts. Selection bias could also have influenced these results if hearing loss varied as a function of the different referral patterns of cases, most of whom were recruited from a specialty clinic, and controls, most of whom were recruited from a primary care clinic. Multivariate analyses in which we adjusted for the site of primary care, however, indicated that the relative odds of hearing impairment for dementia actually increased after controlling for this variable.

Third, since Alzheimer's disease appears to affect cortical and other central structures involved in processing auditory stimuli,^{30,31} it might also involve peripheral structures, such as the cochlea, whose function is reflected in the test of auditory sensitivity that we employed. This mechanism, however, would not seem to explain the age-independent relationship observed between hearing impairment and cognitive dysfunction in nondemented patients.

These results indicate that clinicians need to be especially alert for the presence of hearing impairment in demented patients. Additional studies are needed to define further the pathophysiological and clinical implications of

these results. In particular, the auditory neuropathology and neurophysiology of Alzheimer's disease need to be better characterized, especially in peripheral structures that affect auditory sensitivity. The relationship of hearing impairment to cognitive functioning in non-Alzheimer's-type dementias also needs to be determined. Finally, clinical trials of hearing aids in hearing-impaired patients, both demented and nondemented, would help determine whether hearing impairment is causally related to cognitive dysfunction. Such studies would have important implications for treatment as well.

Our data suggest that hearing impairment may be an important risk factor for having dementia and cognitive dysfunction. If so, correction of hearing impairment would not "prevent" the pathophysiological progression of dementia, but it could potentially ameliorate the symptoms of dementia. This would make the correction of hearing impairment a promising opportunity for the treatment of cognitive dysfunction in elderly persons, particularly since there currently are no means of altering the pathophysiological progression of common dementias such as Alzheimer's disease.

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