Available online at www.sciencedirect.com
SCIENCE DIRECT®
Vision Research 45 (2005) 1013–1020
Vision Research
www.elsevier.com/locate/visres

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

Grover C. Gilmore [a], Alice Cronin-Golomb [b,*], Sandy A. Neargarder [b,c], Sarah R. Morrison [a]

[a] Department of Psychology, Case Western Reserve University, 10900 Euclid Avenue, Cleveland, OH 44106-7123, USA
[b] Department of Psychology, Boston University, 648 Beacon Street, 2nd floor, Boston, MA 02215, USA
[c] Department of Psychology, Bridgewater State College, Hart Hall, Bridgewater, MA 02325, USA

Received 3 August 2004; received in revised form 7 October 2004

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.

Keywords: Alzheimer's disease; Cognition; Masking; Contrast; Aging

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

* Corresponding author. Tel.: +1 617 353 3911; fax: +1 617 358 1380.
E-mail address: alicecg@bu.edu (A. Cronin-Golomb).

0042-6989/$ - see front matter © 2004 Elsevier Ltd. All rights reserved.
doi:10.1016/j.visres.2004.10.017

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.