Visual Impairment and Cognitive Dysfunction in Alzheimer's Disease

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Objective: *To determine whether impaired visual acuity is associated with dementia and cognitive dysfunction in older adults.*

Design: *Paired case-control comparisons of the relative frequencies of visual impairment in demented cases and nondemented controls. Cohort analyses of correlation between visual acuity and cognitive functioning in demented cases.*

Setting: *Internal medicine clinics at two academically affiliated medical centers.*

Participants: *Eighty-seven consecutively selected patients ≥ 65 years of age with mild-to-moderate, clinically diagnosed Alzheimer's disease (cases) and 87 nondemented controls matched to the cases by age, sex, and education.*

Measurements and main results: *The prevalence of visual impairment was higher in cases than in controls [unadjusted odds ratio for near-vision impairment = 2.7 (95% CI = 1.4, 5.2); unadjusted odds ratio for far-vision impairment = 2.1 (95% CI = 1.02, 4.3); odds ratios adjusted for family history of dementia, depression, number of medications, and hearing loss were 2.5 (95% CI = 1.1, 10.5) for near-vision impairment and 1.9 (95% CI = 0.8, 4.6) for far-vision impairment]. When further stratified by quartiles of visual acuity, no statistically significant "dose-response" relationship between vision impairment and dementia risk was observed. Among cases, the degree of visual impairment was significantly correlated with the severity of cognitive dysfunction for both near and far vision (adjusted ps < 0.001).*

Conclusions: *Visual impairment is associated with both an increased risk and an increased clinical severity of Alzheimer's disease, but the increased risk may not be consistent with a progressive dose-response relationship. Further studies are needed to determine whether visual impairment unmasks and exacerbates the symptoms of dementia or is a marker of disease severity.*

Key words: *dementia; Alzheimer's disease; cognition; vision disorders; hearing disorders; aged.* J GEN INTERN MED 1991;6:126-132.

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CURRENT STRATEGIES for the treatment of irreversible dementias often focus on the associated medical conditions that can exacerbate dementia symptoms.[1] Visual impairment and hearing impairment merit particular attention in this regard. They are among the most common health problems of older adults[2, 3] and are often treatable. Most importantly, they have a critical role in learning and other cognitive tasks that might affect functioning in dementia.[4]

Recent studies suggest that hearing impairment exacerbates cognitive dysfunction in Alzheimer's disease[5, 6] and, possibly, other types of dementia.[7] A similar effect could be hypothesized for visual impairment. If impairment of vision contributes to cognitive dysfunction in dementia, one would expect it to be more common in demented individuals. One would also expect the association between visual impairment and dementia to be independent of potentially confounding variables. In addition, one would expect the risk and clinical severity of dementia to increase with poorer vision.[8] Thus, we examined the relationship of visual impairment to the presence and clinical severity of Alzheimer's disease in older adults. We also examined whether combined visual and hearing impairment is associated with an increased risk of dementia relative to an isolated impairment.

METHODS

Subjects

Subjects were recruited and data were collected for a case-control study of hearing impairment and cognitive dysfunction in older adults.[5] Subjects included 100 cases with dementia and 100 age-, sex-, and education-matched, nondemented controls. These individuals were outpatients at the Adult Medicine Clinics at Harborview Medical Center and University Hospital in Seattle, Washington. Study eligibility criteria for both cases and controls were age of at least 65 years; English-speaking; eighth-grade or higher education; and ability to complete audiometric evaluation reliably.

Potential cases and controls fulfilling age, sex, and diagnostic criteria were identified from systematic manual and computer 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 status, as described below.

Cases fulfilling eligibility criteria and consenting to participate were selected consecutively. Cases met National Institute of Neurologic and Communicative Disorders and Stroke/Alzheimer's Disease and Related Disorders Association criteria for the clinical diagnosis of "probable" Alzheimer's disease.[9] All cases also had Mini-Mental State Examination scores of 14 or higher.[10]

When more than one potential control matched a given case on the basis of age, sex, and education, the control subject was randomly selected from among the pool of matching potential controls. To exclude controls with clinically unrecognized dementia, controls were restricted to patients with scores of at least 24 on the Mini-Mental State Examination, since this score has high discriminant validity for dementia or delirium.[10] One patient was excluded from the study on this basis.

Seventy percent of patients approached agreed to participate in the study. Participants and nonparticipants were nearly identical in age, sex, and education level for both cases and controls. The mean (± standard error) patient age was 77 (±0.5) years, 58% were female, and 71% were high school graduates.[5] For cases, participants and nonparticipants had nearly identical Mini-Mental State Examination scores as well. Mini-Mental State Examination scores were usually not available for nonparticipating potential controls.

Of the original 100 pairs of cases and controls, vision testing could not be performed on eight cases and seven controls due to logistic problems. In addition, one case was excluded because of inadequate optotypic-symbol recognition in vision testing. Thus, 87 pairs of cases and controls were included in the analyses reported here.

Informed consent was provided directly by controls and, for cases, by legal guardians, if available, or "patient advocates" (usually spouses or other family members). This study was approved by the University of Washington Human Subjects Division.

Data Collection and Instruments

Subjects had received complete medical evaluations, including histories, physical examinations, and laboratory evaluations, by general internists in the Adult Medicine Clinics. 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 (e.g., family history, head trauma), use of hearing aids and glasses, and supplementary demographic data.[5] Subjects were administered the Mini-Mental State Examination by study personnel. Participants with hearing aids and glasses wore them during administration of the Mini-Mental State Examination. The Mini-Mental State Examination score was used as an indicator of cognitive functioning in separate analyses of demented patients.[11] Clinical audiometry was performed as previously described.[5]

Visual acuity was measured with the Snellen and Rosenbaum methods for far and near vision.[12] After confirmation that patients could recognize optotypic symbols, visual acuity was measured separately for each eye. To avoid measurement bias due to increased latency of response in demented patients, vision testers were instructed to obtain a response for each symbol presented before proceeding to presentation of the next symbol.

Data Analysis

Visual acuity was defined as the uncorrected visual acuity in the better eye. Those with visual acuity less than the median in the control group were considered to have visual impairment.

Similarly, corrected visual acuity was defined as the corrected visual acuity in the better eye. Although equal numbers of cases and controls (93% in each group), reportedly owned glasses, unequal numbers of cases (77%) and controls (93%) brought their glasses to study evaluations. This potential selection bias, in our opinion, precluded meaningful case – control comparisons of corrected vision. However, within-group analyses that incorporated corrected vision were performed on cases who brought their glasses or who did not usually wear glasses. For these analyses, "usual" visual acuity was defined as the corrected visual acuity for those patients who usually wore glasses and as the uncorrected visual acuity for those patients who usually did not wear or never wore glasses.

Patients were considered hearing-impaired if their binaural pure-tone average threshold for speech frequencies was > 30 dB HL.[5, 13]

The crude 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 analyses.

Adjusted odds ratios were calculated with conditional logistic regression[14] and are noted with 95% confidence intervals. In analyzing the interaction between visual impairment and hearing impairment, tests of statistical significance for trend were performed with a likelihood ratio test, using consecutive integers as value codes for the successive categories of normal vision and hearing, isolated visual or hearing impairment, and combined visual and hearing impairment.

For separate analyses of severity of cognitive dys-

TABLE 1

Prevalences of Near-vision Impairment in Demented and Nondemented Patients ($N = 87$ Pairs)*

		Demented Patients: Impaired Vision	Demented Patients: Normal Vision	Total (%)
Nondemented Patients	Impaired Vision	20	12	32 (37%)
	Normal Vision	32	23	55 (63%)
	Total (%)	52 (60%)	35 (40%)	87 (100%)

*Odds ratio of discordant pairs, 32/12 = 2.7 (95% CI = 1.4, 5.2).

TABLE 2

Prevalences of Far-vision Impairment in Demented and Nondemented Patients ($N = 87$ Pairs)*

		Demented Patients: Impaired Vision	Demented Patients: Normal Vision	Total (%)
Nondemented Patients	Impaired Vision	35	11	46 (53%)
	Normal Vision	23	18	41 (47%)
	Total (%)	58 (67%)	29 (33%)	87 (100%)

*Odds ratio of discordant pairs, 23/11 = 2.1 (95% CI = 1.02, 4.3).

TABLE 3

Relative Odds for Dementia of Near-vision Impairment Adjusted for Potentially Confounding Variables

	Model 1	Model 2	Model 3	Model 4	Model 5
Adjusted odds ratio	2.6	2.5	2.7	2.7	2.5
95% confidence interval	1.2, 5.6	1.3, 7.2	1.3, 5.4	1.4, 5.4	1.1, 10.5
Family history of dementia	X*				X
Diagnosis of depression		X			X
Number of prescription drugs			X		X
Hearing impairment				X†	X

*X indicates variable included in model.
†Odds ratio for dementia of hearing impairment adjusted for near-vision impairment = 2.3 (95% CI = 1.1, 4.7).

function among cases, both cognitive functioning and hearing loss were expressed as continuous variables on the basis of Mini-Mental State Examination scores and average pure-tone threshold values, respectively. In these analyses, scores from a partial Mini-Mental State Examination, from which the visually dependent "language" section was deleted, were also incorporated. Multivariate analyses of cases were conducted with multiple linear regression.

In all analyses, odds ratios were taken to be estimates of relative risk, and the two terms are used interchangeably. All tests of significance and confidence intervals are two-tailed. Mean values are noted (± standard error).

RESULTS

Prevalences of Visual Impairment in Demented and Nondemented Patients and Relative Risk of Visual Impairment for Dementia

The median values for near-vision acuity of demented and nondemented patients were 20/200 and 20/100, respectively ($p = 0.01$ by sign test). The median values for far-vision acuity of demented and nondemented patients were 20/70 and 20/60, respectively ($p = 0.18$ by sign test). However, the prevalences of both near- and far-vision impairment, as defined by median values in the nondemented group, were significantly higher in demented than in nondemented patients (Tables 1 and 2).

The increased risk of dementia associated with near-vision impairment remained statistically significant after adjusting for previously recognized risk factors for dementia in this population (family history of dementia, depression, number of medications, and hearing impairment) (Table 3).[5] The adjusted relative risk of dementia for far-vision impairment was also increased, but its 95% confidence interval included 1.0 [adjusted odds ratio = 1.6 (95% CI = 0.7, 3.9)].

When stratified by quartiles of visual acuity, progressive dose–response relationships between visual acuity and the adjusted relative risk of dementia were not observed for either near vision or far vision (Table 4). Although the relative risks for trend were modestly increased, they were not statistically significant.

Visual Acuity and Clinical Severity of Dementia

In analyses of demented patients, poorer uncorrected and poorer usual near- and far-vision acuity were significantly associated with poorer cognitive functioning as measured by the Mini-Mental State Examination (Table 5). These associations remained statistically significant when visually dependent portions of the Mini-Mental State Examination were eliminated from the calculations.

TABLE 4

Dose–Response Relationship of Visual Acuity and Relative Risk of Dementia

Visual Acuity	Adjusted Odds Ratio (95% Confidence Interval)*
Near vision	
<20/50	1.0 (Reference)
20/60–20/80	0.5 (0.1, 2.5)
20/100–20/200	1.5 (0.5, 4.7)
≥20/200	1.8 (0.5, 6.1)
Far vision	
<20/40	1.0 (Reference)
20/40–20/60	0.9 (0.2, 3.5)
20/70–20/100	1.5 (0.3, 6.1)
≥20/200	1.5 (0.3, 6.0)

*Odds ratios were adjusted for family history of dementia, depression, number of prescription drugs, and hearing loss. The adjusted odds ratios for trend were 1.4 (0.9, 2.2) ($p = 0.08$) for near-vision acuity and 1.2 (0.8, 1.7) ($p = 0.4$) for far-vision acuity.

Interaction of Visual Impairment and Hearing Impairment in the Risk of Dementia

For both near and far vision, the risk of dementia was greater when visual impairment occurred in combination with hearing impairment than when either visual or hearing impairment occurred in isolation (Table 6). However, the confidence intervals for combined and isolated impairments overlapped. Nevertheless, the models showed statistically significant trends in the progression from no impairment to either impairment to both impairments.

DISCUSSION

These results provide evidence of an association between visual impairment and the risk and clinical severity of dementia. However, they do not provide evidence that the risk of dementia increases progres-

TABLE 5

Relationship of Visual Acuity to Cognitive Functioning in Demented Patients

	Regression Coefficient		
	Unstandardized (SE)	Standardized	p*
Near vision	0.01 (0.002)	0.34	<0.001
Far vision	0.02 (0.004)	0.37	<0.001
"Usual"† near vision	0.01 (0.004)	0.27	= 0.01
"Usual"† far vision	0.02 (0.006)	0.38	<0.001

*Regression coefficients were adjusted for age, sex, education, family history of dementia, depression, number of prescription drugs, and hearing loss. Positive coefficients indicate that poorer visual acuity was associated with poorer cognitive functioning as measured by the Mini-Mental State Examination.

†Usual visual acuity was defined as the uncorrected visual acuity for patients who usually did not wear glasses and as the corrected visual acuity for patients who usually wore glasses.

TABLE 6

Interaction of Visual Impairment and Hearing Impairment in the Risk of Dementia

Type of Impairment	Adjusted Odds Ratio (95% CI)*
Far vision/hearing	
Neither	1.0 (reference)
Either	3.8 (1.2, 12.0)
Both	4.3 (1.1, 16.5)
Near vision/hearing	
Neither	1.0 (reference)
Either	1.5 (0.6, 3.8)
Both	2.8 (0.7, 10.8)

*Odds ratios were adjusted for family history of dementia, depression, and number of prescription drugs. The adjusted odds ratios for trend were 2.1 (1.1, 4.0) ($p = 0.03$) for near vision/hearing and 2.0 (1.02, 3.8) ($p = 0.04$) for far vision/hearing.

sively with greater visual impairment. We found that the prevalences of both near- and far-vision impairment were significantly greater in demented patients than in non-demented patients. Furthermore, we found that the association between near-vision impairment and dementia remained strong after adjustment for family history of dementia, depression, number of prescription drugs, and hearing impairment. These variables are known risk factors for dementia in this population[5] and, thus, potential confounders. In addition, both uncorrected and usual near- and far-vision acuity were significantly correlated with the severity of cognitive dysfunction among demented patients.

Despite the significant correlation of visual acuity to clinical severity of dementia, these results do not provide compelling evidence of a dose–response gradient between visual acuity and risk of dementia. Specifically, although the relative risk of dementia was modestly increased in the two worst quartiles of visual acuity, the relative risk of dementia was about the same or decreased in the second-best as compared with the best quartile of visual acuity. Conceivably, more compelling evidence of dose-responsiveness might exist if patients were divided into a larger number of strata according to visual acuity. Unfortunately, our sample size was not sufficiently large to permit meaningful analyses of this nature. A lack of dose-responsiveness could suggest either that the associations we observed between vision and risk of dementia were statistical artifacts, or that a nonlinear relationship exists between severity of visual impairment and risk of dementia. In the latter case, the risk of dementia attributable to visual impairment might not be apparent until visual acuity deteriorated beyond a specific threshold and would not necessarily increase with worsening visual acuity.

Given their diminished cognitive reserve, demented persons are thought to be particularly vulnerable to added cognitive dysfunction as a result of comorbid conditions. Thus, if hearing impairment and visual impairment independently increase the risk of dementia, it is plausible to hypothesize that they increase risk further when combined. Our results demonstrate that the risk of dementia is increased by combined hearing and visual impairments; however, it is unclear whether the combination is significantly associated with increased risk relative to a single impairment.

The associations we observed provide epidemiologic support for the hypothesis that visual impairment contributes to cognitive dysfunction in Alzheimer's-type dementia.[8] If visual impairment does contribute to cognitive dysfunction in Alzheimer's disease, it is probably through one of two mechanisms. First, visual impairment may increase confusion and other symptoms of dementia directly through disorientation and impaired learning. This would explain the observed relationship between severity of visual impairment and severity of cognitive dysfunction in patients with Alzheimer's disease. By the same mechanism visual impairment would also tend to "expose" dementia by unmasking symptoms and promoting its diagnosis in marginally compensated patients with previously undetected dementia. This phenomenon would explain the increased risk of dementia associated with visual impairment. Second, visual impairment could exacerbate cognitive dysfunction in dementia indirectly if it predisposed to depression and social isolation.[15,16] Because the effect remained when we adjusted for depression, it is unlikely that depression mediates this association.

In addition, the associations we observed could have occurred if Alzheimer's disease impairs visual acuity. Several studies suggest that the retina, optic nerve, brainstem, and visual cortex are affected structurally or physiologically by Alzheimer's disease.[17-23] The impact of these changes on visual acuity is largely unknown. The steps we took to minimize problems in measuring visual acuity due to response latency and impaired central visual processing lessen the likelihood that these associations reflect Alzheimer's disease–related cortical dysfunction.

Furthermore, near-vision acuity is influenced by centrally mediated functions such as convergence, accommodation, and saccadic eye movements. Thus, if these associations occurred solely as a result of impaired central visual processing, one might have expected to observe significant associations for near-vision acuity, but not for far-vision acuity. However, we found that both near- and far-vision impairments were associated with an increased risk of dementia. Although the risk associated with near-vision impairment was more robust, the correlation with severity of dementia was somewhat greater for far-vision impairment.

In addition, these associations could have been influenced by measurement bias of the Mini-Mental State

Examination in that one of its five sections is administered visually.[24] If this occurred, the severity of dementia would appear to be increased in visually impaired demented patients as a measurement artifact. Our separate analyses of non-visually administered portions of the Mini-Mental State Examination, however, render this possibility unlikely. Finally, these results could have been influenced by selection biases. For example, nondemented patients are more likely than are demented patients to be considered acceptable surgical risks for cataract surgery and other invasive ophthalmologic procedures.

In creating our measure of usual visual acuity, we classified cases as users or nonusers of glasses according to histories obtained from their advocates. Since the reliability of these reports is unknown, these data should be interpreted cautiously. However, any inaccuracy in these reports would be expected to obscure, rather than enhance, the significance of the observed associations.

Further studies are needed to determine whether visual impairment exacerbates cognitive dysfunction in Alzheimer's disease or other dementias. Randomized trials of the efficacy of treatment of vision impairment in improving cognitive functioning of demented patients would provide the most definitive test of this hypothesis.[8] However, randomized trials of visual interventions involving untreated or undertreated control groups would be difficult to justify ethically, given the presumed efficacy of such interventions in enhancing sensory function[25] and other aspects of quality of life.[26] Furthermore, studies would be logistically difficult given the very high exposure rates of older adults to glasses and other vision treatment, as well as the difficulty of quantifying the use of glasses by demented persons. In addition, future studies should include more detailed measures of vision, which might help elucidate the nature of these relationships. Furthermore, studies of larger magnitude will be needed to more fully explore the dose-responsiveness of vision and risk of dementia.

These and other results suggest that both visual impairment and hearing impairment occur with increased frequency in Alzheimer's disease.[5] Treatment of these impairments—often challenging in nondemented older adults—is especially challenging in demented patients. The cognitive dysfunction, physical frailty, social isolation, and depleted financial resources that often accompany dementia render acquisition and use of sensory aids, as well as comprehension of sensory stimuli,[27,28] more difficult. For these reasons, systematic efforts to optimize strategies for correcting sensory impairments in demented patients are needed. A better understanding of vision and hearing in dementia would contribute to the development of such strategies. It would also help elucidate the relationships of vision and hearing to cognition in dementia.

In conclusion, these results suggest visual impairment is associated with both an increased risk and an increased clinical severity of Alzheimer's disease. However, this risk may not have a progressive dose–response relationship with visual impairment. These results are consistent with the hypothesis that visual impairment unmasks and exacerbates cognitive dysfunction in dementia. Further work is needed to determine the extent to which visual impairment causes these effects or is a marker of disease severity.

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REFLECTIONS

Finding the Right Place

WHEN I was first out of my residency, it took me a while to realize that the newest and the best were not necessarily the same thing. Medical theory and practice suffered from as many changes in fashion as stylish clothes and gourmet food. My carefully constructed clinical theology began to reveal its deficiencies. Much of what I had learned was either incomplete or just plain wrong.

In nearly 30 years of practice I've had plenty of good experiences, but I also have made a lot of mistakes. The mistakes give me much to think about. That's because I have more years to look back on than I do to look forward to. My personal search continues for the meaning and purpose of my practice as well as my life, which are inseparable in a physician. And that search requires a special place where I can find peace and renewal.

My spiritual refuge is located on beautiful Marrowstone Island, surrounded by the chilly waters of Puget Sound. It is where I go, actually and in fantasy, in good times as well as in hard times. It is where I can best listen to the inner voices that want to speak, including those from within my own soul.

I walk the beach on a winter morning. The Olympic mountains rise in the early light like awakening giants. A heavy north wind rides over the straits and hits me head-on. The sea celebrates with rollicking waves dotted with brant, scoters, and golden-eyes. A bald eagle soars with the updrafts. Two Columbia black-tail deer meander among some fir trees, pause to look me over, and then resume their browsing.

I turn up my coat collar and think of how good the fire would feel inside my log cabin. But for now I belong here, on this beach, where I watch, listen and shiver.

A screaming gull reminds me of a small white-haired women I saw in consultation last year. I don't recall what was wrong with her. She was worried but not sick. After we had talked for a while she started crying. I wondered if I had said something to offend her. As she reached for a tissue, I asked what was wrong. "Nothing," she replied. "It's just that you're the first doctor who has ever listened to my story."

Rain pelts my face and blurs my glasses. For some reason it reminds me of a middle-aged man from the early years of my practice for whom I made the diagnosis of Lou Gehrig's disease. He asked me if I was sure. I said I was. He asked me if it was fatal. I answered yes. He thanked me and said he just wanted to know. I never saw him again nor do I know what happened to him.

As the wind stiffens, another man pops into mind. He asked me the same question in 1968. I gave him two years to live. He has long since forgiven my arrogance, although he has never let me forget it. His quarterly visits to my office are a form of good-natured revenge.

My stomach rumbles from the coffee I downed before I started this walk. I huddle against a gnarled log that has been trapped in the sand and gravel for many years and still holds fast. I wonder if I am doing as well as that log and what stories are buried among the worm holes and beneath the barnacles in both of our lives.

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