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Enhanced stimulus contrast normalizes visual processing of rapidly presented letters in Alzheimer's disease

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Abstract

Deficient perception and cognition in Alzheimer's disease (AD) has been attributed to slow information processing and attentional disturbance, but an additional explanation may be reduced signal strength. In 21 individuals with probable AD, 29 healthy older and 54 younger adults, we enhanced the contrast level of rapidly-flashed masked letters. The AD group reached identification criterion (80% accuracy), but required significantly higher contrast than the control groups. A source of the prevalent masking deficit may be reduced signal strength arising from dysfunction of retina or visual cortex. Increasing stimulus contrast may be an effective means of enhancing cognitive performance in AD.
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1. Introduction

Alzheimer's disease (AD) is a neurological disorder characterized by significant abnormalities in visual perception and cognition, some of which may arise from lower-level visual deficits (reviewed in Cronin-Golomb & Gilmore, 2003). AD patients demonstrate impairments in the spatial and temporal domains on multiple low-level visual tasks (Cronin-Golomb et al., 1991, Cronin-Golomb, Corkin, & Growdon, 1995; Rizzo, Anderson, Dawson, & Nawrot, 2000). It was found in a large sample of AD patients ($N = 72$) that up to 50% of the variance in performance on cognitive tests of object recognition could be accounted for by performance on one test of basic vision, backward masking, with the next best predictor, contrast sensitivity at low spatial frequencies, accounting for up to 33% of the additional variance in performance (Cronin-Golomb et al., 1995). Pattern masking and low spatial frequency contrast sensitivity were also the vision tests on which deficits in AD were the most prevalent, occurring in 59% and 33% of patients, respectively (Mendola, Cronin-Golomb, Corkin, & Growdon, 1995). Impaired contrast sensitivity has been documented in AD using several methods (Gilmore & Levy, 1991; Neargarder, Stone, Cronin-Golomb, & Oross, 2003), suggesting that reduced luminance sensitivity in patients and hence signal strength on tests of perception and cognition may be a common feature that in turn could account for dysfunction on a variety of tasks of basic vision and visual cognition in this disorder. Here signal strength is conceived as the proximal stimulus propagated in the visual system of the observer. An observer with reduced contrast

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sensitivity, such as an AD patient, will experience a weaker proximal signal.

We focused the present study on delineating the reasons for poor performance on a backward masking test because of the sensitivity of this task to AD. The initial studies described above varied the interval between the presentation of the stimulus and the visual pattern mask, thereby measuring possible slowing of information processing rather than reductions in signal strength. Reduced signal strength alone, however, would lead to poor performance on this type of task. For example, Hellige, Walsh, Lawrence, and Prasse (1979) demonstrated that masking magnitude increased as the stimulus energy in the target was reduced relative to the mask energy. The ubiquity of the contrast sensitivity deficit recorded in AD has prompted us to consider whether signal strength may be especially important to understanding the masking deficit.

This proposition follows from the findings of Gilmore, Seone, Thomas, and Xue (1995), who hypothesized that because light sensitivity declines with age (McFarland, Domey, Warren, & Ward, 1960), impaired performance by older adults on a masking task might be the result of reduced luminance sensitivity rather than slowed processing. On a backward masking task using fixed luminance for target and mask, increasing age (young, middle-aged, and older adults) was associated ($r[55] = .87$) with an increase in the interstimulus interval required to achieve a criterion level (75%) of target identification accuracy. When the interstimulus interval was held constant but the target luminance was increased until participants met a specified criterion level of accuracy, the older adults yielded the same masking magnitude as the young adults. This result suggested that it is the age-related decrease in luminance sensitivity and not slowed information processing that leads to impaired performance on the masking test. The interaction of sensory and cognitive or attentional factors may be particularly important in understanding masking effects in aging populations (Atchley & Hoffman, 2004), including those with the additional visual and cognitive compromise conferred by neurodegenerative disease.

Reduced signal strength may account for disrupted masking performance by AD patients. Changes in contrast sensitivity may result in degradation of the initial percept of the target and consequent impaired ability to detect it. The visual signal, already degraded, would be quite vulnerable to interference from the mask. Further, the onset of AD occurs in later life when even healthy adults experience an age-related decline in light sensitivity (e.g., Eisner, Fleming, Klein, & Mauldin, 1987). We have forwarded a similar argument to account for poor masking performance in Parkinson's disease, another age-related neurodegenerative disorder (Amick, Cronin-Golomb, & Gilmore, 2003).

We hypothesized on the basis of the contrast sensitivity deficit in AD that reduced signal strength is a primary factor in performance on tests of backward masking. Moreover, we predicted that enhancing signal strength would normalize AD performance on this type of task across a range of dementia severity.

2. Methods

This project was part of a large dual-site study of vision and cognition in AD. Recruitment and test procedures and analytic methods were standard across the two sites of the study, Boston University and Case Western Reserve University.

2.1. Participants

The study compared the performance of 21 patients with probable AD (10 men, 11 women), 29 healthy elderly control participants (EC) (10 men, 19 women), and 54 healthy young adult control participants (YC) (31 men, 23 women). Analyses (*t*-tests for homogeneous variances) revealed that the AD and EC groups were comparable in age ($t[48] = 1.02$, $p = .31$). Mean age (standard deviation, SD) was 76.1 (6.1) years for AD; 74.4 (5.2) for EC, and 20.4 (3.4) for YC. All three groups were matched for level of education ($F[2,100] = 1.8$, $p = .18$). Mean education level was 15.2 (3.7) years for AD; 14.5 (3.0) for EC; 13.9 (1.6) for YC.

AD patients were recruited through area hospitals and day programs in Boston and Cleveland and all met NINCDS-ADRDA criteria for probable AD (McKhann et al., 1984). All participants were free of confounding conditions such as depression or other psychiatric disorders as well as ocular abnormalities including glaucoma, cataracts, and macular degeneration as determined from medical reports and detailed neuro-ophthalmological examinations. Dementia severity in the AD group was measured by the Mini Mental State Exam (MMSE) (Folstein, Folstein, & McHugh, 1975). Total scores on the MMSE can range from 0 to 30 with lower scores being indicative of more severe dementia. The mean MMSE score (SD) of our sample was 23.5 (3.1) with scores ranging from 17 to 29, indicative of mild to moderate dementia severity.

EC were recruited from local communities or were caregivers of AD patients. All were free of any signs of dementia (MMSE mean 28.9, SD 1.1). YC were undergraduates at Boston University or Case Western Reserve University and participated as a voluntary experience in one of their courses. All EC and YC participants were free of ocular or other medical abnormalities as determined by health history screening.

No differences in task performance were noted between the Boston and Cleveland samples, and data were accordingly collapsed across sites for analysis.

2.2. Procedure

2.2.1. Acuity

Binocular central acuity was measured using the Lighthouse Near Visual Acuity Test (2nd ed., New York). The letter chart was given at a distance of 16 in., the same distance at which the masking test and chart contrast sensitivity tests were administered. Participants used their own refractive correction. All participants had acuity equal to or better than 20/50 (0.40 LogMAR).

The median acuity score for the AD group was 20/32 (0.20 LogMAR); for the EC group, 20/32 (0.20 LogMAR), and for the YC group, 20/16 (−0.10 LogMAR). Comparison of the frequency of acuities for the AD and EC groups revealed no differences in the distribution of acuities at the level of 20/25 or better vs 20/32 or worse ($\chi^2 = .51$, df = 1, $p = .47$).

2.2.2. Backward masking test of contrast sensitivity

We assessed contrast sensitivity with a backward masking test. We have found such tasks to be very sensitive to AD (Cronin-Golomb et al., 1995) and a more sensitive measure of contrast sensitivity than are standard charts in widespread clinical use. We developed a task that uses a ZEST procedure (Xue, Thomas, Gilmore, & Wilson, 1998) to determine thresholds. ZEST permits the reliable determination of a threshold in relatively few trials, which is advantageous when testing individuals with AD who may suffer from fatigue or inattention earlier in a testing session than their healthy counterparts.

In this test, participants identified briefly presented letters that were followed by a masking stimulus. Letter stimuli appeared on the screen of a Mac G3 computer. Participants were dark adapted for 10 min and the task was performed in a darkened room. The monitor was viewed binocularly from a distance of 16 in. Each of the four target letters H, O, T, and X was .475 in. in height and subtended 1.7° of visual angle. Letters were displayed within a box measuring 256 × 256 pixels, subtending 10.6 by 10.6° of visual angle. This box functioned as a background and was held at a constant 6.9 cd/m^2. Stimuli were presented on the screen for 12 ms followed by a constant interstimulus interval of 59 ms, followed by a visual mask for 506 ms. The luminance of the mask was 25.7 cd/m^2. Stimulus timing was synchronized to the monitor's refresh signal. The visual mask consisted of the target letters H, O, T, and X presented randomly in the mask such that the letters overlapped and filled the entire field. The participant was given the four-choice task to name out loud the letter flashed on the screen. A template with the four target letters was displayed below the monitor to reduce demands on memory for the letter set. The examiner recorded the verbal responses by keyboard.

The luminance of the target letters was varied using a ZEST procedure to establish the luminance required to achieve 80% target identification accuracy. A 2.2 gamma function was used to relate gray level to display luminance. The minimum luminance for this task was 6.9 cd/m^2 and the maximum luminance was 114.8 cd/m^2. Contrast levels were calculated using the Michelson contrast formula, (max Lum − min Lum)/(max Lum + min Lum), where max Lum equaled the luminance of the target and min Lum was the luminance of the constant background.

The masking test was divided into five subtests. In the practice subtest, participants were administered 20 trials and the target letter was presented at the contrast of 85%. This subtest ensured that the participant understood and could perform the task. For each of the subsequent subtests, the only parameter that changed was the contrast level of the target stimulus. The second practice subtest served to orient the participant to the process of threshold measurement. In this task, the target contrast required for the participant to achieve an error rate of 20% (80% accuracy) was determined using the ZEST staircase procedure. The stopping criterion in the threshold estimation was a standard error of 20%. The final threshold estimate was determined on a third subtest that used a stricter stopping criterion of 15% standard error to once again determine the target contrast level required for participants to achieve an error rate of 20%. The latter threshold estimate was used as the estimate of the backward masking threshold. The range in number of trials to obtain thresholds was about 46–48 for each group. Within the same subtest, there followed an immediate presentation consisting of 20 trials presented at the participant's final threshold level to ensure that the threshold estimate was reliable. The fourth subtest consisted of 20 trials presented at the mean YC contrast threshold (14.96% Michelson) collected from a pilot study, in order to estimate differences in performance across groups on a comparable task. Finally, all participants were presented with 10 trials at the maximum contrast of 91% in order to ensure no changes in baseline from the practice task.

2.2.3. Chart test of contrast sensitivity

The Functional Acuity Contrast Test (FACT) was used to assess static contrast sensitivity (Ginsburg, 1996). Although not as sensitive as the masking test, the chart test is used in clinical and research settings and we employed it to demonstrate the comparability of our sample to others reported in the literature.

The participants viewed the chart binocularly from a distance of 16 in. The chart displayed a 9 by 5 array of

circles, the diameter of each circle subtending 1.7° of visual angle. Standard procedures associated with this test were followed. The lighting for the chart was within the recommended luminance of 68–240 cd/m^2. Contrast decreased monotonically in nine steps from left to right with a range of .602 to 2.255 (.59%–25% Michelson contrast), and a log step increment range of 0.109 to 0.176 (SD = .014).

Moving down a column, the gratings increased in spatial frequency, including 1.5, 3, 6, 12, and 18 cycles per degree. In each circle, the gratings were oriented either vertically, tilted 15° to the left or 15° to the right. The participant's task was to indicate verbally or by hand posture the direction in which the lines were oriented. A contrast level was determined for each spatial frequency by finding the minimal perceptible contrast level needed to correctly identify the orientation of the grating for a given row.

3. Results

3.1. Masking

Differences among groups on the masking task were analyzed using a one-way between-group analysis of variance (ANOVA). Because preliminary analyses revealed no gender differences in masking thresholds for any of the three groups, we collapsed results across gender for subsequent analyses. The groups differed significantly for the contrast required to perform the masking task at the 20% error rate ($F[2,101] = 132.3$, $p < .001$) (Fig. 1). AD participants required a mean contrast of 61% (SD 19) whereas the EC and YC groups required a mean contrast of 46% (SD 16) and 14% (SD 5), respectively, to perform the task at the criterion error rate.

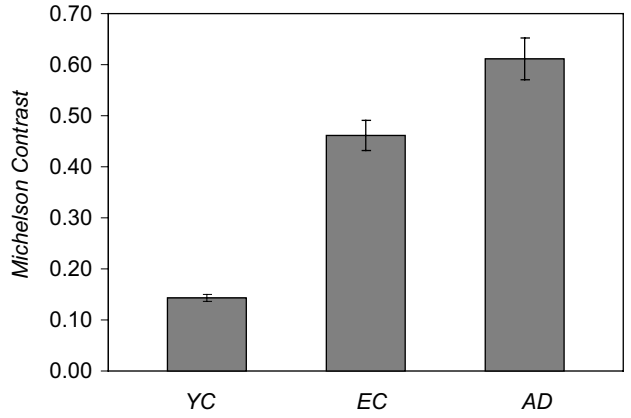

Fig. 1. Mean backward masking thresholds plotted as a function of group. Error bars represent the standard error of the mean. The EC group required significantly higher contrast in order to reach the 80% criterion level when compared to the YC group (a difference of 32%). The AD group required even higher contrast when compared to the EC group (a difference of 15% more).

A priori comparisons using independent groups *t*-tests revealed significant differences between the YC and EC groups ($p < .001$) and between the EC and AD groups ($p = .004$).

To check the reliability of the threshold measure, the participants were given 20 identification trials at their own threshold contrast level. There was no significant difference among the number of errors made by the AD, EC, and YC groups when each person was given stimuli at their own contrast threshold ($F[2,100] = 2.4$, $p = .10$). The AD group had a mean of 75% correct (SD = 17), the EC group had a mean of 83% correct (SD = 15), and the YC group had a mean of 84% correct (SD = 16).

AD masking performance, in percent contrast required for letter identification at criterion, correlated with dementia severity as assessed by the MMSE ($r[19] = -.67$, $p = .001$) and with binocular near acuity (Spearman's $\rho[19] = .50$, $p = .02$). EC masking performance did not correlate with binocular near acuity ($\rho[27] = .11$, $p = .59$). When we eliminated data from individuals with acuity worse than 20/32 (9 AD, 3 EC), the percent contrast required to perform the task at criterion remained the same for the EC (46%, SD 17) and was somewhat reduced from the full-group result for the AD (53%, SD 19). There was no correlation between number of errors and AD dementia severity ($r[18] = -.04$, $p = .88$). There was no correlation between masking thresholds and age for AD ($r[19] = .07$, $p = 0.78$) or YC ($r[52] = .21$, $p = .12$) whereas there was a strong correlation for EC ($r[27] = .39$, $p = .04$).

Of the 21 AD patients, 16 were on cholinergic medications (donepezil, 14; rivastigmine, 1; galantamine, 1), 7 were on statins, and 5 were on non-steroidal anti-inflammatories (NSAIDs). There were no significant differences in masking thresholds for medicated versus non-medicated groups with respect to cholinergics ($t[19] = -.15$, $p = .88$), statins ($t[19] = 1.66$, $p = .11$), or NSAIDs ($t[19] = 1.98$, $p = .06$).

3.2. Contrast sensitivity chart

A mixed design ANOVA with one between-subjects variable (Group) and one within-subjects variable (Spatial Frequency) was conducted to analyze YC, EC, and AD's performance on the FACT assessment. Because a violation of the sphericity assumption was noted, the Huynh–Feldt correction was applied to the data ($\varepsilon = .70$). Results revealed a main effect of group ($F[2,91] = 91.3$, $p < .001$), a main effect of spatial frequency, which was expected because normal contrast sensitivity varies according to spatial frequency ($F[2.8,253.7] = 447.1$, $p < .001$), and a significant interaction between group and spatial frequency ($F[5.6,253.7] = 37.6$, $p < .001$) (Fig. 2). This significant interaction resulted from differences in performance between the YC and EC groups. Removal of the YC group

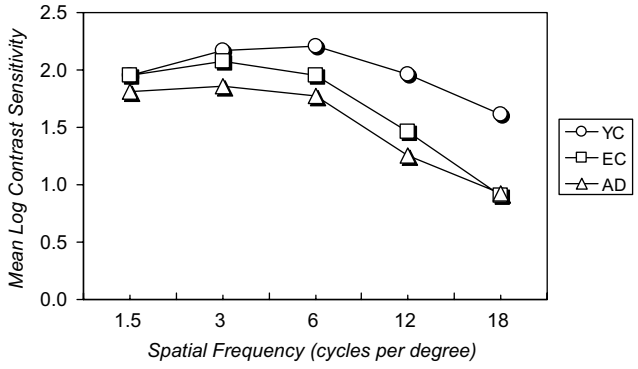

Fig. 2. Mean log contrast sensitivity for the FACT assessment plotted as a function of spatial frequency for the YC, EC, and AD groups. Comparison of the EC and AD groups revealed a significant difference at 1.5 cycles per degree.

from the analysis eliminated the interaction effect but the main effect of group remained ($F[1,38] = 5.27$, $p = .027$). *A priori* contrasts performed via independent groups *t*-tests using the Bonferroni correction ($\alpha = .05/5 = .01$) were conducted to examine differences between the AD and EC groups at each level of spatial frequency. A significant difference between groups was noted for the 1.5 cpd ($p < .001$) FACT condition, with AD having poorer contrast sensitivity than EC at this spatial frequency.

There were significant correlations of performance on the masking test with performance on the FACT at several spatial frequencies. Alpha was adjusted using the Bonferroni correction (.05/5 = .01) to account for multiple comparisons. For the YC group, masking thresholds were correlated with the 12.0 cpd FACT condition ($r[52] = -.43$, $p = .001$). For the EC group, a correlation was noted for the 18.0 cpd condition ($r[24] = -.48$, $p = .01$). For the AD group, correlations were noted for the 3.0 cpd ($r[19] = -.72$, $p = .00$), 6.0 cpd ($r[18] = -.54$, $p = .01$), and 12.0 cpd ($r[14] = -.67$, $p = .01$) FACT conditions. It should be noted that the correlations were likely affected by the number of participants performing at ceiling level on the FACT at the several spatial frequencies, as we discuss elsewhere (Morrison, Gilmore, & Cronin-Golomb, 2004). In general, more YC performed at ceiling than did EC, and more EC than did AD patients. The number performing at ceiling for each spatial frequency was as follows. For YC: 1.5 cpd 41/54, 3.0 cpd 43/54, 6.0 cpd 37/54, 12.0 cpd 16/54, and 18.0 cpd 21/54. For EC: 1.5 18/24, 3.0 10/24, 6.0 3/24, 12.0 1/24, and 18.0 1/24. For AD: 1.5 6/16, 3.0 3/16, 6.0 2/16, 12.0 1/24, and 18.0 0/24.

4. Discussion

The results of this study confirm the hypothesis that individuals with AD can perform normally on a backward masking task of letter identification when the contrast level of the target stimulus is enhanced. The AD group required significantly higher contrast levels to perform the masking task at criterion level than did the control groups. At these adjusted contrast levels, AD patients performed at the same level of accuracy as both control groups, indicating that neither slowed information processing nor a general cognitive deficit was paramount to understanding their poor initial performance. Apparently the vision-related changes that accompany AD can override the normal age-dependency of contrast sensitivity changes as shown with our younger and older control groups. Acuity was correlated with performance on the masking test for the AD group, and we have shown that even subtle group differences in acuity (AD vs. healthy elderly) can affect performance of tests of contrast sensitivity (Neargarder et al., 2003).

The pattern of results from the EC relative to the young adults was similar in kind to the pattern of results from the AD patients relative to the EC, in that the original group differences on the masking test disappeared when each person was given letters to identify at their own contrast threshold. Under this condition, the EC results (mean 83% correct, SD 15) were virtually indistinguishable from the YC results (mean of 84% correct, SD 16). This elimination of the aging effect is consistent with our findings with young and elderly adults (Gilmore et al., 1995), in which we obtained the same mean group thresholds when target luminance was increased until individuals met a specified criterion level of accuracy. In that study, we demonstrated an aging effect when we varied the interstimulus interval between target and mask but removed the effect, when keeping the interstimulus interval constant, by varying signal strength. The earlier study provided direct evidence that the age-related decrease in luminance sensitivity was more important than slowed information processing in explaining performance on the masking test. Although we did not vary interstimulus interval in the present study and so cannot make equivalent claims about the relative importance of speed of processing and luminance sensitivity to masking performance, the results of both studies are quite consistent in demonstrating that enhancing signal strength alone is sufficient to eliminate the normal aging effect on this type of task. Moreover, in the present study we have shown that enhancement of signal strength eliminates not only the age effect, but also the dementia effect.

Masking requires both retinal and cortical processing (Atchley & Hoffman, 2004; Bowen & Wilson, 1994), and deficits in performance on masking tasks may result from dysfunction at the retinal or cortical level in AD. Parkinson's disease (PD), an age-related neurodegenerative disorder arising from abnormalities in brain dopamine levels, is also associated with reductions in dopamine levels in the retina (Harnois & DiPaolo,

1990; Nguyen-Legros, Harnois, DiPaolo, & Simon, 1993). Neurochemical changes in the retina have been used to explain the changes in contrast sensitivity often noted in PD (Bodis-Wollner & Paulus, 1999), including deficient performance on the same masking test described in the present AD study (Amick et al., 2003). Alterations in dopamine levels have been linked to visual abnormalities in a range of disorders, including amblyopia (Gottlob, Charlier, & Reinecke, 1992), cocaine withdrawal (Desai, Roy, Roy, Brown, & Smelson, 1997), phenylketonuria (Diamond & Herzberg, 1996), and schizophrenia (Calvert, Harris, & Phillipson, 1992; Phillipson & Harris, 1985; Shuwairi, Cronin-Golomb, McCarley, & O'Donnell, 2002). AD likewise is associated with reduced dopamine (reviewed in Grossman, 1993; Joyce, 2001). It is noteworthy that dopamine receptors are found in the occipital lobe (Parkinson, 1989; Phillipson, Kilpatrick, & Jones, 1987; Rakic & Lidow, 1995) as well as the retina, in light of the significant neuropathology of visual association cortex and anterior visual structures in this disorder (reviewed in Cronin-Golomb & Gilmore, 2003; Valenti, 2004). Relatively unexplored is the role of the known reductions of acetylcholine in visual dysfunction in AD, though cholinergic neurons and postsynaptic receptors have been found in the retina, lateral geniculate nucleus, and visual cortices (Nobili & Sannita, 1997).

Dorsal stream dysfunction has been implicated in deficient performance on masking tasks (Husain, Shapiro, Martin, & Kennard, 1997; Saccuzzo, Cadenhead, & Braff, 1996). In AD, multiple aspects of dorsal stream function are impaired, including motion and optic flow perception (Mapstone, Steffenella, & Duffy, 2003; O'Brien, Tetewsky, Cushman, Makous, & Duffy, 2001; Tetewsky & Duffy, 1999), and several domains of visuospatial function supported by the parietal lobes (reviewed in Cronin-Golomb, 2001; Cronin-Golomb & Amick, 2001; Cronin-Golomb & Gilmore, 2003). In the present study, we used letter-identification masking, thereby presumably assessing the function of the ventral stream as well. Object and pattern discrimination deficits are common in AD, including difficulties in reading letters and words (reviewed in Cronin-Golomb, 2001). Besides impairments at lower levels of the visual system in AD (reviewed in Valenti, 2004), there is direct disruption of the occipito-temporal pathway important for word and object recognition (Arnold, Hyman, Flory, Damasio, & Van Hoesen, 1991; Braak, Braak, & Kalus, 1989; Brun & Englund, 1981; Hof & Bouras, 1991; Lewis, Campbell, Terry, & Morrison, 1987; Pearson, Esiri, Hiorns, Wilcock, & Powell, 1985; Thompson et al., 2003). There is a 20-fold increase in neurofibrillary tangle density, one of the pathological hallmarks of AD, between primary and parastriate visual cortex (Brodmann area 18) and a further doubling in inferotemporal cortex (area 20) (Lewis et al., 1987). Abnormalities of both the parvocellular and magnocellular input pathways to the cortical visual processing streams are evident in AD (Cronin-Golomb, 2001; Gilmore, Morrison, & Groth, 2004; Kurylo et al., 1994).

In a previous study (Cronin-Golomb et al., 1995), it was found that performance on a letter-identification masking task was the best basic-vision predictor of performance on several object-associated tests in AD. In this case, we varied the length of the interval between target and mask, rather than varying signal strength. Masking performance accounted for 25–50% of the variance in performance on tests of incomplete-picture identification, word reading, picture arrangement, color naming, complex figure copying, and pattern completion. It did not account significantly for variance in performance on tests of spatial localization, though there were fewer such tests administered and therefore the domain was not as well sampled as the object recognition domain. The ability of masking performance to predict cognitive abilities in the domain of object recognition, supported by the occipito-temporal pathway, is important because of the prevalence of these cognitive deficits in AD, which may occur in an individual patient to a greater degree than spatial localization deficits subserved by the occipito-parietal pathway (Kurylo, Corkin, Rizzo, & Growdon, 1996). Impairments in visual cognition in turn predict functional disability in AD (Glosser et al., 2002).

The results of the present study indicate that slowing of information processing or other general cognitive impairment does not necessarily account for deficient performance on tests of masking in AD, because when proximal signal strength is enhanced, performance accuracy can be normalized. Although the relative roles of luminance sensitivity and processing speed remain to be specified, it is clear that signal strength makes an important contribution to backward masking performance. These findings suggest that interventions should be targeted to enhancing the strength of the proximal signal. Environmental modifications that are simple to implement include increasing the contrast of reading materials and other aspects of the immediate visual world through enhanced lighting, reduced glare, use of high-quality print, and the adoption of large-typeface text in reading materials (Dunne, 2004). Enhanced contrast has been shown to normalize the speed of letter identification in AD (Gilmore, Thomas, Klitz, Persanyi, & Tomsak, 1996). In recent work, we demonstrated increased ingestion of food and liquid in severely demented AD patients in long-term care through use of enhanced contrast of dining tableware (Dunne, Neargarder, Cipolloni, & Cronin-Golomb, 2004), and increased food intake together with decreased agitation in the same type of patients using high-contrast tableware and amplified lighting levels (Koss & Gilmore, 1998). Our current finding that overall mental status was correlated with level of masking perfor-

mance suggests that implementing interventions based on signal strength may become more important as dementia becomes more severe. It appears likely that a weakened proximal signal may underlie a number of perceptual, cognitive, and behavioral impairments in AD, and that strengthening the visual signal may prove to be a new and important non-pharmacological avenue for cognitive and functional improvement in this disorder.

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