MOVEMENT PREPARATION IN PARKINSON'S DISEASE
THE USE OF ADVANCE INFORMATION

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SUMMARY
The effects of advance information on movement planning in parkinsonism were assessed by means of movement precuing. Using this technique, the response latencies of identical sets of movements were compared across conditions in which the degree and type of advance movement information were manipulated. Specifically, prior information concerning three movement dimensions (the direction and extent of forthcoming movements, as well as the limb to be used) was or was not provided.

Eight patients with Parkinson's disease and 8 neurologically normal age-matched controls served as subjects. The experiment showed that the elevated reaction times of the parkinsonian subjects are not primarily caused by delays in response selection. Estimates of specification times for each of the three dimensions showed only a modest slowing in parkinsonians. The specification of those movement dimensions unknown before the response signal appears to occur serially, and can occur in a variable order as in normals. Since parkinsonians can initiate movements with shorter latencies when partial or complete information is available, albeit more slowly than normals, we conclude that response selection and specification processes preceding rapid discrete movements are relatively unaffected by the disease. The overall slowness in movement initiation in parkinsonians as compared with normals may in part be caused by excessive delays in motor time and, in general, to those 'input' and/or 'output' processes which are unaffected by advance information.

INTRODUCTION
Debate over the nature of the motor deficits in Parkinson's disease has centred on several issues, including the planning or advance preparation of movement (Marsden, 1982; Sharpe et al., 1983; Bloxham et al., 1984; Stelmach et al., 1987). While it is evident that disruption of movement execution is a principal manifestation of the disease, it is not yet clear if impairment in movement planning lies behind these overt deficits. Parkinson's disease also serves as a window on basal ganglia function, albeit an imperfect one (Marsden, 1982, 1984; Wing and Miller, 1984). The function of these structures in movement processes may be clarified by

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the description of specific deficits in parkinsonism or, conversely, by a failure to find a particular deficit.

The development of new behavioural science techniques in the study of movement in normal populations has allowed the examination of those mental operations which are antecedent to movement itself (Stelmach and Diggles, 1982). We describe in this paper the application of one such method—the movement precuing technique employed by Rosenbaum (1980, 1983), Goodman and Kelso (1980) and Bonnet et al. (1982)—to the study of movement preparation processes in Parkinson's disease.

This experimental paradigm was chosen because it provides insight into the use of advance information concerning an impending movement, a subject of recent debate among those studying Parkinson's disease. Flowers (1978) reported an inability of patients with Parkinson's disease to use a predictive strategy in a tracking task in which the stimulus followed a predictable sinusoidal track, a task which has inherent advance information about the movements to be performed. Bloxham et al. (1984) and Day et al. (1984) have subsequently argued that there is a partial, not a total inability to use predictive strategies in Parkinson's disease. In the experiments of Bloxham et al. (1984) a continuous tracking task and a discrete finger-lifting task were used, with different outcomes. No difference in tracking lag was observed between parkinsonian and control subjects when a predictable track was used, contrary to the finding of Flowers (1978). Bloxham et al. (1984) suggested that the continuous nature of their task could have allowed normal predictive strategies to be employed because no movement initiation was necessary once tracking had commenced. Conversely, the sinusoidal nature of the Flowers' (1978) tracking task required frequent direction changes: since each direction change could be considered as the initiation of a new movement, any specific deficit in movement initiation in Parkinson's disease would result in relatively poor performance. Bloxham et al. (1984) viewed these data as evidence of an inability of parkinsonian subjects to initiate movement on the basis of advance information, although such information could be used perfectly well in guiding the form of an ongoing movement. In support of this conclusion, Bloxham et al. presented data from a second experiment, in which normal subjects could initiate movement more rapidly in a finger-lifting task when advance information was provided as to which finger was to be moved. They found a nonsignificant decrease in the corresponding reaction times of parkinsonian patients, arguing that this was evidence that advance information was not used in the initiation of discrete movements. The degree to which these data support such a conclusion is, however, open to question, and we return to this matter in the Discussion.

Day et al. (1984) also used a tracking task, finding that although the benefit of a predictable track was less marked for Parkinson's disease subjects than for controls, they were able, nevertheless, to use a predictive strategy. They attributed the residual decrement in performance to inaccuracy of execution by parkinsonian subjects rather than to any deficit in strategy.
The present experiment provides new evidence in the unresolved debate over the use made by parkinsonian subjects of advance movement information. In a previous study (Stelmach et al., 1986) it was found that parkinsonians showed no clear evidence of being able to program rapid finger-tapping sequences in the same way as normals, when given a precue as to the number of taps in the forthcoming sequence. They did not show a linear increase in reaction time with increasing sequence length, as did normals, for whom this increase is argued to reflect the length of the motor program. Parkinsonians showed a dissociation of the first and subsequent taps as reflected by an unusually prolonged first intertap interval, and they had an increasing number of sequence length errors (too few or too many taps) as sequence length increased. These three findings together indicate a deficit in the programming of rapid movement sequences in Parkinson's disease.

The current study depicts the temporal structure of parkinsonian movement preparation by analysis of three underlying 'dimensions' of movement: the limb used, the direction of motion and its extent. The time taken to specify values on each of these dimensions is estimated, allowing between-group comparisons of their absolute and relative magnitude. Beyond these estimates of specification time, the method allows inferences to be drawn about the seriality and order of specification, aspects of movement planning which may be altered by Parkinson's disease. When two or more dimensions are not known until the time of movement, are they specified serially or in parallel? Secondly, is there a fixed order in which dimensions must be specified?

Evidence of a second type is provided by the experiment described below which addresses the issue as to whether bradykinesia is accompanied by any slowness of central processing, since the experiment contains a simple reaction time and subsets of choice reaction time conditions. The unique feature of the precuing paradigm is that these conditions all require exactly the same movements, so that differences in response latencies cannot be attributed to variations in the number, complexity, or any other facet of the movements actually made by the subjects. Consequently, inferences can be drawn concerning relative slowing of response selection as opposed to stimulus detection and encoding, and response programming processes.

SUBJECTS AND METHODS

Subjects

Eight adults diagnosed as having Parkinson's disease and 8 age-matched adults with no history of neurological disorder served as subjects in this experiment. All subjects were paid for their participation. The parkinsonian subjects ranged in age from 58 to 76 years, with a mean age of 64.6. Table 1 shows the symptom profile and severity for each of these subjects, all of whom were reported by their neurologist to have no secondary neurological signs not typically associated with Parkinson's disease. Each of these subjects was stabilized on Sinemet in combination with either dopamine agonists or anticholinergic drugs for several years, and was taking medication at the time of testing. One parkinsonian subject was excluded from the study when it became evident in the practice sessions that excessive tremor would preclude adequate performance of the task. Subjects with Parkinson's disease followed their normal medication schedule during testing. The control group ranged in age from 55 to
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TABLE 1. CASE PROFILES OF PARKINSON’S DISEASE GROUP

<table>
<thead>
<tr>
<th>Case</th>
<th>Sex</th>
<th>Age (yrs)</th>
<th>Duration of disease (yrs)</th>
<th>Medication</th>
<th>Symptoms</th>
<th>Laterality</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>M</td>
<td>71</td>
<td>7</td>
<td>Sinemet, Benzhexol</td>
<td>Moderate rigidity, Moderate tremor</td>
<td>Bilateral</td>
</tr>
<tr>
<td>2</td>
<td>F</td>
<td>76</td>
<td>11</td>
<td>Sinemet, Bromocriptine</td>
<td>Moderate rigidity</td>
<td>Bilateral</td>
</tr>
<tr>
<td>3</td>
<td>F</td>
<td>58</td>
<td>7</td>
<td>Sinemet</td>
<td>Severe rigidity, Severe tremor</td>
<td>Bilateral</td>
</tr>
<tr>
<td>4</td>
<td>M</td>
<td>64</td>
<td>17</td>
<td>Sinemet, Benztropine, Bromocriptine</td>
<td>Severe rigidity, Severe bradykinesia</td>
<td>Bilateral, Left</td>
</tr>
<tr>
<td>5</td>
<td>M</td>
<td>65</td>
<td>13</td>
<td>Sinemet, Benzhexol</td>
<td>Moderate rigidity</td>
<td>Bilateral</td>
</tr>
<tr>
<td>6</td>
<td>F</td>
<td>65</td>
<td>4</td>
<td>Sinemet</td>
<td>Moderate rigidity</td>
<td>Bilateral</td>
</tr>
<tr>
<td>7</td>
<td>M</td>
<td>59</td>
<td>5</td>
<td>Sinemet, Benzhexol</td>
<td>Moderate rigidity, Moderate/severe tremor</td>
<td>Bilateral</td>
</tr>
<tr>
<td>8</td>
<td>F</td>
<td>59</td>
<td>16</td>
<td>Sinemet, Pergolide</td>
<td>Moderate/severe rigidity</td>
<td>Bilateral</td>
</tr>
</tbody>
</table>

71 years, with a mean age of 64.8, and were all reported to have no history of neurological disorder or chronic illness.

Apparatus

The experiment was conducted in an experimental chamber free from any distraction. The subject sat in a chair of adjustable height in front of a table on which the apparatus was mounted. The apparatus is depicted in fig. 1. In front of the subject and parallel to the sagittal axis were two rows of five keys, one in front of each arm. The middle key of each row was designated the home key; the other four were target keys. The centre of the near keys was 3.5 cm from the home keys, and that of each far key a further 3.5 cm away. The home and near keys were 1.8 cm squares, while the far keys were 2.6 cm squares (the larger size was intended to compensate for their greater distance from the home keys). The left and right rows were 21 cm apart. The keys were Cherry momentary contact switches with flat surfaces, requiring a force of 125 g for closure. They were set into black styrofoam such that they were flush with the top surface when not depressed.

Stimulus lights corresponding to the target keys were attached to a vertical board covered with black felt situated behind the target keys, about 85 cm away from the subject’s eyes and level with them. The lights were light-emitting diodes approximately 3 mm in diameter. They were spaced so that they subtended an angle of less than 5 deg, permitting all of them to be seen without saccadic eye movements (6.8 x 7.2 cm). The lights were arranged so as to be spatially compatible with the target keys, and are also shown in fig. 1. They were colour-coded in the same way as the target keys so as to maximize compatibility. All target keys and lights were red, while both home keys and lights were yellow.

The experiment was controlled by the laboratory LSI-11 minicomputer which was programmed to present all stimulus lights and warning tones, and to store data on floppy disc for subsequent analysis.
Procedure

Each parkinsonian subject participated in one practice and two experimental sessions, with a minimum of eight blocks of experimental trials administered. Control subjects participated in one practice session and one experimental session. Before data collection each subject performed three practice blocks of 64 trials in which the required movements were rehearsed. Starting with the index fingers of each hand depressing the appropriate home key, the subject was required to move to each of the target keys in a quasirandom order as soon as each target light was illuminated. An intertrial interval of 5 s was used. As in the subsequent experimental sessions, target keys on each side were to be pressed by the ipsilateral index finger. The contralateral finger had to be kept on its home key. The first practice block was performed with full vision of the target keys allowed, so that visual feedback concerning errors of aiming was available. In the second block, the lower half of the visual field was occluded by means of a head-mounted visor, which permitted full view of the stimulus lights. If aiming errors were made, subjects could view the target keys by tilting the head downwards, but they were instructed not to do so until after a trial was completed. Two of the parkinsonian subjects performed the task without the visor, but looked at the keys only occasionally. If the subject made an error (for example, if an incorrect key was pressed), the experimenter drew his or her attention to it if the subject appeared to be unaware that an error had been made.

Following the movement practice blocks, the experimental procedure was described to the subject, and the experiment practice blocks were administered. The sequence for each trial is illustrated in fig. 2. First, the yellow home lights were illuminated as a warning signal. After 1 s the precue lights were illuminated and were accompanied by an auditory signal. The yellow home lights remained illuminated with the precue so as to differentiate the precue from the response signal. The precue combinations comprised 1, 2, 4 or all 8 of the target lights. When 4 lights appeared they precued either arm, direction or extent (e.g., all 4 left lights would indicate that the forthcoming movement would be made with the left arm, but direction and extent remained uncertain). When 2 lights appeared they precued either arm and direction, arm and extent or direction and extent. A single precue light gave
precise information about the required movement, and all 8 lights conveyed no information to the subject about any of the three dimensions. Precues were always valid, so that the response signal was always to one of the precued targets. Subjects were instructed to attend to the precue and to take advantage of this prior information. The precue light(s) remained on for 1000 ms after which all lights were extinguished for a 1000 ms preparation interval. One of the target lights just precued was then reilluminated, serving as the response signal. The subject was required to move to the corresponding target key with the appropriate index finger as rapidly as possible following the presentation of the response signal.

![Diagram of event durations](image)

Fig. 2. Absolute and relative durations of events in a typical trial, beginning at the left. The durations are drawn to scale except for the dependent measures (reaction time (RT) and movement time (MT)) for which the break denotes that these durations vary from one trial to another.

Reaction time was recorded for each trial, and was defined as the interval between the onset of the response signal and departure from the home key. Also recorded was movement time, defined as the interval between departure from the home key and arrival at the target (or any other) key. The response signal remained illuminated until a target key was depressed, or for 6 s, whichever was shorter. Movement before the response signal was recorded as an anticipation error, as was movement within 130 ms of its illumination. Separate error categories were used for both fingers leaving the home key, for failure to leave the home key within 6 s, for slow responses (reaction times or movement times slower than twice the average times for that subject in the final practice session), for incorrect responses (movements to the wrong key), and for failed catch trials. Each block contained four catch trials, identical to the real trials except that no response signal appeared. Their purpose was to discourage anticipation errors. When an error was made, that trial was repeated later in the block so that the required number of correct responses was always made in any given block.

A 5 s intertrial interval was used in the experiment, and a break of between 1 and 2 min was given between blocks. Subjects were permitted a 5 to 10 min break outside the testing chamber every two or three blocks to prevent discomfort and to promote optimal concentration throughout the period of testing.

Three additional measures formed a subsidiary part of the experiment, to be described in a subsequent paper. All subjects were given a separate simple reaction time test before each block, comprising eight trials in which the left or right index finger was raised from the home key in response to the appearance of a reaction signal. Parkinsonian patients also performed a simple alternating tapping task in between blocks, as a measure of bradykinesia. They also rated their degree of control.
over their movements on a 1 to 10 scale before each block. All procedures were explained fully to the subjects, whose written consent was obtained before testing.

RESULTS

Reaction Times

The reaction time results are presented in the following order: overall group differences, comparisons between Uncertainty Levels, and specification times. The following terms will be used in this and subsequent sections: each of the eight conditions are denoted by dimensions which remain to be specified following the response signal (i.e., those not precued), and appear in the text in upper case lettering (e.g., ARM and DIRECTION, in which the subject knows the extent of the movement from the precue, but not which limb to use or how far to move). Uncertainty Level refers to the number of dimensions about which the subject is uncertain before the response signal. Thus, ARM and DIRECTION is one of three conditions in Uncertainty Level Two, since only one dimension—extent—is known from the precue, while two remain unknown.

Group reaction times are shown in fig. 3. Upward arrows indicate movements away from the body, downward arrows those towards the body. Long and short arrows denote long and short movements, respectively, and data for right and left arm movements are distinguished by the use of continuous lines for the former and dashed lines for the latter. A horizontal line depicts the reaction time for each condition averaged across movements involving the different combinations of arm, direction and extent.

Two aspects of the reaction time data presented in fig. 3 are immediately apparent. First, the parkinsonian subjects were notably slower than the controls, with significantly longer response latencies: 573 ms as against 385 ms for the control group (P < 0.05) as revealed by a split-plot analysis of variance. This difference was evident in all eight conditions shown in fig. 3. The second obvious feature of these data is that the parkinsonian subjects appeared to take advantage of the advance information provided about the impending movement. In spite of their overall slowness relative to the controls, they were clearly able to initiate movement sooner with prior knowledge. This is evident from the increase in latencies with increasing uncertainty, an effect also very apparent in fig. 4. Had the parkinsonian subjects been unable to use the advance information to initiate the movements more rapidly, no difference in latencies between Uncertainty Levels would have been evident. The effects on reaction time of target direction, extent and arm, and their interactions with group, were not statistically significant (P > 0.1 in all cases). The largest difference between the two values on any of the dimensions was 19 ms.

We now turn our attention to comparing the Parkinson's disease and control groups on differences between and, subsequently, within Uncertainty Levels. The group means for each Uncertainty Level are presented in fig. 4. Inspection of these points suggests that the group effects are additive and that each data set has a linear trend. No interaction was found between Uncertainty Level and group (P > 0.1).
The linearity of the data for each group was ascertained by trend analysis subsequent to analysis of variance. The linear components were significant for each group ($P < 0.001$). Differences between the slopes for each group were assessed by means of a linearity by group interaction comparison, which showed no difference between the two ($P > 0.1$).

Regression equations for each group were calculated to be $y = 474 + 64.8 \times$ for the Parkinson's disease group, and $y = 305 + 52.9 \times$ for the control group, where $y =$ reaction time and $x = \log_2$ of the number of response alternatives (a figure which is identical to the Uncertainty Level in this experiment). These regression lines have been fitted to the data in fig. 4. This formulation may be expressed alternatively as information transmission rates for the two groups, calculated to be 15.4 and 18.9 bits/s for Parkinson's disease and control groups, respectively. The latency difference between the groups increases only modestly and nonsignificantly as the number of alternatives increases from a simple reaction time condition to
Uncertainty Level

Fig. 4. Group Uncertainty Level latencies. Regression lines have been fitted to the data for each group. △ = parkinsonians (regression equation: RT = 474 + 64.8 \cdot x). ○ = controls (regression equation: RT = 305 + 52.9 \cdot x). 
\( x = \log_{10} \) of number of response alternatives = uncertainty level.

an eight-choice reaction time task. This suggests that the longer reaction times shown by the parkinsonian subjects are not primarily caused by impairment in response selection.

Differences within Uncertainty Levels One and Two are considered next. One unique feature of the precuing technique is that it allows some assessment of the relative time costs of specifying different underlying dimensions of movement, unconfounded by differences in the movements themselves. Do parkinsonians show evidence of differing from normals in these times and their ordering? As can be seen in Table 2, the condition in which only DIRECTION remained to be specified had, on average, the greatest latency for both groups. However, the ordering of the conditions in which only ARM or EXTENT had to be specified was not the same for the two groups. For the parkinsonian subjects, EXTENT was on average shorter than ARM, with the converse true for the control group. Planned comparisons revealed no differences between the latencies for ARM, DIRECTION and EXTENT for either group, however, (\( P > 0.05 \) in all cases). Within Uncertainty Level Two, no differences were found for the Parkinson's disease group, but in the
controls, the latency for ARM and EXTENT was reliably shorter than that for ARM and DIRECTION ($P < 0.05$).

Also shown in Table 2 are the 'specification times' for each of the three dimensions for both groups. These are estimates of the time taken to specify the dimensions as derived from a subtractive procedure involving pairs of latencies. For example, the specification time for DIRECTION may be estimated by subtracting the latency for ARM from that for ARM and DIRECTION. It should be borne in mind that these measures are independent of the relative ordering of the same dimensions within

<table>
<thead>
<tr>
<th>Uncertainty Level 1</th>
<th>Uncertainty Level 2</th>
<th>Specification times</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>A</td>
<td>D</td>
</tr>
<tr>
<td>Controls</td>
<td>350</td>
<td>362</td>
</tr>
<tr>
<td>Parkinsonians</td>
<td>537</td>
<td>538</td>
</tr>
</tbody>
</table>

All times in ms. A = Arm. D = Direction. E = Extent. * AD significantly > AE ($P < 0.05$). ** D significantly > E ($P < 0.05$).

Uncertainty Level One, since they are derived from distinct parts of the data set. Converging evidence for a real difference in the times necessary to specify underlying dimensions of movement would therefore exist if a particular dimension has the longest specification time and the longest latency within Uncertainty Level One. As can be seen in Table 2, there is an identical ordering of these times for the Parkinson's disease group, (DIRECTION > ARM > EXTENT) by these independent measures, the same ordering shown by both groups in the specification times. In the control group, however, the ordering of ARM and EXTENT were reversed in Uncertainty Level One as compared with the specification times. Again, no difference between specification times was found in the Parkinson's disease group, but DIRECTION was significantly longer than EXTENT for the controls ($P < 0.05$). Unlike the latency measures, the longer specification times for the parkinsonians were not statistically different from those of the controls ($P > 0.1$).

Two additional comments about the estimated specification times are in order. First, our data do not contradict the notion that the three dimensions are specified serially by both groups. This conclusion follows from the fact that on only a few occasions is the latency for conditions involving the specification of two dimensions less than the sum of the latency for one of the dimensions and the specification time for the other. Our specification time estimates probably err on the high side since they include a component attributable to the detection and encoding of four stimuli as compared to two, a problem discussed more fully by Rosenbaum (1983). Even if this 'perceptual' component is as small as 10 ms, then the obtained Uncertainty Level Two latencies all exceed the sum of their component Level One latencies and specification times. (The reader may verify this estimate by reference to Table 2.)
Thus it seems that both parkinsonian and control subjects specified the unknown dimensions serially when two of them remained to be specified at the time of movement. In order to argue that serial specification also occurred when all three dimensions were unknown, it is necessary to assume that the perceptual component of our estimated specification times was as high as 35 ms. (The unadjusted specification time for each dimension added to the obtained latency from Uncertainty Level Two for the other two dimensions is between 2 and 35 ms longer than the obtained Uncertainty Level Three latencies: this would constitute evidence of some parallel specification if no allowance for a perceptual component in the specification time were made.) Attributing as much as 35 ms of the estimates of specification time to perceptual processes is not excessive in the light of the data from control experiments presented by Rosenbaum (1983) used to assess this component.

Secondly, parkinsonians and controls were able to specify unknown dimensions in a variable order, since information concerning any of the dimensions was used as fully by parkinsonians as by controls. If, for example, precuing only direction produced latencies no shorter than precuing none of the dimensions, but precuing arm and direction led to latencies shorter than that obtained when arm was precued, then this would suggest that the limb must be specified before the direction of the movement. That this was not the case suggests that Parkinson's disease is not characterized by a rigid hierarchical ordering of response specification, which would clearly be a profound planning deficit.

**Movement Times**

The group movement times are shown in fig. 5. The parkinsonian subjects were markedly slower than the controls: 541 and 282 ms on average, respectively ($P < 0.02$). Most of this difference can be attributed to the parkinsonian's bradykinesia, but terminal inaccuracy also contributed since keys depressed at the very edge of the key surface were not triggered as rapidly as when they were hit in the centre. This was because the finger was partially obstructed by the flush surrounding surface when the finger hit the border between the key and the adjacent surface. Decreased accuracy in ballistic aiming has been demonstrated in Parkinson's disease by Flowers (1975), and our subjects were observed to hit the edge of the key surface more frequently than the controls.

The extent, direction and arm used in the movement all produced significant main effects ($P < 0.05$ in all cases) such that the short movements were faster than the long ones by 69 and 34 ms for control and Parkinson's disease groups, respectively, those towards the body took longer than those away from it (by 25 and 53 ms), and movements of the right arm were faster than those with the left (by 18 and 77 ms). None of these factors interacted with group.

**Error Patterns**

The parkinsonian subjects had a higher overall error rate than controls, 18.69 compared with 12.03%. The error rates are shown in Table 3 by category and group.
FIG. 5. Group movement times. The data are depicted in the same manner as in fig. 3.

TABLE 3. GROUP DATA FOR ERRORS

<table>
<thead>
<tr>
<th>Incorrect responses</th>
</tr>
</thead>
<tbody>
<tr>
<td>Slow response</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Controls</td>
</tr>
<tr>
<td>Parkinsonians</td>
</tr>
</tbody>
</table>

All figures are percentages of total trials. L = long movements performed when short movements were required. S = short movements performed when long movements were required.

Note that the largest source of errors for both groups was 'slow responses'. Since reaction times and movement times were evaluated not with respect to an arbitrary limit but by a criterion derived for each subject, the higher slow response rate for the parkinsonians reflects greater variability in speed of responding and moving than that shown by the controls. The second major source of errors for both groups was
'errors of extent', of which the majority were short movements made when long movements were required. Although a small number of anticipation errors were made by both groups, none of the catch trials was failed by any subject.

**DISCUSSION**

In discussing these results, we emphasize that the movements studied were brief, rapid and discrete, and that no inferences about preparatory processes in more sustained or complex movements are implied. The parkinsonian subjects used in this study made more errors than the controls, moved more slowly, and had longer response latencies. In other respects, however, their motor behaviour showed little deficit: use of advance information to initiate movement with shorter latencies, serial specification of movement dimensions (at least in the case where two were not precued) and the ability to specify movement dimensions in a flexible order.

Would similar results have been forthcoming had the Parkinson's disease group been unmedicated during testing? We suggest that while movement times and errors would probably have increased, the reaction time (RT) data would most likely have increased by only a fixed amount across conditions. First, the total testing period of approximately 4 h covered all parts of the subject's medication schedules, so that the data cannot be regarded as pure 'medicated' performance. Secondly, the three experiments reported by Rafal *et al.* (1984) show consistently additive RT effects of withdrawing medication, using experimental tasks and procedures similar to our own. Thus a greater overall RT difference, but no interaction with conditions, would have been the most likely result with completely unmedicated parkinsonian subjects.

The experiment reported here had an outcome different from that of Bloxham *et al.* (1984) who concluded that 'parkinsonian patients have no difficulty in using prior information to plan in advance the form of a movement, but do have difficulty in using this information to initiate or select a movement'. In our experiment, the decreases in RT with more advance knowledge were no less for the parkinsonian subjects than for the controls, in a task using aiming movements of the hand. The discrepancy is not explained by the continuous/discrete dichotomy advanced by Bloxham *et al.*, since the hand movements required of our subjects were clearly discrete, nor by differing medication status of subjects in the two studies. Inspection of the data for experiment two of Bloxham *et al.* suggests that their conclusion may be somewhat overstated. With both a 250 ms and a 2000 ms delay, parkinsonian reaction times decreased when prior information was available. Indeed, the 46 ms decrease in the 2000 ms delay condition is comparable to the 61 ms difference between Uncertainty Levels Zero and One in our own study, in which a 2000 ms delay was also used. The high values of cell standard errors in the Bloxham *et al.* study prevent significant differences from being found, but may also reflect a lack of power in their design. Our own data are clearly consistent with the view that
parkinsonians can indeed select and initiate discrete movements more rapidly on the basis of advance information.

The limited number of significant differences within Uncertainty Levels and between specification times stands in contrast to the findings of similar experiments using young adults (Goodman and Kelso, 1980; Rosenbaum, 1980; Bonnet et al., 1982) in which extent has proved to have a reliably shorter latency. The ordering of DIRECTION, ARM, and EXTENT is consistent for the parkinsonian group, whether assessed by specification times or by relative differences between the values for the three Uncertainty Level One conditions, and shows EXTENT to require the shortest specification time, as in these earlier studies. While the Uncertainty Level One data for our control group does not show EXTENT as being shortest, it is significantly shorter than DIRECTION as judged by specification times and, in combination with ARM, is significantly shorter than DIRECTION and ARM within Uncertainty Level Two. It should be noted that not all subjects in either group had the same ordering of RTs within Uncertainty Levels or specification times. We see nothing in these data to suggest that specification times are differentially affected in Parkinson's disease. On the contrary, parkinsonian specification times were slowed proportionately less than response latencies as a whole (31% as opposed to 49%). Moreover, parkinsonian subjects retain the ability to specify dimensions in a variable order as argued earlier, an important characteristic of flexible strategies for movement.

It might be argued that the very similar specification times (and comparisons within Uncertainty Levels) for the three dimensions reflect an insensitivity of the method to any actual differences between specification times. Such a criticism was made by Goodman and Kelso (1980), particularly for highly compatible stimulus-response relationships such as that used here. We discount this possibility since reliable differences, especially the finding of lower specification times for EXTENT, have been found previously both in a different laboratory (Bonnet et al., 1982) and in our own using the same apparatus and methods (G. E. Stelmach, N. A. Goggin and A. Garcia-Colera, 1987).

The additive nature of the difference between the latencies for the two groups at different Uncertainty Levels (fig. 4) is strongly reminiscent of the data provided by Rafal et al. (1984), who showed no slowing of memory scanning in parkinsonians when unalleviated by medication compared with an alleviated condition. Similarly, no deficit was found between the cognitive components of orientation of attention in the visual field, or preparation for a manual movement when the subjects were untreated. These data of Rafal et al. (1984) have similarities with our Uncertainty Level data. Both show the normal latency increases with task difficulty (memory set size or Uncertainty Level). These increases are not disproportionately steep for the untreated condition or for parkinsonians in comparison to controls, but do show higher intercepts. A common interpretation for these findings would be that slowing of 'input' and/or 'output' stages (i.e., stimulus detection and classification, response programming and production) is a manifestation of the disease, but intervening
processes involving memory searching or response selection are not, since effects are additive (Theios, 1975). Reaction times measured by movement initiation include 'motor time' (the time between the first occurrence of EMG activity and onset of movement. The abnormal EMG patterns documented in parkinsonism—inability to modulate the amplitude of the initial burst (Hallett and Khoshbin, 1980) and repetitive bursts of agonist and, sometimes, antagonist activity (Baroni et al., 1984)—suggest a selective slowing in motor time. In support of this interpretation are our recalculations of Schneider's (1968) simple and choice RT data (SRT and CRT, respectively), which show that motor time was disproportionately slower in Parkinson's disease than in normals (SRT: 26.4%, CRT: 89.0%) while only modest increases were evident in premotor time (SRT: 10.7%; CRT: 16.0%). At least part of the higher intercept seen in our parkinsonian Uncertainty Level data and in the 'unalleviated' memory-scanning data of Rafal et al. (1984) can be ascribed to slower force production following improperly regulated EMG signals, but a full account of elevated response latencies in parkinsonism is still needed.

ACKNOWLEDGEMENTS

This work was supported by funding from the National Institute of Neurological Diseases and Stroke, grant no. NS17421-US Public Health Service. We thank Dr Henry Peters for referring patients.

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(Received October 14, 1985. Revised February 11, 1986. Accepted February 20, 1986)