Neuropsychology
1997, Vol. 11, No. 1, 13–29

0894-4105/97/$3.00

Inhibition of Return and Visuospatial Attention in Healthy Older Adults and Individuals With Dementia of the Alzheimer Type

Mark E. Faust and David A. Balota
Washington University

Covert orienting of visuospatial attention in response to peripherally presented cues was assessed in healthy younger and older adults and those with dementia of the Alzheimer type (DAT) during a simple detection task. The results yield both an age-related increase (Experiments 1 and 2) and a DAT-related increase (Experiment 2) in the facilitatory effect of a single peripheral cue on detection. By contrast, equivalent inhibition of return (i.e., a slowing of target detection at previously cued locations) was observed for all 3 groups when a 2nd cue was presented at central fixation. Results suggest that both healthy older adults and individuals with DAT experience changes in the posterior attention system thought to subserve visuospatial attention. Results also suggest limitations on the generality of inhibitory deficits in healthy aging and DAT.

One important function of visuospatial attention is to orient attention to a particular location in the visual field in response to the appearance of an object (e.g., Posner, 1980). The visuospatial attention system can be thought of as a filter, enhancing detection of stimuli at locations that are currently attended (e.g., Bashinski & Bacharach, 1980; Hawkins et al., 1990; Müller & Findlay, 1988) and inhibiting detection of stimuli at locations that were previously attended (i.e., inhibition of return; e.g., Posner & Cohen, 1984; Posner, Rafal, Choate, & Vaughan, 1985). Although a number of studies have examined age-related changes in aspects of selective attention such as selection for action (e.g., Spieler, Balota, & Faust, 1996), susceptibility to distracting information from to-be-ignored locations, sensory modalities (e.g., Hartley, 1993; McDowd, Oseas-Kreger, & Filion, 1995), or visual search (e.g., Plude & Doussard-Roosevelt, 1989), a much smaller number of studies have examined visuospatial orienting in healthy older adults (see Hartley, 1992, for a recent review). Furthermore, few researchers have used experimental tasks to examine dementia of the Alzheimer type (DAT)-related changes in selective attention in general, and fewer still have specifically examined visuospatial orienting (see Nebes, 1992, and Parasuraman & Haxby, 1993, for recent reviews).

In this article we explore automatic covert orienting of visuospatial attention in healthy older adults and individuals with DAT. In two experiments, we measured cuing effects to assess how well the appearance of a peripheral stimulus would capture visuospatial attention and to assess the strength of bias against reorienting attention to a location that had just been attended to (i.e., inhibition of return). Assessment of the bias against reorienting attention in healthy older adults and DAT individuals will provide important tests of hypotheses regarding a relative preservation of the posterior attention system underlying visuospatial attention and a generalized breakdown in inhibitory mechanisms in these populations.

Mark E. Faust and David A. Balota, Department of Psychology, Washington University.

The research reported in this article was conducted while Mark E. Faust was a research associate at the Department of Psychology and a research fellow at the Alzheimer's Disease Research Center (ADRC; P50 AG05681). Funding was provided by Grants R01 AG10193 and P01 AG03991 from the National Institute on Aging and by ADRC gift funds. We thank Kris Kuhlemeier and Renee Frederickson for their help in testing the participants and Richard Ferraro for assistance in the planning stages of Experiment 2. We also thank Leonard Berg and John Morris and the physicians associated with the ADRC for providing their time and expertise in diagnosing individuals with dementia of the Alzheimer type and Martha Storandt, J. Philip Miller, and Betsy Grant for providing descriptive psychometric data on the older individuals. Finally, we thank Jay Pratt and Alan Hartley for comments on drafts of this article.

Correspondence concerning this article should be addressed to Mark E. Faust, who is now at the Department of Psychology, University of South Alabama, Life Sciences Building, Room 320, Mobile, Alabama 36688-0002.

Automatic Covert Orienting of Visuospatial Attention

Although visuospatial attention and gaze are typically oriented toward the same location (e.g., Abrams & Dobkin, 1994; Remington, 1980), visuospatial attention can be oriented covertly in the absence of overt eye movements (e.g., Posner, 1980; Posner, Nissen, & Ogden, 1978). In fact, numerous studies have shown that when participants are asked to keep gaze constant, relatively automatic and involuntary covert shifts of visuospatial attention are induced by a luminance increment (i.e., an exogenous cue) in the periphery (e.g., Jonides, 1981; Jonides & Yantis, 1988; Müller & Findlay, 1988; Müller & Rabbitt, 1989). Under these conditions, participants typically are faster to detect a target that appears at an exogenously cued location than when it appears at an uncued location (e.g., Eriksen & Hoffman, 1973; Eriksen & Yeh, 1985; Jonides, 1981; Posner, 1980). Furthermore, studies of younger adults have shown that the advantage of cued locations over uncued

locations appears quickly (typically within 100 ms) and decreases over time with somewhat longer cue–target onset asynchronies in the 300- to 500-ms range (e.g., Maylor, 1985; Müller & Findlay, 1988; Müller & Rabbitt, 1989; Posner & Cohen, 1984). This peripheral cuing effect is an indication of the ability to automatically orient visuospatial attention to a location.

Inhibition of Return

Covert orienting of attention in response to peripheral visual cues does not always result in faster detection of targets at cued locations. For example, when enough time is allowed to elapse before presentation of the target (e.g., Maylor, 1985; Posner & Cohen, 1984), or a second cue is presented in a new location (e.g., Posner et al., 1985; Rafal, Calabresi, Brennon, & Sciolto, 1989), detection of targets is typically slowed for targets presented in originally cued locations. This decrement in the ability to detect targets at a previously covertly attended location has been thought to reflect a bias in the visuospatial attention system to not return attention to previously attended locations and, accordingly, has been termed *inhibition of return* (e.g., Abrams & Dobkin, 1994; Berlucchi, Tassinari, Marzi, & Di Stefano, 1989; Maylor & Hockey, 1987; Posner & Cohen, 1984).

As noted earlier, inhibition of return has been observed after intrinsic and extrinsic orientation of attention away from an originally cued peripheral location. Inhibition of return observed after long cue–target intervals, in the absence of any intervening stimuli, presumably is attributable to a spontaneous, intrinsically generated orienting of attention back to central fixation (e.g., Abrams & Dobkin, 1994; Maylor, 1985; Posner & Cohen, 1984; Remington, 1980). By contrast, inhibition of return after a second exogenous cue presumably is attributable to attention being reflexively drawn away from the originally cued peripheral location (e.g., Posner et al., 1985; Rafal et al., 1989). In either case, the peripheral location becomes a previously attended location after a reorientation of attention back to fixation, and inhibition of return then is observed.

Neural Systems and Visuospatial Attention

Recent evidence indicates that a posterior attention system (see Posner & Petersen, 1990, for a review) associated with the parietal cortex, thalamus, and superior colliculus is associated with orienting of visuospatial attention. For example, lesions of the thalamus result in deficits in focusing or engaging attention (Rafal & Posner, 1987); lesions of the posterior parietal lobe result in deficits in disengaging attention (Posner, Walker, Friedrich, & Rafal, 1984); and lesions of the superior colliculus and surrounding midbrain areas result in deficits in shifting of attention (Posner, Cohen, & Rafal, 1982; Posner et al., 1985). Moreover, inhibition of return also has been associated with the posterior attention system, specifically with midbrain structures (e.g., the superior colliculus, basal ganglia, and substantia nigra) associated with generating saccades, and the tectopulvinar visual pathway that projects from the retina to the superior colliculus, pulvinar, and parietal cortex (e.g., Clohessy, Posner, Rothbart, & Vecera, 1991; Posner et al., 1985; Rafal et al., 1989; Rafal, Henik, & Smith, 1991).

Visuospatial Attention in Healthy Aging

Several studies have shown age-related differences in the ability to localize targets under conditions of distraction during visual search (e.g., Plude & Doussard-Roosevelt, 1989; Plude & Hoyer, 1986) and in aspects of controlled orienting of attention using central symbolic cues such as rightward or leftward pointing arrows (e.g., Greenwood, Parasuraman, & Haxby, 1993; Hartley, Kieley, & Slabach, 1990; Hoyer & Familiant, 1987; Madden, 1983; Nissen & Corkin, 1985). Interestingly, however, reflexive orienting in response to peripheral cues has been found to be relatively well preserved in healthy older adults (e.g., Greenwood et al., 1993; Hartley, 1993; Hartley & Kieley, 1995, Experiments 1 and 2; Hartley et al., 1990, Experiment 3; Robinson & Kertzman, 1990), except when visual processing of distractors is required (e.g., Madden, 1990, 1992; Madden, Connelly, & Pierce, 1994).

The results of studies of inhibition of return in healthy older adults are somewhat less clear. Connelly and Hasher (1993, Experiment 1) found an inhibition of return effect in younger adults. They also found that healthy older adults were actually faster to respond when a target repeated its location across successive displays, an effect in opposition to inhibition of return. Note that there were no explicit task demands in their study requiring participants to return attention to the central location. Therefore, if the older adults typically did not choose to reorient to the central fixation between displays, they would be expected to show a facilitation when the target repeated locations (i.e., repetition priming; Pratt, 1996). More recently, Hartley and Kieley (1995, Experiments 1 and 2) reported equivalent inhibition of return effects in younger and older adults using a more traditional double-cue paradigm in which attention was drawn explicitly back to central fixation by a second cue.

The fact that healthy older adults produce similar peripheral cue effects and similar inhibition of return effects has led to the view that the posterior attention system thought to underlie visuospatial attention is relatively well preserved in healthy aging (e.g., Hartley, 1993). By contrast, the breakdowns in inhibitory mechanisms associated with selection for action and the anterior attention system (e.g., prefrontal cortex, thalamus, and anterior cingulate cortex; Corbetta, Miezin, Shulman, & Petersen, 1993; Pardo, Pardo, Janer, & Raichle, 1990) seem to be more pronounced (e.g., Dempster, 1992; Hartley, 1993; Spieler et al., 1996). However, given the few studies examining automatic covert orienting in healthy older adults, as well as the conflicting results of Connelly and Hasher (1993) and Hartley and Kieley (1995), further exploration of automatic covert orienting in healthy older adults seems warranted.

Visuospatial Attention in DAT

It is more difficult to assess the current state of knowledge of visuospatial attention in DAT (see Parasuraman & Haxby,

1993, for a recent review). There is growing literature documenting fairly large deficits in aspects of selective attention thought to be mediated by an anterior attention system (e.g., Fletcher & Sharpe, 1986; Lezak, 1995; Spieler et al., 1996). However, as might be predicted from the finding of parietal lobe degeneration early on in DAT (e.g., Haxby et al., 1986), there do seem to be DAT-related changes in visuospatial attention. Three studies have reported that DAT individuals produced larger cue effects than healthy older adults when centrally presented arrows (i.e., controlled orienting of attention) were used (Nissen, Corkin, & Growdon, 1981; Oken, Kishiyama, Kaye, & Howieson, 1994; Parasuraman, Greenwood, Haxby, & Grady, 1992; cf. Freed, Corkin, Growdon, & Nissen, 1989). However, we could find only one study in the literature that examined peripheral cuing in DAT. Parasuraman et al. found increased peripheral cue effects (i.e., reflexive orienting of attention) in DAT individuals, but only when participants performed a letter discrimination task, not when they performed a simple detection task.

Therefore, the available evidence supports the conclusion that individuals with DAT suffer from a breakdown in automatic orienting of visuospatial attention under some conditions. Such evidence also supports the suggestion that DAT-related changes in visuospatial attention may not be as great as those reported for other aspects of selective attention (e.g., Spieler et al., 1996). However, it is difficult to directly compare deficits across tasks. Nevertheless, because there are no published reports of inhibition of return in DAT, to our knowledge, an examination of inhibition of return in DAT would provide important converging evidence regarding the relative preservation of the posterior attention system in DAT.

Inhibition of Return and Inhibitory Function

In addition to providing converging evidence about the relative preservation of the (posterior) visuospatial attention system in healthy older adults and DAT individuals with DAT, we designed our experiments to examine issues regarding inhibitory changes in healthy aging and DAT. The phenomenon of inhibition of return is relevant to recent proposals that normal aging, and DAT, is marked by decrements in inhibitory processes. Hasher and Zacks (1988; Zacks & Hasher, 1994) proposed that normal aging results in declines in inhibitory cognitive processes. There are many demonstrations of inhibitory breakdowns in normal aging across a wide range of experimental paradigms (e.g., Duchek, Balota, Faust, & Ferraro, 1995; Hamm & Hasher, 1992; Hartman & Hasher, 1991; Hasher, Stoltzfus, Zacks, & Rypma, 1991; McDowd et al., 1995; Spieler et al., 1996; Zacks & Hasher, 1994).

The results of a growing number of studies have suggested that breakdowns in inhibitory processing also may play an important role in cognitive changes that occur during the early stages of DAT. Studies of intrusion and perservative errors (e.g., Butters, Granholm, Salmon, Grant, & Wolfe, 1987; Fuld, Katzman, Davies, & Terry, 1982; Loewenstein et al., 1991), selective attention (e.g., Faust, Balota, & Ducheck, 1995, 1996; Spieler et al., 1996; Sullivan, Faust, & Balota, 1995), word reading and rhyme decisions (e.g., Balota & Duchek, 1991; Balota & Ferraro, 1993, 1996), and sentence comprehension (e.g., Faust, Balota, Duchek, Gernsbacher, & Smith, in press) have provided evidence consistent with the notion that individuals with DAT suffer from breakdowns in inhibitory processes above and beyond those produced by healthy aging. For the most part, these studies have concentrated on relatively high-level inhibitory processes that presumably are responsible for deactivating potentially distracting task-irrelevant information. It is unclear whether these inhibitory breakdowns will be as apparent for tasks associated with the posterior visuospatial attention system, where inhibitory mechanisms should be tied more to the selective filtering of information for further processing.

Inhibition of Return and Visual Search

Finally, an examination of inhibition of return in healthy older adults and DAT individuals also may provide an important preliminary step toward a better understanding of impaired visual search in these populations (e.g., Madden & Plude, 1993; Panicker, Greenwood, Parasuraman, & Haxby, 1993; Plude & Doussard-Roosevelt, 1989; Plude & Hoyer, 1986). However, even though inhibition of return is generally thought to reflect a bias against attending to a previously attended location under conditions of exogenous peripheral cues (Posner & Cohen, 1984; Rafal et al., 1989), it is unclear how inhibition of return is involved in more global visuospatial functioning such as visual search (e.g., Klein, 1988, 1989; Tipper, 1992; Tipper, Driver, & Weaver, 1991; Tipper, Weaver, Jerreat, & Burak, 1994; Wolfe & Pokorny, 1990).

Summary

The results of studies assessing automatic visuospatial orienting in healthy older adults (e.g., Greenwood et al., 1993; Hartley, 1993; Hartley et al., 1990, Experiment 3; Robinson & Kertzman, 1990), suggest that the posterior attention system responsible for reflexive covert orienting of visuospatial attention is relatively well preserved in healthy aging, at least relative to other aspects of selective attention thought to depend more on an anterior attention system (e.g., Dempster, 1992; Hartley, 1993; Spieler et al., 1996). Although there is some evidence for breakdowns in the posterior attention system in DAT (e.g., Parasuraman & Haxby, 1993), breakdowns in other aspects of selective attention that depend on an anterior attention system appear to be more impaired in individuals with DAT (e.g., Fletcher & Sharpe, 1986; Spieler et al., 1996).

Turning to the phenomenon of inhibition of return, although the results of the most extensive study to date suggest little in the way of age-related changes in inhibition of return (Hartley & Kieley, 1995), there is some inconsistency in the literature regarding the possibility of age-related differences in inhibition of return effects in healthy older adults (e.g., Connelly & Hasher, 1993), and there are no published studies of inhibition of return in DAT. Age- or

DAT-related changes in inhibition of return would suggest that proposals of relative preservation of visuospatial attention in these groups would need to be reevaluated. Furthermore, it has been proposed that healthy older adults (e.g., Hasher & Zacks, 1988; McDowd et al., 1995; Zacks & Hasher, 1994) and individuals with DAT might suffer from a generalized breakdown in inhibitory function (e.g., Balota & Ferraro, 1993, 1996; Faust et al., in press). An examination of inhibition of return in healthy aging and DAT will provide a test of the boundary conditions of this hypothesis. We therefore examined the timecourse of cue validity and inhibition of return effects following a peripheral cue in younger adults, healthy older adults, and individuals with DAT both in the absence (i.e., intrinsic reorienting; Experiment 1) and the presence (i.e., extrinsic reorienting; Experiment 2) of a second exogenous cue.

Experiment 1

Experiment 1 was designed to address peripheral cuing effects in a simple detection task for healthy older adults and individuals with DAT (e.g., Greenwood et al., 1993; Parasuraman et al., 1992) and, more important, to examine the decline of peripheral cuing effects over time and the possible emergence of inhibition of return effects in these groups following intrinsic reorientation of attention. To do this, we used a variant of a task developed by Posner and colleagues (Posner, 1980; Posner & Cohen, 1984; Posner et al., 1978). Participants fixed their gaze on a central location and detected the appearance of targets in one of two peripherally located boxes following a brightening of the outline of one of the boxes. On most trials the target appeared in the cued location (i.e., the valid condition), and on the remaining trials the target appeared in the box on the contralateral side (i.e., the invalid condition). Also included as a control condition were trials in which both peripheral locations were cued.

To assess the time course of peripheral cuing effects, we varied the interval between the onset of the cue and the onset of the target (stimulus onset asynchrony [SOA]), with 40% of the trials having a 100-ms SOA and 40% having an 800-ms SOA. The final 20% of the trials had a 1,500-ms SOA, which acted as de facto catch trials (i.e., trials without a target) for the shorter SOA conditions. At the short (100-ms) SOA, the cue was expected to facilitate target detection (Posner, 1980). To the extent that participants spontaneously reoriented attention back to central fixation at the long (800-ms) SOA, cue validity effects should decrease and inhibition of return effects should emerge.

Method

Participants

Thirty-five individuals with DAT and 51 healthy older adults were recruited from the Washington University Medical School Alzheimer's Disease Research Center (ADRC). An additional 25 younger adults (aged 25 years or less) were recruited from the Washington University community and were paid $10 for their effort. Healthy older adults and individuals with DAT were seen by a physician and were screened for the neurological, psychiatric, and medical disorders that could cause dementia. The inclusionary and exclusionary criteria for a diagnosis of DAT have been described in detail elsewhere (e.g., Morris, McKeel, Fulling, Torack, & Berg, 1988) and conformed to those outlined in the criteria of the work group of the National Institute of Neurological and Communicative Disorders and Stroke and the Alzheimer's Disease and Related Disorders Association (McKhann et al., 1984). Diagnostic accuracy for Alzheimer's disease has been reported to be high (e.g., 96%; Alzheimer's disease was confirmed in 102 of 106 consecutive autopsies in individuals with DAT; Berg & Morris, 1994) when these criteria were used.

Dementia severity for each individual with DAT recruited from the ADRC was staged according to the Washington University Clinical Dementia Rating (CDR) scale (Hughes, Berg, Danziger, Coben, & Martin, 1982; Morris, 1993). According to this scale, a score of 0 indicates no cognitive impairment, a score of 0.5 indicates questionable or very mild dementia, a score of 1 indicates mild dementia, and a score of 2 indicates moderate dementia. At the ADRC, a CDR 0.5 rating has been found to accurately indicate the earliest stages of DAT (Morris et al., 1991).

Data from 2 individuals with DAT were removed because more than 15% of their responses were slower than the 2,000-ms cutoff set for this experiment (see the description of outlier screening procedures in the *Results* section). Furthermore, there was one anomalous individual in the DAT group whose cue validity effect was more than 4 *SD*s from the overall mean in the negative direction (i.e., this participant was markedly slower on valid than invalid trials). Freed et al. (1989) also found similar anomalous responders among individuals with mild DAT using a similar task and argued for a subtype of attentional breakdown in DAT. Because we could only identify one such individual, we chose to drop this anomalous responder from the analyses.

The DAT group consisted of 18 individuals with very mild DAT and 14 with mild DAT. The results from 25 younger adults, 51 healthy older adults, and 32 individuals with DAT were retained for analysis. The young group had a mean age of 21.2 years (SD = 2.6, range = 18–25), the healthy old group had a mean age of 76.7 years (SD = 8.6, range = 61–90), and the DAT group had a mean age of 72.4 years (SD = 7.3, range = 54–95).

Psychometric Test Performance

Eighty-one of the 83 participants recruited from the ADRC whose response latency data met inclusionary criteria also participated in a 2-hr battery of psychometric tests designed to assess psychological functions, including language, memory, and intelligence. Table 1 shows the results of a subset of the tasks included in this battery as a function of group (i.e., healthy old or DAT). Because some participants did not finish some of the tasks, the sample size varied somewhat across tasks. Memory was assessed with the Wechsler Memory Scale (WMS; Wechsler & Stone, 1973) Associates subscale (paired-associates learning) and the Logical Memory subscale (surface-level story memory). General intelligence was measured with the Information and Digit Symbol subtests of the Wechsler Adult Intelligence Scale (WAIS; Wechsler, 1955). Participants also received the Word Fluency test, on which they were required to name as many words beginning with a specified letter (P or S) in a 60-s time period (Thurstone & Thurstone, 1949). Participants also completed the Boston Naming Test (Kaplan, Goodglass, & Weintraub, 1983). As shown in Table 1, the performance of the participants with DAT was poorer than that of the healthy old group on all tests. Because the young participants were recruited from another source, they were not given the psychometric battery.

Materials and Design

All stimuli were presented by an IBM AT-compatible computer fitted with a VGA graphics card on a standard VGA monitor in 640 × 350 pixel mode. Participants viewed displays at an approximate distance of 75 cm. The basic display consisted of two white boxes (1.75 cm on each side, or 1.3° of visual angle) on a dark background, centered vertically on the monitor, and presented 8.25 cm (6.25°) to the right and left of center. A central fixation cross (1 cm or 0.75°) also was presented. The peripheral boxes and central fixation cross were always visible. Cues were presented by tripling the width of the lines forming one of the boxes. The target was a highly visible red asterisk (0.75 cm or 0.6°) centered in one of the boxes, and it remained visible until a response was made. The displays were similar to those of Posner and Cohen (1984), but with two important modifications: Posner and Cohen (1984) included a central box instead of the central cross used in Experiment 1, and we allowed the peripheral cues (i.e., the brightening of the peripheral boxes) to remain visible until response onset or the 3,000-ms display limit.

The cue–target SOA was either 100 ms (40%), 800 ms (40%), or 1,500 ms (20%). As noted earlier, the 1,500-ms SOA trials were designed to act as de facto *catch* trials (Posner, 1980; Posner & Cohen, 1984). Because their purpose was to reduce anticipatory responses by the participants, we did not analyze them. Figure 1 depicts the succession of events in the conditions of Experiment 1.

There were six cue–target combinations: (a) valid right, both the cue and the target were on the right (30%); (b) valid left, both the cue and the target were on the left (30%); (c) invalid right, the cue was on the left and the target was on the right (10%); (d) invalid left, the cue was on the right and the target was on the left (10%); (e) both right, the cues were simultaneously on both sides and the target was on the right (10%); and (f) both left, the cues were simultaneously on both sides and the target was on the left (10%). Thus, the cue validity, based only on the valid and invalid cue trials, was 75%. The both-cue trials were included as a neutral control on which to base a cost–benefit analysis (Posner, 1980, 1982; cf. Jonides & Mack, 1984).

Table 1
Scores on Selected Psychometric Tests for Healthy Older Adults and Individuals With DAT in Experiment 1

Test measure	Group: Healthy old (n = 51)	Group: DAT (n = 30)[a]	F	df
WMS Associates				
M	13.69	9.76	14.56**	1,78
SD	4.51	4.26		
WMS Logical Memory				
M	8.99	5.33	18.49**	1,79
SD	3.57	3.91		
WAIS Information				
M	21.03	15.98	18.82**	1,79
SD	4.45	5.96		
WAIS Digit Symbol				
M	47.63	34.18	21.57**	1,79
SD	10.76	15.23		
Word Fluency				
M	31.58	25.90	5.19*	1,79
SD	11.39	9.81		
Boston Naming Test				
M	55.02	46.07	15.17**	1,79
SD	5.37	14.91		

Note. DAT = dementia of the Alzheimer type; WMS = Wechsler Memory Scale; WAIS = Wechsler Adult Intelligence Scale.
[a]WMS Associates included 29 individuals with DAT.
*$p < .05$. **$p < .001$.

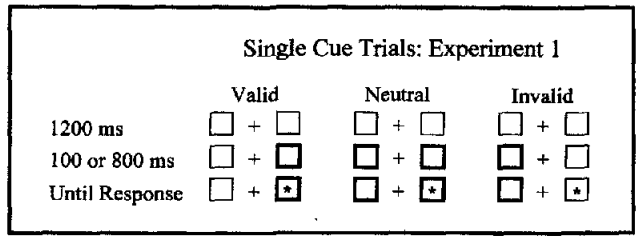

Figure 1. Depiction of the displays and their timing for the single peripheral cue trials included in Experiment 1.

There were seven blocks of 50 trials each, 120 trials at each of the experimental (i.e., 100- and 800-ms) SOAs and 60 trials at the 1,500-ms SOA, which acted as de facto catch trials for the shorter SOAs. The first block was viewed as practice and was therefore not analyzed. There were 12 experimental observations for both the invalid and neutral conditions in each of the four within-subjects cells that were produced by fully crossing target position (i.e., right or left) and SOA (i.e., 100 or 800 ms). There also were 36 valid-cue experimental observations for each of the four within-subjects cells produced by fully crossing target position and SOA.

Procedure

The task was explained verbally to participants. Participants sat approximately ¾ m from the computer monitor and pressed the left mouse button with the index finger of their right hand on detection of the target (red asterisk) while keeping their gaze fixated on the central fixation cross. Eye movements were monitored by the experimenter via a magnifying cosmetic mirror mounted on top of the monitor. Trials on which visible eye movements were detected were removed from the response latency analysis. There was a short break between each block of 50 trials. Each block contained all conditions in their proper proportions randomly intermixed. Overall task time was approximately 35–40 min. This task was part of a 2-hr battery.

The basic display containing the central fixation cross and the two peripheral boxes was visible throughout a block of trials. Each trial began with the onset of a cue display (cues remained visible throughout the trial). The target appeared in one of the boxes either 100 ms, 800 ms, or 1,500 ms after the onset of the cue display and remained until a response was made or 3,000 ms elapsed, whichever came first. Although targets remained visible for 3,000 ms, the predetermined cutoff for responses was 2,000 ms, and all responses greater than 2,000 ms (i.e., the timeout criterion) were removed from further consideration in analyses of response latency. Following a response, the screen then was returned to the basic display for a 1,200-ms intertrial interval. If participants responded before the onset of the target or within 100 ms following the onset of the target (i.e., an early response), or after 3,000 ms following the onset of the target (i.e., a timeout), an error message was presented on the screen for an additional 700 ms just before the intertrial interval.

Results

Response latencies for trials on which an early response, a timeout, or a visible eye movement occurred were removed from further consideration (see the *Procedure* section). We

then computed an overall mean and standard deviation for each participant and removed any responses more deviant than 2.5 *SD*s from the mean. The removal of early and timeout responses resulted in removal of 4.3%, 4.9%, and 6.4% of the experimental trials for the young, healthy old, and DAT groups, respectively. Using the remaining correct responses, we calculated the median for each participant in each experimental condition and submitted these values to further statistical analyses.

General Analysis of Response Latencies

The mean and standard deviation of participants' median response latencies for the experimental trials are presented in Table 2. There was an overall main effect of group, $F(2, 105) = 24.72$, $p < .001$. The young group ($M = 369$ ms) was faster overall than the healthy old group ($M = 479$ ms), $F(1, 74) = 43.07$, $p < .001$, which in turn was marginally faster than the DAT group ($M = 517$ ms), $F(1, 81) = 3.53$, $p = .064$. The long SOA condition yielded faster latencies than the short SOA condition, $F(1, 105) = 153.57$, $p < .001$. Responses to targets presented in the left box ($M = 471$ ms) were slower than when targets were presented in the right box ($M = 459$ ms), $F(1, 105) = 12.45$, $p = .001$. Furthermore, response latencies decreased across SOAs more for targets presented in the right box (51 ms) than for those presented in the left box (42 ms), $F(1, 105) = 5.25$, $p = .024$. Target position interacted marginally with group, $F(2, 105) = 2.69$, $p = .073$, with the DAT group ($M = 17$ ms), $F(1, 50) = 14.07$, $p < .001$, and the healthy old group ($M = 11$ ms), $F(1, 31) = 2.15$, $p = .153$, detecting targets faster in the right box than the left. By contrast, the young group exhibited little lateral asymmetry ($M = -1$ ms), $F(1, 24) = 0.28$, $p = .599$. Given that the overall target position effect was a small one ($M = 11$ ms) and did not interact with any combination of variables containing the cue validity variable (all Fs < 1.2), we collapsed across the variable of target position in the analyses of cue effects that follow.

Cue Validity Effects

Costs and benefits. Both costs (i.e., the slowing of detection in the invalid vs. the neutral conditions), $F(1, 105) = 30.17$, $p < .001$, and benefits (i.e., the facilitation of detection in the valid vs. the neutral conditions), $F(1, 105) = 22.20$, $p < .001$, decreased with SOA. There were significant overall costs ($M = 27$ ms), $F(1, 105) = 40.47$, $p < .001$, and significant overall benefits ($M = 20$ ms), $F(1, 105) = 49.41$, $p < .001$, at the 100-ms SOA, but no costs or benefits at the 800-ms (Ms = 0 and 3-ms SOA, respectively; all Fs < 1). More important, there were no interactions of costs or benefits with group at the 100-ms SOA (all Fs < 1.97, ps $> .19$). Thus, there were relatively equivalent costs and benefits at the 100-ms SOA that did not vary significantly across groups. We therefore turn to an analysis of overall cue validity effects (i.e., costs plus benefits), avoiding the potential problems associated with interpreting neutral conditions in cued attention tasks (e.g., Jonides & Mack, 1984).

Overall cue validity effects (costs plus benefits). Figure 2 shows response latency as a function of group (i.e., young, healthy old, and DAT), cue validity (i.e., valid vs. invalid), and cue–target SOA (i.e., 100 or 800 ms). Valid trials were faster than invalid trials, $F(1, 105) = 54.28$, $p < .001$. Cue validity interacted with group, $F(2, 105) = 4.22$, $p = .017$, and with SOA, $F(1, 105) = 89.86$, $p < .001$. Validly cued targets were detected faster than invalidly cued targets (47 ms) at the short SOA, $F(1, 105) = 134.49$, $p < .001$, but not at the long SOA (-3 ms; $F < 1$). Finally, there was a significant Group × SOA × Cue interaction, $F(2, 105) = 3.26$, $p = .042$, indicating that cue validity effects varied across SOAs differently for the different groups. Planned comparisons to determine the effects of age and DAT on the cue validity effects are reported next.

Age and cue validity effects. The overall Group (young vs. healthy old) × SOA × Cue interaction approached significance, $F(1, 74) = 3.69$, $p = .059$. Younger and healthy older adults detected validly cued targets faster than invalidly cued targets at the short (100-ms) SOA, $F(1, 74) = 93.61$, $p < .001$. This cue validity effect increased with age (Ms = 26 and 57 ms for younger and healthy older adults, respectively; see Figure 2), $F(1, 74) = 9.13$, $p = .003$. Furthermore, both groups produced significant facilitatory cue effects at the short SOA (all Fs > 18, all ps $< .001$). By contrast, both younger and healthy older adults detected invalidly cued targets faster than validly cued targets (i.e., inhibition of return; Ms = -13 and -7 ms for younger and healthy older adults, respectively) at the long (800-ms) SOA, $F(1, 74) = 4.43$, $p = .039$. The inhibition of return effect did not differ between groups ($F < 1$). However, when tested separately, the young group produced signifi-

Table 2
Participants' Median Response Latencies in Experiment 1

	100-ms SOA						800-ms SOA					
	Valid		Neutral		Invalid		Valid		Neutral		Invalid	
Group	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
Young	375	50	385	63	402	74	356	49	352	53	344	52
Healthy old	477	78	499	79	534	107	461	86	452	77	454	90
DAT	519	125	543	127	567	115	484	99	493	108	496	109

Note. SOA = stimulus onset asynchrony; DAT = dementia of the Alzheimer type.

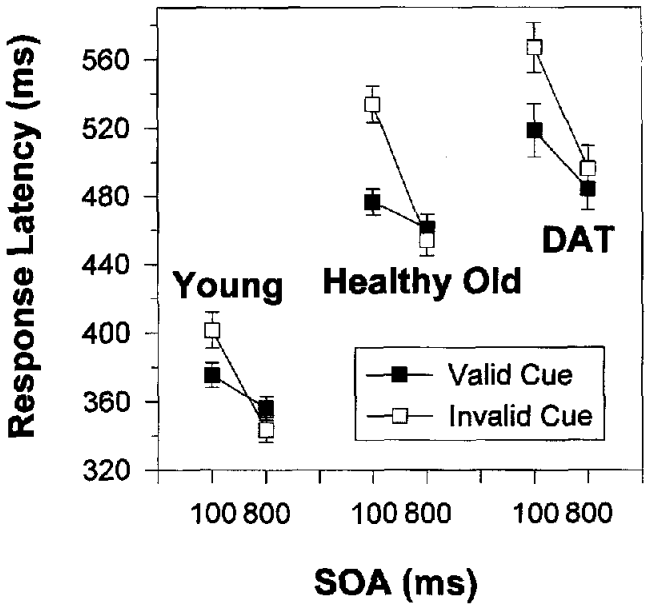

Figure 2. Mean of median response latencies as a function of cue validity, stimulus onset asynchrony (SOA), and group in Experiment 1. Error bars indicate standard errors. DAT = dementia of the Alzheimer type.

cant inhibition of return, $F(1, 24) = 8.13$, $p = .009$, but the healthy old did not ($F < 1.5$).

The fact that the test of the healthy old group in isolation did not yield significant inhibition of return should be viewed with caution because the power for detecting a 10- to 15-ms inhibition of return effect was approximately .50–.75. These results demonstrate clearly that both groups were able to take advantage of cues at the short SOA to improve detection of targets. Moreover, these results are consistent with the hypothesis that both younger and healthy older adults spontaneously reoriented attention back to fixation in the interval between the short and long SOAs in a similar manner.

DAT and cue validity effects. The overall Group (healthy old vs. DAT) × SOA × Cue interaction was significant, $F(1, 81) = 4.61$, $p = .035$. As can be seen in Figure 2, cue validity effects were reduced less across SOA for the individuals with DAT than for the healthy older adults. Both healthy older adults and individuals with DAT produced cue validity effects at the short SOA (*M*s = 57 and 48 ms, respectively), which differed from zero in a combined analysis, $F(1, 81) = 117.76$, $p < .001$, and did not differ across groups ($F < 1$). By contrast, healthy older adults produced a 7-ms inhibition of return effect, whereas individuals with DAT produced a 12-ms cue validity effect (i.e., valid targets faster than invalid targets) at the 800-ms SOA. These opposing effects were marginally different, $F(1, 81) = 3.51$, $p = .064$. These results suggest that individuals with DAT were relatively unimpaired in their ability to automatically orient covert attention but that they did not reorient attention as much as the healthy older adults in the 800-ms interval tested.

General Slowing and Cue Validity Effects

The absolute size of the cue effects for an individual was related to his or her overall speed of response at the short SOA ($r = .43$), $t(106) = 4.88$, $p < .001$. Thus, differences in overall speed among participants might have influenced the results. To correct for possible distortions of the response latency scale due to individual or group differences in overall speed (e.g., Faust, Balota, & Ferraro, 1996; Spieler et al., 1996), we divided each participant's condition medians by his or her overall mean. This transformation had the effect of scaling each individual's cue effect as a proportion of his or her overall mean. Cue validity effects, in terms of proportion of overall speed, are presented in Figure 3.

The analysis of proportional cue effects was essentially the same as that for the raw difference scores; therefore, only the analysis of raw latencies is presented fully. For example, the change in the proportional cue effect across SOAs for the DAT group (.077) was smaller than the change across SOAs for the healthy old group (.133) depicted in Figure 3, $F(1, 81) = 5.36$, $p = .023$. However, there was one important difference between the analysis of median latencies and proportions. Although the Group (healthy old vs. DAT) × SOA × Cue interaction was significant in both the median latency and proportion analyses, the Group (young vs. healthy old) × SOA × Cue interaction that was marginally significant when median response latencies were considered was not significant when proportional cue effects were considered, $F(1, 74) = 1.55$, $p = .217$, indicating that both younger and healthy older adults produced similar reductions in cue effects across SOAs when proportional cue effects were considered. The age-related increase in cue effects at the short SOA remained significant in the analysis of proportions, $F(1, 74) = 7.31$, $p = .008$.

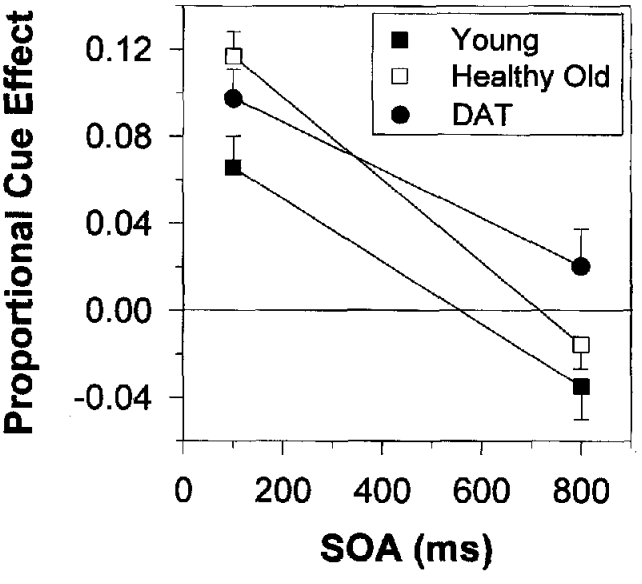

Figure 3. Proportional cue effect (i.e., invalid minus valid, divided by overall mean) as a function of stimulus onset asynchrony (SOA) and group for Experiment 1. Error bars indicate standard errors. DAT = dementia of the Alzheimer type.

Eye Movement Errors

Figure 4 shows the proportion of eye movement errors as a function of group. Overall, the groups differed in the amount of errors made, $F(2, 105) = 8.17, p < .001$. Planned comparisons revealed age-related increases in eye movement errors, $F(1, 74) = 6.62$, $p = .012$, and DAT-related increases in eye movement errors, $F(1, 81) = 5.43$, $p = .022$.

Discussion

All groups produced reliable costs and benefits at the short SOA, reflecting the benefit to target detection of a valid cue and the cost to detection of an invalid cue. Although no reliable group differences emerged for costs or benefits considered in isolation, the cue validity effect (i.e., costs plus benefits) increased with age but not dementia. This finding is consistent with a previous report of an age-related increase in peripheral-cue validity effects using a detection task (i.e., Hartley & Kieley, 1995, Experiment 1) but is inconsistent with the majority of studies that have reported no age-related differences in peripheral-cue effects but that have used discrimination tasks (e.g., Greenwood et al., 1993; Hartley et al., 1990). It is unclear under what conditions age-related increases in peripheral-cue effects will be observed or what components (i.e., costs or benefits) are more affected. Our results were based on nonsignificant trends toward age-related increases in both costs and benefits, which were statistically reliable only in an analysis of costs plus benefits.

Of greater interest was the extent to which cue validity effects decreased across SOAs and the extent to which inhibition of return emerged. The results are similar to those of previous studies (e.g., Posner & Cohen, 1984) in that we found cue validity effects at the short SOA that declined across SOAs for all groups. Cue validity effects decreased similarly over the 700-ms interval between the 100- and the 800-ms SOA for the younger and healthy older adults. Furthermore, the young and healthy old groups did not differ in performance at the 800-ms SOA. Therefore, a conservative interpretation of these results would hold that both groups spontaneously reoriented attention over time similarly.

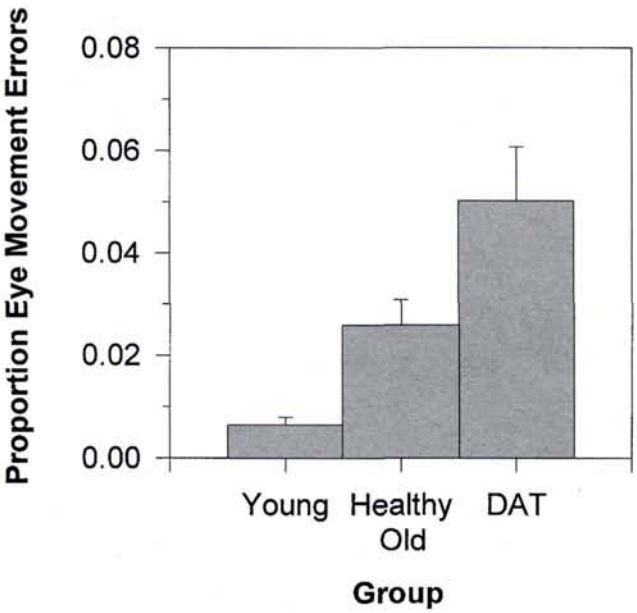

Figure 4. Proportion of eye movement errors as a function of group for Experiment 1. Error bars indicate standard errors. DAT = dementia of the Alzheimer type.

In contrast to the equivalent spontaneous reorientation of attention with age, cue validity effects were reduced to a lesser extent for individuals with DAT than for healthy older adults across SOAs when both raw latencies or proportions were considered. One potential explanation of this is that, as proposed by Parasuraman et al. (1992), the early stages of DAT results in a deficit in the ability to disengage attention. If individuals with DAT find it harder to disengage attention, they also would be slower to reorient attention. However, a specific deficit in the ability to disengage covert attention from a location should have produced DAT-related increases in costs (e.g., Posner et al., 1984), which were not observed in our results. (A breakdown in spontaneous reorientation in the absence of a stimulus [either cue or target] is not synonymous with a breakdown in the ability to disengage.) Spontaneous reorienting involves disengaging and shifting attention from one location to another and control mechanisms to initiate such operations in the absence of an external stimulus. Therefore, the results suggest that individuals with DAT are simply slower to spontaneously reorient in the absence of an exogenous stimulus to draw covert attention from a currently attended location.

Evidence of inhibitory breakdowns across groups was found in the analysis of eye movement errors. Participants in Experiment 1 were actually faced with two tasks. First, they were to detect targets while keeping their gaze fixated on a central location. However, given the reflexive nature of overt saccades toward objects that appear suddenly in the periphery (e.g., Rafal et al., 1991), this amounted to requiring a second task of inhibition of normal reflexive saccades. Consistent with proposals of breakdowns in inhibitory mechanisms in healthy aging and DAT (e.g., Faust et al., in press; Hasher & Zacks, 1988), failures to inhibit reflexive saccades increased both with age and DAT in Experiment 1. This result is consistent with studies demonstrating breakdowns in the control of eye movements in DAT (e.g., Fletcher & Sharpe, 1986). Eye movement errors are discussed further in the General Discussion section.

Experiment 2

In Experiment 1, individuals with DAT were slower than healthy older adults to spontaneously reorient attention. This amounted to a change in the strategic control of processes leading to inhibition of return rather than any breakdown in the inhibitory mechanisms underlying inhibition of return. As discussed in the introduction, there is evidence to indicate that both age and DAT result in a general breakdown in inhibitory mechanisms (e.g., Balota & Duchek, 1991; Balota & Ferraro, 1993, 1996; Faust et al., in press;

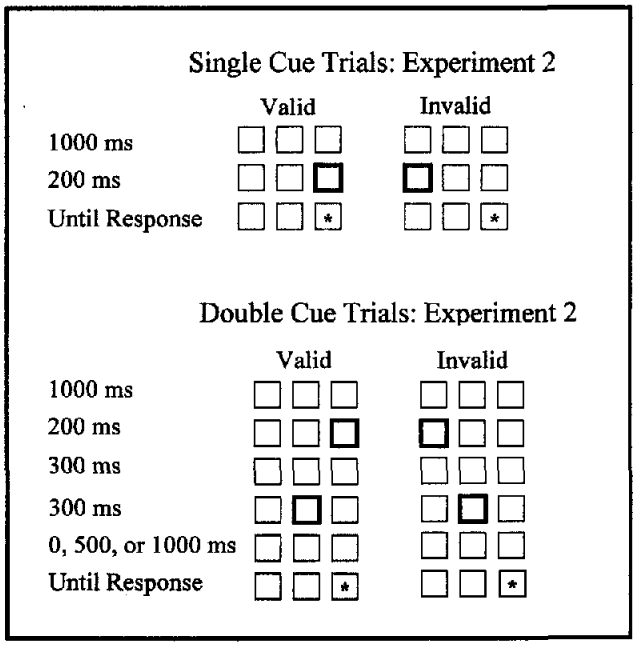

Figure 5. Depiction of the displays and their timing for the single- and double-cue trials included in Experiment 2. Single-cue trials included a brightening of a peripheral box, and double-cue trials included both the brightening of a peripheral box followed by a brightening of the central box.

Hasher & Zacks, 1988; Sullivan et al., 1995). Age- and DAT-related changes in inhibition of return would provide further evidence regarding general inhibitory breakdowns in these populations. On the other hand, preserved inhibition of return in healthy older adults and DAT individuals would support the view that the posterior attention system is relatively preserved in these populations and would provide information about the boundary conditions for inhibitory breakdowns.

To provide an explicit test of the inhibitory mechanisms underlying inhibition of return, we used a task previously reported by Posner et al. (1985; see also Rafal et al., 1989) in which a second exogenous cue was used to elicit reflexive reorienting of attention to central fixation. As in Experiment 1, participants fixated their gaze on a central location and detected the appearance of targets in one of two peripherally located boxes following a brightening of one of the two boxes (see Figure 5). On half the trials, the target appeared shortly after the peripheral cue, just as in Experiment 1. However, on the other half of the trials, a second cue (i.e., the brightening of the centrally located box) was presented. On these double-cue trials, the target appeared in the previously cued location half the time (i.e., valid double-cue trials) and the side opposite the other half of the time (i.e., invalid double-cue trials).

The single-cue trials were predicted to yield a traditional cue validity effect, and the double-cue trials were predicted to yield a reversal of the cue validity effect (i.e., inhibition of return) because of attention being drawn back to the center by the second cue. Once attention was reflexively reoriented to the center, the natural tendency for previously attended locations to be inhibited should have become apparent in subsequent detection performance (Posner et al., 1985).

Method

Participants

Twenty-five young adults (aged 25 years or younger), 48 healthy old adults, and 52 individuals with DAT were recruited using the same procedures as in Experiment 1. Using the same inclusion and exclusion criteria for Experiment 1, we removed data from 1 healthy old adult and 9 individuals with DAT because more than 15% of their responses were deemed too slow for further analysis. The young group had a mean age of 19.3 years (SD = 1.3, range = 17–23), the healthy old group had a mean age of 77.3 years (SD = 9.6, range = 58–93), and the DAT group had a mean age of 73.4 years (SD = 8.9, range = 53–91).

Psychometric Test Performance

Eighty-eight of the 90 participants recruited from the ADRC whose response latency data met inclusionary criteria also participated in a 2-hr battery of psychometric tests designed to assess psychological functions, including language, memory, and intelligence. Table 3 shows the results of a subset of the tasks included in this battery as a function of group (i.e., healthy old or DAT). Because some participants did not finish some of the tasks, sample size varied somewhat across tasks. Memory was assessed using the WMS Associates subscale (paired-associates learning) and the Logical Memory subscale (surface-level story memory). General intelligence was measured with the Information and Digit Symbol subtests of the WAIS. Participants also completed the Word Fluency test, on which they were required to name as many words

Table 3
Scores on Selected Psychometric Tests for Healthy Older Adults and Individuals With DAT in Experiment 2

Test measure	Group: Healthy old (n = 45)	Group: DAT (n = 43)[a]	F	dfs
WMS Associates				
M	14.88	7.59	89.14**	1,83
SD	3.82	3.22		
WMS Logical Memory				
M	9.62	3.67	85.38**	1,86
SD	2.91	3.13		
WAIS Information				
M	21.51	13.56	49.10**	1,86
SD	4.19	6.29		
WAIS Digit Symbol				
M	44.53	26.28	44.66**	1,83
SD	11.28	13.89		
Word Fluency				
M	32.07	18.52	43.17**	1,85
SD	10.71	8.26		
Boston Naming Test				
M	55.89	42.67	43.10**	1,86
SD	4.04	12.86		

Note. DAT = dementia of the Alzheimer type; WMS = Wechsler Memory Scale; WAIS = Wechsler Adult Intelligence Scale.
[a]WMS Associates and WAIS Digit Symbol included 40 individuals with DAT, and Word Fluency included 42 individuals with DAT.
**$p < .001$.

as they could beginning with a specified letter (*P* or *S*) in a 60-s time period, and the Boston Naming Test. As shown in Table 3, the performance of the DAT group was poorer than that of the healthy old group on all tests. Because the young were recruited from another source, they were not given the psychometric battery.

Materials and Design

The task used was similar to that of Posner et al. (1985; see also Rafal et al., 1989). The apparatus and displays were identical to those of Experiment 1, with three important exceptions. First, the peripheral boxes were placed closer to the central fixation box (i.e., 5.75 cm or 4.4°, instead of 8.25 cm or 6.25°). We did this to reduce the number of eye movement errors made by the DAT group. Second, the central fixation cross was replaced by a central box identical to the peripheral boxes. The brightening of the central box in double-cue trials was accomplished in the same manner that peripheral boxes were brightened. Third, unlike in Experiment 1, the cues used in Experiment 2 were of a fixed duration (i.e., peripheral cues with a 200-ms duration, central cues with a 300-ms duration, and a 300-ms uncued intervening interval for double-cue trials). Half the trials contained a single cue, and the other half included two cues: the first peripheral and the second central. Figure 5 depicts the succession of events in the various conditions of Experiment 2.

For single-cue trials, there were six cue–target combinations: (a) valid right, both the cue and the target were on the right (37.5%; 54 observations); (b) valid left, both the cue and the target were on the left (37.5%; 54 observations); (c) invalid right, the cue was on the left and the target was on the right (6.25%; 9 observations); (d) invalid left, the cue was on the right and the target was on the left (6.25%; 9 observations); (e) neutral right, there was no cue and the target was on the right (6.25%; 9 observations); and (f) neutral left, there was no cue and the target was on the left (6.25%; 9 observations). Thus, the cue validity, based only on the single-cue trials, was 85.7%. For the double-cue trials, there were four equally likely cue–target conditions: valid right (36 observations), valid left (36 observations), invalid right (36 observations), and invalid left (36 observations). Validity for double-cue trials was determined by the location of the first (peripheral) cue. Each double-cue condition had three equally likely SOAs (800, 1,300, or 1,800 ms following the onset of the first cue). There were four blocks of 96 trials each. The first block was viewed as practice and was not analyzed.

Procedure

The procedure was similar to that of Experiment 1. Participants performed a practice block of 96 trials that contained all conditions in their proper proportions randomly intermixed. Overall task time was approximately 35–40 min and consisted of four blocks of 96 trials each, with short intervening breaks. Experiments 1 and 2 were included as part of two different experimental batteries that were conducted approximately 1 year apart.

Figure 5 depicts the succession of events in the various conditions of Experiment 2. Each trial began with the onset of a basic display containing three boxes along the horizontal meridian. The onset of the first cue occurred 600 ms following onset of the three boxes (no cue was actually presented for neutral trials). The first cue lasted 200 ms. On half the trials (i.e., single-cue trials), the target appeared at the offset of the first cue (i.e., an SOA of 200 ms) and remained visible until a response occurred or the 10-s display limit was reached, whichever came first. On the other half of the trials (i.e., double-cue trials), a second cue was presented in the center box 300 ms following the offset of the first cue. The second central cue lasted 300 ms. The target then appeared either at the offset of the second cue (i.e., an SOA of 800 ms, in which the SOA is measured from the onset of the first cue), 500 ms later (i.e., an SOA of 1,300 ms), or 1,000 ms later (i.e., an SOA of 1,800 ms) and remained until a response occurred or the 10-s display limit was reached, whichever came first. The screen was then blanked for a 1,000-ms intertrial interval. As in Experiment 1, responses longer than 2,000 ms (i.e., the timeout criterion) were considered outliers and were removed from further consideration in response latency analyses.

Results

As in Experiment 1, response latencies for trials on which an early response, a timeout, or a visible eye movement occurred were removed from further consideration. We then computed an overall mean and standard deviation for each participant and removed any responses greater than 2.5 *SD*s. (The results on eye movement errors are discussed in more detail later.) The removal of early and timeout responses resulted in removal of 3.6%, 4.1%, and 5.5% of the experimental trials for the young, healthy old, and DAT groups, respectively. Using the remaining correct responses, we calculated the median for each participant in each experimental condition and submitted these values to further statistical analysis.

General Analysis of Response Latencies

Table 4 shows the mean and standard deviation of participants' median response latencies for the experimental trials, and Figure 6 shows the response latencies for the valid and invalid trials as a function of SOA (200, 800, 1,300, or 1,800 ms). Overall, there was an effect of group, $F(2, 102) = 16.74$, $p < .001$. The young group ($M = 382$ ms) was faster than the healthy old group ($M = 451$), $F(1, 70) = 18.45$, $p < .001$, which was faster than the DAT group ($M = 492$), $F(1, 88) = 18.45$, $p = .014$. Response latencies were faster for double-cue trials than for single-cue trials, $F(1, 112) =$

Table 4
Participants' Median Response Latencies in Experiment 2

	Group					
	Young		Healthy old		DAT	
Group	*M*	*SD*	*M*	*SD*	*M*	*SD*
Single-cue trials						
Valid	391	65	446	76	475	82
Neutral	415	72	470	85	517	99
Invalid	403	82	478	91	528	112
Double-cue trials						
800-ms SOA						
Valid	394	65	471	68	510	96
Invalid	375	76	450	84	489	90
1,300-ms SOA						
Valid	375	58	443	75	494	105
Invalid	340	58	412	71	450	90
1,500-ms SOA						
Valid	350	57	431	71	471	92
Invalid	332	59	416	71	453	90

Note. DAT = dementia of the Alzheimer type; SOA = stimulus onset asynchrony.

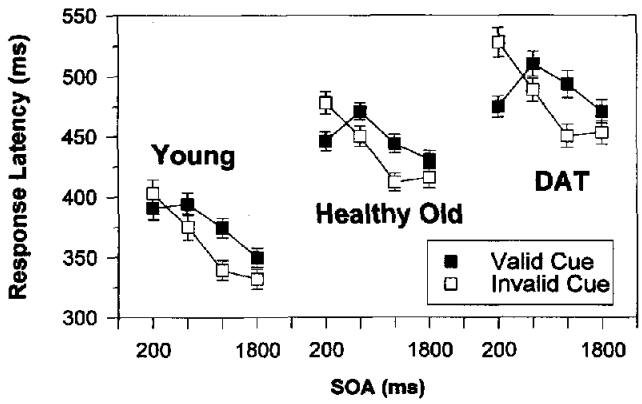

Figure 6. Mean of median response latencies as a function of cue validity, stimulus onset asynchrony (SOA), and group for Experiment 2. Error bars indicate standard errors. DAT = dementia of the Alzheimer type.

148.97, $p < .001$, and also were faster for targets presented in the right visual hemifield than for targets presented in the left visual hemifield, $F(1, 112) = 11.95$, $p = .001$. As in Experiment 1, the groups differed in the magnitude of hemifield asymmetry, $F(2, 112) = 3.52$, $p = .033$. There was no right visual hemifield advantage for the young group, $M = -1$ ms, $F(1, 24) = 0.16$, $p = .694$; but the healthy old, $M = 17$ ms, $F(1, 46) = 17.36$, $p < .001$, and the DAT group, $M = 7$ ms, $F(1, 42) = 4.11$, $p = .049$, did produce a right hemifield advantage.

Single-Cue Trials

An analysis of the single-cue trials revealed that response latencies for valid trials were faster than for invalid trials, $F(1, 112) = 101.36$, $p < .001$. The cue validity effect was greater for the healthy old group ($M = 32$ ms) than for the young group ($M = 12$ ms), $F(1, 70) = 5.79$, $p = .019$, and greater for the DAT group ($M = 53$ ms) than for the healthy old group, $F(1, 88) = 5.87$, $p = .017$. There were no overall costs (i.e., invalid minus neutral trials), $F(1, 112) = 1.26$, $p = .265$, and no Group × Cost interaction, $F(2, 112) = 2.17$, $p = .119$. However, there were overall benefits (i.e., neutral minus valid trials), $F(1, 112) = 94.59$, $p < .001$, which interacted with group, $F(2, 112) = 3.90$, $p = .023$. Thus, the age- and DAT-related increases in costs plus benefits were carried mainly by differences in benefits.

Double-Cue Trials

In contrast to the results for the single-cue trials, response latencies for valid double-cue trials were slower than response latencies for invalid double-cue trials (i.e., an inhibition of return effect), $F(1, 112) = 137.92$, $p < .001$. The size of the inhibition of return effect varied across SOAs, $F(2, 224) = 12.65$, $p < .001$, but not age or DAT (all Fs < 1). A Newman-Keuls analysis revealed that the inhibition of return effect was greater for the 1,300-ms SOA ($M = 37$ ms) than for either the 800-ms SOA ($M = 21$ ms), $q(2, 220) = 5.38$, $p < .001$, or the 1,800-ms SOA ($M = 17$ ms), $q(3, 224) = 6.72$, $p < .001$. The inhibition of return effect was reliable at the 1,300-ms SOA for the young, $F(1, 24) = 111.56$, $p < .001$, healthy old, $F(1, 46) = 49.54$, $p < .001$, and DAT, $F(1, 42) = 51.22$, $p < .001$, groups (Ms = 35, 32, and 43 ms, respectively). However, there was no modulation of the inhibition of return effect by group, $F(2, 112) = 1.44$, $p = .240$. Planned comparisons to determine the effects of age and dementia on the cue validity effects are reported next.

General Slowing and Cue Validity Effects

As in Experiment 1, we corrected for possible distortions of the response latency scale attributable to individual or group differences in overall speed by dividing each participant's condition medians by his or her overall mean. Cue validity effects, in terms of proportion of overall speed, are presented in Figure 7. The analysis of proportional cue effects was essentially the same as that for the raw difference scores. Of particular interest is the fact that the age-related increase in cue effects for the single-cue trials remained significant, $F(1, 70) = 5.92$, $p = .018$, and that the DAT-related increase remained marginally significant, $F(1, 88) = 3.84$, $p = .053$. Moreover, each group still produced a reliable inhibition of return effect that did not differ reliably across groups.

Eye Movement Errors

Figure 8 shows the proportion of eye movement errors as a function of group. Overall, the groups differed in the number of errors made, $F(2, 112) = 8.75$, $p < .001$. Planned comparisons for age and DAT effects revealed that the young group made fewer eye movement errors than did the healthy old group, $F(1, 70) = 4.63$, $p = .035$, which made fewer eye movement errors than the DAT group, $F(1, 88) = 8.25$, $p = .005$.

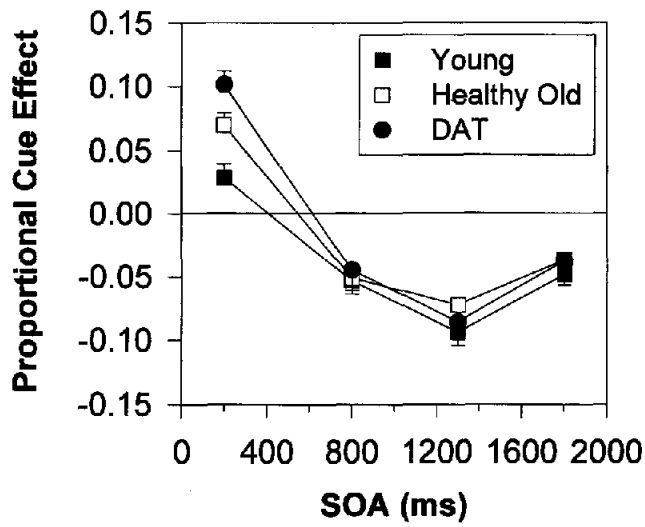

Figure 7. Proportional cue effect (i.e., invalid minus valid divided by overall mean) as a function of stimulus onset asynchrony (SOA) and group for Experiment 2. Error bars indicate standard errors. DAT = dementia of the Alzheimer type.

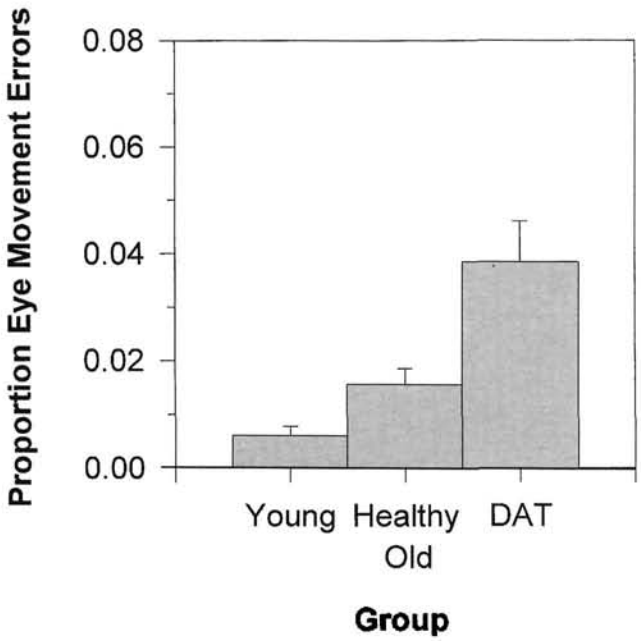

Figure 8. Proportion of eye movement errors as a function of group for Experiment 2. Error bars indicate standard errors. DAT = dementia of the Alzheimer type.

Discussion

The results of the single-cue conditions of Experiment 2 yielded no reliable costs. This was expected given the relatively automatic nature of the peripheral cue effects being tested, but it also might have been because the neutral condition contained no exogenous cue and therefore might have lacked a general alerting component that was present in the valid and invalid conditions. The neutral single-cue trials might have been slowed somewhat due to this lack of an alerting component, and therefore costs might have been underestimated.

The results for the younger adults are similar to those of previous studies (e.g., Posner et al., 1985) in that we found a cue validity effect for single-cue trials and inhibition of return for double-cue trials. There was a modest increase in the cue validity effect with age that remained even when overall speed was taken into account. Furthermore, the cue validity effect increased with DAT. The group differences in cue validity effects for single-cue trials were not, however, matched by differences in the inhibition of return effect for double-cue trials. This was somewhat surprising because decrements in inhibitory processing with age and DAT have been reported in the literature in a wide variety of paradigms (e.g., Balota & Duchek, 1991; Balota & Ferraro, 1993, 1996; Butters et al., 1987; Faust et al., in press; Hamm & Hasher, 1992; Hartman & Hasher, 1991; Hasher et al., 1991; Loewenstein et al., 1991; Spieler et al., 1996; Sullivan et al., 1995). However, our results regarding age are consistent with those of Hartley and Kieley (1995), who also found no age differences in inhibition of return.

The lack of group differences in inhibition of return stand in contrast to the age- and DAT-related increases in failures to inhibit reflexive saccades to the periphery in Experiments 1 and 2. The inhibition of reflexive saccades is thought to be mediated by the frontal lobes (e.g., Roberts, Hager, & Heron, 1994). Consequently, increases in the failure to inhibit reflexive saccades with age and DAT in the presence of equivalent, more posteriorly mediated inhibition of return in these groups suggest that frontal systems, associated with more controlled aspects of inhibitory function, are differentially impaired in these groups. A potential counterargument would be that individuals with DAT, and perhaps even healthy older adults, either have more difficulty allocating cognitive resources to the inhibition of reflexive saccades or simply have more difficulty keeping this task demand active in working memory. Either way, such a view would still fit with the more general view of breakdowns in the higher order control of inhibitory processing.

General Discussion

Our experiments yielded five basic results. First, individuals with DAT were slower than healthy older adults to spontaneously reorient attention in the absence of a second exogenous cue in Experiment 1. Because inhibition of return requires orientation of attention away from a location, these results suggest that individuals with DAT will be slower to produce inhibition of return effects. However, because the size of the cue validity effect did not reach a stable asymptote by the 800-ms SOA for the individuals with DAT, the results of Experiment 1 do not bear on the question of the relative preservation of the processes underlying inhibition of return in DAT. Second, the results of Experiment 2 clearly yield equivalent inhibition of return in healthy younger and older adults and individuals with DAT under conditions of extrinsically driven reorienting (i.e., a second exogenous cue). Third, overall cue validity effects (i.e., costs plus benefits) increased with age in both experiments and with DAT in Experiment 2. Fourth, the results of both experiments indicate that controlled inhibition of reflexive eye movements was impaired in healthy older adults and individuals with DAT. Finally, although both healthy older adults and individuals with DAT demonstrated a bias for targets appearing in the right visual hemifield to be detected faster than those appearing the left visual hemifield in both Experiments 1 and 2, younger adults did not.

Inhibition in DAT and Healthy Aging

A major goal of the current research was to explore inhibitory processes at a lower level in the cognitive system than have been probed by previous researchers reporting inhibitory deficits in healthy aging (e.g., Duchek et al., 1995; Hamm & Hasher, 1992; Hartman & Hasher, 1991; Hasher et al., 1991; Spieler et al., 1996) and in DAT (e.g., Balota & Duchek, 1991; Spieler et al., 1996; Sullivan et al., 1995). Inhibition of return reflects a bias against reorienting attention to a previously attended location and is involved in the selective filtering of information early in the visual system. Furthermore, inhibition of return is associated with the posterior visuospatial attention system underlying overt and

covert orienting of visuospatial attention (e.g., Clohessy et al., 1991; Posner et al., 1985; Rafal et al., 1989, 1991; however, see Johnson, 1990, for a discussion of possible frontal mediation of overt and covert attention). Therefore, the finding of equivalent inhibition of return effects for individuals with DAT, healthy older adults, and younger adults in Experiment 2 supports the idea that there is a relative preservation in healthy aging and DAT of inhibitory mechanisms associated with the posterior attention system that are responsible for early filtering of information.

The equivalent inhibition of return effects observed across groups in Experiment 2, presumably reflecting equivalent filtering of previously attended locations early in the information-processing system, stand in contrast to age- and DAT-related breakdowns in the inhibitory mechanisms associated with selection for action. For example, Spieler et al. (1996) found that healthy older adults and individuals with DAT experienced more interference while performing the Stroop color naming task (Stroop, 1935). The Stroop color naming task emphasizes selection of an attribute of a word (i.e., its color) to drive the appropriate response. The ability to control interference from the to-be-ignored word identity in the Stroop task has been tied to the frontal lobe and the anterior attention system (e.g., Corbetta et al., 1993; Dempster, 1992; Lezak, 1995; Pardo et al., 1990).

Of further interest was the finding of both age- and DAT-related increases in the failure to inhibit reflexive eye movements in response to peripheral cues. Individuals with DAT have been found to be deficient at inhibiting reflexive overt saccades in response to peripheral stimulation (e.g., Fletcher & Sharpe, 1986). Roberts et al. (1994) recently proposed that breakdowns in the ability to inhibit reflexive saccades are indicative of a more general breakdown (presumably associated with the frontal lobes) in the ability to deactivate response alternatives that become activated in working memory relatively automatically. Therefore, the finding of equivalent inhibition of return in DAT and healthy aging in Experiment 2 suggests that inhibitory breakdowns in these groups are associated more with the anterior attention system and top-down control of processes associated with the control of a response (i.e., selection for action and the inhibition of prepotent responses) than with the posterior attention system associated with the early filtering of information from to-be-ignored locations. The current results also are consistent with proposals that inhibitory deficits observed in healthy aging most likely do not reflect a breakdown in a single global inhibitory ability (e.g., Kramer, Humphrey, Larish, & Logan, 1994).

Age and Cue Validity Effects

Although the results of Experiment 2 regarding inhibition of return suggest that inhibitory mechanisms associated with the posterior attention system are relatively unimpaired in healthy aging, the results from the single-cue trials in Experiment 2 and from the short SOA in Experiment 1 do exhibit age-related changes in covert orienting. Healthy older adults produced cue effects that were larger than those produced by younger adults in both experiments, even after overall differences in speed were controlled. Because there were no age-related increases in costs or benefits alone in Experiment 1, and no groups produced reliable costs in Experiment 2, there is no evidence of specific components underlying the current findings of age-related increases in costs plus benefits.

However, our results are consistent with the results of several studies in the literature demonstrating that age-related increases in overall cue effects likely are to be found during simple detection tasks (e.g., Hartley & Kieley, 1995, Experiment 1; cf. Robinson & Kertzman, 1990, for a counterexample, but with only 7 older adults), but not during discrimination tasks (Greenwood et al., 1993; Hartley & Kieley, 1995, Experiment 2; Hartley et al., 1990, Experiment 3). The increased likelihood of finding age-related increases in overall cue effects for detection tasks may be related to age-related breakdowns in the localization of targets during visual search (e.g., Plude & Doussard-Roosevelt, 1989). From this perspective, the current finding of age-related increases in peripheral cue effects likely is due to a breakdown in the posterior attention system that results in a reduced ability to localize objects as they appear in the visual field. It remains to be seen whether the pattern emerging in the literature of age-related changes in peripheral cue effects using detection but not discrimination tasks will hold up under more direct examination. Furthermore, the reason for such a distinction remains unclear. One possible explanation is that relatively small age-related differences in detection performance may be masked during the later, more variable stages of processing required for discrimination.

DAT and Cue Validity Effects

In contrast to the age-related increases in the overall cue validity effect (i.e., costs plus benefits) in Experiments 1 and 2, there were DAT-related increases in the overall cue validity effects only in Experiment 2. One possible explanation for this DAT-related increase in overall cue validity effects in Experiment 1, but not Experiment 2, comes from a consideration of task differences between the two experiments interpreted relative to the findings of Parasuraman et al. (1992). Parasuraman et al. found that individuals with DAT produced a cue validity effect that was approximately twice that of healthy older adults at a 200-ms SOA using a discrimination task and peripheral cues. A closer consideration of the single-cue condition of Experiment 2 reveals that even though participants were asked to simply detect a target following a cue, they still had to discriminate whether the next luminance change was in actuality a target or a second cue of a double-cue trial. Thus, because of random intermixing of the single- and double-cue trial types in Experiment 2, single-cue trials had a discrimination component. Our results are therefore consistent with those of Parasuraman et al. (1992) in that when task demands emphasized discrimination of targets from cues, DAT-related increases in the overall cue validity effect were observed.

DAT and Disengaging Visuospatial Attention

As discussed earlier, Parasuraman et al. (1992) found that under conditions of a 200-ms SOA, peripheral cues, and a discrimination task, the cue validity effect for individuals with DAT was larger than that for healthy older adults. Furthermore, this increased cue validity effect was correlated ($r = -.54$) with cerebral hypometabolism in the superior parietal area in the individuals with DAT. Because the parietal lobes have been associated with the component process of disengagement of visuospatial attention from a particular location (Posner et al., 1984), and because Parasuraman et al. also found dementia-related increases in costs but not in benefits, Parasuraman et al. proposed a disengagement deficit for DAT. However, using a detection task, we found no group differences in costs in Experiment 1 and no reliable costs for any group in Experiment 2. The current results therefore are consistent with proposals that individuals with DAT will produce increased overall cue validity effects when a target discrimination is required, but this will not always be accompanied by an increase in costs.

Age and Detection Asymmetry

Experiments 1 and 2 both yielded an unexpected age-related difference in detection asymmetry. Whereas younger adults detected targets equally well in both visual hemifields, older adults detected targets faster when presented in the right visual hemifield than in the left visual hemifield. Moreover, individuals with DAT did not differ from healthy older adults in this respect. Robinson and Kertzman (1990) also reported an age-related increase in the lateral asymmetry of the detection of targets during a peripheral cuing experiment. The current results are consistent with two interrelated proposals in the literature. First, several studies have shown greater age-related declines in scores on psychometric tests thought to rely primarily on right rather than left hemisphere function (e.g., Goldstein & Shelly, 1981; Klisz, 1978), and therefore it has been suggested that the right hemisphere may age more rapidly than the left. However, studies directly examining perceptual field asymmetries using brief presentation of targets at lateral locations have shown little evidence in support of such a claim (e.g., Cherry, Hellige, & McDowd, 1995; Hoyer & Rybash, 1992; Nebes, Madden, & Berg, 1983). However, none of these studies used simple detection as a task. Second, there is evidence in the literature for an age-related decline in the efficiency of the interhemispheric transfer of information (e.g., Moes, Jeeves, & Cook, 1995). Furthermore, studies examining age-related changes in the structure of the corpus callosum have indicated decreases in callosal area or length (e.g., Hayakawa, Konishi, Matsuda, Kuriyama, & Konishi, 1989). Therefore, another possible explanation of the age-related detection asymmetry in our results is that the transfer of information from the right hemisphere to the left hemisphere (required for the right hand to respond to targets presented in the left visual field) slows with age.

One way to differentiate between these two hypotheses would be to have participants respond with both their left and right hands. If the interhemispheric transfer hypothesis is correct, older adults should show a reversal in the detection asymmetry when responding with their left hand (i.e., right visual field targets responded to more slowly) because the hemisphere controlling response had shifted. The right hemisphere aging hypothesis would predict little or no change in detection asymmetry as a function of response hand. Therefore, given that we did not have participants use their left hands to respond, both explanations fit our results equally well.

Local Versus Global Cortical Integrity and Inhibition

Finally, one important way theories of cognitive neuroscience vary is in how diffusely elementary cognitive operations are proposed to be distributed in the central nervous system (Mesulam, 1981, 1990; Posner, 1988; Posner, Petersen, Fox, & Raichle, 1988). Parasuraman and Nestor (1993) recently argued for the possibility that "some cognitive operations operate normally in the AD [Alzheimer's disease] brain because they are subserved by localized neural modules that are not affected markedly by pathological processes that affect communication *between* modules" (p. 98). They based this argument on the results of several recent studies indicating that the pathology in DAT is regionally systematic (Damasio, Van Hoesen, & Hyman, 1990; Kemper, 1984) and can be characterized as a cortical disconnection syndrome in which corticocortical connections are particularly disrupted (Morrison et al., 1990). From this perspective, inhibitory processes that rely heavily on subcortical computations, as location-based inhibition of return seems to, may be more likely to be relatively well preserved in DAT than are inhibitory processes that rely on distributed computations among a wide range of cortical maps.

References

Abrams, R. A., & Dobkin, R. S. (1994). Inhibition of return: Effects of attentional cuing on eye movement latencies. *Journal of Experimental Psychology: Human Perception and Performance, 20,* 467–477.

Balota, D. A., & Duchek, J. M. (1991). Semantic priming effects, lexical repetition effects, and contextual disambiguation effects in healthy aged individuals and individuals with senile dementia of the Alzheimer type. *Brain and Language, 40,* 181–201.

Balota, D. A., & Ferraro, F. R. (1993). A dissociation of frequency and regularity effects in pronunciation performance across young adults, older adults, and individuals with senile dementia of the Alzheimer type. *Journal of Memory and Language, 32,* 573–592.

Balota, D. A., & Ferraro, F. R. (1996). Lexical, sublexical, and implicit memory processes in healthy young and healthy older adults and in individuals with dementia of the Alzheimer type. *Neuropsychology, 10,* 82–95.

Bashinski, H. S., & Bacharach, V. R. (1980). Enhancement of perceptual sensitivity as the result of selectively attending to spatial locations. *Perception & Psychophysics, 28,* 241–248.

Berg, L., & Morris, J. C. (1994). Diagnosis. In R. D. Terry, R. Katzman, & K. Bick (Eds.), *Alzheimer's disease* (pp. 9–25). New York: Raven Press.

Berlucchi, G., Tassinari, G., Marzi, C. A., & Di Stefano, M. (1989). Spatial distribution of the inhibitory effect of peripheral non-informative cues on simple reaction time to non-fixated visual targets. *Neuropsychologia, 27,* 201–221.

Butters, N., Granholm, E., Salmon, D. P., Grant, I., & Wolfe, J. (1987). Episodic and semantic memory: A comparison of amnesic and demented patients. *Journal of Clinical and Experimental Neuropsychology, 9,* 479–497.

Cherry, B. J., Hellige, J. B., & McDowd, J. M. (1995). Age differences and similarities in patterns of cerebral hemispheric asymmetry. *Psychology and Aging, 10,* 191–203.

Clohessy, A. B., Posner, M. I., Rothbart, M. K., & Vecera, S. P. (1991). The development of inhibition of return in early infancy. *Journal of Cognitive Neuroscience, 3,* 345–354.

Connelly, S. L., & Hasher, L. (1993). Aging and inhibition of spatial location. *Journal of Experimental Psychology: Human Perception and Performance, 19,* 1238–1250.

Corbetta, M., Miezin, F. M., Shulman, G. L., & Petersen, S. E. (1993). A PET study of visuospatial attention. *Journal of Neuroscience, 13,* 1202–1226.

Damasio, A. R., Van Hoesen, G. W., & Hyman, B. T. (1990). Reflections on the selectivity of neuropathological changes in Alzheimer's disease. In M. F. Schwartz (Ed.), *Modular deficits in Alzheimer-type dementia* (pp. 83–100). Cambridge, MA: Bradford.

Dempster, F. N. (1992). The rise and fall of the inhibitory mechanism: Toward a unified theory of cognitive development and aging. *Developmental Review, 12,* 45–75.

Duchek, J., Balota, D. A., Faust, M. E., & Ferraro, F. R. (1995). Inhibitory processes in young and older adults in a picture-word task. *Aging and Cognition, 2,* 1–11.

Eriksen, C. W., & Hoffman, J. E. (1973). The extent of processing of noise elements during selective encoding from visual displays. *Perception & Psychophysics, 14,* 155–160.

Eriksen, C. W., & Yeh, Y. (1985). Allocation of attention in the visual field. *Journal of Experimental Psychology: Human Perception and Performance, 11,* 583–597.

Faust, M. E., Balota, D. A., Duchek, J. M., Gernsbacher, M. A., & Smith, S. (in press). Inhibitory control during sentence comprehension in individuals with dementia of the Alzheimer type. *Brain and Language.*

Faust, M. E., Balota, D. A., & Duchek, J. M. (1995, November). *Interference and negative priming: Flanker task performance in dementia of the Alzheimer type (DAT) and normal aging.* Poster presented at the 36th Annual Meeting of the Psychonomic Society, Los Angeles.

Faust, M. E., Balota, D. A., & Duchek, J. M. (1996, April). *Interference and negative priming: Local-global attention in aging and dementia.* Poster presented at the Sixth Biannual Cognitive Aging Conference, Atlanta, GA.

Faust, M. E., Balota, D. A., & Ferraro, F. R. (1996). *Cognitive slowing, cognitive speed, and response latency.* Manuscript submitted for publication.

Fletcher, W. A., & Sharpe, J. A. (1986). Saccadic eye movement dysfunction in Alzheimer's disease. *Annals of Neurology, 20,* 464–471.

Freed, D. M., Corkin, S., Growdon, J. H., & Nissen, M. J. (1989). Selective attention in Alzheimer's disease: Characterizing cognitive subgroups of patients. *Neuropsychologia, 27,* 325–329.

Fuld, P. A., Katzman, R., Davies, P., & Terry, R. D. (1982). Intrusions as a sign of Alzheimer dementia: Chemical and pathological verification. *Annals of Neurology, 11,* 155–159.

Goldstein, G., & Shelly, C. (1981). Does the right hemisphere age more rapidly than the left? *Journal of Clinical Neuropsychology, 3,* 65–78.

Greenwood, P. M., Parasuraman, R., & Haxby, J. V. (1993). Changes in visuospatial attention over the adult lifespan. *Neuropsychologia, 31,* 471–485.

Hamm, V. P., & Hasher, L. (1992). Age and the availability of inferences. *Psychology and Aging, 7,* 56–64.

Hartley, A. A. (1992). Attention. In F. I. M. Craik & T. Salthouse (Eds.), *Handbook of aging and cognition* (pp. 3–49). Hillsdale, NJ: Erlbaum.

Hartley, A. A. (1993). Evidence for the selective preservation of spatial selective attention in old age. *Psychology and Aging, 8,* 371–379.

Hartley, A. A., & Kieley, J. M. (1995). Adult age differences in the inhibition of return of visual attention. *Psychology and Aging, 10,* 670–683.

Hartley, A. A., Kieley, J. M., & Slabach, E. H. (1990). Age differences and similarities in the effects of cues and prompts. *Journal of Experimental Psychology: Human Perception and Performance, 16,* 523–537.

Hartman, M., & Hasher, L. (1991). Aging and suppression: Memory for previously relevant information. *Psychology and Aging, 6,* 587–594.

Hasher, L., & Zacks, R. T. (1988). Working memory, comprehension, and aging: A review and a new view. *Psychology of Learning and Motivation, 22,* 193–225.

Hasher, L., Stoltzfus, E. R., Zacks, R. T., & Rypma, B. (1991). Age and inhibition. *Journal of Experimental Psychology: Learning, Memory, and Cognition, 17,* 163–169.

Hawkins, H. L., Hillyard, S. A., Luck, S. J., Mouloua, M., Downing, C. J., & Woodward, D. P. (1990). Visual attention modulates signal detectability. *Journal of Experimental Psychology: Human Perception and Performance, 16,* 802–811.

Haxby, J. V., Grady, C. L., Duara, R., Schlageter, N. L., Berg, G., & Rapoport, S. I. (1986). Neocortical metabolic abnormalities precede non-memory cognitive deficits in early Alzheimer-type dementia. *Archives of Neurology, 43,* 882–885.

Hayakawa, K., Konishi, Y., Matsuda, T., Kuriyama, M., & Konishi, K. (1989). Development and aging of brain midline structures: Assessment with MR imaging. *Radiology, 172,* 171–177.

Hoyer, W. J., & Familiant, M. E. (1987). Adult age differences in the rate of processing expectancy information. *Cognitive Development, 2,* 59–70.

Hoyer, W. J., & Rybash, J. M. (1992). Age and visual field differences in computing visual-spatial relations. *Psychology and Aging, 7,* 339–342.

Hughes, C. P., Berg, L., Danziger, W. L., Coben, L. A., Martin, R. L. (1982). A new clinical scale for the staging of dementia. *British Journal of Psychiatry, 140,* 566–572.

Johnson, M. H. (1990). Cortical maturation and the development of visual attention in early infancy. *Journal of Cognitive Neuroscience, 2,* 81–95.

Jonides, J. (1981). Voluntary versus automatic control over the mind's eye. In J. Long & A. Baddeley (Eds.), *Attention and performance IX* (pp. 187–203). Hillsdale, NJ: Erlbaum.

Jonides, J., & Mack, R. (1984). On the cost and benefit of cost and benefit. *Psychological Bulletin, 96,* 29–44.

Jonides, J., & Yantis, S. (1988). Uniqueness of abrupt visual onset in capturing attention. *Perception & Psychophysics, 43,* 346–354.

Kaplan, E., Goodglass, H., & Weintraub, S. (1983). *Boston Naming Test scoring booklet.* Philadelphia: Lea & Febiger.

Kemper, T. (1984). Neuroanatomical and neuropathological changes in normal aging and in dementia. In M. Albert (Ed.), *Clinical neurology of aging* (pp. 9–52). New York: Oxford University Press.

Klein, R. M. (1988). Inhibitory tagging system facilitates visual search. *Nature, 334,* 430–431.

Klein, R. M. (1989). [Inhibitory tagging in visual search: Three failures to replicate]. Unpublished raw data.

Klisz, D. (1978). Neuropsychological evaluation in older persons. In M. Storandt, I. C. Siegler, & M. F. Elias (Eds.), *The clinical psychology of aging* (pp. 71–95). New York: Plenum.

Kramer, A. F., Humphrey, D. G., Larish, J. F., & Logan, G. D. (1994). Aging and inhibition: Beyond a unitary view of inhibitory processing in attention. *Psychology and Aging, 9,* 491–512.

Lezak, M. D. (1995). *Neuropsychological assessment* (3rd ed.). New York: Oxford University Press.

Loewenstein, D. A., D'Elia, L., Guterman, A., Eisdorfer, C., Wilkie, F., LaRue, A., Mintzer, J., & Duara, R. (1991). The occurrence of different intrusive errors in patients with Alzheimer's disease, multiple cerebral infarctions, and major depression. *Brain and Cognition, 16,* 104–117.

Madden, D. J. (1983). Aging and distraction by highly familiar stimuli during visual search. *Developmental Psychology, 19,* 499–507.

Madden, D. J. (1990). Adult age differences in the time course of visual attention. *Journal of Gerontology, 45,* P9–P16.

Madden, D. J. (1992). Selective attention and visual search: Revision of an allocation model and application to age differences. *Journal of Experimental Psychology: Human Perception and Performance, 18,* 821–836.

Madden, D. J., Connelly, S. L., & Pierce, T. W. (1994). Adult age differences in shifting focused attention. *Psychology and Aging, 9,* 528–538.

Madden, D. J., & Plude, D. J. (1993). Selective preservation of selective attention. In J. Cerella, W. J. Hoyer, J. Rybash, & M. L. Commons (Eds.), *Adult information processing: Limits on loss* (pp. 273–300). San Diego, CA: Academic Press.

Maylor, E. A. (1985). Facilitatory and inhibitory components of orienting in visual space. In M. I. Posner & O. S. M. Marin (Eds.), *Attention and performance XI* (pp. 189–204). Hillsdale, NJ: Erlbaum.

Maylor, E. A., & Hockey, R. (1987). Effects of repetition on the facilitatory and inhibitory components of orienting in visual space. *Neuropsychologia, 25,* 41–54.

McDowd, J. M., Oseas-Kreger, D. M., & Filion, D. L. (1995). Inhibitory processes in cognition and aging. In F. N. Dempster & C. J. Brainerd (Eds.), *Interference and inhibition in cognition* (pp. 363–400). San Diego, CA: Academic Press.

McKhann, G., Drachman, D., Folstein, M., Katzman, R., Price, D., & Stadlan, M. (1984). Clinical diagnosis of Alzheimer's disease: Report of the NINCDS-ADRDA Work Group under the auspices of the Department of Health and Human Services Task Force on Alzheimer's Disease. *Neurology, 34,* 39–44.

Mesulam, M. M. (1981). A cortical network for directed attention and unilateral neglect. *Annals of Neurology, 10,* 309–325.

Mesulam, M. M. (1990). Large-scale neurocognitive networks and distributed processing for attention, language, and memory. *Annals of Neurology, 28,* 597–613.

Moes, P., Jeeves, M. A., & Cook, K. (1995). Bimanual coordination with aging: Implications for interhemispheric transfer. *Developmental Neuropsychology, 11,* 23–40.

Morris, J. C. (1993). Clinical dementia rating. *Neurology, 43,* 2412–2414.

Morris, J. C., McKeel, D. W., Fulling, K., Torack, R. M., Berg, L. (1988). Validation of clinical diagnostic criteria for Alzheimer's disease. *Annals of Neurology, 24,* 17–22.

Morris, J. C., McKeel, D. W., Jr., Storandt, M., Rubin, E. H., Price, J. L., Grant, E. A., Ball, M. J., & Berg, L. (1991). Very mild Alzheimer's disease: Informant-based clinical, psychometric, and pathologic distinction from normal aging. *Neurology, 41,* 469–478.

Morrison, J. H., Hof, P. R., Campbell, M. J., DeLima, A. D., Voigt, T., Bouras, C., Cox, K., & Young, W. G. (1990). Cellular pathology in Alzheimer's disease: Implication for corticocortical disconnection and differential vulnerability. In S. I. Rapoport, H. Petit, D. Leys, & Y. Christen (Eds.), *Imaging, cerebral topography, and Alzheimer's disease* (pp. 19–40). Berlin: Springer-Verlag.

Müller, H. J., & Findlay, J. M. (1988). The effect of visual attention on peripheral discrimination thresholds in single and multiple element displays. *Acta Psychologia, 69,* 129–155.

Müller, H. J., & Rabbitt, P. M. A. (1989). Reflexive and voluntary orienting of visual attention: Time course of activation and resistance to interruption. *Journal of Experimental Psychology: Human Perception and Performance, 15,* 315–330.

Nebes, R. D. (1992). Cognitive dysfunction in Alzheimer's disease. In F. I. M. Craik & T. Salthouse (Eds.), *Handbook of aging and cognition* (pp. 373–446). Hillsdale, NJ: Erlbaum.

Nebes, R. D., Madden, D. J., & Berg, W. D. (1983). The effect of age on hemispheric asymmetry in visual and auditory identification. *Experimental Aging Research, 9,* 87–91.

Nissen, M. J., & Corkin, S. (1985). Effectiveness of attentional cueing in older and younger adults. *Journal of Gerontology, 40,* 185–191.

Nissen, M. J., Corkin, S., & Growdon, J. H. (1981, August). *Attentional focusing in patients with Alzheimer's disease.* Paper presented at the annual conference of the American Aging Association, Chicago.

Oken, B. S., Kishiyama, S. S., Kaye, J. A., & Howieson, D. B. (1994). Attention deficit in Alzheimer's disease is not simulated by an anticholinergic/antihistaminergic drug and is distinct from deficits in healthy aging. *Neurology, 44,* 657–662.

Panicker, S., Greenwood, P. M., Parasuraman, R., & Haxby, J. V. (1993, November). *Cued visual search in Alzheimer's disease.* Poster presented at the annual meeting of the Society for Neuroscience, Washington, DC.

Parasuraman, R., Greenwood, P. M., Haxby, J. V., & Grady, C. L. (1992). Visuospatial attention in dementia of the Alzheimer type. *Brain, 115,* 711–733.

Parasuraman, R., & Haxby, J. V. (1993). Attention and brain function in Alzheimer's disease: A review. *Neuropsychology, 7,* 242–272.

Parasuraman, R., & Nestor, P. G. (1993). Preserved cognitive operations in early Alzheimer's disease. In J. Cerella, J. Rybash, W. J. Hoyer, & M. L. Commons (Eds.), *Adult information processing: Limits on loss* (pp. 77–111). San Diego, CA: Academic Press.

Pardo, J. V., Pardo, P. J., Janer, K. W., & Raichle, M. E. (1990). The anterior cingulate cortex mediates processing selection in the Stroop attentional conflict paradigm. *Proceedings of the National Academy of Sciences, 87,* 256–259.

Plude, D. J., & Doussard-Roosevelt, J. A. (1989). Aging, selective attention, and feature integration. *Psychology and Aging, 4,* 98–105.

Plude, D. J., & Hoyer, W. J. (1986). Age and the selectivity of visual information processing. *Psychology and Aging, 1,* 4–10.

Posner, M. I. (1980). Orienting of attention. *Quarterly Journal of Experimental Psychology, 32,* 3–25.

Posner, M. I. (1982). Cumulative development of attentional theory. *American Psychologist, 32,* 53–64.

Posner, M. I. (1988). Structures and functions of selective attention. In T. Boll & B. K. Bryant (Eds.), *Master lectures in psychology* (pp. 173–202). Washington, DC: American Psychological Association.

Posner, M. I., & Cohen, Y. (1984). Components of visual orienting. In H. Bouma & D. G. Bouwhuis (Eds.), *Attention and performance X: Control of language processes* (pp. 531–556). Hillsdale, NJ: Erlbaum.

Posner, M. I., Cohen, Y., & Rafal, R. D. (1982). Neural systems control over spatial orienting. *Philosophical Transaction Royal Society of London Series B, 2908,* 187–198.

Posner, M. I., Nissen, M. J., & Ogden, W. C. (1978). Attended and unattended processing modes: The role of set for spatial location. In H. L. Pick & I. J. Saltzman (Eds.), *Modes of perceiving and processing information* (pp. 137–161). Hillsdale, NJ: Erlbaum.

Posner, M. I., & Petersen, S. E. (1990). The attention system of the human brain. *Annual Review of Neuroscience, 13,* 25–42.

Posner, M. I., Petersen, S. E., Fox, P. T., & Raichle, M. E. (1988). Localization of cognitive operations in the human brain. *Science, 240,* 1627–1631.

Posner, M. I., Rafal, R. D., Choate, L. S., & Vaughan, J. (1985). Inhibition of return: Neural basis and function. *Cognitive Neuropsychology, 2,* 211–228.

Posner, M. I., Walker, J. A., Friedrich, F. J., & Rafal, R. D. (1984). Effects of parietal lobe injury on covert orienting of visual attention. *Journal of Neuroscience, 4,* 1863–1874.

Pratt, J. (1996). *Inhibition of return in discrimination tasks.* Unpublished doctoral dissertation, Washington University, St. Louis, MO.

Rafal, R. D., Calabresi, P. A., Brennon, C. W., & Sciolto, T. K. (1989). Saccade preparation inhibits reorienting to recently attended locations. *Journal of Experimental Psychology: Human Perception and Performance, 15,* 673–685.

Rafal, R., Henik, A., & Smith, J. (1991). Extrageniculate contributions to reflex visual orienting in normal humans: A temporal hemifield advantage. *Journal of Cognitive Neuroscience, 3,* 322–328.

Rafal, R. D., & Posner, M. I. (1987). Deficits in visual spatial attention following thalamic lesions. *Proceedings of the National Academy, 84,* 7349–7353.

Remington, R. (1980). Attention and saccadic eye movement. *Journal of Experimental Psychology: Human Perception and Performance, 6,* 726–744.

Roberts, R. J., Jr., Hager, L. D., & Heron, C. (1994). Prefrontal cognitive processes: Working memory and inhibition in the antisaccade task. *Journal of Experimental Psychology: General, 123,* 374–393.

Robinson, D. L., & Kertzman, C. (1990). Visuospatial attention: Effects of age, gender and spatial reference. *Neuropsychologia, 28,* 291–301.

Spieler, D. H., Balota, D. A., & Faust, M. E. (1996). Stroop performance in younger adults, healthy older adults and individuals with senile dementia of the Alzheimer's type. *Journal of Experimental Psychology: Human Perception and Performance, 22,* 461–479.

Stroop, J. R. (1935). Studies of interference in serial verbal reactions. *Journal of Experimental Psychology, 18,* 643–662.

Sullivan, M. P., Faust, M. E., & Balota, D. A. (1995). Identity negative priming in old adults and individuals with dementia of the Alzheimer type. *Neuropsychology, 9,* 537–555.

Thurstone, L. E., & Thurstone, T. G. (1949). *Examiner manual for the SRT Primary Mental Abilities Test.* Chicago: Science Research Associates.

Tipper, S. P. (1992). Selection for action: The role of inhibitory mechanisms. *Current Directions in Psychological Science, 1,* 105–109.

Tipper, S. P., Driver, J., & Weaver, B. (1991). Object-centered inhibition of return of visual attention. *Quarterly Journal of Experimental Psychology, 43A,* 289–298.

Tipper, S. P., Weaver, B., Jerreat, L. M., & Burak, A. L. (1994). Object-based and environment-based inhibition of return of visual attention. *Journal of Experimental Psychology: Human Perception and Performance, 20,* 478–499.

Wechsler, D. (1955). *Manual: Wechsler Adult Intelligence Scale.* New York: Psychological Corporation.

Wechsler, D., & Stone, C. P. (1973). *Manual: Wechsler Memory Scale.* New York: Psychological Corporation.

Wolfe, J. M., & Pokorny, C. W. (1990). Inhibitory tagging in visual search: A failure to replicate. *Perception & Psychophysics, 48,* 357–362.

Zacks, R. T., & Hasher, L. (1994). Directed ignoring: Inhibitory regulation of working memory. In D. Dagenbach & T. H. Carr (Eds.), *Inhibitory processes in attention, memory, and language* (pp. 241–264). San Diego, CA: Academic Press.

Received February 8, 1996
Revision received June 26, 1996
Accepted July 30, 1996 ■