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Sustained Attention in Mild Alzheimer's Disease

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Abstract

The vigilance decrement in perceptual sensitivity was examined in 10 patients with mild Alzheimer's disease (AD) and 20 age-matched controls. A visual high-event rate digit-discrimination task lasting 7.2 min. (six 1.2 min blocks) was presented at different levels of stimulus degradation. Previous studies have shown that sensitivity decrements (d′) over time at high-stimulus degradation result from demands on effortful processing. For all degradation levels, the overall level of vigilance (d′) was lower in AD patients than in controls. All participants showed sensitivity decrement over blocks, with greater decrement at higher degradation levels. AD patients exhibited greater sensitivity decrement over time at the highest degradation level they all could perform relative to control participants. There were no concomitant changes in either response bias (C) or response times. The results indicate that mild AD patients have overall lower levels of vigilance under conditions that require both automatic and effortful processing. Mild AD patients also exhibit a deficit in the maintenance of vigilance over time under effortful processing conditions. Although the sample of AD patients was small, results further suggest that both possible and probable AD patients had greater sensitivity decrement over time at the highest degradation level than did control participants, but only probable AD patients had lower overall levels of vigilance. In the possible AD patients as a group, the decrement in vigilance occurred in the absence of concurrent deficits on standard attentional tasks, such as the Stroop and Trail Making tests, suggesting that deficits in vigilance over time may appear earlier than deficits in selective attention.

Alzheimer's disease (AD) is a progressive, degenerative brain disorder, the most usual and early clinical manifestation of which is memory impairment (Baddeley, Della Sala, Logie, & Spinnler, 1986; Morris & Kopelman, 1986). Attentional functions, particularly selective and divided attention, are also affected early in the course of AD (Parasuraman & Haxby, 1993). Although sustained attention has been less extensively studied, this aspect of attention is worthy of further investigation in relation to dementia, for several reasons. First, clinical observations indicate that demented patients have difficulty concentrating and fatigue easily (Lezak, 1995). Altered sustained attention in AD patients may also contribute to their deficits in other cognitive functions. Furthermore, everyday activities such as safe driving (Ball, 1997; Duchek, Hunt, Ball, Buckles, & Morris, 1997, 1998; Hunt, Morris, Edwards, & Wilson, 1993; Parasuraman & Nestor, 1991) and prevention of falls (Buchner & Larson, 1987) may depend on intact maintenance of attention.

Several different experimental paradigms have been used to investigate sustained attention, including signal detection (Bushnell & Rice, 1999), serial reaction (Robbins, 1998), response

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inhibition (Anderson, Fenwick, Manly, & Robertson, 1998), and vigilance tasks (Mackworth, 1948). The Continuous Performance Test (CPT), which has been widely used in neuropsychological studies of brain-damaged patients, can also be considered within the vigilance paradigm (Rosvold, Mirsky, Sarason, Bransome, & Beck, 1956). Of these various experimental paradigms, the most extensive theoretical and empirical work has been done on vigilance (Davies & Parasuraman, 1982). Vigilance refers to the ability to detect small changes occurring at random intervals in the environment (Mackworth, 1948). It is usually tested in repetitive monotonous tasks in which the participant is asked to detect the appearance of infrequent and unpredictable targets. A cardinal aspect of sustained attention as assessed by vigilance tasks is the *vigilance decrement*, or the decline in the detection rate of critical targets with time on task. The application of signal detection theory (Green & Swets, 1966) has further refined the assessment of vigilance decrement by differentiating between a decrement in perceptual sensitivity (d′) over time and a shift in response criterion (β; Parasuraman, 1979). Sensitivity decrement over time occurs in high-event-rate tasks with targets that place a load on working memory (Parasuraman, 1979), on perceptual discriminability (Neuchterlein, Parasuraman, & Jiang, 1983), or both (Parasuraman & Mouloua, 1987). The importance of event rate and working memory as factors contributing to sensitivity decrement has been confirmed in a recent meta-analysis of several vigilance studies (see, Howe, Warm, & Dember, 1995). The sensitivity decrement may reflect limitations in the allocation of processing resources to the monitoring of the visual display and to target discrimination (Parasuraman, 1979).

A few previous studies have examined vigilance in relation to AD (for a review, see Perry & Hodges, 1999). Using the CPT, Alexander (1973) reported that demented patients had lower detection rates than controls. However, this study did not examine vigilance decrement. Three pharmacological studies using versions of the CPT also found that AD patients had lower detection rates than controls, but again did not report data on vigilance decrement (Linesetal., 1991; Sahakian, Jones, Levy, Gray, & Warburton, 1989; Sunderland et al., 1987). Nebes and Brady (1993) tested young, old, and mild to moderate AD patients using a self-paced four-choice reaction-time task, in which participants had to detect the appearance of a square in four different locations on a computer screen. Mean response times (RTs) increased over time on task in all three groups, but the rate of increase in RTs over time was equivalent in the old and mild AD groups. Previous research has shown that vigilance decrement is more likely to occur with low-target probabilities and experimenter-paced tasks (Davies & Parasuraman, 1982). The testing conditions used by Nebes and Brady may therefore not have been optimal to find vigilance decrements. Brazzelli, Cocchini, Della Sala, and Spinnler (1994), using a modification of the Mackworth Jump Clock test (Mackworth,1961), claimed to find evidence for a sensitivity decrement in mild AD patients. However, the results of this study are surprising for several reasons. First, no sensitivity decrement over time was observed for the control group, precluding comparison of the extent of vigilance decrements in the AD versus control groups. Second, and perhaps more seriously, the AD patients as a group performed at chance levels on the two last blocks of test (five of their AD patients detected no target at all on the last block). For these reasons, the Brazzelli et al. study provides only limited evidence of the effects of AD on vigilance decrement. Finally, Baddeley, Cocchini, Della Sala, Logie, and Spinnler (1999) reported a significant vigilance decrement over time in probable AD patients compared to controls. These authors suggested that working memory load may be an important factor in explaining the vigilance decrement in AD patients.

Two measures can typically be derived from vigilance tasks: (a) *Overall vigilance* is dependent on arousal and is influenced by numerous factors, such as time of day, lack of sleep, or fatigue (see Davies & Parasuraman, 1982, for an exhaustive review of the factors influencing overall vigilance). This aspect of vigilance is reflected in the average level of detection performance over the entire duration of a vigilance task; (b) the *vigilance decrement* refers to the

deterioration over time in perceptual sensitivity (Parasuraman, 1986). This more fundamental aspect of vigilance reflects the ability to maintain attention to a critical stimulus as a function of time on task; the task is divided in blocks containing equivalent numbers of trials, and the decrement is calculated as the difference in sensitivity between successive blocks (typically between the last and the first blocks of test).

The aim of this study was to determine whether there are changes in overall vigilance and vigilance decrement over time in mild AD patients compared to age-matched, nondemented controls. We used the paradigm developed by Neuchterlein et al. (1983), which, by using high-event-rate visual stimulus presentation under conditions of moderate to high-stimulus degradation, shows performance decrements in healthy participants after as little as 5 to 8 min. This task has a high perceptual load but no working memory load (Neuchterlein et al., 1983). This study was based on the assumption that when stimuli are degraded visually by adding random noise, perceptual sensitivity (d′) in detecting a critical target shows a rapid decline. Parasuraman (1985) used a dual-task probe RT technique to assess the capacity demands of this task, and found that effortful processing, as assessed by probe RT, increased in the second compared to the first half of the task for degraded stimuli. Hence, decrements in d′ at high levels of stimulus degradation result from demands on the allocation of processing capacity to target discrimination (Neuchterlein et al., 1983). We predicted that mild AD patients would show greater sensitivity decrement under the effortful processing conditions characteristic of high-stimulus degradation. The major rationale was to titrate task difficulty so that AD patients and controls were compared on a wide range of levels of stimulus degradation, thereby increasing the likelihood of finding one level in which neither of the two groups would perform at chance or ceiling, so that their performance could be compared. Hence, five levels of stimulus degradation were used to examine whether there were any changes in overall vigilance or vigilance decrement or both in mild AD. Although we had a small sample of mild AD patients, we also examined whether there were any differences in overall vigilance or vigilance decrement or both in possible versus probable AD patients, relative to each other and to control participants. Although possible and probable AD usually reflect different degrees of diagnostic certainty, some studies suggest that they may also reflect differing degrees of AD severity, with possible AD patients being less impaired than probable AD patients (e.g., Hughes, Perkins, Wright, & Westrick, 2003; Ikeda et al., 1994; Perry, Watson, & Hodges, 2000).

METHOD

Participants

Ten patients with mild AD (6 men, 4 women), and 20 control participants (10 men, 10 women) were studied. There were twice as many control participants as there were mild AD patients because the control group data was available. Although we had planned to test 20 mild AD patients, this goal had to be revised because of the difficulty finding patients that satisfied the strict health selection criteria. Dementia severity was assessed with the Mini-Mental State Examination (MMSE; Folstein, Folstein, & McHugh, 1975). All patients had MMSE scores ≥ 20, and were mildly demented (Range: 20–28). Five patients met National Institute of Neurological and Communicative Disorders and Stroke/Alzheimer's Disease and Related Disorders Association criteria for probable AD (4 men, 1 woman); the other 5 met criteria for possible AD (2 men, 3 women; McKhann et al., 1984). The clinical diagnosis of *probable* AD requires a deficit in two areas of cognition, one of which is memory, and progressive deterioration of cognitive function. The clinical diagnosis of *possible* AD requires evidence for an isolated memory impairment and exclusion of other causes of dementia. Three of the patients who were originally diagnosed as possible AD went on a year later to satisfy the criteria for probable AD. Among the remaining 2 possible AD patients, one was very mild and high functioning at the time of testing; his MMSE score was 28, which is well within the normal

range. He was included in the study because he showed cognitive decline at 6 months follow-up, and because of his strong positive family history (2 first-degree and 3 second-degree relatives were affected; the diagnosis of AD was autopsy confirmed for one of the second-degree relatives). For all AD patients, diagnosis was based on clinical history, medical laboratory tests, neurologic and psychiatric evaluations to exclude all other possible causes of dementia or other disorders that could affect brain function (for a detailed description of participants' recruitment and selection criteria, see Duara et al. 1983). All patients were normotensive and on no medication at the time of testing. Therefore, none of the participants were, or had been, on cholinergic or other type of treatment before or at the time of testing. This is important, as cholinergic treatment may affect attentional functions (Sarter, Givens, & Bruno, 2001). All AD patients had Hachinski ischemia scores of less than 4 (Hachinski et al., 1975). Control participants were screened for optimal health as described extensively in a previous study (Duara et al., 1983); they had no current or past medical illness, or history of psychiatric or neurological disorder. All controls were unmedicated and normotensive. In all participants, vision was normal or corrected to normal (20:20/25). Control participants and mild AD patients were matched on age and education (Table 1).

Standard Neuropsychological Tests

The Wechsler Adult Intelligence Scale (WAIS; Wechsler, 1955), the Stroop Color Word Test (Golden, 1978), and the Trail Making (Reitan, 1958) test were administered as standardized measures of general intellectual, visuospatial, verbal, and attentional functions. For the WAIS, we also report the WAIS Verbal Sum of Scaled Scores and WAIS Performance Sum of Scaled Scores, because these scores are not age-corrected. Tests of immediate and delayed recall for stories (Logical Memory [LM]) and figures (Visual Reproduction [VR]) from the Wechsler Memory Scale were used as standard measures of nonverbal–visual and verbal memory (Wechsler, 1945; Russell, 1975). In addition, the MMSE (Folstein et al., 1975), and the Mattis Dementia Rating Scale (MDRS; Mattis, 1976), were administered to AD patients as measures of dementia severity. Although the MMSE may not be the best measure to assess severity in high-functioning mild AD patients, other tests suffer from similar flaws (e.g., Mungas & Reed, 2000). We included the MMSE to provide a measure of comparability of our AD patients' severity with that of patients in other studies, as has been recommended (McKhann et al., 1984; Perry & Hodges, 1999). For similar purposes, we included the MDRS, a more comprehensive and probably more sensitive measure of cognitive functioning in mild AD patients (Zec, 1993). The diagnosis of AD, however, was based on a wide battery of neuropsychological tests.

Standard test scores for the AD patients and control participants are shown in Table 1. Patients with mild AD did not differ from control participants on age and education. AD patients had lower scores within the mild range, relative to control participants, on two measures of dementia severity, namely the MMSE and the MDRS. Mild AD patients also had decreased performance on all tests of general intellectual (WAIS IQ), visual (WAIS Performance IQ, WAIS Performance Sum of Scaled Scores), and verbal function (WAIS Verbal IQ, WAIS Verbal Sum of Scaled Scores), as well as on tests of verbal and nonverbal–visual memory (immediate and delayed LM and VR) compared to control participants. They also differed from control participants on all measures of attentional function (Stroop and Trail Making). Although we consider these tests as tapping attention, more particularly selective attention and attention shifting, other authors consider them as tapping visuomotor speed, or the ability to resist interference (see Lezak, 1995).

Differences in standard test scores for AD patients of differing diagnosis (possible vs. probable AD) relative to each other and to controls were also examined (Table 2). Patients with possible and probable AD were matched to controls for age and education. Possible and probable AD

patients had lower scores relative to controls on the MMSE and on the MDRS; probable AD patients also had lower scores on the MDRS, but not on the MMSE, relative to possible AD patients. Probable AD patients had decreased performance on tests of general intellectual (WAIS IQ) and visual function (WAIS Performance IQ, WAIS Performance Sum of Scaled Scores) relative to controls; they also had lower scores than possible AD patients on tests of visual function (WAIS Performance IQ, WAIS Performance Sum of Scaled Scores). Possible AD patients did not differ from control participants on tests of general intellectual (WAIS IQ) and visual function (WAIS Performance IQ, WAIS Performance Sum of Scaled Scores). Moreover, none of the groups differed on tests of verbal function (WAIS Verbal IQ, WAIS Verbal Sum of Scaled Scores). Both probable and possible AD patients had decreased scores on tests of verbal and nonverbal–visual memory (both immediate and delayed LM and VR) relative to controls; probable and possible AD patients did not differ on any of the memory measures. Probable AD patients also had decreased performance relative to controls and possible AD patients on measures of attention (Stroop Words, Stroop Colors, Trails A, Trails B). Probable AD patients also differed from controls, but not from possible AD patients, on the Stroop Interference. Possible AD patients did not differ from controls on any of the attentional measures.

Sustained Attention Digit-Discrimination Task

Materials—Black on white digits were presented on a screen placed 1 m in front of the seated participant. At this distance, digits subtended a visual angle of 8° vertically and of 6° horizontally. To reduce visual persistence, a mask (formed with rows of "+++" on a transparency) was placed behind the screen where the stimuli were presented. A template of the target stimulus, of dimensions 6 × 7.5 cm, was mounted on black cardboard and positioned on the table in front of the participants. This template was to be used in the event that AD patients would forget which target they were looking for. No patient had any problem recalling test instructions and the target throughout the testing period.

Stimuli were degraded by decreasing the slide to lens distance using custom-made lenses (DBL Labs, St. Joseph, MN). The power of the correcting lenses (pc), that is, the diopter required to restore the image to the focused level was used to quantify the degree of stimulus degradation. For pc values of 2.00, digits appeared almost focused, for pc values of 2.50, they appeared moderately blurred (Parasuraman, Nestor, & Greenwood, 1989), and for pc values of 3.00 or more, digits appeared highly blurred. In this study, stimuli were degraded at pc levels 2.00, 2.25, 2.50, 2.75, and 3.00. These degradation levels were determined empirically in pilot studies as described in the Procedure section.

Apparatus—Sustained attention tests were controlled by a PC connected to a box with two response buttons. Degraded digits were projected using a Kodak Ektagraphic IIIAT slide projector (Kodak, Rochester, NY) with a tachistoscopic shutter (Gerbrands, Arlington, MA) placed on a Kodak projection Ektagraphic FF zoom lens of 100 to 150 mm (F 3.5), with a focal length of 15.2 cm (6 in.; Kodak, Rochester, NY). To diffuse the light source, a Kodak Wratten gelatin neutral density filter of grade 1.00 was placed in front of the tachistoscopic shutter (Kodak, Rochester, NY).

Procedure—The sustained attention task used in this experiment and originally described by Neuchterlein et al. (1983) requires participants to attend to visually presented digits, which are perceptually degraded. The degradation levels used in the actual experiment were determined empirically such as to yield initial minimum hit rates of 80% and maximum false alarm rates of 20% within three practice runs for each participant. Another requirement was that all participants could perform the task. During the practice and experimental testing sessions, the lights were turned off.

All participants first received a practice run with 81 undegraded stimuli. Before each degradation level, participants received a minimum of one and a maximum of three practice runs (81 stimuli each) at the level of degradation to be used. Degradation levels were presented in counterbalanced order. During the practice period, feedback concerning the performance (hit and false alarm rates) was provided at the end of each run. If the 80% hits/20% false alarms criterion was not reached within three practice runs, the task at that level and all higher degradation levels was discontinued. All control participants reached the 80% hits/20% false alarms criterion on the first practice run; 4 mild AD patients reached criterion on the second or third practice runs, 1 patient for the degradation level 2.00, 1 patient for the degradation level 2.25, and 2 patients for the degradation level 2.50. This effect was related to the order in which the degradation levels were administered. Patients who started with lower degradation levels needed less practice at higher degradation levels, whereas participants who started with the higher degradation levels first, needed more practice to reach criterion at these levels. However, as required, all participants reached criterion at the highest level of image degradation within 243 trials (three practice runs). All mild AD patients were able to complete the degradation levels 2.00, 2.25, and 2.50. However, only 4 mild AD patients (3 possible and 1 probable AD) could complete the degradation level 2.75, and only 3 (possible AD) could complete the degradation level 3.00. Therefore, for this study, we considered only the degradation levels that all participants could complete (pc 2.00, 2.25, 2.50). During the main sustained attention task, participants were not provided with feedback on task performance.

Digits were presented one at a time for a duration of 100 msec, at a rate of 1 digit per second. Targets (the digit “0”) were presented on 25% of the trials. Nontargets (digits 1 through 9) were presented equally often on the remaining trials. The only constraints were that two identical nontargets were never presented one after another, and that targets were preceded by nontargets an equal number of times. For each degradation level, six test runs of 81 stimuli were presented continuously, so that testing at each degradation level included 486 stimuli, 120 of which were targets. Testing at each degradation level lasted 7.2 min. Participants indicated the presence of targets by pressing the right-hand-side button of a box with their preferred hand. Because of the fast stimulus presentation rates, no response was required for nontargets.

The testing session lasted about 1 hr. After one run with the undegraded condition, the first degradation level was administered. At midsession participants had a break of at least 10 min, after which the other degradation levels were administered. During the testing sessions, the experimenter sat behind the participant where he could not be seen, because this may affect performance (Davies & Parasuraman, 1982).

We did not use a head restrainer, because we were interested in overall vigilance and the vigilance decrement in “natural” conditions. However, all participants were instructed to pay attention to the display at all times, and all participants complied with the instructions during the entire experimental session.

Statistical Procedures

Sensitivity was calculated from hit and false alarm rates using a measure of detectability (d′), derived from signal detection theory (Green & Swets, 1966). Median RTs to targets were also calculated. Response bias was measured using C, following a standard procedure (Snodgrass, 1988). Sensitivity, hits (proportion of correct detections to total number of targets), false alarms (total number of nontargets incorrectly identified to total number of distractors), response bias, and median RTs were computed separately for each test block, and the means for test Blocks 1 and 2, 3 and 4, and 5 and 6 (blocks of 162 stimuli each) were used for statistical analyses. Results from other studies using this paradigm indicated the need for averaging across successive blocks to reduce statistical noise. Results were analyzed using three-way analyses

of variance (ANOVAs) with one between factor: group (mild AD patients, control participants), and repeated measures on two within-group factors: degradation level (pc 2.00, 2.25, 2.50) and block (1, 2, and 3; Kendall & Stuart, 1961).

To evaluate differences in AD patients of differing severity (possible AD vs. probable AD) relative to each other and to control participants, the three-way ANOVAs with one between-group factor (possible AD, probable AD, control participants) and repeated measures on two within-group factors (degradation level and block) were rerun for analyses of sensitivity (d′), response bias, and RTs. To determine between-group differences for main group effects, Bonferroni–Dunn post hoc *t* tests were used (Snedecor & Cochran, 1980). For significant interactions involving the group factor, groups were compared two by two using ANOVAs.

The overall level of vigilance was computed for each degradation level as the mean performance for all test blocks. Sustained attention decrements were calculated as the difference between mean sensitivity on Blocks 5 and 6 and mean sensitivity on Blocks 1 and 2 for each degradation level. Decrement scores, therefore, correspond to negative values. Correlations between standard cognitive tests and both overall vigilance and vigilance decrements at the maximum degradation level that all participants could perform (pc 2.50) were computed in mild AD patients and old controls by using the Pearson method (Snedecor & Cochran, 1980).

RESULTS

Sustained Attention Digit-Discrimination Task

To determine whether there were any order effects, preliminary analyses of sensitivity (d′), hits, false alarms, response bias, and RTs were carried out using four-way ANOVAs with two between factors (order, group) and two within-group factors (block, degradation level). We did not consider the four-way interactions, because we didn't have enough participants in some of the cells for meaningful interpretation. All analyses indicated that there were no significant main effects, two- or three-way interactions, due to order at the exception of a significant Degradation Level × Group × Order interaction for false positives, $F(6, 40) = 2.99$, $p = .02$, power = .85. However, ANOVAs for the mild AD and the control participants separately indicated that the interaction was not significant in both groups (both *p*s > .18). Therefore, subsequent analyses are based on the data collapsed across the different orders.

Sensitivity (d)—The mean sensitivity values for AD patients and controls are shown in Figure 1. The main group effect was significant, $F(1, 28) = 17.57$, $p < .0005$, power = .98, indicating that mild AD patients had lower overall vigilance than control participants. d′ decreased with higher degradation levels, $F(2, 56) = 9.25$, $p < .0005$, power = .97, and with time on task, $F(2, 56) = 7.99$, $p < .001$, power = .95. The interaction between degradation level and block, $F(4, 112) = 5.80$, $p < .0005$, power = .98, indicated that the sensitivity decrement over time was greater at the higher degradation levels. The only other significant interaction was the Degradation Level × Block × Group interaction, $F(4, 112) = 5.40$, $p < .001$, power = .97, indicating that the decrease in sensitivity over blocks for higher degradation levels was more pronounced in AD patients than in controls.

Figure 1 indicates that this interaction might be significant not because of a greater sustained attention decrement over time at the highest degradation level, but because of an increase in sensitivity at the degradation level pc 2.25. We therefore analyzed the simple effect of block at each degradation level using the Satterthwaite's procedure described by Winer (1971). This procedure was used to correct the error-term degrees of freedom for pooled sources of variance. We found that, for both AD patients and controls, there was no significant block effect for the degradation levels 2.00 and 2.25, with $F(2, 36) = 2.56$, *ns*, and $F(2, 36) = 2.53$, *ns*, in AD

patients; and with $F(2, 76) = .56$, *ns*, and $F(2, 76) = 1.94$, *ns*, in controls, respectively. However, a significant block effect was found in both groups at the degradation level 2.50, with $F(2, 36) = 5.21$ for AD patients, and $F(2, 76) = 5.79$ for controls, both *p*s < .05. These results suggest therefore that the significant Degradation Level × Block × Group interaction is due to a greater decrease in sensitivity at the degradation level pc 2.50 in mild AD patients than in controls. This was confirmed by an ANOVA on sensitivity decrement scores, defined as the difference in mean sensitivity between the late (5 and 6) and early blocks (1 and 2). This analysis gave significant effects for degradation level, $F(2, 56) = 9.39$, $p < .0005$, power = .97, and for the Degradation Level × Group interaction, $F(2, 56) = 8.54$, $p < .001$, power = .96, indicating that mild AD patients had more marked sensitivity decrement than controls under the high degradation condition. Other effects were not significant. Mean decrement scores ± *SD* at the highest degradation level were −.129 ± .25 in control participants and −.427 ± .37 in mild AD patients.

The analyses of sensitivity were rerun to investigate differences in overall vigilance and in vigilance decrement in possible AD, probable AD patients, and control participants (Table 3). We only report details for main effects and interactions involving group. The pattern of results was similar in all respects to that obtained for the entire mild AD group. The main group effect was significant, $F(2, 27) = 14.27$, $p = .0001$, power = .98. Bonferroni–Dunn post hoc *t* tests suggested that probable AD patients had lower overall vigilance than possible AD patients ($p = .005$) and old controls ($p = .0001$); possible AD patients and old controls did not differ ($p = .10$). The Degradation Level × Group interaction was significant, $F(4, 54) = 2.56$, $p = .03$, power = .76, and suggested that probable AD patients had greater vigilance decrement at higher degradation levels relative to controls, $F(2, 46) = 5.83$, $p = .006$, power = .85; the other comparisons were not significant. The Degradation Level × Block × Group interaction was significant, $F(4, 92) = 3.43$, $p = .02$, power = .96; both possible, $F(4, 92) = 5.77$, $p = .0003$, power = .98, and probable AD patients, $F(4, 92) = 3.43$, $p = .01$, power = .84, had greater vigilance decrement over time on task at higher degradation levels relative to controls; probable and possible AD patients did not differ.

Examination of the simple effect of block at each degradation level using the Satterthwaite's procedure (Winer, 1971), suggested that in possible AD patients there was a marginally significant block effect at degradation level 2.50, $n = 5$, $F(2, 16) = 2.71$, $p < .10$. The effect of block was not significant in probable AD patients, probably because of the small number of participants, $n = 5$; $F(2, 16) = 2.46$, $p > .10$. The block effect at degradation levels 2.00 and 2.25 was not significant in neither group (all *p*s > .10). The analysis of sensitivity decrement scores, defined as the difference in mean sensitivity between the late (5 and 6) and early blocks (1 and 2), gave a similar pattern of results to that obtained for the entire group of AD patients. In particular, the Degradation Level × Group interaction, $F(4, 54) = 4.17$, $p = .005$, power = .90, showed that both probable, $F(2, 46) = 6.19$, $p = .004$, power = .87, and possible AD patients, $F(2, 46) = 6.44$, $p = .003$, power = .89, had more marked sensitivity decrement than controls under the high level of degradation condition; possible and probable AD patients did not differ. Other effects involving group were not significant.

Hit rate—(Table 4). Significant main effects were obtained for group, $F(1, 28) = 7.05$, $p < .05$, power = .73, degradation level, $F(2, 56) = 6.42$, $p < .005$, power = .89, and block, $F(2, 56) = 5.99$, $p < .005$, power = .86, suggesting that hit rates were lower in the AD group, at higher degradation levels, and in the later time blocks. The Degradation Level × Group interaction was marginally significant, $F(2, 56) = 2.97$, $p = .059$, power = .56, indicating that mild AD patients showed a tendency for lower hit rates at higher degradation levels. The Degradation Level × Block interaction was significant, $F(4, 112) = 3.69$, $p < .01$, power = .87, indicating that hit rate decreased more over time at higher degradation levels. The marginally significant interaction of Degradation Level × Block × Group, $F(4, 112) = 2.43$, $p = .05$, power = .68,

suggested that mild AD patients tended to have lower hit rates over time on task at higher degradation levels relative to controls. All other interactions were not significant.

False positives—(Table 4). The main effects of group, $F(1, 28) = 12.79$, $p < .005$, power = .93, and degradation level were significant, $F(2, 56) = 6.45$, $p < .005$, power = .89, suggesting that false positives were higher in the AD group and that for all participants they increased with higher degradation levels. The Degradation Level × Group interaction was marginally significant, $F(2, 56) = 2.61$, $p = .08$, power = .50, suggesting that mild AD patients tended to make more false positives at higher degradation levels relative to controls. The main block effect was not significant, $F(2, 56) = 2.12$, $p > .05$, indicating that false positive rate did not change over time. The Degradation Level × Block interaction, $F(4, 112) = 4.49$, $p < .005$, power = .93, and the Degradation Level × Block × Group interactions were significant, $F(4, 112) = 5.46$, $p < .001$, power = .97, indicating that mild AD patients had higher increases in false positives over time with higher degradation levels than did controls.

Response bias (C)—(Table 4). The main group effect was not significant, $F(1, 28) < 1$, indicating that there was no overall difference in response bias between AD patients and controls. The main degradation level was significant, $F(2, 56) = 3.94$, $p < .05$, power = .69, indicating that response bias became more conservative with higher degradation levels. The Degradation Level × Group interaction was marginally significant, $F(2, 56) = 3.10$, $p = .05$, power = .58, suggesting that mild AD patients tended to have more conservative response bias at higher degradation levels relative to controls. However, ANOVAs within the mild AD and the control groups separately indicated that the main degradation level effect was not significant in either group (both *p*s > .10). All other main effects and interactions were not significant.

The analyses were rerun to investigate differences in response bias between possible AD, probable AD patients, and controls (Table 3). The results were similar to those obtained for the entire group of mild AD patients (means ± *SD* are presented in Table 3), except that now the Degradation Level × Group interaction was significant, $F(4, 54) = 2.65$, $p = .04$, power = .70; response bias in both the possible, $F(2, 46) = 3.34$, $p = .044$, power = .60, and the probable AD patients, $F(2, 46) = 3.58$, $p = .036$, power = .64, was more conservative with higher degradation levels relative to controls; probable and possible AD patients did not differ. However, power analyses suggested low reliability for these findings, and ANOVAs within the possible and probable AD groups separately suggested that the main degradation level effect was not significant in either group (both *p*s > .10). All other effects involving group were not significant.

RT—(Table 4). Variances in mild AD patients and control participants at each degradation level and for each block did not differ significantly by *F* tests (all *p*s > .10), and the underlying distributions for both groups were normal (Kolmogorov–Smirnov *z* values testing for the normality of the distributions ranged between .35 and .86; all *p*s > .10).

The main effects of degradation level, $F(2, 56) = 7.85$, $p < .001$, power = .94, and block, $F(2,56) = 8.12$, $p < .001$, power = .95, were significant, suggesting that RT increased at higher degradation levels and with time on task, but did not differ between groups (main effect of group: $F(1,28) < 1$. A trend for the Block × Group interaction, $F(2,56) = 2.51$, $p = .09$, power = .48, suggested that mild AD patients showed a trend for slower RTs with time on task. All other effects were not significant.

The analyses were rerun to investigate differences in RTs in possible AD, probable AD patients, and control participants (Table 3). The pattern of results was similar in all respects to that described previously, except that the Block × Group interaction was significant, $F(4, 54) = 2.80$, $p = .03$, power = .73, suggesting that probable AD patients were slower than controls

over time on task, $F(2, 46) = 5.08$, $p = .01$, power = .79; the other comparisons were not significant. All other main effects or interactions involving group were not significant.

Within-Task and Intertask Analyses

As discussed previously, the defining feature of vigilance performance is the vigilance decrement, defined in our study as the difference in sensitivity (d′) between the last two and first two blocks of the task. We examined whether our finding of greater sensitivity decrement over time in mild AD patients was related to either the overall vigilance level (defined as the mean level of d′ over all blocks) or to performance on the other neuropsychological tasks included in our study. In control participants, the sensitivity decrement and overall vigilance scores were significantly intercorrelated ($n = 20$, $r = .59$, $p < .01$), indicating that participants with lower vigilance levels had greater sensitivity decrement over time. In mild AD patients, however, the correlation was not significant ($n = 10$, $r = .38$, $p > .05$), indicating that in this group the vigilance decrement was not associated with overall vigilance. Nevertheless, it remained possible that *initial* differences in perceptual ability might be responsible for the greater sensitivity decrement in the AD group. To evaluate this possibility, we examined group differences in d′ values on the practice trials (given prior to the main vigilance task) at the degradation level at which increased sensitivity decrement in the AD group was found (pc 2.50). Mild AD patients and controls did not differ in hits ($t = 1.11$, $p > .05$), false alarms ($t = -1.83$, $p > .05$) or d′ ($t = 1.31$, $p > .05$). If the increased vigilance decrement in the AD group reflected a perceptual deficit, this should have been apparent as a significant group difference in the practice task-accuracy scores. However, no such difference was found. We then examined whether the initial level of performance on the vigilance task—d′ in the first of the six blocks of the task—was predictive of sensitivity decrement over blocks. The d′ on the first experimental block at the degradation level 2.50 was not significantly correlated with the sensitivity decrement either in the control participants ($n = 20$; $r = .11$, $p > .05$) or the mild AD group ($n = 10$; $r = .15$, $p > .05$). The results of these analyses examining the relation between initial perceptual difficulty and sensitivity decrement are remarkably similar and clearly show that the sensitivity decrement cannot be explained exclusively in terms of a perceptual deficit in the AD patients.

We also examined the correlations between the two vigilance scores—overall vigilance and sensitivity decrement—and performance on standard tests of general intellectual and attentional function. In mild AD patients, significant correlations were found between overall vigilance and both the WAIS Performance IQ and the WAIS Performance Sum of Scaled Scores (see Table 5). This result suggests that in mild AD patients, higher levels of vigilance were associated with better visuospatial performance. A trend for both Trails A and Trails B to be correlated with overall vigilance was also found in mild AD patients, but both correlations failed to reach statistical significance (both *p*s = .06). No other significant correlations were found between standard neuropsychological test scores and overall vigilance or sensitivity decrement in either the mild AD or the healthy control groups (Table 5).

It is possible that some of the correlations may have failed to reach statistical significance because of the low number of participants tested. Moreover, if the *p* values are adjusted for the number of comparisons, none of the previously mentioned correlations are significant ($p < .005$).

DISCUSSION

The findings of this study indicate that three main differences distinguished mild AD patients from controls. First, mild AD patients could not perform what are generally considered high-stimulus degradation levels (pc 2.75), whereas controls could easily perform at even higher levels (e.g., pc 3.00; see Berardi, Parasuraman, & Haxby, 2001; Parasuraman et al., 1989).

Second, mild AD patients showed a decrease in overall vigilance at low and moderate degradation levels (pc 2.00, 2.25, and 2.50), suggesting a decrease in tonic arousal. Third, mild AD patients showed greater sensitivity decrement over time relative to the control group at moderate degradation levels, indicating a deficit in the maintenance of vigilance over time under effortful processing conditions (Neuchterlein et al., 1983; Parasuraman, 1985). Because significant effects of block were found at the moderate degradation level of pc 2.50 in both groups, the task was optimal at this level to demonstrate vigilance decrement and compare its extent between groups.

Mild AD patients differed from controls on overall levels of performance on the vigilance task. It has been suggested that the decrease in overall vigilance levels may reflect a global reduction in tonic arousal (e.g., Parasuraman, 1985, 1986). Tonic arousal is a general state affecting the ability of an individual to carry out various functions of attention, among which is remaining vigilant (Parasuraman, 1985, 1986). Overall vigilance levels covary directly with changes in tonic arousal induced endogenously (fatigue, boredom) or exogenously (environmental stressors) (see Parasuraman, 1985, 1986). As a consequence, overall levels of vigilance may be more variable and less consistently reliable across studies than the vigilance decrement that is not affected by the same factors (e.g., Parasuraman, 1986; Poulton, 1977). Perhaps for this reason, our finding of decreased overall vigilance in patients with mild AD is consistent with some previous studies of vigilance in senile dementia (e.g., Alexander, 1973; Sahakian et al., 1989; Sunderland et al., 1987), but not with others (Lines et al., 1991; Nebes & Brady, 1993). The decreased overall performance in mild AD patients may partly reflect a greater perceptual difficulty, which might in turn be responsible for their greater sensitivity decrement over time on task. However, a number of analyses showed that the greater vigilance decrement in the AD patients could not be attributed to pretask or initial differences in perceptual difficulty. Therefore, although mild AD patients may have general perceptual deficits, our results suggest that the vigilance decrement in this task was not due to a perceptual deficit in the discrimination of visually degraded digits.

A cardinal feature of vigilance performance is the decrement in performance over time (Davies & Parasuraman, 1982; Warm, 1984). The vigilance decrement can be indexed reliably in high-processing load tasks by a decrement over time in sensitivity (d′; Neuchterlein et al., 1983; Parasuraman, 1979; See et al., 1995). Our results indicated a deficit of mild AD patients maintaining vigilance over time under conditions of moderate stimulus degradation, as revealed by the significant Degradation Level × Block × Group interaction for the sensitivity results. Previous studies using dual-task methods suggest that sensitivity decrement under such conditions may reflect limitations in the sustained allocation of effortful or controlled processing resources to visual detection (Neuchterlein et al., 1983; Parasuraman, 1985). The finding of increased sensitivity decrement in mild AD contrasts with the effects of normal adult aging on vigilance. Previous reports using the same vigilance task as in this study have found equivalent declines in sensitivity over time on task in young and healthy older adults (Berardi, Gaillard, Haxby, Greenwood, & Parasuraman, 1992; Berardi et al., 2001; Parasuraman et al., 1989). Thus, mild AD affects the vigilance decrement, whereas healthy aging does not, at least in individuals up to about 70 years of age.

The vigilance decrement may result either from a sensitivity (d′) decrement, from a change in response bias (C), or both (Davies & Parasuraman, 1982). Observers in vigilance tasks tend to become more cautious with time on task (Davies & Parasuraman, 1982). Therefore, as the task proceeds, participants may tend to respond “yes” to a target, only when they are absolutely sure that it has been presented. This increase in response criterion is associated with decrements in both correct and false detections, but not with a change in sensitivity. Use of d′, which combines both misses and false recognitions, allows one to determine whether differences between groups reflect a sensitivity decrement or a shift in response bias. Optimally, vigilance

decrements over time should be reflected in a decrease in sensitivity (d′) rather than in a change in response bias. Mild AD patients had greater sensitivity decrement over time relative to controls, but this decrement was not associated with concomitant changes in response bias, as there were no differences over time on task between groups (both the Block × Group and the Degradation level × Block × Group interactions were not significant for response bias, both *p*s > .35). Therefore, although mild AD patients made overall more false alarms than old controls and had a higher rate of false alarms over time on task at higher degradation levels, our results suggest that the differential effects of image degradation over time on task in mild AD patients are due to a change in perceptual sensitivity and not to a change in response criterion.

Response time data suggest that there were no differences between mild AD patients and controls. Response times increased, as expected, over time on task and for higher degradation levels, but there were no significant between-group differences. This finding may be surprising because RTs are often found to be slower in mild AD, but these changes are more commonly seen in choice RT tasks that involve target discrimination than in simple RT tasks, such as the one used in this study, that involve target detection (Ferris, Crook, Sathananthan, & Gershon, 1976; Nestor, Parasuraman, & Haxby, 1991; Pirozzolo, Christensen, Ogle, Hansch, & Thompson, 1981). The mild AD patients evaluated in this study were high-functioning, highly educated, and rigorously screened for health status. The task was fast paced and required that participants respond rapidly. Although participants did not know when a target would appear, they were ready and prepared to respond. These factors may have minimized changes in RTs in this simple detection task between mild AD patients and control participants. Other studies of simple RT in similar AD patients have found comparable results (Nestor et al., 1991; Parasuraman, Greenwood, Haxby, & Grady, 1992; Parasuraman & Nestor, 1993).

Our finding of a significant vigilance decrement in mild AD at moderate levels of stimulus degradation differs from that of other studies, probably because of methodological differences in the tasks and procedures used. Lines et al. (1991) did not find a main effect of time on task for both percentage of hits and false alarms, suggesting that their task was probably too easy for both mild AD patients and controls; they used higher target probabilities and longer stimulus presentations than those used in this study. Sustained attention decrements are more likely to occur with low target probabilities and shorter stimulus durations (Davies & Parasuraman, 1982).

Nebes and Brady (1993) found that AD patients had overall longer RTs than old controls. However, the mean increase in RT across blocks was equivalent in both groups, indicating that there was no greater decrement in sustained attention in mild AD patients than in normal controls. Although Nebes and Brady's task can be classified as high event rate (> 30 events per min), processing load was lower than in this study, and because their task was self-paced, the number of trials per block (and therefore event rate) varied across and within participants, with some of their AD patients getting lower event rates than the AD patients included in this study. Finally, because of the participants' low error rates, trends over time for accuracy data were not reported. It is possible that in mild AD patients, given adequate difficulty of the vigilance task, accuracy may be a more sensitive measure of vigilance decrement than RTs, as our results seem to suggest.

Brazzelli et al. (1994), on the other hand, reported significant decreases in sensitivity in patients with AD. However, AD patients performed just above chance on the first block, and their performance decreased at chance levels on the second and third blocks for both hits and false alarms. Five out of 12 AD patients detected no target at all on the last block. It is uncertain whether, in these conditions, the patients fully understood the task. Dementia severity could perhaps account for their findings. However, because dementia severity in the study by

Brazzelli et al. was not evaluated using the MMSE, or other standard measures, the severity of their patients' conditions cannot be compared with that of the patients tested in our study. Their participants' low performance on the task, however, may suggest that their patients were in more advanced stages of disease than the participants included in this study.

Baddeley et al. (1999) suggested that the vigilance decrement in AD may occur only when the executive control processes of working memory are involved. These authors showed that a successive discrimination, but not a simultaneous discrimination task (both of which were matched for initial levels of performance), was associated with greater vigilance decrement in mild AD patients relative to old controls. They interpreted this finding as suggesting that the successive discrimination task imposed a greater load on the executive processes of working memory than the simultaneous task, and that maintaining the representation of a visual stimulus over time is an effortful task that places demands on executive processes. According to Baddeley et al. (1999), the vigilance decrement may be linked to short-term memory rehearsal, especially in the case of visual information. The task used in this study, however, did not impose a load on working memory, but had a high load on perceptual discriminability (see Neuchterlein et al., 1983), suggesting that working memory load may not be the only factor affecting the vigilance decrement. Future studies will need to determine the precise role of working memory deficits in the vigilance decrement of mild AD patients.

Although the AD sample in this study was small, our results further suggest that possible and probable AD patients had significant decrements in vigilance over time at higher degradation levels relative to controls. However, only probable AD patients had lower overall vigilance levels relative to controls; possible AD patients and controls did not differ. The vigilance decrement in the possible AD patients was found to co-occur with deficits in episodic memory, although deficits in other attentional tasks were not yet evident (e.g., Stroop Color Word Test, Trail Making Test). These results suggest that deficits in vigilance over time in these patients may occur earlier than deficits on selective attention tasks. In contrast, in probable AD patients, deficits in overall vigilance and in vigilance decrement co-occurred with deficits in memory and in all standard attentional tasks used in this study (Stroop Color Word Test, Trail Making Test). It is interesting to note that Perry et al. (2000) found that minimal AD (equal to our possible AD) patients were impaired relative to controls on tests of episodic memory and on two tests of selective attention, among which was the Stroop Color Word Test. However, minimal AD patients did not differ from controls on two sustained attention tasks. Perry et al. examined only overall vigilance and not the vigilance decrement, although he suggested that the vigilance decrement may be a more sensitive measure than overall vigilance. Indeed, in this study, the vigilance decrement over time was successful in differentiating the performance of possible (or minimally) impaired AD patients from controls, whereas overall vigilance was not. There was a suggestion that the MMSE scores of the possible AD patients in this study were slightly lower than those reported for the minimal AD patients in the study by Perry et al. However, the minimal AD patients in their study had deficits on the Stroop task, where as our possible AD patients did not, suggesting that our possible AD patients may have been more high-functioning or less impaired or both. The MMSE may not be the most sensitive measure of disease severity in mild AD (e.g., Mungas & Reed, 2000). Nevertheless, our results indicate, with those of Grady et al. (1988) and Perry et al. that attentional deficits occur early in mild AD, and extend these previous findings by suggesting that changes in vigilance over time in possible AD patients as a group may occur before deficits on standard tests of selective attention. These results also suggest that the vigilance decrement over time should be systematically examined in mild AD patients.

In both the possible and probable AD patients, similar to the results for the entire mild AD group, the vigilance decrement was due to a decrease in sensitivity (d′) over time on task at higher degradation levels, and not to a change in response bias. Moreover, similar to the entire

AD group, no main effect of group on RTs was found. However, the RTs of the probable AD patients increased with time on task to a greater rate than in control participants.

Although the results on possible and probable AD patients are preliminary and need to be replicated on larger patient samples, the power analyses for the significant sensitivity (d′) results were generally in the acceptable range, suggesting that these findings should be reliable. Future studies will need to determine whether the group pattern observed in this study also consistently characterizes possible AD patients individually.

In healthy participants and mild AD patients, no correlation was found between overall vigilance or sensitivity decrement and standard tests of attentional function. Previous studies in larger samples of healthy participants, however, have suggested a relation between overall vigilance and standard tests of attention (Berardi et al., 2001). These correlations may have failed to reach statistical significance because of the small number of participants. In contrast, no relation has generally been found between the vigilance decrement and performance on standard cognitive tests in both healthy participants and in patient groups (Berardi et al., 2001; Davies, Jones, & Taylor, 1984; Rueckert & Grafman, 1996). The vigilance decrement is a measure of vigilance over time. It may therefore not correlate well with other standard cognitive tasks because these are *overall* measures of performance and not measures of performance decrement over time. In addition, at least for the healthy old controls, the attentional load needed in some standard cognitive tasks may not be sufficiently high.

Our results also indicate that in healthy participants there was no relation between vigilance performance and intelligence, similarly to what has been reported in other studies (Berardi et al., 2001; Davies et al., 1984; Tomporowski & Simpson, 1990). Vigilance decrements, however, have been reported in mentally retarded individuals (Tomporowski & Simpson, 1990) suggesting that intelligence may affect vigilance performance only in patients with brain damage. In our mild AD group, a significant relation was found only between overall vigilance and the WAIS Performance IQ. Similar correlations were not significant for Full Scale IQ, or for sensitivity decrement, providing only partial support for a relation between intelligence and sustained attention ability in patients with cerebral impairment. The nonsignificant correlations obtained in control participants, and the minimal correlations obtained in AD patients, may suggest that intelligence is not related to vigilance performance per se. Intelligence and sustained attention may be related only because both reflect disease-related changes. Alternatively, the correlations in this study may also have failed to reach statistical significance due to the low number of participants.

Although the neural systems involved in vigilance tasks are not well specified, two popular attentional models suggest that overall vigilance may be mediated by ascending reticulothalamic pathways in interaction with the prefrontal and posterior parietal cortices (Mesulam, 1981; Posner & Petersen, 1990). A right hemisphere dominance in the capacity to maintain an alert or vigilant state has been suggested by simple RT studies (e.g., De Renzi & Faglioni, 1965; Howes & Boller, 1975; Whitehead, 1991). Although these findings have not always been replicated because of imprecise categorization of lesions within the right or left hemispheres, as pointed out by Audet, Mercier, Collard, Rochette, and Hebert (2000), human studies on patients with brain lesions restricted to the right frontal cortex have demonstrated that these lesions affect overall vigilance (Rueckert & Grafman, 1996; Wilkins, Shallice, & McCarthy, 1987). These effects are generally observed for both the accuracy of target detection (Rueckert & Grafman, 1996; Wilkins et al., 1987) and for RTs (Rueckert & Grafman, 1996).

Functional brain imaging studies in healthy participants have further suggested that overall vigilance may involve a subcortico-cortical system relying on the right prefrontal cortex (Cohen, Semple, Gross, Holcomb, Dowling, & Nordahl, 1988; Cohen, Semple, Gross, King,

& Nordahl, 1992; Coull, Frith, Frackowiak, & Grasby, 1996; Deutsch, Papanicolaou, Bourbon, & Eisenberg, 1987; Fassbender et al., 2004; Lewin et al., 1996; Pardo, Fox, & Raichle, 1991), the parietal lobes (Coull et al., 1996; Fassbender et al., 2004; Pardo et al., 1991), the anterior cingulate, the thalamus, and brainstem structures (Coull, Frackowiak, & Frith, 1998; Kinomura, Larsson, Gulyas, & Roland, 1996; Paus et al. 1997; Sturm et al., 1999). There is some evidence from functional brain imaging studies suggesting that the brain regions involved in overall vigilance may also be involved in the vigilance decrement, as activation in these same regions has been shown to decrease over time on task (Coull & al., 1998; Paus et al., 1997). Human brain lesion studies also suggest that lesions of the frontal and parietal cortices, but not of the anterior cingulate, are associated with the vigilance decrement (Koski & Petrides, 2001; Rueckert & Grafman, 1996, 1998).

In light of previous human brain lesion and functional brain imaging studies, our findings of overall decreased vigilance and of greater vigilance decrement over time in mild AD suggest that the brain systems underlying vigilance performance could be affected early in the disease process. After the medial temporal structures, brain pathology spreads to neocortical associative regions such as parietal, temporal, and frontal lobes (Kemper, 1984). Resting state positron emission tomography studies have shown early reductions in regional cerebral metabolic rates for glucose (rCMRglc) and increased rCMRglc asymmetry in the frontal lobes of mild AD patients (Grady et al., 1990; Haxby, Duara, Grady, Cutler, & Rapoport, 1985; Haxby et al., 1990; Kumar et al., 1991), as well as a decreased number of intercorrelations between homologous regions within the frontal lobes and between the frontal and parietal lobes (Horwitz, Grady, Schlageter, Duara, & Rapoport, 1987).

The disruption of two neural systems may be consistent with the vigilance deficits observed in mild AD patients. First, animal studies have shown that the basal forebrain cholinergic system provides major innervation to the prefrontal and parietal cortices, and this system has been involved in sustained attention (Baxter & Chiba, 1999; Sarter et al., 2001). Mild AD is associated with a loss of innervation of the frontal cortex from the nucleus basalis (Arnold, Hyman, Flory, Damasio, & Van Hoesen, 1991; Coull, 1998; Lawrence & Sahakian, 1995; Muir, 1997), and with a loss of long cortico-cortical connections between the frontal and parietal lobes leading to a frontal disconnection (Morrison et al., 1990). Second, animal and human studies have demonstrated that the noradrenergic system is involved in vigilance (see Coull, 1998). Early brain impairment in subcortical nuclei such as the locus coeruleus and corresponding cortical projection areas (Aston-Jones, 1985; Bondareff, Mountjoy, & Roth, 1982; Chan Palay, 1989; Palmer, Wilcock, Esiri, Francis, & Bowen, 1987; Posner & Petersen, 1990) may also be implicated in the vigilance deficits observed in mild AD. Further studies are needed to more precisely address the relation of overall vigilance and vigilance decrement with structural or functional brain changes or both in mild AD.

In conclusion, this study indicates that mild AD patients have decreased overall vigilance relative to age-matched control participants, which may reflect their lower level of tonic arousal. Mild AD patients also have greater sensitivity decrement over time at moderate levels of stimulus degradation, indicating a deficit in the maintenance of sustained attention over time in effortful processing conditions. These results provide a controlled demonstration of the common clinical observation that mild AD patients have difficulty concentrating and maintaining consistent levels of performance over long periods of time. It is possible that such a sustained attention deficit may contribute to or compound other cognitive deficits in mild AD, particularly in task conditions that require effortful processing.

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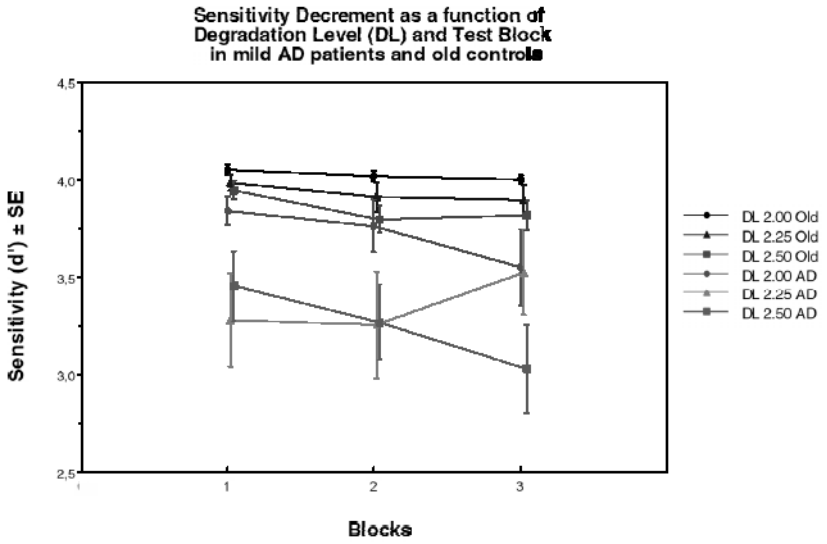

FIGURE 1.

Mean values ± SE for sensitivity (d′) as a function of test block at each degradation level (2.00, 2.25, and 2.50) in mild AD patients and in old healthy participants (controls).

TABLE 1

Characteristics of Mild AD Patients and Control Participants

Variable	Controls[a]	Mild AD[b]	p Value
Age	67 ± 4.4	67 ± 8.9	*ns*
Education	17 ± 3.2	15 ± 3.3	*ns*
MMSE	29.5 ± .64	24.2 ± 2.3	.0001
MDRS	141 ± 3.8	124 ± 10.4	.0001
General intellectual ability			
WFSIQ	128 ± 7.2	111 ± 19.6	.002
WPIQ	124 ± 8.2	104 ± 18.1	.0003
WVIQ	129 ± 9.8	116 ± 20.6	.03
WPSSS	51.9 ± 7.2	36.3 ± 12.6	.0002
WVSSS	81.4 ± 9.9	69 ± 18.6	.02
Memory tests			
Logical memory			
Immediate	21.85 ± 5.27	8.70 ± 3.89	.0001
Delayed	16.95 ± 5.98	3.00 ± 3.37	.0001
Visual reproduction			
Immediate	9.05 ± 2.72	3.50 ± 2.46	.0001
Delayed	8.35 ± 2.85	2.20 ± 2.78	.0001
Attention tests			
Stroop Words	97 ± 10.1	76.4 ± 26.8	.005
Stroop Colors	65 ± 7.3	49 ± 17.5	.001
Stroop Interference	39 ± 10.9	25 ± 12.9	.006
Trail Making A (time)	39 ± 11.1	73.9 ± 45.8	.003
Trail Making B (time)	75 ± 17.5	249.8 ± 56.5	.0001

Note. AD = Alzheimer's patients; MMSE = Mini-Mental State Examination; MDRS = Mattis Dementia Rating Scale; WFSIQ = Wechsler Adult Intelligence Scale (WAIS) Full Scale IQ; WPIQ = WAIS Performance IQ; WVIQ = WAIS Verbal IQ; WPSSS = WAIS Performance Sum of Scaled Scores; WVSSS = Wechsler Verbal Sum of Scaled Scores.

[a] $n = 20$.

[b] $n = 10$.

TABLE 2

Characteristics of Possible AD, Probable AD Patients, and Control Participants

Variable	Controls[a]	Possible AD[b]	Probable AD[b]	Overall p Value	Subgroup p Values		
					Possible AD Versus Controls	Probable AD Versus Controls	Possible AD Versus Probable AD
Age	67 ± 4.4	66 ± 5.8	67 ± 11.9	*ns*	*ns*	*ns*	*ns*
Education	17 ± 3.2	14 ± 3.6	15 ± 3.3	*ns*	*ns*	*ns*	*ns*
MMSE	29.5 ± .64	25.2 ± 1.9	23.2 ± 2.4	.0001	.0001	.0001	*ns*
MDRS	141 ± 3.8	132 ± 6.8	116 ± 7.0	.0001	.002	.0001	.0001
General intellectual ability							
WFSIQ	128 ± 7.2	120 ± 20.8	103 ± 16.2	.001	*ns*	.0007	*ns*
WPIQ	124 ± 8.2	115 ± 14.1	92 ± 14.5	.0001	*ns*	.0001	.0004
WVIQ	129 ± 9.8	121 ± 24.7	111 ± 17.0	.06	*ns*	*ns*	*ns*
WPSSS	51.9 ± 7.2	45.2 ± 10.8	27 ± 6.5	.0001	*ns*	.0001	.0003
WVSSS	81.4 ± 9.9	74 ± 23.9	63 ± 11.7	.03	*ns*	*ns*	*ns*
Memory tests							
Logical memory							
Immediate	21.85 ± 5.27	9.20 ± 4.71	8.20 ± 3.35	.0001	.0001	.0001	*ns*
Delayed	16.95 ± 5.98	2.60 ± 2.51	3.40 ± 4.34	.0001	.0001	.0001	*ns*
Visual reproduction							
Immediate	9.05 ± 2.72	4.60 ± 2.41	2.40 ± 2.19	.0001	.004	.0001	*ns*
Delayed	8.35 ± 2.85	3.00 ± 3.32	1.40 ± 2.19	.0001	.002	.0001	*ns*
Attention tests							
Stroop Words	97 ± 10.1	92 ± 20.4	61 ± 24.7	.0003	*ns*	.0002	.001
Stroop Colors	65 ± 7.3	58 ± 17.4	39 ± 13.2	.0002	*ns*	.0001	.003
Stroop Interference	39 ± 10.9	29 ± 14.3	21 ± 11.2	.01	*ns*	.009	*ns*
Trail Making A (time)	39 ± 11.1	51 ± 16.2	97 ± 56.1	.0003	*ns*	.0002	.002
Trail Making B (time)	75 ± 17.5	125 ± 48.4	374 ± 175.2	.0001	*ns*	.0001	.0001

Note. Subgroup *p* values are Bonferroni–Dunn post hoc *t* tests (corrected for multiple comparisons). AD = Alzheimer's patients; MMSE = Mini-Mental State Examination; MDRS = Mattis Dementia Rating Scale; WFSIQ = Wechsler Adult Intelligence Scale (WAIS) Full Scale IQ; WPIQ = WAIS Performance IQ; WVIQ = WAIS Verbal IQ; WPSSS = WAIS Performance Sum of Scaled Scores; WVSSS = WAIS Verbal Sum of Scaled Scores.

[a] $n = 20$.

[b] $n = 5$.

TABLE 3

Means and Standard Deviations for Sensitivity (d′), Response Bias (C), and Response Times (msec) in Probable Alzheimer's Disease (AD), Possible AD, and Control Participants

	Stimulus Degradation Level																	
	DL 2.00						DL 2.25						DL 2.50					
Test Block	1		2		3		1		2		3		1		2		3	
	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD
Sensitivity (d′)																		
Probable AD	3.71	.25	3.56	.52	3.48	.74	3.08	.53	3.18	.54	3.35	.68	3.07	.42	2.80	.37	2.65	.17
Possible AD	3.97	.13	3.97	.19	3.62	.55	3.47	.95	3.33	1.2	3.70	.74	3.84	.41	3.74	.39	3.41	.87
Controls	4.05	.12	4.02	.12	4.00	.09	3.98	.17	3.91	.35	3.90	.35	3.95	.22	3.80	.32	3.82	.35
Response bias (C)																		
Probable AD	.07	.09	.03	.18	.08	.12	.15	.24	.22	.37	.31	.30	.19	.30	.22	.47	.26	.52
Possible AD	.18	.07	.16	.10	.16	.20	.30	.21	.32	.21	.22	.03	.13	.16	.12	.14	.23	.19
Controls	.21	.04	.21	.07	.21	.06	.21	.07	.23	.13	.21	.12	.22	.12	.21	.18	.25	.17
Response times (msec)																		
Probable AD	381	35	416	56	410	39	400	29	418	28	433	46	407	25	423	31	436	35
Possible AD	374	28	383	25	382	39	389	56	399	51	379	32	377	43	384	33	397	44
Controls	380	39	379	37	379	41	400	43	394	39	407	48	402	39	416	47	416	47

Note. Mean values for sensitivity, response bias, and response times, as a function of test block at each degradation level (2.00, 2.25, and 2.50) in probable AD, possible AD, and control participants. DL = degradation level.

TABLE 4

Means and Standard Deviations for Hits (%), False Alarms (%), Response Bias (C), and Response Times (msec) in Mild AD Patients and Control Participants

	Stimulus Degradation Level																	
	DL 2.00						DL 2.25						DL 2.50					
Test Block	1		2		3		1		2		3		1		2		3	
	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD	M	SD
Hits (%)																		
Mild AD	.96	.004	.96	.01	.94	.04	.89	.12	.87	.15	.91	.11	.93	.07	.90	.11	.86	.12
Control	.97	.004	.96	.01	.96	.01	.96	.01	.95	.05	.95	.05	.96	.03	.95	.04	.94	.05
False alarms (%)																		
Mild AD	.02	.01	.03	.04	.04	.04	.04	.03	.04	.03	.03	.02	.04	.03	.05	.04	.06	.05
Control	.01	.01	.01	.01	.01	.003	.02	.01	.02	.01	.02	.01	.02	.01	.02	.02	.02	.01
Response bias (C)																		
Mild AD	.12	.10	.09	.15	.12	.16	.22	.23	.27	.29	.27	.21	.16	.23	.17	.33	.24	.37
Control	.21	.04	.21	.07	.21	.06	.21	.07	.23	.13	.21	.12	.22	.12	.21	.18	.25	.17
Response times (msec)																		
Mild AD	378	30	400	45	396	40	395	43	409	40	406	47	392	37	404	36	417	43
Control	380	39	379	37	379	41	400	43	394	39	407	48	402	39	416	47	416	47

Note. Mean values for hits, false alarms, response bias, and response times, as a function of test block at each degradation level (2.00, 2.25, and 2.50) in mild Alzheimer's Disease (AD) patients and in control participants. DL = degradation level.

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TABLE 5

Correlations Between Vigilance Performance and Standard Tests of Cognitive Function in Mild Alzheimer's Disease (AD) and in Control Participants

Test	Overall Vigilance		Vigilance Decrement	
	Controls[a]	AD[b]	Controls[a]	AD[b]
General cognitive function				
WFSIQ	.15	.54	.35	−.03
WVIQ	−.02	.43	.10	−.03
WPIQ	−.04	.65*	−.14	−.008
WVSSS	−.04	.50	.02	.03
WPSSS	.24	.73*	.31	.07
Attention				
Stroop Words	.22	.45	−.004	−.008
Stroop Color	.008	.44	.03	−.22
Stroop Interference	.09	−.01	.10	−.09
Trail Making A (time)	−.27	−.61	−.09	−.05
Trail Making B (time)	.03	−.60	.11	.17

Note. Overall vigilance represents the mean level of global sensitivity for the degradation level 2.50. Vigilance decrement represents the decrement at degradation level 2.50; the vigilance decrement was calculated as the difference between mean sensitivity on Blocks 5 and 6 versus mean sensitivity on Blocks 1 and 2. WFSIQ = Wechsler Adult Intelligence Scale (WAIS) Full Scale IQ; WVIQ = WAIS Verbal IQ; WPIQ = WAIS Performance IQ; WVSSS = WAIS Verbal Sum of Scaled Scores; WPSSS = WAIS Performance Sum of Scaled Scores.

[a] $n = 20$.

[b] $n = 10$.

* $p < .05$.