Neuropsychology
1994, Vol. 8, No. 2, 218-226

0894-4105/94/$3.00

Component Analysis of a Rhythmic Finger Tapping Task in Individuals With Senile Dementia of the Alzheimer Type and in Individuals With Parkinson's Disease

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The present experiment examined different components of motor control that may be impaired in normal aging, senile dementia of the Alzheimer type (SDAT), and Parkinson's disease (PD). Specifically, A. M. Wing and A. B. Kristofferson's (1973) formal quantitative model of rhythmic finger tapping was used to obtain estimates of central timekeeping and response execution components of timing control. Subjects included young college students, healthy older adults, nondemented individuals with PD, and individuals with very mild and mild SDAT. Individuals with mild SDAT exhibited a breakdown in the central timekeeping mechanism but not in the execution of the response. Both very mild SDAT and PD individuals did not show any deficits in the two timing mechanisms relative to age-matched healthy controls. Finally, there was no effect of normal aging on timing control in this task. This study underscores the importance of examining issues of motor control in SDAT as a function of separate processing components and stages of disease progression.

Senile dementia of the Alzheimer type (SDAT) is characterized by a relatively generalized breakdown in cognitive performance. Although the deficits observed in Alzheimer's disease appear to encompass most aspects of cognitive performance, it is has been shown that isolatable aspects of cognitive performance appear to deteriorate at different rates as the disease progresses (see, e.g., Balota & Duchek, 1991). The isolation of such subcomponents in SDAT is important because it will allow a better understanding of the cognitive breakdowns in these individuals and potentially help in the development of better screening procedures. Finally, such an analysis may also provide useful information regarding models of healthy cognitive functioning (e.g., see Balota & Ferraro, 1993).

Although there is an extensive literature delineating cognitive deficits in SDAT individuals in the areas of attention, memory, and language (see Nebes, 1989, for a review), one component of performance that has received relatively little attention is the area of motor control. Moreover, the few available reports addressing motor control in SDAT have provided little evidence regarding specific subcomponents of performance.

In the present study, an attempt was made to utilize an experimental paradigm that was specifically developed to isolate separate processes in motor control. This investigation involved a simple finger tapping task and relied on the formal analytic model of performance in this task that was developed by Wing and Kristofferson (1973). In this task, subjects are required to tap their finger in rhythm with a computer-generated tone. After the tone is terminated, the subject continues tapping at the same rate for some period of time. The major dependent measure of interest in this task is the variability in the interresponse interval (IRI).

According to Wing and Kristofferson's (1973) model, the variation of the IRI can be broken down into two independent components: clock delay variance and motor delay variance. Clock delay refers to the delay interval between the internal trigger for response i and the internal trigger for the next response ($i + 1$). Motor delay refers to the delay interval between the internal trigger for a response and its actual execution, that is, the time taken to execute a response after the signal to respond has been initiated.

Wing and Kristofferson's (1973) model predicts that the variation in the motor delay component should produce a negative correlation between adjacent intervals. The notion is that if the motor delay that begins a response interval is long, then the subsequent interval will be shorter and vice versa. Given that the model predicts statistical dependence among the IRIs (i.e., adjacent IRIs will be negatively correlated), the motor delay variance is calculated from the negative lag-one covariance, and the clock variance can be estimated by subtracting the motor variance from the total variance. (For mathematical derivations and a fuller exposition of the model, see Wing, 1980; Wing & Kristofferson, 1973).

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This work was supported by National Institute on Aging Grant PO1 AG03991. Thanks are extended to the Clinical and Psychometric Cores of the Washington University Alzheimer's Disease Research Center for their diagnostic and testing assistance. We would like to especially acknowledge John Morris for his diagnostic assistance with the Parkinson's patients. We also thank Lisa Connor for helping in the early stages of this project and Virginia Buckles and three anonymous reviewers for comments on an earlier draft of this article.

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The important point for the present research is that within Wing and Kristofferson's (1973) model, clock delay and motor delay represent different components of the motor control system. The motor delay component is viewed as a function of a motor implementation system. However, the clock delay component is viewed as a central timekeeping mechanism. Thus, this model affords a componential analysis of the processes involved in simple rhythmic tapping across healthy older individuals and those with SDAT.

As already noted, there are relatively few data available regarding the components of timing control in SDAT. In one study of finger tapping performance, it was found that SDAT individuals were significantly slower and produced more variability in IRIs than a group of healthy controls (Muller, Weisbrod, & Klingberg, 1991). However, Muller et al. did not decompose the variability of the IRIs into clock and motor delay estimates. Therefore, there is no indication whether the breakdown in the control of timing was due to a deficit in the central timing mechanism, response execution, or both.

One might expect a breakdown in the central timing mechanism in SDAT given the rather widespread nature of the cognitive deficits associated with the disease. If one considers the timekeeping mechanism as being important in the coordination of inputs from various levels within the processing system, then it is possible that a disruption in this system would lead to deficits across most cognitive tasks that demand such coordination.

Thus, one of the major goals of the present study was to isolate different components of motor control (i.e., the central timekeeping mechanism and response execution components) at various stages of SDAT. The inclusion of two levels of SDAT was crucial for understanding the rate of change in these components across different levels of disease progression. Moreover, the finger tapping task places minimal demands on other cognitive operations, such as memory and language, and hence is ideally suited for testing with SDAT individuals in early and mild stages of disease progression. Because individuals with "pure" SDAT do not appear to exhibit a substantial motor breakdown relative to other patient groups, such as those with Parkinson's disease (PD), the former group may exhibit a deficit in the central timekeeping mechanism but not necessarily in the execution of the response. Furthermore, one might expect that the deficit in the central timekeeping mechanism may change as a function of the stage of SDAT.

In addition, a group of young college students, two groups of healthy older individuals, and a group of medicated, nondemented PD patients were also included. There has been much discussion in the aging literature regarding the attribution of cognitive deficits in normal aging to a generalized slowing of processing (Cerella, 1990; Myerson, Ferraro, Hale, & Lima, 1992; Salthouse, 1985a, 1985b). According to one version of the generalized slowing model, all cognitive processes are slowed at a constant rate. The clock delay estimate obtained from Wing and Kristofferson's (1973) model affords a unique opportunity to test one potential mechanism underlying the observed general slowing. Furthermore, there is very little literature on healthy older adults over 80 years of age (e.g., Johansson, Zarit, & Berg, 1992). This group is of particular interest in light of arguments that SDAT may merely represent accelerated normal aging. Thus, just as it was important to include two levels of disease progression, it was also useful to include two levels of healthy aging. Finally, the PD group was included to assess whether a disease condition that produces a breakdown in motor function would differentially affect the two components of timing relative to normal aging and SDAT. Although this same experimental paradigm has been used with PD subjects in previous studies (e.g., Ivry & Keele, 1989; Keele & Ivry, 1987; Wing, Keele, & Margolin, 1984), there has not been any comparison of clock or motor delay variation in PD relative to early stages of SDAT.

Method

Subjects

A total of 167 subjects were recruited from the Washington University Alzheimer's Disease Research Center (ADRC) for this study. All participants were originally screened for depression, hypertension, reversible dementias, and other disorders that could potentially produce cognitive impairment. The inclusionary and exclusionary criteria for SDAT are consistent with the criteria of the National Institute of Neurological and Communicative Disorders and Stroke and the Alzheimer's Disease and Related Disorders Association (NINCDS–ADRDA; McKhann, Drachman, Folstein, Katzman, Price, & Stadlan, 1984). The severity of dementia was staged according to the Washington University Clinical Dementia Rating (CDR) scale (Berg, 1988; Hughes, Berg, Danziger, Coben, & Martin, 1982). According to this scale, CDR 0, 0.5, 1, and 2 represent no dementia, questionable or very mild dementia, mild dementia, and moderate dementia, respectively. The label *questionable dementia* (CDR 0.5) was used for individuals with very mild dementia. In fact, the majority of these individuals (11 of 16) either progressed to more severe stages of SDAT or had the diagnosis of Alzheimer's disease confirmed upon autopsy (Morris, McKeel, Fulling, et al., 1988; Rubin, Morris, Grant, & Vendegna, 1989).

The CDR is based on a 90-min interview with both the subject and a collateral source. This interview assesses the subjects' cognitive abilities in the areas of memory, orientation, judgment and problem solving, community affairs, home and hobbies, and personal care. The original interview is conducted by one board-certified physician (neurologist or psychiatrist) and is videotaped for review by a second physician. Both the reliability of the CDR and validation of the diagnosis with this research team have been excellent and well-documented (Berg et al., 1990; Burke et al., 1988; Morris, McKeel, Fulling, et al., 1988; Morris, McKeel, Price, et al., 1988). Presently, 103 of 107 individuals diagnosed with SDAT have had Alzheimer's disease confirmed upon autopsy (J. C. Morris, personal communication, April 14, 1993).

Of the 167 participants recruited from the Washington University ADRC, 34 were healthy older controls (CDR = 0) under age 80 (mean age = 70.3); 36 were healthy older controls (CDR = 0) over age 80 (mean age = 84.6); 31 were diagnosed with very mild dementia (CDR = 0.5; mean age = 72.9); and 41 were diagnosed with mild or moderate dementia (CDR = 1 or 2; mean age = 73.9). The 25 individuals with PD were not cognitively impaired (CDR = 0; mean age = 68.8); however, they all were taking medication (e.g., Sinemet) to control their Parkinson symptoms.

According to the Hoehn–Yahr scale (Hoehn & Yahr, 1967), 75% of the PD individuals were in Stage 1 or 2.

In addition, 24 college-aged participants (mean age = 20.3) were recruited for this study. These participants were paid $5 per hour for their participation.

Psychometric Testing

Each participant from the ADRC was administered a 2-hr comprehensive psychometric battery that assesses various aspects of memory, psychomotor performance, and language. Memory and language performance was assessed with the Boston Naming Test and the Wechsler Memory Scale (Paired Associate Learning, Logical Memory, forward and backward Digit Span, Mental Control; Wechsler & Stone, 1973), the Benton Visual Retention Test (picture memory; Benton, 1963), and the Word Fluency Test (Thurstone & Thurstone, 1949). Intelligence was assessed with the following subtests of the Wechsler Adult Intelligence Scale (WAIS): Information, Comprehension, Block Design, and Digit Symbol (Wechsler, 1955). Perceptual motor performance was assessed with the BVRT copy test and Part A of the Trail Making Test (Armitage, 1946).

Psychometric performance as a function of subject group is displayed in Table 1. Following the overall analysis reflected by the main effect of group, separate t tests were used to compare each subject group's performance with the healthy controls under 80. The data in Table 1 reflect only those subjects (n = 131) whose data did not violate the assumptions of Wing and Kristofferson's (1973) model (see the Results section for further discussion). In general, performance across all tests declined as the severity of dementia increased. Also, the healthy older controls over 80 showed somewhat poorer performance on a few of the tests, and the PD group showed similar psychometric performance to the age-matched healthy controls under 80.

Apparatus

Testing was conducted with an Apple IIe microcomputer interfaced with a Mountain Hardware Clock card to measure response latency to the nearest millisecond.

Procedure

All subjects were seated in front of a computer with the index finger of their dominant hand resting on a response key mounted on a key pad. Each trial consisted of a series of 50-ms tones generated from the computer, which were presented every 550 ms. Subjects were instructed to tap in rhythm with the tones and to continue tapping after the tones had stopped. After 13 tones were presented, the tones stopped and subjects continued tapping on their own for an additional 31 key presses. At this time the instruction "STOP TAPPING" was displayed on the computer screen, and the subjects ceased tapping. Feedback was then displayed regarding the mean IRI and the standard deviation of the IRI.

To ensure that subjects understood the instructions, one or two practice trials were presented. The practice trials were followed by six "acceptable trials." In this task, unacceptable trials were defined as those in which the standard deviation of the IRI exceeded 100 ms. Thus, some subjects engaged in more than six trials to reach the criterion of six acceptable trials. The mean percentage of unacceptable trials out of the total number of trials given for each subject group is as follows: young college students (0.8%), healthy controls under age 80 (4.2%), healthy controls over age 80 (4.3%), subjects with very mild SDAT (4.8%), subjects with mild SDAT (8.6%), and subjects with PD (4.3%). Although the mild SDAT group had a greater percentage of unacceptable trials than the other subject groups, this difference did not reach significance, $F(5, 143) = 1.37$, $MS_e = 90.96$, $p = .24$. The unacceptable trials were not included in any of the data analyses.

Results

A major assumption of Wing and Kristofferson's (1973) model is that the variation in motor delay time will produce a negative correlation among adjacent response intervals. In applying the model to the present data, this assumption was violated in some cases. That is, the motor delay estimates for some subjects yielded a positive correlation across the six trials. The data from these subjects were discarded from all of the following analyses. This resulted in the following numbers of subjects and mean ages per group: college-age subjects (n = 18; mean age = 20.6); healthy controls under 80 (n = 30; mean age = 69.5); healthy controls over 80 (n = 31; mean age = 84.6); subjects with very mild SDAT (n = 28; mean age = 72.8); subjects with mild to moderate SDAT (n = 22; mean age = 73.5); and subjects with PD (n = 20; mean age = 68.6). The percentage of subjects in each group showing positive covariance estimates was as follows: 25% of the college-age subjects; 12% of the healthy controls under 80; 14% of the healthy controls over 80; 10% of the subjects with very mild SDAT; 46% of the subjects with mild to moderate SDAT; and 20% of the subjects with PD. Similar percentages of violations have been reported elsewhere (see Ivry & Keele, 1989). The loss of subjects was greatest in the group with mild to moderate SDAT, and the second highest loss of subjects was in the college-age group. The lowest loss was in the very mildly demented group. Hence, it does not appear that the loss of subjects was due simply to the fact that positive covariance estimates across trials were systematically related to dementia severity. (We return to this issue later.)

One-way analyses of variance across the six groups of subjects were conducted for (a) the mean IRI across the six trials, (b) the mean standard deviation of the IRI, (c) the estimate of clock delay variability, and (d) the estimate of motor delay variability. The means and standard deviations for each of these variables are presented in Table 2 as a function of subject group.

There was no effect of group for either the mean IRI, $F(5, 143) = 1.45$, $MS_e = 343.14$, $p > .21$, or the mean standard deviation of the IRI, $F(5, 143) = 1.37$, $MS_e = 68.24$, $p > .24$. However, there was a marginally significant main effect of group for the clock delay variability, $F(5, 143) = 2.15$, $MS_e = 65.89$, $p < .07$, and a significant main effect of group for the motor delay variability, $F(5, 143) = 2.41$, $MS_e = 38.00$, $p < .04$. Because the major focus of the present work was on these latter two estimates of variability, we present specific group comparisons for clock delay and motor delay variability separately.

Table 1
Psychometric Performance as a Function of Subject Group

Test	Healthy controls		SDAT				
	Under 80	Over 80	Very mild	Mild	PD	*F*	*df*
WMS Logical Memory							
M	10.17	7.94*	5.21*	1.71*	8.35	34.22***	4, 120
SD	2.97	2.24	3.10	1.31	3.08		
TMT Part A							
M	39.69	54.37	53.79	59.00	57.50	1.83	4, 118
SD	16.30	23.61	28.58	49.56	57.50		
WAIS Information							
M	21.72	20.19	16.43*	9.94*	21.10	21.15***	4, 119
SD	3.83	4.60	5.80	5.35	3.02		
WAIS Block Design							
M	34.28	28.97	24.67*	13.50*	30.85	13.63***	4, 118
SD	8.93	7.75	11.39	11.11	8.73		
WAIS Digit Symbol							
M	50.90	40.30*	36.57*	19.63*	41.70*	14.67***	4, 118
SD	12.78	13.46	13.02	17.01	10.88		
BVRT (no. correct)							
Delay							
M	6.66	5.63	4.50*	2.06*	5.90	17.13***	4, 118
SD	1.74	1.94	2.03	2.08	1.74		
Copy							
M	9.83	9.30	9.18	6.31*	9.65	11.64***	4, 118
SD	0.54	1.18	1.33	4.24	0.59		
Boston Naming Test							
M	57.07	51.40*	48.93*	37.13*	55.35	16.33***	4, 118
SD	2.95	8.44	10.30	14.19	3.34		
WMS Mental Control							
M	7.79	7.26	6.21*	4.53*	7.70	7.04***	4, 120
SD	1.59	1.98	2.70	3.45	1.72		
WMS Paired Associate Learning							
M	14.34	13.26	10.39*	5.47*	13.60	18.94***	4, 120
SD	4.02	3.54	4.15	3.20	3.35		
BVRT (no. of errors)							
Recall							
M	5.34	7.83*	11.14*	17.88*	6.95	23.09***	4, 118
SD	2.96	3.98	5.64	6.47	3.50		
Copy							
M	0.21	0.80	0.86	8.44*	0.40	12.00***	4, 118
SD	0.68	1.69	1.41	11.47	0.68		
Word Fluency Test							
M	34.93	29.68	26.29*	14.00*	27.10	9.28***	4, 119
SD	12.93	10.94	9.85	9.80	12.03		
WMS Digit Span							
M	12.52	11.58	10.71*	9.06*	11.55	6.24**	4, 120
SD	2.43	2.16	2.00	2.93	2.63		

Note. SDAT = senile dementia of the Alzheimer type; PD = Parkinson's disease; WMS = Wechsler Memory Scale; TMT = Trail Making Test; WAIS = Wechsler Adult Intelligence Scale; BVRT = Benton Visual Retention Test.
* $p < .01$ (for group comparison with healthy controls under 80). ** $p < .001$. *** $p < .0001$.

Clock Delay Estimates

As shown in the fourth column of Table 2, the group with mild to moderate SDAT produced the greatest variability in clock delay. The clock delay variability for the very mildly demented SDAT individuals was very similar to the healthy controls over 80 and the healthy controls under 80. Post hoc *t* tests confirmed that the mildly to moderately demented SDAT group produced significantly greater variability in clock delay than the healthy controls under 80, $t(50) = 3.06$, $p < .01$; the healthy controls over 80, $t(51) = 2.51$, $p < .02$; and the very mildly demented group, $t(48) = 2.24$, $p < .03$. The PD subjects' clock time was slightly greater than the healthy controls under 80 and slightly lower than the mildly to moderately demented SDAT group, although these differences were not significant, $t(48) = 1.32$, $p > .19$, and $t(40) = 1.98$, $p > .05$, respectively. Finally, to examine the effect of normal aging on the variability in clock delay, we

Table 2
Mean Interresponse Intervals (IRIs), Clock Delay Estimates, and Motor Delay Estimates as a Function of Subject Group

Group	*n*	IRI (in ms)		Clock delay	Motor delay
		M	*SD*		
College-age	18				
M		543.00	29.00	24.10	11.30
SD		13.84	8.85	8.26	5.63
Healthy controls <80	30				
M		538.00	30.00	21.10	14.00
SD		11.97	8.91	9.29	6.53
Healthy controls >80	31				
M		549.00	32.80	23.40	15.30
SD		19.94	7.03	7.03	6.39
Very mild SDAT	28				
M		545.00	29.20	22.90	11.50
SD		23.89	9.00	9.54	5.71
Mild to moderate SDAT	22				
M		549.00	33.40	28.30	11.70
SD		22.86	11.16	8.34	8.42
PD	20				
M		541.00	29.20	24.20	10.40
SD		22.55	7.89	7.92	4.91

compared the college-age group with the healthy controls under 80 and the healthy controls over 80. There was no significant difference across any of these groups in clock delay (all *p*s > .27).

Motor Delay Estimates

As shown in the fifth column of Table 2, the motor delay estimates were very similar for the college-age controls, the group with very mild SDAT, the group with mild to moderate SDAT, and the group with PD. In contrast, the two older healthy control groups produced the greatest variability in motor delay.

We first conducted post hoc comparisons between the healthy controls under 80 and the other subject groups. There were no significant differences in the estimates of motor delay between the healthy controls under 80 and (a) the college-age controls, $t(46) = 1.45$, $p > .15$; (b) the healthy controls over 80, $t(59) = 0.80$, $p > .43$; (c) the very mildly demented individuals, $t(56) = 1.51$, $p > .14$; and (d) the mildly to moderately demented individuals, $t(50) = 1.20$, $p > .24$. We then compared the healthy controls over 80 with the other subject groups. There was a significant difference in the estimates of motor delay between the healthy controls over 80 and (a) the college-age controls, $t(49) = 2.21$, $p < .05$, and (b) the very mildly demented individuals, $t(57) = 2.37$, $p < .05$. However, there was no significant difference in motor delay estimates between the healthy controls over 80 and the mildly to moderately demented individuals, $t(51) = 1.93$ $p > .05$.

The PD subjects were included in this study to represent a group with a disease that could affect output aspects of the motor system. It is interesting to note that the PD group produced reliably less variability in motor delay than the age-matched healthy controls under 80, $t(48) = 2.07$, $p < .05$. Clearly, there is no evidence of a breakdown in variability in motor delay in medicated PD individuals compared with age-matched controls.

Psychometric Performance and Clock and Motor Delay Estimates

In an attempt to examine how the central timing and motor implementation components are related to other aspects of cognitive processing, we calculated correlations among psychometric test performance and clock and motor delay estimates. These data are displayed in Tables 3 and 4 as a function of subject group. In general, these data indicate that only a few correlations were reliable, and these were only for the healthy controls under 80 and only for the clock delay estimate. Thus, the central timekeeping and motor implementation components do not appear to be related to other aspects of processing as measured by the psychometric tests.

Violations of the Model

As previously noted, the assumption of a negative correlation among adjacent response intervals was violated in some cases. Specifically, 46% of the mildly to moderately demented SDAT group showed positive covariance estimates and were discarded from the analyses. Given that

Table 3
Correlations Between Psychometric Test Performance and Clock Delay Estimate as a Function of Subject Group

Test	Healthy Controls		SDAT		PD
	Under 80	Over 80	Very mild	Mild to moderate	
WMS Logical Memory	−.21	.12	−.40	.10	.10
TMT Part A	.45	.12	.16	−.44	.04
WAIS Information	−.23	−.12	−.25	−.15	−.11
WAIS Block Design	−.40	−.19	−.37	−.14	.21
WAIS Digit Symbol	−.26	.07	−.29	−.001	−.09
BVRT delay[a]	−.61**	−.10	−.32	.02	.27
BVRT copy[a]	−.48*	.07	−.33	.08	.06
Boston Naming Test	−.49*	−.01	−.07	−.02	.09
WMS Mental Control	−.30	.05	−.40	.15	−.39
WMS Paired Associate Learning	−.16	.18	−.30	.23	.23
BVRT recall[b]	.54*	.02	.44	.07	−.14
BVRT copy[b]	.44	−.14	.34	−.05	.04
Word Fluency Test	−.19	.00	−.31	.01	−.21
WMS Digit Span	−.36	.12	−.22	.16	−.11

Note. SDAT = senile dementia of the Alzheimer type; PD = Parkinson's disease; WMS = Wechsler Memory Scale; TMT = Trail Making Test; WAIS = Wechsler Adult Intelligence Scale; BVRT = Benton Visual Retention Test.
[a] No. correct. [b] No. of errors.
* $p < .01$. ** $p < .001$.

Table 4
Correlations Between Psychometric Test Performance and Motor Delay Estimate as a Function of Subject Group

Test	Healthy controls		SDAT		
	Under 80	Over 80	Very mild	Mild to moderate	PD
WMS Logical Memory	−.24	−.30	.07	−.22	.00
TMT Part A	.11	−.13	−.16	−.18	−.02
WAIS Information	−.13	.15	−.19	−.36	−.07
WAIS Block Design	−.07	.07	.17	−.32	−.00
WAIS Digit Symbol	−.05	.19	−.02	−.04	.12
BVRT delay[a]	.06	−.00	−.02	−.29	−.47
BVRT copy[a]	−.14	−.04	−.02	.05	.23
Boston Naming Test	.06	−.12	−.35	−.22	−.01
WMS Mental Control	−.14	.09	−.29	.14	.19
WMS Paired Associate Learning	−.17	−.15	−.26	.31	−.09
BVRT recall[b]	−.02	−.04	−.13	.37	.38
BVRT copy[b]	.12	.05	−.01	−.18	−.24
Word Fluency Test	.06	.11	−.41	−.02	−.02
WMS Digit Span	−.11	.24	−.44	.14	−.01

Note. SDAT = senile dementia of the Alzheimer type; PD = Parkinson's disease; WMS = Wechsler Memory Scale; TMT = Trail Making Test; WAIS = Wechsler Adult Intelligence Scale; BVRT = Benton Visual Retention Test.
[a] No. correct. [b] No. of errors.

only this group showed a deficit in clock delay, this raises some concern. Therefore, a comparison was made between "valid" mild to moderate SDAT subjects (i.e., subjects with negative covariance estimates, $n = 22$) and "invalid" mild to moderate SDAT subjects (i.e., subjects with positive covariance estimates, $n = 19$) in terms of age, psychometric performance, and IRI. The valid and invalid subjects did not differ in terms of age (mean age = 73.5 and 74.4, respectively), $t(39) = 0.43$. There was a significant difference between them on two of the psychometric tests. The invalid subjects performed worse than the valid subjects on Part A of the Trail Making Test (116.33 vs. 59.00, respectively), $F(1, 26) = 8.81$. $p = .006$, and the Boston Naming Test (37.13 vs. 25.06, respectively), $F(1, 31) = 5.47$, $p = .03$. Also, there was a marginally significant difference between valid and invalid subjects on WMS Mental Control, $F(1, 30) = 3.97$, $p = .06$.

At first glance, there appears to be very little difference in psychometric performance between the valid and invalid subjects. However, all three of these psychometric tests have been shown to produce a considerable breakdown in mild SDAT (LaBarge, Balota, Storandt, & Smith, 1992; Storandt, Botwinick, Danziger, Berg, & Hughes, 1984). Thus, it is possible that the invalid subjects were somewhat more cognitively impaired than the valid subjects. Furthermore, the mean IRI of the invalid subjects was significantly faster (531 ms) than that of the valid subjects (549 ms), $t(39) = 2.03$, $p = .05$, indicating that the invalid subjects were having more difficulty with the timing of the task. However, this does not minimize the finding of impaired clock delay in the valid SDAT subjects. Instead, the results indicate that the less affected SDAT subjects who validly could perform the task showed a clock delay deficit relative to the very mild SDAT group, healthy controls, and nondemented PD individuals. In fact, if anything, this finding emphasizes the importance of testing subject groups at various points in the disease progression.

Discussion

The purpose of this study was to identify two different components of timing control that may be impaired in SDAT: a central timekeeping mechanism and a response execution mechanism. In addition, a group of nondemented individuals with PD disease and two groups of healthy older adults were included to examine the influence of a motor-related disease and normal aging on timing control. We first discuss the performance of the SDAT individuals.

Timing Control and SDAT

The results from the finger tapping task with SDAT individuals are straightforward. The mildly to moderately demented SDAT individuals produced a deficit in the central timekeeping mechanism but did not produce any deficit in the response execution mechanism relative to the other subject groups. The very mildly demented SDAT individuals did not show any deficit in the timekeeping mechanism. In fact, the clock delay estimates for the very mildly demented SDAT individuals are very similar to those of the healthy older controls under 80. Thus, this study indicates that the central cognitive component of timing control is impaired only in the mild stage of SDAT and that the implementation of the timing response remains relatively unimpaired, even in mildly demented individuals.

Given the global nature of the deficits associated with Alzheimer's disease, it is not surprising that there is also a breakdown in the central timing mechanism of motor control. It is important to emphasize that Ivry and Keele (1989) argued that the timekeeping mechanism of the cerebellum is indeed central. Therefore it is not limited only to the motor system but also controls timing functions of the perceptual and cognitive system. This central mechanism is called into play by the motor or perceptual and cognitive systems whenever a time-related function is needed. In this light, one may speculate about the impact that a deficient timekeeping mechanism might have on cognitive performance in SDAT. It is clear that many actions we perform are the result of the coordination of multiple cognitive elements. For example, let us assume that the execution of a particular response requires timed coordination between two successive cognitive elements (Element A and Element B). Furthermore, suppose that Element A takes 100 ms to complete before Element B is called into play. If there is a disruption in a central timekeeping mechanism, it is quite possible that on some occasions Element B would start before Element A was completed, and thus some competition between elements might disrupt aspects of performance.

In Ivry and Keele's (1989) investigation of the impact of different neurological deficits on timing control, they found

that only a group of patients with cerebellar damage showed a deficit in the central timekeeping parameter. Unfortunately, there was no indication of the cognitive status of the cerebellar group and there was no group of demented subjects in their study. Therefore, it is difficult to make any comparisons with Ivry and Keele's study in reference to the issue of dementia. However, on the basis of their data from the finger tapping task and from a perceptual task that involved the discrimination of different timed intervals, Ivry and Keele argued that this central timing mechanism is a function of the integrity of the cerebellum. On the other hand, the impaired central timing mechanism in Parkinson's disease has been related to basal ganglia dysfunction. Thus, Ivry and Keele further argued that an impairment in clock delay may also be apparent when there is basal ganglia dysfunction, as in PD, because the basal ganglia is part of the neural circuit involved in timing. Although they argued that the cerebellum plays the major role in timing functions, any change in a structure (e.g., basal ganglia) that provides some type of input into this circuit could contribute to an impairment in central timing. However, there is a recent report of impaired central timing and motor implementation in nonmedicated PD (Pastor, Jahanshahi, Artieda, & Obeso, 1992). Thus, the neural subsystems involved in timing are not entirely clear.

Interestingly, there are both clinical data suggesting that cerebellar function may be impaired in SDAT (Huff et al., 1987) and pathological indications of amyloid plaques in the cerebellum (Braak et al., 1989; Mackenzie et al., 1991). However, given the diffuse cortical damage associated with SDAT, it is difficult to speculate about the role of the cerebellum in central timing deficits in SDAT. In this light, it is interesting to further note that the variability in clock delay for the present SDAT group (28.3) was much less than that for the cerebellar group (38.1) and more similar to that of the cortical group (30.1) in Ivry and Keele's (1989) study.

There are several studies in the literature reporting intact motor learning in SDAT (e.g., Grafman et al., 1990; Heindel et al., 1989; Knopman & Nissen, 1987; however, see, Ferraro, Balota, & Connor, 1993). These tasks typically involve SDAT subjects learning some type of simple motor response, such as a serial reaction time task or pursuit-rotor task. The results of such studies indicate that SDAT individuals show similar improvement in performance compared with controls (e.g., a decrease in reaction time) across trials. At first glance, these studies may seem inconsistent with the present results of an impaired central timing mechanism in SDAT. However, there are several reasons why these results may not be inconsistent. For example, Ferraro et al. (1993) found evidence that individuals with mild SDAT do exhibit a deficit in implicit memory performance in a serial reaction time task. Thus, a deficit in clock delay in mild SDAT is not entirely inconsistent with the motor learning literature. Also, it is possible that these motor tasks do not rely heavily on the repetitive timing of motor control, as estimated in the present study. Possibly, it is the repetitive nature of the response that is crucial to obtain an influence of the central timekeeping mechanism on performance. Moreover, even though SDAT individuals do show evidence of skill learning across trials, their overall performance is typically worse than controls. Therefore, even if there is some generalized skill learning of certain motor tasks in SDAT, there may still be an impairment in the central timing of motor control. Again, this emphasizes the importance of examining such cognitive processes across stages of disease progression. It is possible that individuals with very mild SDAT show intact motor learning skills yet individuals with mild SDAT do not (see Ferraro et al., 1993).

Timing Control and Parkinson's Disease

The issue of impairment in timing control in PD is somewhat equivocal from previous studies. First, it is apparent that PD individuals are deficient in time estimation and temporal discrimination when unmedicated yet become more accurate when medicated (Artieda, Pastor, Lacruz, & Obeso, 1992; Pastor, Artieda, Jahanshahi, & Obeso, 1992). In addition, there are case reports of impaired clock time in the finger tapping task with PD patients when comparing the affected and the unaffected hand (Wing, Keele, & Margolin, 1984), and before medication versus after medication (Keele & Ivry, 1987), yet no impairment in the variability in motor delay. Furthermore, in a repetitive wrist movement task, Pastor, Jahanshahi, et al. (1992) reported that both clock delay and motor delay were impaired in a group of nonmedicated, nondemented PD individuals.

The results from the finger tapping task with the PD subjects in the present study are clear. First, in terms of the clock delay variability, the PD individuals showed a slight deficit (although nonsignificant) in the timekeeping function compared with the age-matched healthy older controls (under 80). Ivry and Keele (1989) reported similar findings in their study comparing different neurological groups. That is, their PD group also showed slightly more variability in clock delay (27.7) than the healthy older controls (24.3), yet this difference also was not statistically significant. Thus, both Ivry and Keele's study and the present study indicate that there may be a slight breakdown in the central timekeeping component of motor control in Parkinson's disease. Moreover, in the present study and in Ivry and Keele's study, the PD subjects were medicated at the time of testing. Thus, the slight breakdown in clock delay in a nondemented group of PD individuals may be due to the effect of medication and thus may underestimate the impaired central timekeeping mechanism in PD.

The issue of variability in motor delay in PD in this study is also clear. The present results indicate that there was significantly less variability in motor delay in the PD group compared with the healthy older controls under 80. Again, this difference is most likely due to the control of PD symptoms by appropriate medication (e.g., Sinemet). In addition, Ivry and Keele (1989) reported slightly less variability in motor delay in their medicated PD group (9.3) compared with their elderly controls (11.0), although this difference did not reach significance. On the other hand,

Pastor, Jahanshahi, et al. (1992) found impaired motor estimates in nonmedicated PD individuals in a repetitive wrist movement task. Thus, it is possible that the motor implementation system is affected in nonmedicated, nondemented PD individuals. Clearly, the present study and Ivry and Keele's study indicate that nondemented, medicated PD subjects do not produce any breakdown in the implementation of a timed response, at least as measured with the finger tapping paradigm.

Application of Wing and Kristofferson's (1973) Model

In the present study, there were violations of the negative covariance assumption of the model in each subject group. The largest percentage of subjects was discarded from the critical mildly to moderately demented SDAT group. Furthermore, there appeared to be differences on some of the more dementia-sensitive psychometric tests between the valid and invalid subjects in this SDAT group, along with evidence that the invalid subjects' mean IRI was significantly faster than that of the valid subjects. This pattern may indicate that the more affected SDAT individuals had more difficulty maintaining the timing of the task, perhaps because of a loss of memory for the rhythm. In any case, one may question the validity of Wing and Kristofferson's model, especially when applied to patient groups. Violations of this assumption have been reported elsewhere and tend to be highest for various patient groups (e.g., Ivry & Keele, 1989; Pastor, Jahanshahi, et al., 1992). Ivry and Keele reported positive covariance estimates for 26% of their cerebellar group and Pastor, Jahanshahi, et al. reported 30%–60% violations in PD patients. Thus, the application of the model and hence the validity of the clock and motor delay estimates with various patient groups should be interpreted with caution (also see Pastor, Jahanshahi, et al., 1992).

Conclusions

The purpose of this study was to examine the influence of SDAT, PD, and normal aging on components of a relatively simple rhythmic finger tapping task. The results clearly indicate that there is a breakdown in the central timekeeping mechanism (i.e., clock delay) in mild SDAT, yet there is no impairment in the execution of the timed response (i.e., motor delay). One may be inclined to conclude that there will be a breakdown in timekeeping whenever a cognitive deficit is present. However, judging by the results of the present study, this does not appear to be so. Individuals with very mild SDAT did not show any breakdown in either timekeeping or response implementation, even though they produced consistent breakdowns in psychometric performance. Moreover, the healthy controls over 80 did not show an impairment in the central timekeeping mechanism relative to the mild SDAT group. Thus, the performance of healthy adults over 80 was clearly different from that of a younger mildly demented group in the finger tapping paradigm.

Although there are cognitive deficits associated with normal aging, the results of this study indicate that there was no impairment in clock delay or motor delay as a function of normal aging (i.e., when healthy older controls were compared with college-age subjects; there was some impairment in motor delay for the healthy controls over 80, however). A similar finding of no age differences has been reported by Greene and Williams (1993). On the other hand, Ivry and Keele (1989) reported an impairment in clock delay for their older controls relative to their younger controls. Unfortunately, there was no indication of the cognitive status of the older group in their study. The finding of no age differences in the present study is interesting in light of recent arguments for a generalized slowing in normal aging (Cerella, 1990; Myerson et al., 1992; Salthouse, 1985a, 1985b). The argument has been made that the deficits seen in normal aging across different tasks are due to a general slowing of the cognitive operations necessary to perform those tasks. Thus, it appears that the variability in the central timing of a simple response is not affected by an age-related general slowing factor.

Finally, the breakdown in the central mechanism only occurred at the mild to moderate stages of the disease in the absence of any response execution deficit. Of course, it is also possible that there will eventually be a breakdown in the implementation system in more severe stages of SDAT. In this light, this study underscores the importance of examining issues of motor control in SDAT as a function of (a) separate processing components and (b) various stages of disease progression.

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Received September 29, 1992
Revision received April 21, 1993
Accepted June 29, 1993 ■