

## Implicit Memory and the Formation of New Associations in Nondemented Parkinson's Disease Individuals and Individuals with Senile Dementia of the Alzheimer Type: A Serial Reaction Time (SRT) Investigation

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Using the serial reaction time (SRT) task developed by Nissen and Bullemer (1987, *Cognitive Psychology*, 19, 1-32), implicit memory performance was examined in four groups of subjects: nondemented healthy aged individuals; nondemented Parkinson's disease individuals; very mildly demented senile dementia of the Alzheimer type (SDAT) individuals; and mildly demented SDAT individuals. The SRT task involved four blocks of a repeated 10-item keypress sequence that tapped general skill development along with a fifth block of a nonrepeated sequence that presumably reflected the impact of switching from a learned set of associations (developed during the first four blocks) to a novel sequence. The increase in response latency from the fourth repeated block to the fifth nonrepeated block was used as the reflection of implicit learning. The results revealed preserved implicit memory performance in the very mildly demented individuals compared to that of the age-matched control individuals. However, the mildly demented SDAT individuals and the nondemented Parkinson's disease individuals showed reliably less implicit learning, compared to the age-matched control individuals. Differences between the past studies using the SRT task to tap implicit memory performance in SDAT individuals and the present study are discussed in some detail. We conclude that nondemented Parkinson's disease individuals and mildly demented SDAT individuals produce some deficit in the formation of new associations in implicit memory, as measured by the SRT task. © 1993 Academic Press, Inc.

Within the past decade, there has been considerable interest in the notion that there are separate and empirically dissociable memory systems (e.g., Cohen & Squire, 1980; Squire, 1986; Tulving, 1983). Among

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the numerous classification systems employed to identify separate memory systems, the terms *implicit* and *explicit* memory have been widely used. Explicit memory is reflected by tasks in which information retrieval is accomplished via conscious recollection of an earlier event. For example, results obtained from a recall or recognition test are typically viewed as reflecting explicit memory performance. By contrast, implicit memory is reflected by tasks in which the retrieval of information is accomplished via a task that does not demand conscious recollection of an earlier episode. For example, results obtained from repetition priming experiments and word-stem completion experiments are viewed as reflecting implicit memory performance (e.g., Roediger, 1990; Schacter, 1987).

Of particular interest regarding the distinction between implicit and explicit memory performance is the finding that certain clinical populations (e.g., amnesics) reveal a dissociation between these two types of memory performance (Squire, 1986). That is, amnesic individuals are deficient on tasks which require explicit memory, yet their performance is comparable to healthy control subjects on tests which tap implicit memory. The dissociation in amnesic individuals has been viewed as some of the strongest evidence in support of distinct memory systems (e.g., Squire, 1986).

A similar pattern of dissociation has also been found in individuals diagnosed with senile dementia of the Alzheimer's type (SDAT; dementia onset after age 60). SDAT individuals produce rather large breakdowns on explicit memory tests like episodic recognition tests but produce as much repetition priming as healthy controls on implicit tests such as speeded mirror-reversed sentence reading (Moscovitch, Winocur, & McLachlan, 1986). These results run counter to the arguments outlined by Squire (1986), who suggested that individuals with SDAT might be more likely, at least as compared to more focal amnesics, to produce a breakdown on procedural/implicit memory tasks such as repetition priming. In Squire's taxonomy of memory, declarative (or, for simplicity, what we are referring to as explicit) memory primarily relies upon systems located within the medial temporal as well as diencephalic brain regions. A lesion to these areas, as in amnesia, produces deficits in explicit memory. Presumably, procedural (or what we refer to as implicit) memory is more distributed and encompasses numerous anatomical systems. In fact, Squire specifically argues that one should find implicit memory deficits only in clinical populations which sustain global brain damage, as some have argued is the case in SDAT (e.g., Salmon, Shimamura, Butters, & Smith, 1988).

As noted, one implicit memory measure that appears to be preserved in SDAT is repetition priming (e.g., Balota & Duchek, 1991; Moscovitch *et al.*, 1986). Although there is some controversy, a number of researchers have argued that the repetition priming effect is at least in part the

result of a preexisting memory representation of the stimulus remaining activated throughout the interval between the first and second presentations of that stimulus (Ratcliff, Hockley, & McKoon, 1985). Both the Balota and Duchek study, which employed word naming, and the Moscovitch *et al.* study, which employed mirror-reversed sentence reading, support the notion that implicit memory, as indexed via repeated exposures to items that have preexisting representations in memory, is relatively well preserved in SDAT individuals.

In addition to implicit tasks that tap preexisting memory representations (such as repetition priming), there are also implicit tasks that appear to tap the learning of new associations (see Howard, 1988, for a review). It is quite possible that, at least relatively early in the disease progression, SDAT individuals produce normal repetition priming effects that tap preexisting memory representations, but these individuals may produce deficits on implicit tasks that demand the formation of new associations.

Nissen and Bullemer (1987) developed the serial reaction time (SRT) task to specifically tap the learning of new associations. In the SRT, subjects are seated at a computer screen with their index and middle fingers of each hand resting on four keys of a keyboard. Four horizontal lines appear on the screen directly above each of the four keys. On each trial, an asterisk appears at one of the four screen locations and subjects are instructed to press the key corresponding to the asterisk's position each time the asterisk appears above it. A total of five blocks of 100 trials per block are typically administered. The first four blocks are identical to one another, each containing 10 repetitions of a 10-item sequence of screen locations. Because a sequence of this length exceeds the normal digit span, subjects should be unable to rehearse the sequence and hence are unlikely to notice that the sequence is being repeated. On Block 5, the sequence of asterisks is pseudorandom, with the only constraint being that an asterisk cannot appear at two consecutive screen locations.

There are two indices of performance in the SRT task that are particularly important. First, the latency difference across the first four blocks has been viewed as reflecting both generalized skill learning that occurs as subjects become familiar with the requirements of the task and any benefit in performance attributable to the repeating sequence. Second, the latency difference between Blocks 4 and 5 has been viewed as reflecting implicit learning. The rationale is that if subjects have stored the associations between the items in the repeated 10-item sequence across Blocks 1 to 4 and this facilitates performance across these blocks, then there should be an increase in response latency in Block 5, compared to Block 4, when the stimulus sequence becomes pseudorandom. Thus, slower response latencies in Block 5 compared to Block 4 is the measure of implicit learning in this task.

The SRT task is particularly well suited for use with a variety of patient

populations since its cognitive and motor demands are quite modest. The majority of the results gained from SRT performance on the numerous patient populations reported thus far supports the notion that the learning of new associations, as tapped by this task, appears relatively preserved (Grafman, Weingartner, Newhouse, Thompson, Lalonde, Litvan, Molchan, & Sunderland, 1990; Knopman & Nissen, 1987). For example, implicit learning as reflected by SRT performance appears to be preserved in individuals diagnosed with Korsakoff's syndrome amnesia (Nissen, Willingham, & Hartman, 1989) and AIDS dementia (Shulman, Dhanwan, Miller, Clifford, & Posner, 1991). The only subject groups revealing deficits in implicit learning when performing the SRT are those individuals diagnosed with Huntington's disease (Knopman & Nissen, 1991) or progressive supranuclear palsy (Grafman *et al.*, 1990).

It is also noteworthy that when college students perform the SRT task within a dual-task situation (e.g., simultaneously counting the number of audible tones they hear while performing the SRT task), implicit learning performance is disrupted (Nissen & Bullemer, 1987). This pattern is important because it supports the notion that implicit learning in the SRT task does demand attentional resources for satisfactory performance. In this light, because of evidence of attentional dysfunction in SDAT (e.g., Nestor, Parasuraman, & Haxby, 1991), one might expect a breakdown in implicit memory as indexed by the SRT task in these individuals.

Interestingly, however, both Knopman and Nissen (1987) and Grafman *et al.* (1990) have recently argued that there is no breakdown in implicit learning, as reflected by SRT task performance, in SDAT individuals. Unfortunately, there are aspects of both studies that may compromise this conclusion. First consider the Grafman *et al.* study. In this study, both implicit and explicit memory functioning were examined in several subject populations including healthy nondemented controls, dementia of the Alzheimer's type (DAT; onset prior to age 60) individuals, individuals diagnosed with progressive supranuclear palsy (PSP), and elderly depressed individuals. Grafman *et al.* compared the performance of the DAT group to the performance of the PSP group on the SRT task. While the DAT group showed an increase in response latency from the last block containing the repeating sequence to the block containing a random sequence, the PSP individuals surprisingly showed a decrease in response latency. Although the overall Block-by-Group interaction was reliable, it was unclear whether this was due to a reliable decrease in response latency between the last repeated sequence block and the random sequence block in the PSP group and/or a reliable increase in response latency in the SDAT individuals. Moreover, it would have been useful if Grafman *et al.* compared the DAT performance to an age-matched nondemented control group. It is quite possible that the DAT individuals would not have benefited as much as an age-matched control group from the re-

peating sequence. Finally, inspection of Fig. 4a from Grafman *et al.* reveals that the overall response latencies of the DAT subjects are considerably faster than the latencies of the SDAT individuals from the Knopman and Nissen (1987) study. In fact, the Grafman *et al.* DAT subjects' latencies are as fast as the very mildly demented subjects' in the present experiment (see Fig. 1). Since the performance of a control group was not reported on the SRT in this experiment, it is difficult to estimate degree of dementia. Thus, it is possible that these DAT individuals were relatively early into the disease progression. In order to address this possibility, a group of individuals who are in early stages of disease progression (very mild SDAT) along with a group of mildly demented SDAT individuals were included in the present study.

Knopman and Nissen (1987) also reported "normal" implicit memory in SDAT individuals as indicated by SRT task performance. As these authors readily admit, however, there are some aspects of their data that may compromise this conclusion. For example, although the overall Group-by-Block interaction failed to reach significance, the overall increase in RT from Block 4 to Block 5 for the SDAT group is considerably smaller than the corresponding increase in RT for the healthy control group (110 ms versus 181 ms, respectively). Moreover, of the 28 SDAT subjects that participated, 9 (32%) failed to show any increase in Block 5 latency as compared to Block 4 latency. Thus, these 9 subjects produced no evidence of implicit learning.

Because of the concerns with the Grafman *et al.* (1990) and Knopman and Nissen (1987) studies and because of the theoretical importance of tapping implicit performance via the development of new associations in SDAT individuals, we further investigated implicit learning using the SRT task in groups of healthy nondemented aged adults along with individuals diagnosed with either very mild (questionable) or mild SDAT. An important aspect of the present experiment is the inclusion of two different levels of AD severity. Thus, it will be possible to examine the difference in implicit memory performance at different stages of disease progression. This is of particular interest in light of the Grafman *et al.* results described above.

The present experiment also examined a group of nondemented Parkinson's Disease (PD) individuals. The inclusion of a group of PD individuals is relevant in the context of the present experiment because results of some implicit memory tests like word-fragment completion, mirror-reversed tracking, mirror-reading, and pursuit-rotor tracking reveal preserved performance in nondemented PD individuals (e.g., Bondi & Kaszniak, 1991; Frith, Bloxham, & Carpenter, 1986; Taylor, Saint-Cyr, & Lang, 1990). On the other hand, performance on other tests of implicit memory like the *Tower of Toronto* puzzle, the fragmented picture completion task, and maze learning (e.g., Bondi & Kaszniak, 1991; Saint-

Cyr, Taylor, & Lang, 1988; Wallesch, Karnath, Papagno, Zimmerman, Dueschl, & Lucking, 1990) produce substantial deficits in nondemented PD individuals. Thus, it appears that the implicit memory deficits associated with nondemented PD individuals are inconsistent from test to test. However, it is noteworthy to mention that Bondi and Kaszniak found preserved implicit memory performance in nondemented PD individuals on tests of mirror-reading and pursuit-rotor tracking. Moreover, these authors claimed that these tests, like the SRT task, tap processes related to perceptual-motor skill learning. If their argument is correct, then we should find preserved implicit memory performance in our sample of nondemented PD individuals on the SRT task.

## METHODS

### *Subjects*

A total of 85 subjects participated. All subjects were recruited from the Washington University Alzheimer's Disease Research Center (ADRC). The participants were originally screened for depression, severe hypertension, reversible dementias, and any other disorders that could affect cognitive performance. Inclusionary and exclusionary criteria for SDAT conform to those outlined in the NINCDS-ADRDA criteria (McKhann, Drachman, Folstein, Katzman, Price, & Stadlan, 1984). Dementia severity for each participant was staged in accordance with the Washington University Clinical Dementia Rating (CDR) scale (Berg, 1988; Hughes, Berg, Danziger, Coben, & Martin, 1982). In this scale a score of 0 indicates no dementia; a score of .5 indicates very mild, or "Questionable," dementia; a score of 1 indicates "Mild" dementia; and a score of 2 indicates "Moderate" dementia.

The CDR is based on a 90-min interview that assesses cognitive ability in areas including memory, orientation, problem solving, community affairs, hobbies, and personal care. Both the patient and his or her collateral source (e.g., spouse, child) participate in the interview. One of eight board-certified physicians (four neurologists and four psychiatrists) conducted these interviews, which were all videotaped and subsequently reviewed by a second physician for reliability. The diagnosis of AD by this research team has been excellent, with 89/92 (97%) individuals diagnosed with SDAT indeed having AD confirmed at autopsy (Berg, Smith, Morris, *et al.*, 1990; Burke, Miller, Rubin, *et al.*, 1988; Morris, McKeel, Fulling, Torack, & Berg, 1988; Morris, McKeel, Price, *et al.*, 1988).

Of the 85 participants in the present study, 43 were diagnosed with no dementia (i.e., CDR = 0). Of these, 17 had a diagnosis of Parkinson's disease (mean age = 69 years) and 26 (mean age = 70 years) were healthy age-matched control individuals (CDR = 0). As noted, the PD individuals were cognitively unimpaired (nondemented, CDR = 0), although most required medications (e.g., Sinemet) that are designed to assist in the control of the disease. Of the remaining participants, 27 were diagnosed with very mild dementia (mean age = 73 years), and 15 were diagnosed with mild dementia (mean age = 74 years). It is important to note here that there is now evidence to suggest that in another study of this subject population that a subset of individuals (11/16) originally classified as having very mild dementia (CDR = .5) actually progressed to a more severe stage of SDAT over the course of 84 months or had SDAT positively confirmed at autopsy (Rubin, Morris, Grant, & Vendega, 1989). This finding supports the view that a diagnosis of very mild SDAT indicates an early stage in the disease progression.

### *Psychometric Test Performance*

Each participant enrolled in the ADRC is administered a 2-hr battery of psychometric tests designed to assess psychological functions including memory, language, psychomotor performance, and intelligence. Memory performance was assessed via the following: Wechsler Memory Scale (WMS; paired-associate learning; Wechsler & Stone, 1973), Benton Visual Retention Test (picture memory; Benton, 1963), WMS Logical Memory (surface-level story memory), and WMS forward and backward digit span. Intelligence was assessed using the following subtests of the Wechsler Adult Intelligence Scale (WAIS): Information, Comprehension, Block Design, and Digit Symbol (Wechsler, 1955). Visual Perceptual-Motor performance was assessed by the Benton Copy Test and Trail Making Form A. In the Benton Copy Test, participants must copy a geometric figure; in Trail Making Form A, participants connect numerically ordered dots that result in a specified pattern (Armitage, 1946). Participants also received the WMS Mental Control test which evaluates the ability to quickly produce a well-rehearsed letter or digit sequence, such as the alphabet, in a specified amount of time. Participants also received the Word Fluency test, which addresses processes associated with lexical retrieval (Thurstone & Thurstone, 1949). In this task, subjects are required to quickly generate as many words beginning with a specified letter (P or S) in an allotted time period (60 sec per letter). As shown in Table 1, the results of the psychometric tests revealed the expected effect of CDR level with the only exception being Benton Copy Errors. Specifically, performance consistently becomes poorer as dementia severity increases.

### *Apparatus*

All testing was performed using an Apple IIe microcomputer that was interfaced with a Mountain Hardware clock card accurate to the nearest millisecond.

### *Procedure*

Procedures were similar to those of Knopman and Nissen (1987). Subjects sat approximately 30 cm from the Apple IIe monitor. Subjects rested their index and middle fingers of the left hand on the 3 and 5 keys and the index and middle fingers of their right hand on the 7 and 9 keys. Four horizontal lines appeared at the bottom of the computer screen which were physically aligned with the four keys. On each trial an asterisk (\*) appeared at one of the four possible monitor locations and subjects were instructed to quickly press the key which corresponded to the location of the asterisk on the monitor. Each asterisk remained on the screen until the subject responded. If the subject responded incorrectly, the asterisk remained at that screen location until the correct key was struck. There was a 500-ms delay between each key press and the presentation of the next asterisk. The asterisk never appeared at the same location on successive trials. Subjects practiced the task at their own pace until it was clear that they understood the instructions. Following practice, each subject received five blocks of 100 key presses, for a total of 500 key presses across the entire experiment. In Blocks 1-4, a 10-item sequence repeated itself a total of 10 times. In Block 5 the sequence of asterisks was presented in a pseudorandom order. The experiment lasted approximately 20-25 min.

### *Design*

The design is a 4 (Control, nondemented PD, very mild SDAT, and mild SDAT) by 5 (Block) mixed-factor design.

TABLE 1  
Mean (and Standard Deviation) Psychometric Test Performance for Each Subject Group

Test	Subject Group				F value
	Healthy elderly controls	Nondemented PD	Very mild SDAT	Mild SDAT	
Logical Memory	10.52 (2.73)	8.59 (3.06)	5.30 (3.12)	2.60 (1.64)	31.04†
Trails Form A	38.04 (13.20)	58.65 (28.68)	56.07 (28.24)	64.53 (21.50)	5.02**
WAIS Information	22.16 (3.89)	21.23 (3.23)	16.52 (5.83)	11.93 (4.73)	18.71†
WAIS Block Design	34.48 (7.43)	30.94 (8.36)	24.15 (11.60)	21.00 (7.32)	9.29†
WAIS Digit Symbol	52.12 (12.74)	42.94 (11.18)	35.78 (12.66)	31.00 (6.21)	13.76†
Benton Delay (No. correct)	6.60 (1.47)	6.06 (1.85)	4.37 (2.08)	2.93 (1.79)	15.81†
Benton Copy (No. correct)	9.92 (.40)	9.76 (.44)	9.22 (1.34)	9.53 (.64)	3.12*
Boston Naming Test	56.80 (2.42)	56.00 (3.54)	49.15 (10.34)	41.13 (13.24)	13.42†
Mental Control	7.92 (1.44)	7.71 (1.76)	5.96 (2.81)	6.20 (2.21)	4.86**
Associate Recall	14.72 (4.02)	14.24 (3.72)	10.48 (4.20)	6.47 (2.29)	18.41†
Benton Recall (errors)	5.40 (2.48)	6.47 (3.52)	11.52 (5.71)	15.00 (4.72)	20.14†
Benton Copy (errors)	.12 (.60)	.29 (.59)	.81 (1.42)	.47 (.64)	2.49
Word Fluency (S + P)	34.80 (13.62)	30.59 (13.51)	26.33 (9.74)	19.93 (7.20)	5.79**
Digit Span (F + B)	12.72 (2.39)	11.65 (2.98)	10.59 (1.99)	10.27 (1.79)	5.09**

Note. SDAT indicates senile dementia of the Alzheimer's type, PD indicates Parkinson's disease, F value is from Group main effect, and the degrees of freedom associated with this value are 3 and 80.

\*  $p < .05$ .

\*\*  $p < .01$ .

\*\*\*  $p < .001$ .

†  $p < .0001$ .

## RESULTS

Unless otherwise stated, all analyses on response latencies are based on log-transformed scores. Figure 1 presents the mean of the median response latencies as a function of Group and Block. There are several aspects of Fig. 1 to consider: First, across all subject groups, response latency decreased at a similar rate across Blocks 1 to 4. This pattern



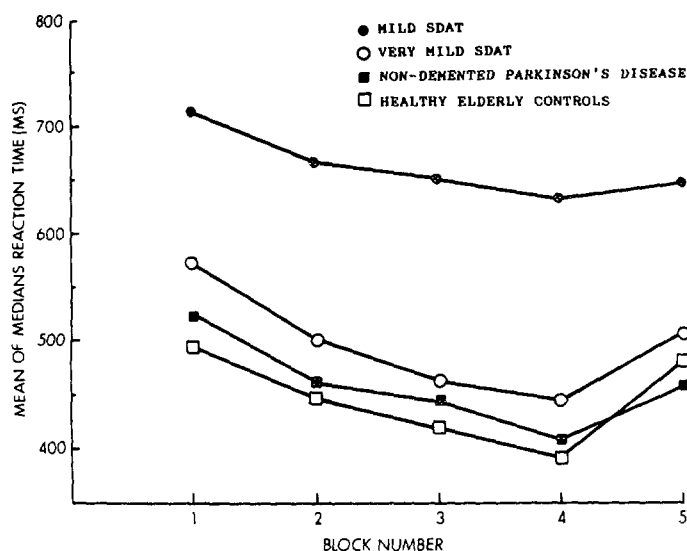


FIG. 1. Mean of median reaction time (in ms) as a function of Subject Group and Block.

could be due to subjects learning the sequence and/or becoming familiar with the demands of the SRT task. Second, turning to the indicant of implicit learning, although overall response latencies appear to increase between Block 5 and Block 4, this difference appears to be smaller for the mildly demented individuals compared to both the healthy control individuals and the very mildly demented individuals. Finally, the PD individuals appear to also show some reduction in latency difference between Blocks 4 and 5 compared to that of the healthy control individuals.

The above observations were supported by a 4 (Group)  $\times$  5 (Block) mixed-factor ANOVA. This analysis yielded main effects of Group,  $F(3, 81) = 3.20$ ,  $MSe = .002$ ,  $p < .03$ , and Block,  $F(4, 324) = 46.61$ ,  $MSe = .104$ ,  $p < .0001$ . More importantly, there was also a reliable Group-by-Block interaction,  $F(12, 324) = 2.04$ ,  $MSe = .005$ ,  $p < .03$ .

In order to further investigate the Group-by-Block interaction, separate ANOVAs were performed on Blocks 1 through 4 and Blocks 4 and 5. The analysis on Blocks 1 through 4, which we will refer to as the *Generalized Skill* analysis, is somewhat ambiguous with respect to the underlying mechanism because it may reflect both the benefit from generalized skill learning along with benefit from the repetition of the sequence. More importantly, the analysis of Blocks 4 and 5, which we will refer to as the *Implicit Learning* analysis, reflects the interference caused by switching from a well-practiced sequence to a pseudorandom sequence.

The *Generalized Skill* analysis was a 4 (Group)  $\times$  4 (Block) mixed-factor ANOVA. This analysis yielded main effects of Group,  $F(3, 81) =$

3.23,  $MSe = .280$ ,  $p < .03$  and Block,  $F(3, 243) = 74.21$ ,  $MSe = .130$ ,  $p < .0001$ . The Group-by-Block interaction did not reach significance,  $F(9, 243) = 1.72$ ,  $MSe = .003$ ,  $p = .08$ . Separate one-way ANOVAs were performed for each group across Blocks 1–4. Each analysis revealed a reliable decrease in response latency across Blocks 1 to 4: nondemented healthy aged controls,  $F(3, 75) = 21.86$ ,  $MSe = .002$ ,  $p < .0001$ ; nondemented PD individuals,  $F(3, 48) = 27.87$ ,  $MSe = .001$ ,  $p < .0001$ ; very mildly demented individuals,  $F(3, 78) = 41.57$ ,  $MSe = .002$ ,  $p < .0001$ ; mildly demented individuals,  $F(3, 42) = 5.20$ ,  $MSe = .002$ ,  $p < .01$ .

The *Implicit Learning* analysis was a 4 (Group)  $\times$  2 (Block) mixed-factor ANOVA and resulted in main effects for Group,  $F(3, 81) = 3.47$ ,  $MSe = .125$ ,  $p < .02$  and Block,  $F(1, 81) = 46.27$ ,  $MSe = .149$ ,  $p < .01$ . More importantly, there was also a reliable Group-by-Block interaction,  $F(3, 81) = 3.06$ ,  $MSe = .009$ ,  $p < .04$ . Across groups, the nondemented healthy aged adults displayed the greatest amount of implicit learning (88 *ms*), followed by the very mildly demented group (62 *ms*), the nondemented PD group (51 *ms*), and the mildly demented group (15 *ms*).

To further investigate the influence of disease status (i.e., PD and SDAT) on implicit learning, separate analyses were performed comparing each group to the nondemented healthy control individuals. The comparison of the nondemented healthy aged adults to the very mildly demented SDAT individuals only revealed a main effect of Block,  $F(1, 51) = 43.78$ ,  $MSe = .005$ ,  $p < .01$ . However, neither the Group main effect nor the Group-by-Block interaction was significant (both  $ps > .42$ ). This analysis suggests no difference in the amount of implicit learning displayed by the nondemented healthy aged adults and the very mildly demented SDAT individuals. Results from the remaining two analyses, however, were substantially different. In the comparison between the healthy control group and the nondemented PD group, there was no main effect of Group,  $F(1, 41) < 1$ , but there was a reliable main effect of Block,  $F(1, 41) = 63.94$ ,  $MSe = .002$ ,  $p < .01$ , and, more importantly, there was a reliable Group-by-Block interaction,  $F(1, 41) = 5.50$ ,  $MSe = .002$ ,  $p < .03$ . This interaction indicated that the nondemented PD individuals produced *less* implicit learning than the healthy control individuals. The results of the comparison between the healthy control individuals and the mildly demented individuals yielded main effects of Group,  $F(1, 39) = 7.55$ ,  $MSe = .04$ ,  $p < .01$  and Block,  $F(1, 39) = 37.21$ ,  $MSe = .002$ ,  $p < .01$ , along with a reliable Group-by-Block interaction,  $F(1, 39) = 15.15$ ,  $MSe = .002$ ,  $p < .01$ . These results clearly indicate that the mildly demented individuals produced decreased implicit memory as measured by the SRT task. Finally, an analysis of implicit learning for the two impaired groups, the PD individuals and the mildly demented SDAT individuals, was conducted to compare the degree of impairment in these two groups. The analysis revealed a reliable effect of Group,  $F(1, 30) = 5.74$ ,  $MSe =$

.046,  $p < .03$ , a reliable effect of Block,  $F(1, 30) = 21.11$ ,  $MSe = .001$ ,  $p < .01$ , and a marginally reliable Group-by-Block interaction,  $F(1, 30) = 3.79$ ,  $MSe = .001$ ,  $p = .061$ . This marginal interaction indicates that the SDAT group appears to be more impaired in implicit learning performance than the PD group.

Table 2 displays the mean percentage correct performance as a function of Group and Block. There are three observations that should be made from Table 2. First, accuracy was overall very high. This is quite important because it indicates that this task can be performed with high accuracy even by the mildly demented individuals. Second, across Blocks 1 through 4, accuracy increased across all subject groups. Third, accuracy decreased slightly across Blocks 4 and 5 for all groups.

Results of an ANOVA yielded a main effect of both Group,  $F(3, 81) = 3.92$ ,  $MSe = 209.56$ ,  $p < .05$ , and Block,  $F(4, 324) = 3.11$ ,  $MSe = 28.32$ ,  $p < .05$ , while the Group-by-Block interaction did not approach significance,  $F(12, 324) < 1$ . In order to provide information regarding implicit learning, a separate ANOVA was conducted on the accuracy data from Blocks 4 and 5. This ANOVA yielded main effects of Group,  $F(3, 81) = 4.18$ ,  $MSe = 56.08$ ,  $p < .01$ , and Block,  $F(1, 81) = 7.13$ ,  $MSe = 15.99$ ,  $p < .01$ ; however, the Group-by-Block interaction did not approach significance,  $F(3, 81) < 1$ . Of course, one must be cautious in drawing any strong conclusions from the accuracy data because it appears that all groups are close to ceiling on this measure.

Table 3 displays Pearson-product moment correlations for each subject group as a function of Psychometric Test performance and the implicit learning measure from the SRT task, that is, Block 5 RT minus Block 4 RT. These correlations were performed in order to determine how the factors tapped by psychometric test performance were related to implicit learning performance. One aspect of Table 3 that is immediately apparent is the relative lack of reliable correlations between the measure of implicit memory and the psychometric test functions for the nonde-

TABLE 2  
Mean Percentage Correct as a Function of Subject Group and Block

Group	Block				
	1	2	3	4	5
Healthy elderly controls	96.7	97.3	98.1	97.8	97.0
Nondemented PD	93.2	93.1	92.4	95.5	93.0
Very mild SDAT	90.3	91.2	93.1	93.0	91.6
Mild SDAT	89.7	92.4	89.5	95.1	93.0

*Note.* SDAT indicates senile dementia of the Alzheimer's type; PD indicates Parkinson's disease.

TABLE 3  
Pearson-Product Moment Correlations for Each Subject Group as a Function of  
Psychometric Test Performance and Amount of Implicit Learning

Test	Subject group			
	Healthy elderly controls	Nondemented PD	Very mild SDAT	Mild SDAT
Logical Memory	.24	-.03	.35	-.42
Trails Form A	-.25	-.11	-.43*	-.44
WAIS Information	-.14	.41	-.13	.25
WAIS Block Design	-.19	-.23	.57**	.22
WAIS Digit Symbol	.10	.18	.56**	.04
Benton Delay (No. Correct)	-.12	-.35	.27	.40
Benton Copy (No. Correct)	.05	-.26	.38*	-.21
Boston Naming Test	.18	.06	-.04	.36
Mental Control	.02	-.10	.10	.05
Associate Recall	.26	.20	.26	.35
Benton recall (errors)	.19	.26	-.34	.40
Benton copy (errors)	-.05	.24	-.40*	.21
Word Fluency (S + P)	.12	-.14	.19	.10
Digit Span (F + B)	.06	.16	-.06	.34

*Note.* Implicit learning is indicated by the Block 5-4 latency difference. SDAT indicates senile dementia of the Alzheimer's type; PD indicates Parkinson's disease.

\*  $p < .05$ .

\*\*  $p < .01$ .

mented healthy aged, nondemented PD, and the mildly demented SDAT individuals. The only subject group to reveal any reliable correlations between implicit learning and psychometric test performance is the very mildly demented group.<sup>1</sup> Close inspection of those psychometric tests which revealed reliable correlations (Trails A, WAIS Block, WAIS Digit-Symbol, Benton Copy Form D-Number Correct, Benton Recall Errors,

<sup>1</sup> Recently, LaBarge, Balota, Storandt, and Smith (1992) revealed a similar pattern of correlations among their very mildly demented group with measures of psychometric test performance and Boston Naming Test performance. They interpreted this pattern as indicating that the rather widespread and interrelated disruption of cognitive functions are characteristic of the very early stages of SDAT. The present pattern of correlations extends this finding to include implicit memory performance as measured by the SRT task. One caveat, however, concerning the various correlations is the fact that the nondemented healthy aged individuals are quite accurate on Benton Copy Errors while the mildly demented individuals are quite inaccurate on Logical Memory. As a result of these ceiling and floor effects, correlations obtained between dementia severity and results of these two psychometric tests should be viewed cautiously due to the limited ranges obtained. Group performance on the remaining psychometric tests, however, falls within acceptable ranges and therefore correlations with these tests are informative.

Benton Copy Errors) indicate that these tests rely more on perceptual-motor aptitude for completion than on verbal aptitude. Furthermore, it is interesting to note that most of the psychometric test measures that did not correlate with implicit learning performance are tests which presumably tap into explicit memory functioning (e.g., WMS paired-associate memory, Benton Visual Retention test, WMS Logical Memory, WMS digits forward and backward). The lack of a reliable correlation between the psychometric tasks that tap explicit memory performance and the implicit memory measure from the SRT task is important because it suggests that differences in performance across groups are not simply due to differences in the involvement of explicit memory processes.

## DISCUSSION

The results from the current experiment can be summarized as follows: Using the SRT task of Nissen and Bullemer (1987), the present results demonstrate relatively comparable implicit learning in healthy nondemented aged individuals and in very mildly demented SDAT individuals. However, there does appear to be some breakdown in implicit learning in nondemented PD individuals and an even greater breakdown in mildly demented SDAT individuals. We will first discuss the SRT performance in the PD individuals and will then turn to a discussion of the results from the mildly demented SDAT individuals.

### *SRT Task Performance and Parkinson's Disease*

The present results suggest that nondemented PD individuals do not develop new associative/implicit memories at a normal rate in the perceptual-motor domain, at least as measured by the SRT task. However, before accepting this conclusion, one might ask whether the PD individuals used in the present sample were indeed nondemented. In fact, if one considers the psychometric data displayed in Table 1, one can see some slight breakdowns in certain task compared to the healthy control individuals. There are two points to note about this pattern: First, the PD individuals were overall more similar on the psychometric battery to the healthy control individuals than to the very mildly demented individuals. This is important, because even the very mildly demented individuals produced no evidence of a breakdown in implicit learning. Second, it is interesting to note that the PD individuals produced overall response latencies that were relatively fast and quite similar to the healthy control individuals. Thus, the breakdown in implicit learning in this group is not simply due to an overall impairment in task performance.

As mentioned in the *Introduction*, the results from previous investigations addressing implicit memory performance in nondemented PD individuals have been mixed, with some studies revealing preserved function

and others producing evidence for implicit memory deficits. For instance, Saint-Cyr *et al.* (1988) found that nondemented PD individuals were impaired on the *Tower of Toronto* (similar to the *Tower of Hanoi*) procedural memory task. Likewise, Bondi and Kaszniak (1991) found that nondemented PD individuals were impaired on the skill learning component of the fragmented picture completion test, and Wallesch *et al.* (1990) found that nondemented PD individuals were impaired on a test of maze learning in which only a specified section of the maze was visible to participants. The SRT task can now be added to this list of tasks that appear to produce breakdowns in implicit learning in nondemented PD individuals. Conversely, several studies have found preserved implicit memory performance in nondemented PD individuals in tasks including mirror-reversed tracking (Frith *et al.*, 1986), word-fragment priming (Taylor *et al.*, 1990), mirror-reversed reading, and pursuit-rotor tracking (Bondi & Kaszniak, 1991). At this level, the present results would appear to be inconsistent with the arguments by Bondi and Kaszniak, who suggested that the SRT task taps operations that are similar to the operations tapped by the mirror-reading and pursuit-rotor tracking tasks, and hence, should not produce an implicit memory impairment in nondemented PD individuals. Although we would agree that these tasks, on the surface, appear to tap similar perceptual motor skill operations, the breakdown observed in the present data suggests that (a) the tasks do not tap the same underlying structures and/or (b) the tasks differ in sensitivity to breakdowns in the same underlying structures.

Turning to possible underlying neural mechanisms, the results from the PD individuals may be viewed as suggesting a role of the frontal-striatal system that has already been implicated in a wide variety of cognitive task performance in PD individuals (e.g., Freedman & Oscar-Berman, 1989; Sullivan & Sagar, 1989, 1991; Saint-Cyr *et al.*, 1988; Taylor, Saint-Cyr, & Lang, 1986, 1990). Moreover, evidence indicating that Huntington's disease patients are deficient on the SRT task (e.g., Knopman & Nissen, 1991) and that attentional resources are necessary for adequate performance on the SRT task is consistent with the notion of an important role for frontal areas. While frontal lobe dysfunction has not typically been associated with *early* stages of SDAT, it is noteworthy to mention that some SDAT individuals at autopsy have had deficits associated with PD, such as nigral degeneration. This occurs despite the fact that our SDAT individuals are clinically screened for Parkinsonian symptoms at the time of entry (see Morris *et al.*, 1988). The marginally reliable Group (PD vs. mild SDAT) by Block (4 vs. 5) interaction points out that the impairment in performance of the PD group on the SRT task was somewhat less than the impairment of the mildly demented SDAT group. Of course, it is possible that the PD individuals could have an attentional deficit that is mediated by the frontal/striatal complex and this deficit is

simply more pronounced in the SDAT group. Although this is possible, we believe that the most parsimonious account of the present results is that the *primary* substrate underlying the breakdown in PD individuals is the frontal/striatal complex, whereas the *primary* substrate underlying the more severe breakdown in AD individuals is the rather widespread involvement of neural structures, in which frontal structures are only a subset of the many relevant structures involved in this disease.

### *SRT Task Performance and SDAT*

We now turn our attention to the results from the SDAT individuals. Inspection of Fig. 1 reveals that our mildly demented individuals displayed little (15 ms) implicit learning as measured by the difference between Block 5 and Block 4 performance. This pattern differs from Grafman *et al.* (1990) and Knopman and Nissen (1987), who both investigated SRT task performance and argued that there were no impairments in implicit learning in SDAT individuals. Discrepancies between implicit memory studies in SDAT individuals have also been reported in tests of word-stem completion, with some investigators finding performance preserved in SDAT individuals (Grosse *et al.*, 1990) and others finding that performance is impaired (e.g., Bondi & Kazsniak, 1991; Heindel, Salmon, Shults, Walicke, & Butters, 1989; Keane, Gabrielli, Fennema, Growdin, & Corkin, 1991). We shall now attempt to resolve the apparent discrepancy across the studies that address SRT task performance in SDAT individuals.

As described previously, the conclusions reached by Grafman *et al.* (1990) and Knopman and Nissen (1987) should be interpreted with caution. In Grafman *et al.*, the overall response latency of the DAT individuals is considerably faster than both the DAT individuals' performance in the Knopman and Nissen study as well as from the SDAT individuals who participated in the present experiment. It is possible that the DAT individuals who participated in the Grafman *et al.* study were more similar to the very mildly demented individuals who participated in the present experiment. As is evident from Fig. 1, implicit memory performance is relatively preserved in the very mildly demented group. By comparison, the mildly demented group revealed less implicit memory performance than the very mildly demented group (15 ms versus 64 ms, respectively).

The Knopman and Nissen (1987) conclusion of intact implicit learning in the SRT task should also be interpreted cautiously. As noted earlier, 9/28 (32%) of the SDAT individuals tested by Knopman and Nissen failed to display even minimal implicit learning. That is, these 9 individuals actually produced faster response latencies in Block 5 as compared to Block 4. Furthermore, the SDAT individuals, as a group, in the Knopman

and Nissen study revealed lower overall implicit memory performance, compared to that of the control subjects (110 *ms* versus 181 *ms*, respectively, for the AD and the control subjects). Thus, instead of accepting the null hypothesis, we feel that the Knopman and Nissen results, along with those from the present study, support the contention that there is a breakdown in implicit memory in mildly demented SDAT individuals, as reflected by SRT task performance.

The finding of a breakdown in implicit learning in SDAT individuals in the SRT task is quite important because this task presumably taps the learning of new associations in the perceptual motor domain. As noted earlier, Squire (1986) has suggested that one might obtain such breakdowns in SDAT individuals because of the rather widespread involvement of neural structures in AD. Squire has argued that such a widespread involvement is more likely to tap the structures involved in procedural (or what we refer to as implicit) learning. In addition, there is clear evidence of attentional breakdowns in these SDAT individuals, and, as Nissen and Bullemer (1987) originally documented, attention appears to be important for implicit learning as measured by the SRT task. Thus, based on both Squire's arguments regarding the widespread involvement of neural structures involved in procedural memory tasks and Nissen and Bullemer's arguments regarding the attentional requirements demanded by the SRT task, one should expect a breakdown in performance in SDAT individuals, as the present results clearly indicate.

Thus, if the task demands the formation of new associations, as measured by SRT task performance, then it is likely that mildly demented SDAT individuals will produce some deficit. However, if the task primarily demands reactivation of previously existing representations as in mirror-reversed reading (see Moscovitch *et al.*, 1986) or in simple repetition priming (see Balota & Duchek, 1991), at least mildly demented SDAT individuals will produce relatively little deficit. In this light, it is important to emphasize here that one must be cautious to restrict one's arguments regarding cognitive functioning in SDAT individuals to particular stages of disease progression. We would not be surprised to find some breakdown in even simple repetition priming effects in more severe stages of dementia, in which preexisting memory structures begin to deteriorate. As shown in the present study, one might be led to considerably different conclusions based on performance from a very mildly demented group of individuals versus a mildly demented group of individuals. This is precisely why we feel that it is paramount to address task performance at various levels of disease progression.

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