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Procedural Memory in Parkinson's Disease: Impaired Motor But Not Visuoperceptual Learning *

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ABSTRACT

A current model proposes that memory consists of two functionally separate systems that have different neurological substrates. Declarative memory appears to be dependent on the diencephalic medial temporal lobe system whereas some speculate that the basal ganglia may be a neurological substrate for procedural memory. This study tested the role of the basal ganglia in regulating different types of procedural skills by comparing performance on a motor and a visuoperceptual skill learning task. Twenty Parkinson's (PD) patients and 20 normal control subjects performed two procedural learning tasks (rotary pursuit and mirror reading) and one declarative learning task (paired associates) over 3 days. The results showed that PD patients were not impaired on mirror reading or paired associate learning. On rotary pursuit, performance levels on day 1 were similar between groups, but the PD group showed less improvement across days than controls. However, only patients with more advanced symptoms of PD showed impaired rotary pursuit learning, and this could not be attributed directly to deficits in primary motor or general cognitive function. These findings suggest that the underlying processes/procedures for procedural learning are specific to the task, and are supported by different neuroanatomical systems.

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It has been suggested that memory is composed of two functionally separate knowledge systems (Cohen & Squire, 1980). Declarative memory appears to be dependent on the diencephalic medial temporal lobe system (Squire & Cohen, 1984), and some have suggested that the basal ganglia may regulate procedural functions (Heindel, Butters, & Salmon, 1988; Martone, Butters, Payne, Becker, & Sax, 1984; Mishkin & Petri, 1984). This study tests the basal ganglia hypothesis of procedural memory by examining whether only certain types of procedural learning are controlled by the neostriatum.

The procedural-declarative model emerged from observations that amnesics show normal performance on procedural tasks (e.g., priming, mirror reading, rotary pursuit, the Tower of Hanoi puzzle), but they are impaired on tests of declarative memory (e.g., recall, recognition) (Brooks & Baddeley, 1976; Cohen, 1984; Cohen & Squire, 1980; Gordon, 1988; Graf, Squire, & Mandler, 1984; Moskovitch, 1984; Squire & Cohen, 1984). Similarly, studies with normal subjects show that certain experimental conditions differentially affect performance on tests of procedural and declarative memory (Graf & Mandler, 1984; Graf & Schacter, 1987; Jacoby & Dallas, 1981; Schacter & Graf, 1986a).

The neurological substrate(s) for procedural functions is(are) not well defined. Primate studies suggest the neostriatum may mediate "habit" formation, putatively the output of procedural memory (Mishkin & Petri, 1984). Some studies of patients with Huntington's Disease (HD), which primarily involves damage to the caudate nucleus, have been interpreted as revealing a double dissociation between procedural and declarative memory as HD patients show performance deficits on procedural but not declarative tasks and amnesic patients show the opposite pattern of effects (Heindel et al., 1988; Martone et al., 1984).

Although these results provide support for the basal ganglia's role in regulating procedural memory, several studies have found deficits in both types of learning with HD (Butters, Wolf, Martone, Granholm, & Cermak, 1985; Heindel et al., 1988; Martone et al., 1984; Saint-Cyr, Taylor, & Lang, 1988). The problem in interpreting these results is that HD is frequently associated with structural and metabolic changes in frontal and frontotemporal cortex (Stober, Wussow, & Schimrigk, 1984; Tanahasi et al., 1985). While abnormalities in metabolism of the frontal cerebral cortex have been reported in Parkinson's disease (PD) (Perlmutter & Raichle, 1985) and pathology may extend beyond the basal ganglia in demented PD patients (Marsden, 1984; Taylor, Saint-Cyr, & Lang, 1986), structural abnormalities are uncommon especially when patients are not demented. Therefore, PD appears to more specifically affect the basal ganglia, allowing for a more direct assessment of the basal ganglia's role in procedural memory.

The present experiment was designed to examine the basal ganglia hypothesis of procedural memory in PD. Support for separate memory systems in PD is limited as most studies have not directly compared performance on skill learning with performance on tests of declarative memory. One exception is a

study reporting impaired procedural learning in PD patients on a Tower puzzle task but normal performance on recall and recognition tests of declarative memory (Saint-Cyr et al., 1988). In contrast, another study showed normal performance on tests of declarative (i.e., recall and recognition) and procedural memory (i.e., lexical priming and rotary pursuit learning) in a nondemented PD group (Heindel, Salmon, Shults, Walicke, & Butters, 1989). Thus, the role of the basal ganglia in regulating various kinds of skill learning is unclear.

In the present study, we compared the performance of normal controls and PD patients with minimal cognitive deficits on one test of declarative memory (paired associate learning) and two tests of procedural memory, one visuoperceptual (mirror reading) and the other motor (pursuit rotor learning). The comparison between visuoperceptual and motor tasks is a crucial issue for our understanding of procedural memory as the acquisition of skills in patients with basal ganglia involvement may depend on processes that are specific to motor-based tasks. Few studies have contrasted performance on different types of procedural memory tests. A notable exception is the Heindel study (1989) reporting impaired rotary pursuit learning but normal lexical priming in HD patients and the opposite pattern of effects in Alzheimer's disease patients.

Dissociations among procedural memory tasks contrasts with the procedural-declarative view and suggests the possibility that some processes or procedures are specific to the task. This is consistent with a proceduralist account of memory (Kolers & Roediger, 1984) where knowledge is defined by the processes or procedures applied to stimuli. This view may better account for the observations that under some circumstances amnesics can retrieve newly learned facts and form new associations, which presumably require the use of a declarative or semantic memory system (Glisky, Schacter, & Tulving, 1986; Graf et al., 1984; Hirst, Johnson, Phelps, & Volpe, 1988; Schacter, Harbluk, & McLachlan, 1984), but are not able to learn a procedural task such as the Tower of Hanoi puzzle (Butters et al., 1985).

If the procedural-declarative model is correct, we expect PD patients to show impaired learning on mirror reading and rotary pursuit but normal performance on paired associate learning. Alternatively, if visuoperceptual and motor procedural skills are associated with different processes or procedures, PD patients should be impaired on rotary pursuit learning but show normal mirror reading performance.

METHODS

Subjects

Twenty patients with Parkinson's Disease (PD) and 20 neurologically intact control subjects were studied. Table 1 provides descriptive information on these subjects. PD patients and control subjects were right-handed and matched for age and educational level. The PD group consisted of 80% males and 20% females and in the control group there were 45% males and 55% females. Sex was not, however, related to performance on

Table 1.
Characteristics of control and Parkinson's subjects.

	Control Group			Parkinson's Group		
	M	*SD*	Range	*M*	*SD*	Range
Age	66.7	9	51-82	65.8	6	54-77
Education	12.4	2	8-15	12.2	2	4-15
Duration of Disease (years)				6.2	7	<1-26
Age of Disease Onset				59.0	9	44-76
NYU Disability Scale[1]				18.1	15	1-47

[1] Scores on the New York University Disability Scale are sums across all items with a score of zero reflecting no disability and a score of 100 representing complete disability. Scores of 40 or greater suggest considerable disability.

any of the experimental tests.

All PD patients were outpatients and all but one was on Parkinsonian medications at the time of testing. Nineteen PD patients were being treated with dopaminergic drugs and seven of these patients were also taking anticholinergic drugs. All patients had a clinical diagnosis of PD. Subjects were excluded from the study if they evidenced other neurological diagnoses such as stroke, epilepsy, history of alcoholism, or head injury.

A board-certified neurologist (E.M.) assessed Parkinsonian status using two different instruments. Stage of the disease was assessed using the Hoehn and Yahr (1967) severity measure. Thirty-five percent of the PD patients were classified in Stage 1 (mild, unilateral involvement), 20% in Stage 2 (bilateral involvement), and 45% in Stage 3 (mild to moderate gait disturbance). No patients were classified in Stage 4 or 5 (marked gait disturbance, or confined to bed or wheelchair). On the New York University (NYU) Disability Scale (Lieberman, 1974) most patients showed no rigidity (70%), tremor (55%), dyskinesia (95%), postural abnormality (68%), or gait problems (75%). As for bradykinesia, 60% of the patients showed impaired motor speed and 85% showed impaired amplitude of movement. Only three patients received total scores on this scale that reflected considerable disability (40 or greater).

Table 2.
Ancillary testing for control and Parkinson's subjects.

	Control Group			Parkinson's Group		
	M	*SD*	Range	*M*	*SD*	Range
Mini Mental State	29.3	1	27-30	27.8*	2	25-30
Wechsler Memory Quotient	121.7	13	99-143	107.1*	14	83-137
Information (WAIS-R)[1]	12.5	2	9-16	11.5	2	6-16
Block Design (WAIS-R)[1]	8.6	2	4-13	6.7*	2	4-11
Line Orientation[2]	27.6	4	19-33	25.1	4	18-32
Beck Depression Inventory[3]	5.5	4	0-14	11.1*	8	1-29

* $p<.01$ for *T* tests comparing controls and PD subjects
[1] Scale scores ($M = 10 + 3$)
[2] Scores corrected for age (normal scores are greater than 20)
[3] Range for normal scores is 0 to 9

All subjects were given a battery of neuropsychological tests to describe their cognitive functioning more broadly. Table 2 shows that there were no significant differences between the groups on the Information subtest from the Wechsler Adult Intelligence Scale-Revised (WAIS-R) (Wechsler, 1981) or Judgement of Line Orientation (Benton, Hamsher, Varney, & Spreen, 1983). Although the PD group performed more poorly on the Mini Mental State Exam (Folstein, Folstein, & McHugh, 1975) ($F(1,38)=9.35$, $p<.01$), all scores were within the normal range. The PD group also performed more poorly on the Block Design subtest of the WAIS-R ($F(1,38)=8.39$, $p<.01$) and on the Wechsler Memory Scale (WMS)(Wechsler, 1945) than control subjects ($F(1,38)=11.78$, $p<.01$) which was indicative of mild cognitive decline; in all cases except Block Design, however, performance remained within normal limits. On the Beck Depression Inventory (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961), the PD group was more depressed than controls ($F(1,38)=8.07$, $p<.01$), but scores on this scale were not related to performance levels or learning on any of the experimental tasks.

Procedures

The tasks used to assess procedural learning were mirror reading and rotary pursuit. Declarative learning was examined with a verbal paired-associates learning task. Performance was always measured over 3 days. For *mirror reading*, similar words and procedures were used as reported previously (Cohen & Squire, 1980). On each day five blocks of 10 unique word triads were presented backwards using a slide projector. Word triads were never repeated across blocks or days. The time to pronounce the triads phonetically and the number of errors (i.e, phonetic errors or the absence of a response within 120 s) were recorded. If subjects produced a syllable that was phonetically correct in isolation, but not in that particular word, they received a cue as to the way it should sound in that word. The examiner also repeated the part of the word that was said correctly when subjects evidenced difficulty. The *rotary pursuit* task was administered over three blocks each day. Each block consisted of six trials of 30-s duration. All subjects received two trials each at 30, 45, and 60 revolutions per min (rpm). Subjects held the stylus with their right hand, and were instructed to hit a target as the disc revolved. For each block, the time on target was averaged across the two trials at each rpm. Control and PD subjects were not matched on initial performance levels so as to allow for an examination of the possible differential effects of task difficulty on skill learning. As we will report, however, there were no differences between control and PD subjects in their initial level of performance regardless of rpm condition. For the *paired associates* task (Cohen & Squire, 1980), ten unrelated word pairs were presented three times each day with cued recall after each presentation; subjects were cued with the stimulus word of a pair and asked to provide the response. The number of words correctly recalled per presentation was recorded.

RESULTS

Rotary Pursuit

An analysis of variance (ANOVA) was performed on the number of seconds on target. A mixed-model design was used with group as the between factor, and day, block, and rpm as repeated factors. As can be seen in Figure 1, the PD

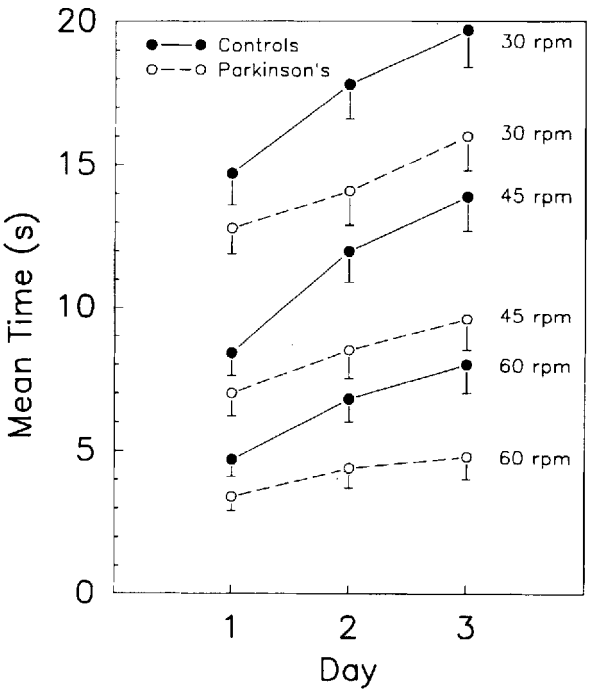

Fig. 1. Rotary pursuit learning in control and Parkinson's subjects. Means and standard errors.

group held the stylus on the target for less time than the control group ($F(1,38)=5.05$, $p<.05$). Although both groups showed a similar amount of learning across blocks ($F(2,76)=27.59$, $p<.001$), the PD group showed less learning across days ($F(2,76)=10.47$, $p<.001$). Impaired learning in PD patients was found in all rpm conditions when compared with control subjects using the difference between performance on day one and day three ($F(1,38)=7.11$, $p<.01$ for 30 rpm; $F(1,38)=10.80$, $p<.01$ for 45 rpm; $F(1,38)=9.04$, $p<.01$ for 60 rpm). These findings were explained by the PD group's significantly poorer performance only on day two ($F(1,38)=5.7$, $p<.05$) and on day three ($F(1,38)=6.54$, $p<.025$). Thus, the PD group's poorer rotary pursuit learning could not be attributed to initial differences between groups in performance level on day one. Furthermore, there were no statistically reliable differences between groups in performance level in any of the rpm conditions on day one during the first block of trials.

Both groups held the stylus on the target less as rpms increased ($F(2,37)=212.20$, $p<.001$). While the effect of rpm did not differ between groups, it did vary as a function of block ($F(4,152)=3.62$, $p<.01$) and day ($F(4,152)=12.19$, $p<.001$). Specifically, for all subjects more learning occurred

across blocks in the 30 rpm condition than the 45 or 60 rpm conditions, and over days more learning occurred in the 30 and 45 rpm conditions than the 60 rpm condition. These findings demonstrate that the amount of skill learning for both groups is dependent on task difficulty. Finally, for both groups, the amount of learning across blocks decreased from day one to day three ($F(4,152)=5.34$, $p<.001$).

Mirror Reading

Separate ANOVAs were performed on the mean time (seconds) to respond and the number of errors in mirror reading. A mixed model design was used with group as the between factor, and block and day were the repeated factors. As can be seen in Figure 2, the analyses showed that the average time to respond was similar between groups on all days ($p>.05$), and the PD group demonstrated the same amount of learning as controls both across blocks ($F(4,152)=24.6, p<.001$) and over days ($F(2,76)=115.30, p<.001$). Table 3 shows

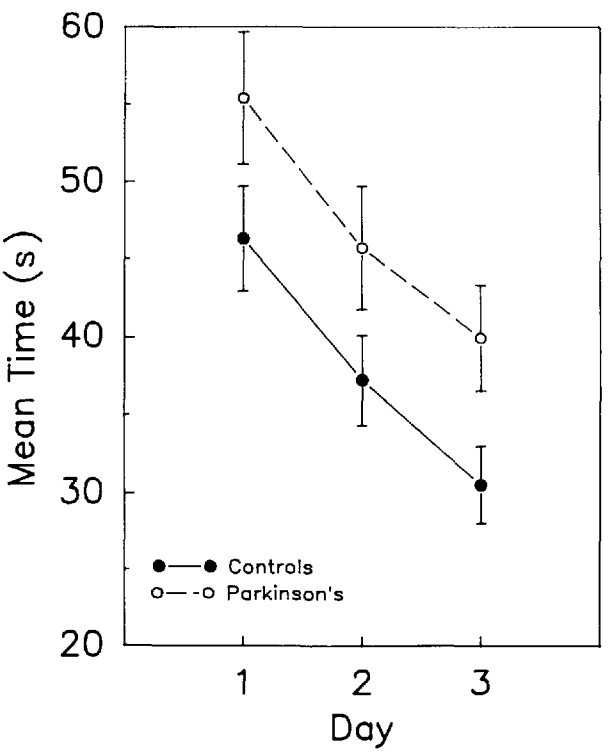

Fig. 2. Mirror reading learning in control and Parkinson's subjects. Means and standard errors.

Table 3.
Mirror reading: Mean (Standard Error) percentage of errors across days.

	Control Group	Parkinson's Group
DAY 1	0.3 (0.1)	1.0 (0.4)
DAY 2	0.2 (0.1)	0.5 (0.4)
DAY 3	0.0 (0.0)	0.1 (0.1)

that these trends were also found for the number of errors with both groups increasing their accuracy across blocks ($F(4,152)=12.49, p<.001$) and over days ($F(2,76)=15.41, p<.001$). One exception, however, was that group performance varied as a function of block ($F(4,152)=2.55, p<.05$). This interaction was simply due to the PD group's greater improvement in accuracy across blocks only on the first day of testing ($F(4,152)=2.46, p<.05$) because their initial level of performance in the first block was lower relative to the control group. Otherwise, the performance accuracy and improvement in accuracy was similar between groups on the second and third days.

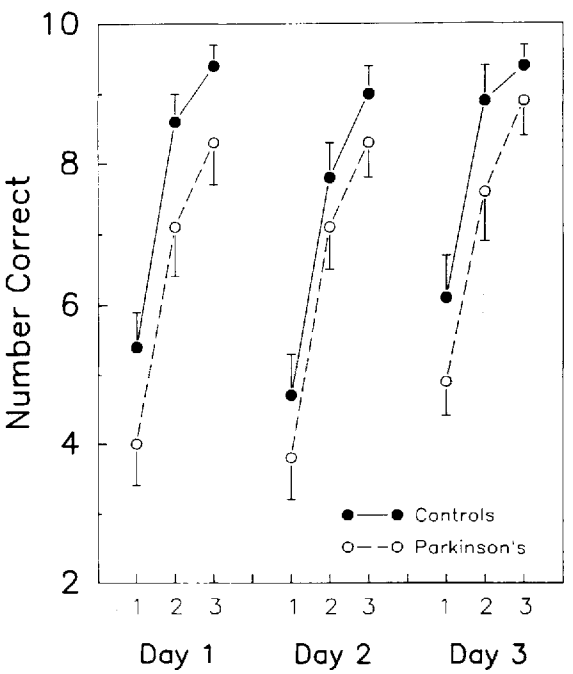

Fig. 3. Paired associate learning in control and Parkinson's subjects. Means and standard errors.

Paired Associates

An ANOVA was performed on the number of correct responses using a mixed-model design with group as the between factor and day and block as the repeated factor. The analyses found no difference between groups in the number of correct responses regardless of block or day of testing. As can be seen in Figure 3, learning also did not vary between groups. The PD group learned as rapidly as the control group across blocks ($F(2,76)=183.38$, $p<.001$) and over days ($F(2,76)=8.35$, $p<.001$). The rate of learning across blocks also was similar between groups within each day.

Relationships Among Experimental Tasks

A dissociation between groups on one task but not on others does not necessarily imply that the tasks reflect independent processes (Dunn & Kirsner, 1988). Impaired function in a particular process common to both tasks may or may not impair performance level depending on other task variables. To investigate this possibility, performances on the three experimental tasks were intercorrelated separately for PD and control subjects. No significant relationships were found among tasks in the amount of learning between day one and day three for either group. For PD patients, there also were no significant correlations among tasks in absolute performance level. For control subjects as well, performance level on paired associates was not related to performance on any other tasks, but performance levels on mirror reading (seconds) and rotary pursuit were correlated ($r=-.52$, $-.57$, and $-.64$, $p<.025$, for days 1, 2, and 3, respectively) such that faster mirror reading was associated with better rotary pursuit performance. These findings suggest that although similar processes may have influenced performance levels of control (but not PD) subjects on the two procedural tasks, skills involved in learning mirror reading are different from those used in rotary pursuit learning for both PD and control subjects.

Correlates of Rotary Pursuit Learning

To explore some of the possible underlying mechanisms for the deficits in rotary pursuit learning, we correlated performance on a clinical test of general perceptual organization (Block Design), a test of memory function (WMS), and a test of general cognitive function (Mini Mental State Exam) since these skills may partially explain the PD patient's slower learning rate. Specifically, we examined whether the pattern of rotary pursuit learning differed for PD and controls depending on how well they performed on the Block Design subtest of the WAIS-R, the WMS, and the Mini Mental State Examination. Using separate regression analyses with repeated measures and controlling statistically for age, these tests did not explain differences between groups in absolute performance (i.e., group X ancillary test interaction) or the amount of learning (i.e., group X ancillary test X day interaction). Thus global tests of cognitive functioning could not account for differences found in this study between PD and controls in the rate of rotary pursuit learning.

Early and Advanced PD

The question arises as to whether patients with more advanced PD showed deficits on both procedural tasks as well as on paired associate learning. Although we would expect advanced PD patients to show more primary motor dysfunction, cognitive dysfunction in PD also is frequently reported (Benecke, Rothwell, Dick, Day, & Marsden, 1987; Harrington & Haaland, 1989; Sanes, 1985; Sheridan, Flowers, & Hurrell, 1987), and might be evidenced by impaired skill learning on some or all tasks. Because age of PD onset and disease duration were not related to performance on any of the experimental tasks, summed scores on the NYU Disability Scale were correlated with task performance. Using separate regression analyses with repeated measures and controlling statistically for age, ratings on the NYU Disability Scale were not correlated with the level of performance or the amount of learning (across blocks or days) on paired associates or mirror reading (time or errors). In contrast, as symptoms of PD worsened (i.e., rigidity, tremor, bradykinesia), patients showed less time on the target ($F(1,17)=8.94$, $p<.01$) and less learning across days ($F(2,34)=3.46$, $p<.05$) in the rotary pursuit task regardless of rpm condition. These findings demonstrate that normal patterns of learning for the PD group on paired associates and mirror reading were not related to disease severity in this sample of PD patients. However, impaired rotary pursuit learning clearly was related to disease severity. This finding was due to the relationship of bradykinesia but not tremor or rigidity to the amount of rotary pursuit learning ($r=-.45$, $p<.05$ for 45 rpm; $r=-.52$, $p<.025$ for 60 rpm).

Follow up analyses compared PD patients with ratings on the NYU Disability Scale in the lower (scores less than 10) and upper (scores greater than 20) 35th centile. There were no differences between these groups in age, disease duration, age of disease onset, general cognitive functioning (i.e., Mini Mental State, WMS, WAIS-R subtests), spatial skills, or amount of tremor and rigidity. The advanced PD group did show more severe bradykinesia. Figure 4 shows that early PD patients ($n=7$) showed more learning across days in comparison to advanced PD patients ($n=7$) ($F(2,22)=6.27$, $p<.01$). However, because initial performance levels were not equated between early and advanced PD groups, differences in primary motor function could potentially explain these findings.

To equate the two PD groups on initial performance level, we compared rotary pursuit performance on day 1 (and also the first block of day 1) using different rpm conditions for each group. The initial performance levels on day 1 of the early PD group at 45 rpm ($M=9.6$, $SD=3.1$) and the advanced PD group at 30 rpm ($M=9.7$, $SD=3.4$) did not differ significantly. Similarly, the initial performance levels on day 1 of the early PD group at 60 rpm ($M=4.9$, $SD=1.6$) and the advanced PD group at 45 rpm ($M=4.2$, $SD=2.7$) did not differ significantly. An analysis of the difference in learning between day 1 and day 3 using the 45 rpm condition for the early PD group and the 30 rpm condition for the advanced PD group showed significantly less learning for the advanced PD group ($F(1,12)=7.33$, $p<.02$), although the advanced group did show some

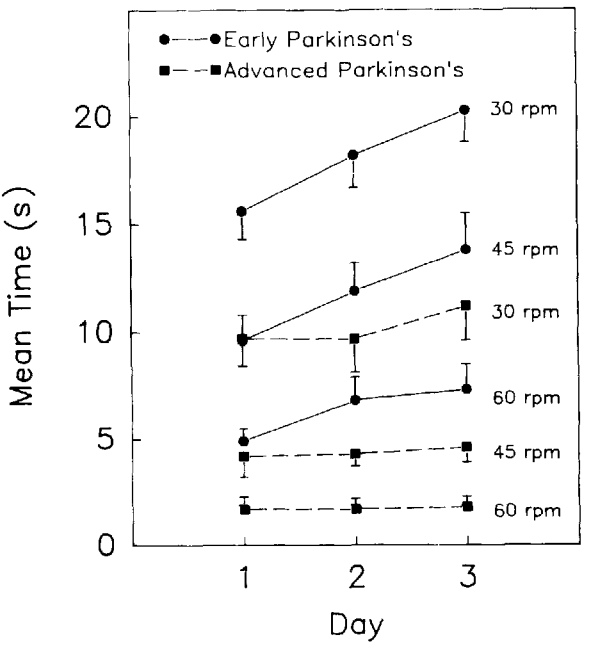

Fig. 4. Rotary pursuit learning in Parkinson's subjects with early and advanced symptoms of the disease. Means and standard errors.

learning ($F(2,10)=8.00, p<.01$). A similar test using the 60 rpm condition for the early PD group and the 45 rpm condition for the advanced PD group only approached significance ($F(1,12)=4.30, p=.06$). However, the early PD group showed significant learning in the 60 rpm condition ($F(2,10)=7.05, p<.025$) whereas the advanced PD group showed no learning in the 45 rpm condition ($p>.05$). These findings indicate that, when initial performance levels were equated between the PD groups, disease severity accounts for deficits in the processes that regulate skill learning. Further as the task requirements become more difficult, the strategies that advanced PD patients use break down. This is consistent with Flowers (1976) who suggests that PD patients utilize qualitatively different strategies than normal controls or patients with intention tremor in executing simple aiming movements, and this becomes more apparent as task difficulty increases.

DISCUSSION

Our results were not entirely consistent with the predictions from the procedural-declarative model as no dissociation was found between paired

associate learning and learning on a visuoperceptual procedural task in PD patients. Rather, impaired performance in the PD group was found only on a motor learning task and only for patients with more advanced symptoms of the disease. This is the first study to report performance differences among procedural tasks in PD patients. There is some indirect evidence for a dissociation among procedural tasks as some studies of HD patients have reported impaired mirror reading and rotary pursuit learning (Heindel et al., 1988; Martone et al., 1984) whereas others show normal priming in HD (Shimamura, Salmon, Squire, & Butters, 1987). The only direct evidence for a dissociation among procedural tasks (Heindel et al., 1989) showed that HD patients were impaired on rotary pursuit learning but not lexical priming while Alzheimer patients showed the opposite pattern of performance. These findings point to the differences among procedural tasks in the underlying component cognitive processes and suggest that structures of the neostriatum do not regulate all aspects of skill learning. This is consistent with recent formulations of the procedural-declarative model whereby procedural memory is viewed as an aggregate of many skills that are not necessarily dependent on one neuroanatomical system (Squire, 1987). The view that specific processes or procedures are dependent on separate neuroanatomical systems may also better account for some data from amnesia studies (Butters et al., 1985; Gliskey et al., 1986; Schacter et al., 1984) and may suggest that the diencephalic medial temporal lobe system is not the neuroanatomical substrate for declarative memory per se. Rather, this area may regulate certain component processes frequently associated with declarative tasks as well as aspects of some procedural tasks.

Much of the debate concerning single versus multiple memory systems has centered on comparisons between declarative memory tasks and procedural memory tasks as exemplified by priming paradigms (Jacoby, 1984; Schacter, 1987; Schacter & Graf, 1986a, 1986b; Smith, Butters, White, Lyon, & Granholm, 1988). By comparison, there has been little focus upon the nature of other procedural tasks on which amnesics or Alzheimer patients frequently demonstrate normal performance but HD or PD patients show impairment. Our findings clearly show that mirror reading and rotary pursuit learning can be disassociated and, therefore, must differ in one or more component processes.

These considerations suggest the possibility that learning may require the interaction of both declarative and procedural skills. This approach does not necessarily negate the possibility of separate procedural and declarative memory systems but rather emphasizes their interaction as tasks likely differ in the degree to which these two systems are involved. For instance, mirror reading may not be a general procedure that is constant regardless of the characteristics of the text (see Jacoby, 1984). Rather, several experiments (see Kolers & Roediger, 1984) have demonstrated that the skill of mirror reading is dependent on the physical and semantic features of the text, implying that procedural knowledge may be so task specific that it cannot be separated from declarative

knowledge, at least in this paradigm.

In our study, if mirror reading performance was partially dependent upon declarative memory, PD patients could adopt a strategy whereby they rely more on declarative processes than controls, in which case we would not expect to see a difference between learning on the paired associates task and mirror reading tasks. There is some evidence for this speculation as performance levels of PD patients were not correlated on the two procedural tasks whereas for controls performance was correlated. However, because the amount of learning on the three tasks was not related for either group this implies that processes involved in learning were specific to the task.

Whether motor learning is strictly procedural, in contrast to mirror reading, priming, and puzzle tasks which may involve both procedural and declarative processes, is open to question. A recent study suggests that in one particular motor learning task procedural and declarative memory were interactive (Fendrich, Healy, & Bourne, 1988). In this study, after neurologically intact subjects learned digit sequences on a computer keypad they were retested one month later. Subjects were able to discriminate old sequences from new sequences, and typing speed was faster for old sequences only if they were correctly classified on the recognition test.

Our results suggest that the basal ganglia subserves motor learning but not necessarily visuoperceptual procedural learning. The basal ganglia, however, should not be considered in isolation, as learning complex motor skills is clearly dependent upon a wide variety of areas which are neuroanatomically connected to form parallel systems (Goldman-Rakic, 1988). In addition, the role different parts of the basal ganglia play in regulating movement must also be examined because the neuroanatomical connections of the putamen and the caudate nucleus are different. Because the caudate nucleus has more neocortical input than the putamen, it may be more important for cognitive processing (Selemon & Goldman-Rakic, 1985). In the present study, this issue is interesting because our early PD group (who showed no motor learning deficits) was comparable to PD patients who showed putamen but not caudate abnormalities in positron emission tomography studies assessing dopamine distribution (Nahmias, Garrett, Firnau, & Lang, 1985). Our advanced group, however, may be more likely to have abnormalities in the caudate nucleus as the severity of their symptoms was greater.

In contrast to our results, Heindel et al. (1989) found no rotary pursuit learning deficits in a sample of nondemented PD patients. Because the dementia and the disease severity ratings used in this study were different from ours, it is difficult to directly compare the two. Unlike the nondemented PD group in the Heindel et al. study, our PD patients showed evidence of mild cognitive decline. Most studies, however, have evaluated dementia using a variety of dementia scales which may not be sensitive to the types of mild cognitive deficits reported in the present study. Despite these deficits, performance on general tests of cognitive function were not related to performance or learning on any of the

experimental tasks and could not explain the differences found between patients with early and more advanced symptoms of the disease. Contrary to our findings, others (Heindel et al., 1989) have found no relationship of tremor, rigidity, and bradykinesia with rotary pursuit learning, although these findings were not reported separately for nondemented and demented PD patients. Our observation that disease severity was related to rotary pursuit learning, might seem to suggest that impaired rotary pursuit learning could be attributed to poor primary motor skills. We do not believe that this is the case because when initial performance levels were equated, motor learning deficits were still observed. These results are consistent with the current experimental literature which suggests that along with primary motor dysfunction, PD is a disorder in the programming of movements (Benecke et al, 1987; Bloxham, Mindel, & Firth, 1984; Flowers, 1976; Harrington & Haaland, 1989; Sharpe, Cermak, & Sax, 1983). Therefore, we believe that the motor learning deficits seen in the advanced PD group are likely related to deficits in the cognitive processes specifically required to learn motor tasks.

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