

The control of bimanual aiming movements in Parkinson's disease

GEORGE E STELMACH, CHARLES J WORRINGHAM*

From the Motor Behavior Laboratory, University of Wisconsin-Madison, Madison, Wisconsin, USA

SUMMARY The control of unimanual and bimanual aiming movements by Parkinson's disease and control subjects was examined. Despite greater bimanual movement initiation asynchrony and overall bradykinesia, the Parkinson's disease subjects were affected by the experimental manipulations in the same way as controls. Symmetrical and, more especially, asymmetrical bimanual movements required more preparation time and were executed more slowly by both groups than were unimanual movements. Both groups also showed temporal linkage of movements to targets of different extents—movements which have different movement times when performed unimanually, as well as of the faster and slower limbs. A majority in both groups over-compensated for asynchrony in bimanual movement initiation by modulation of movement times, but there was no group difference in this tendency. The results are discussed in terms of underlying motor control processes and with regard to previous evidence for impaired control of simultaneous movements in Parkinson's disease.

A notable characteristic of normal movement is the ability to execute two actions at the same time. If individuals with a given movement disorder retain the ability to perform certain movements individually, but experience difficulty in doing so simultaneously, it suggests that there is a high level deficit in the integration of two or more motor programmes. Clinically, just such a movement deficit has been described in relation to Parkinson's disease, and several studies have also documented this impairment,^{1 9 17 20 21} although the results of Perret¹⁶ are not clear-cut.

Of these, the study by Benecke *et al*¹ is the only one to have used rapid, discrete movements, thereby circumventing problems of differential attention demands and of fatigue from repetitive movements, both of which complicate the interpretation of earlier studies. These workers required Parkinson's disease and control subjects to make rapid elbow flexion

movements, rapid finger flexion movements, and an isometric "squeezing" action, each independently. Subjects also performed the elbow flexion movement simultaneously with each of the other two manoeuvres. In addition to slower execution (movement time) than controls in each individual task, the Parkinson's disease subjects showed a further slowing when performing the pairs of movements, especially when the same limb was used. (A much smaller degree of additional slowing was present when the tasks were performed with both limbs at the same time.) The authors concluded that such slowing was evidence of a "pure motor deficit in the performance of two different movements at the same time", and portrayed the underlying cause as a processing deficit, namely difficulty in superimposing one motor programme on another.

What is intriguing about this hypothesis is that it leads to the prediction that there would be differing degrees of impairment in different types of simultaneous movement, depending on the extent to which it is necessary to utilise two distinct motor programmes. If the movements have some elements which are programmed in common, then relatively little impairment may be seen, despite the fact that their overt characteristics are different (for example, the direction, extent and accuracy of each).

Address for reprint requests: George E Stelmach, Motor Behavior Laboratory, University of Wisconsin-Madison, Madison, WI 53706, USA.

*Present address: Motor Behavior Laboratory, Department of Kinesiology, University of Michigan, 401 Washtenaw Avenue, Ann Arbor, MI 48104-2219, USA.

Received 27 March 1987 and in revised form 19 August 1987.
Accepted 29 August 1987

One class of movements in which there is evidence for a common programming element is bimanual aiming movements to different targets, performed together. In normals, the duration of single movements to targets has long been known to have a lawful relationship to the distance to the target and its size.⁷ When movements to two different targets are made together, however, this law no longer applies to each limb separately. Kelso^{10,11} and Marteniuk *et al*¹⁵ have shown that these movements are performed nearly simultaneously, as if controlled as a single unit. The limb which is moving to the easier target moves more slowly than when moving to the same target unimanually. While the movements are different in some respects (direction, extent, degree of precision), the timing appears to be regulated in common for the two limbs. It is therefore of interest to see whether Parkinson's disease subjects plan and perform such movements with an additional speed decrement relative to controls (over and above bradykinesia effects), as well as to examine other aspects of bimanual coordination.

In the experiment described below, we examined the performance of simultaneous movements of Parkinson's disease subjects and controls, using this bimanual movement paradigm, with a view to determining if Parkinson's disease adversely affects control at several levels. Specifically, we posed the following questions:

- (1) Are movement latencies and speeds slower in Parkinson's disease than control subjects for bimanual as opposed to unimanual movements? An undue slowing in bimanual performance would be evidence of a general difficulty in preparing and executing simultaneous movements.
- (2) Do Parkinson's disease subjects show the same type of temporal linkage of bimanual (especially asymmetrical) movements as do controls? (By temporal linkage we mean the tendency towards

simultaneous performance of movements which have different time-courses when performed independently.) Any departure from the pattern for controls would indicate a difficulty in *integrating* two otherwise separate motor programmes.

- (3) Do Parkinson's disease and control subjects differ with respect to the asynchrony of movement onset and termination in bimanual tasks? Whether or not the overall structure of these movement pairs is impaired, there may be a deficit evident in the degree to which Parkinson's disease and controls can synchronise movement initiation and termination.

Methods

Subjects

The Parkinson's disease group comprised six females and four males, with an average age of 64 years (SD = 7.1). These subjects had been diagnosed as having Parkinson's disease but no other neurological disease. A profile of these subjects is given in table 1. The control group was made up of six females and four males who had no evidence of any neurological disease. Their average age was 64.9 years (SD = 7.3). All subjects performed a bradykinesia assessment test, in which the index finger was tapped across a 20 cm sector painted on a wooden board for a period of 20 s. The Parkinson's disease subjects' scores are also shown in table 1. The Parkinson's disease group had significantly lower scores than controls on this measure, the group scores averaging 34.9 and 44.25 complete cycles, respectively ($p < 0.05$), the Parkinson's disease group therefore showed objective evidence of bradykinesia in addition to the symptoms shown in table 2. While somewhat heterogeneous with regard to severity and symptoms, all of the Parkinson's disease subjects had noticeable impairments at the time of testing. Six of the 10 Parkinson's disease subjects experienced considerable difficulty with writing, walking or getting up from a chair at the time of testing. Parkinson's disease subjects continued medication according to their normal

Table 1 Profile of Parkinson's disease subjects

Sub No.	Sex	Age (yr)	Duration of Parkinson's disease	Bradykinesia Test Score (l/r)	Predominant symptoms	Medications
1	F	50	21	29/34	Moderate tremor	Sinemet, Amantadine, Bromocriptine
2	F	60	1	28/31	Moderate rigidity	Sinemet, Bromocriptine
3	F	66	9	28/25	Severe tremor	Sinemet, Bromocriptine
4	F	63	9	37/33	Moderate tremor	Sinemet
5	M	61	17	36/40	Severe rigidity	Sinemet, Artane, Bromocriptine
6	F	67	6	44/52	Moderate rigidity	Sinemet, Bromocriptine
7	M	65	8	16/18	Mild rigidity, moderate tremor	Sinemet
8	M	73	22	43/34*	Severe tremor	Sinemet
9	M	75	2	40/39	Moderate rigidity and tremor	Sinemet
10	F	60	18	43/46	Mild tremor, moderate rigidity	Sinemet, Pergolide

*Subject 8 was unable to perform the test without his finger intermittently touching the middle sector of the board (see text). The scores therefore overestimate his performance. l, left hand; r, right hand.

Table 2 Group reaction and movement times (mean values, between subject SDs in parentheses) for the three types of movement

		<i>Bimanual</i>		
		<i>Unimanual</i>	<i>Symmetrical</i>	<i>Asymmetrical</i>
RT	Controls:	388 (50)	425 (47)	463 (50)
	Parkinson's disease group:	477 (135)	514 (196)	565 (237)
MT	Controls:	247 (84)	294 (96)	341 (111)
	Parkinson's disease group:	379 (148)	473 (258)	551 (265)

RT, reaction time; MT, movement time.

schedule. Since testing took a total of about 4 hours spread over 2 days, the data represent all stages of the subjects' medication cycles.

Apparatus

The experimental task was to make lateral arm movements away from the midline under unimanual and bimanual conditions. The apparatus used for these movements comprised two yellow "home" keys (from which all movements started) and four red target keys. These were arranged in the frontal plane and were mounted on ball-bushings set into a horizontal panel so that they could only move vertically. A "snap-action" momentary contact microswitch was mounted beneath each ball-bushing shaft on the underside of the panel. This arrangement ensured that the microswitch would be closed even if the very edge of the target key were struck. The home keys were located either side of the subject's midline, with the short and long target keys centred on points 10.5 and 21.0 cm lateral to the home keys. The diameter of the home keys was 1.5 cm, while the short and long keys were 5.0 and 7.0 cm in diameter, respectively. These relatively large targets were chosen to ensure that excessive accuracy requirements did not prevent the Parkinson's disease subjects from performing the task without visual guidance. The short and long keys had indices of difficulty (IDs) of 2.07 and 2.58, respectively.⁷

Six light-emitting diodes (LEDs) were mounted on a vertical surface at eye level in front of the subject. They corresponded in colour and layout to the home and target keys, and were used to signal to the subject which movement or pair of movements to make and when to make it. The LED array spanned 8 cm, so that no saccadic eye movements were necessary in order for subjects to see which LED(s) had been illuminated. Control over the sequence of trials was undertaken with a mini-computer (LSI 11-03), as was the response timing, data collection and reduction.

Procedure

Each movement began with the subject's index fingers resting on their respective home keys. The two LEDs corresponding to the home keys were illuminated for 1 second to warn the subject that a trial was about to begin. One second later, one or two target LEDs were illuminated, depending on whether unimanual or bimanual movements were required on that block of trials. This signalled the subject to move the index finger(s) as rapidly as possible to the target(s) corresponding to the LED(s). The target LED(s) remained illuminated until a target key was struck. The subject then returned his or her finger(s) to the starting position ready for the next trial. For bimanual movements, no instructions

were given as to the simultaneity of movement initiation or termination, beyond the restriction that one hand could not complete its movement before the other began. Thus the temporal organisation of these movement pairs was spontaneous, not imposed by instructions. During the testing sequence outlined below, subjects were permitted to take rest breaks of a few minutes at approximately 20 minute intervals.

Design

Reaction time (RT) and movement time (MT) for each hand were the dependent measures. The former was defined as the interval between the response signal onset and initiation of movement as defined by the release of the home key microswitch. Movement time began with movement initiation and ended with the arrival of the finger at the target key, as indicated by switch closure. Testing was split over two sessions on separate days. On the first day subjects began by practising each of the four possible unimanual movements (left long, left short, right long, right short) and each of the bimanual movement combinations (left long and right long, left long and right short, left short and right short, left short and right long) eight times each with full vision.

The purpose of these 64 practice trials was to acquaint the subjects with the required movements and to allow them to learn the target locations before introducing the stimulus sequence, which was then explained to them. From this point on, all movements were made with the subjects wearing a visor which occluded the lower part of the visual field so that the target keys and hands could not be seen. Subjects could tilt the head downward to locate the home keys between trials and to see the extent of any error after missing a target. Four additional blocks of practice trials were then undertaken: two unimanual and two bimanual. Each consisted of 34 trials with the four possible combinations each appearing eight times in a random sequence. Two of these were catch trials in which no response signal followed the illumination of the warning LEDs. These served to discourage subjects from anticipating the response signal.

From the final practice blocks of unimanual and bimanual movements, average RTs and MTs were obtained. An upper limit was set at twice the mean RT or MT for each subject from these blocks. A trial was classified as a "slow response" error if either the RT or MT exceeded these criterion times. If a subject initiated a movement before the response signal or with a RT of less than 120 ms, the trial was designated an anticipation error. Striking the wrong target was classified as an incorrect response, and a trial was designated as a "both hand moved" error in unimanual trials if the contralateral hand also left the home key. Trials on which errors were

made were reinserted at a random point in the same block, for a total of 32 correct trials.

Eight blocks of 32 unimanual movement trials and eight blocks of 32 bimanual movement trials then followed in an alternating pattern. These were split between the first and second days of testing. The second day began with practice blocks of 10 unimanual and 10 bimanual movements which were not analysed.

RT and MT data were first averaged for each subject and for each condition and arm, and were then used in a split-plot analysis of variance. The RT and MT analyses were performed on log-transformed data because of unequal variances, but all the effects reported below (both significant and non-significant at the 0.05 level) were also found with the corresponding analyses of untransformed data. Effects involving long and short movements (Extent) are mentioned only in the context of the three levels of the factor which we call Movement Type: unimanual, symmetrical bimanual and asymmetrical bimanual movements.

Results

The results are presented in four parts. We first consider the overall reaction time and movement time data for the three movement tasks. Next we present data on the extent to which the two limbs appeared to be controlled as a single unit. Finally we consider trial-by-trial asynchrony in movement initiation and termination, and group error patterns.

Reaction and movement times

The control and Parkinson's disease subjects took longer to initiate movements in the symmetrical bimanual task than in the corresponding unimanual movements, each group showing an average decrement of 37 ms. Similarly, both groups initiated asymmetrical bimanual movements later than symmetrical bimanual pairs, by 38 ms for the controls and by 51 ms for the Parkinson's disease group (the absolute group RTs averaged across arm and extent are shown in table 2). This effect of Movement Type on RT was statistically significant ($p < 0.0001$), but there was no interaction between Movement Type and Group ($p > 0.5$), showing that in the planning of bimanual movements, Parkinson's disease subjects and controls were slowed similarly when compared to unimanual performance.

Movement times, which are also shown in table 2, were longer for both groups for symmetrical bimanual than for unimanual movements by an average of 47 ms for controls and 94 ms for Parkinson's disease subjects, and longer again for asymmetrical movements than for their symmetrical counterparts by 47 ms for controls and 78 ms for Parkinson's disease subjects. In addition, the Parkinson's disease group had significantly longer MTs than controls overall ($p < 0.05$). The Movement Type effect was significant ($p < 0.001$), but the Movement Type by

Group interaction was not ($p > 0.5$). Thus while the average MT increments were indeed somewhat greater for Parkinson's disease subjects than for controls, this trend was attributable largely to a single subject (Parkinson's disease subject 8) who was markedly slower under bimanual conditions, and to a lesser extent Parkinson's disease subject 4, and did not attain statistical significance. For the other eight Parkinson's disease subjects, the additional time needed to execute the bimanual as opposed to the unimanual movements was the same as for controls, averaging 71 ms in each case. This considerable overlap between Parkinson's disease and control subjects is clear from inspection of fig 1, in which the difference in the MTs between the three levels of the Movement Type factor are presented for each subject. (The values for subjects 8 and 4 are, respectively, the highest and second highest pairs of points on the right of fig 1.) While it seems that *some* individuals with Parkinson's disease experience an unusual prolongation of bimanual movements of the type studied here (we discuss some individual results later), it does not seem to be a general feature of the disease.

Temporal linkage between simultaneous movements

The second question posed in this study was whether Parkinson's disease subjects, like normals, spontaneously reorganise the timing of individual movements when these are performed together so that they appear to be performed as a single unit, a phenomenon we refer to as "temporal linkage". The clearest

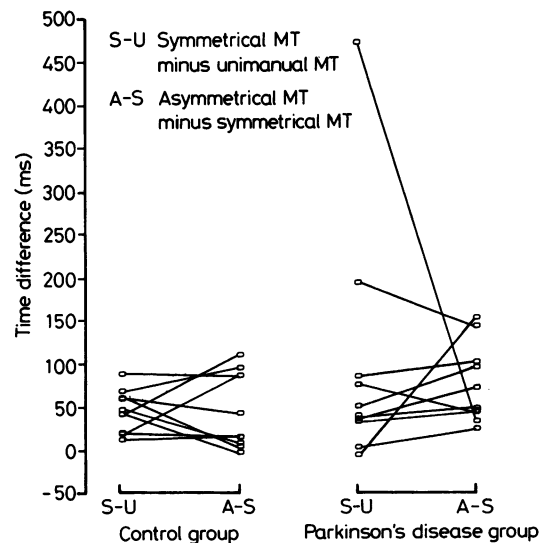


Fig 1 Differences between the movement times of individual subjects for the three movement types. Lines connect the two difference scores for each subject.

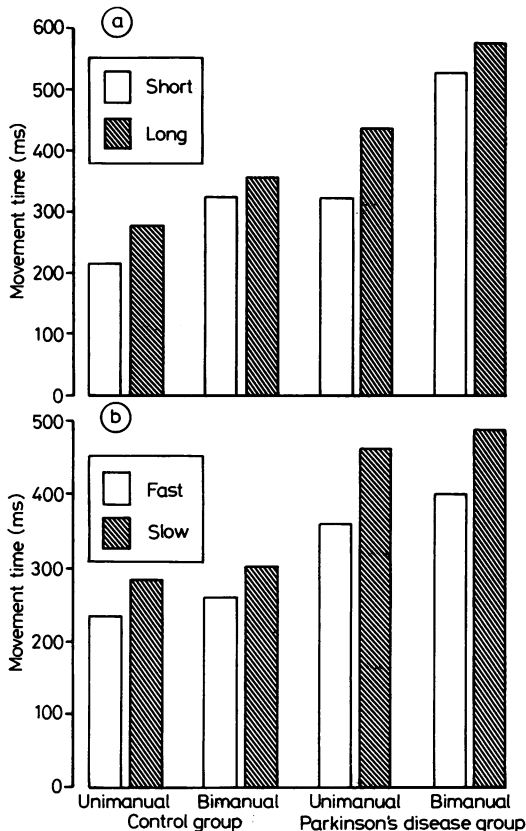


Fig 2 Group mean movement times for (a) long and short movements performed separately (unimanual) or together (bimanual asymmetrical) and for (b) movements of the same extent performed by the faster and slower arms.

way to assess this is to compare the difference between the MTs for long and short movements performed separately with those performed together. When performed separately, long movements took, on average, 63 ms longer than short movements for the control group, and 115 ms longer for the Parkinson's disease subjects. This effect for Extent was significant ($p < 0.001$), but the interaction between Group and Extent was not ($p > 0.5$). When the same pairs of asymmetrical movements were made together, however, the difference between long and short MTs was reduced to 32 ms (controls) and 49 ms (Parkinson's disease subjects) as depicted in fig 2a. This reduction was statistically significant, as shown by the interaction of Extent and Movement Type ($p < 0.001$). The magnitude of this reduction in the MTs of each hand in bimanual movements was not significantly different between the two groups, there being no Group by Movement Type by Extent interaction ($p > 0.1$). While the absolute difference in the MTs of

long and short movements performed bimanually is still greater for Parkinson's disease subjects than controls, by 19 ms, it should be noted that both the absolute and relative *reduction* in this difference from the corresponding unimanual values is greater for the Parkinson's disease group (a 70 ms or 59% reduction) than for the control group (a 31 ms or 49% reduction).

A parallel assessment of temporal linkage was also made by contrasting the MTs of the slower and faster hands for movements of the same extent. If, for example, a given subject's unimanual MT for the left hand is on average 30 ms less than that for the right hand, but the difference is only 15 ms when the movements are made together, this would provide evidence for temporal linkage in symmetrical, as well as asymmetrical bimanual movements. To test this possibility, unimanual and bimanual symmetrical MTs were compared using Group, Movement Type (symmetrical or unimanual) and Arm (fast or slow) as factors in an analysis of variance. The group MTs for this comparison are shown in fig 2b. These mean differences declined for both groups: from 26 to 18 ms in the controls, and from 40 to 26 ms in the Parkinson's disease group: a 31% and 35% drop, respectively. This reduction was statistically significant, as shown by the interaction of Movement Type and Arm ($p < 0.01$). As was the case for the previous comparison involving asymmetrical movements, there was no interaction between Group, Movement Type and Arm ($p > 0.5$), confirming that the groups did not differ in the extent of the reduction in MT differences between the faster and slower arms for symmetrical bimanual movements. Together, these results indicate that simultaneous bimanual movements to targets tend to be organised as a single unit, just as in normals.

Movement initiation and termination asynchrony

In order to determine how the two groups compared with respect to the asynchrony of movement initiation in the bimanual movement pairs, the absolute difference between left and right arm RTs for each trial in the two bimanual conditions was first obtained. (This trial-by-trial absolute difference is the most sensitive measure of movement initiation asynchrony. Any asynchrony can be obscured by taking the signed RT difference trial-by-trial, or the unsigned difference averaged over trials, unless the same arm is always the first to start moving.) These values were then averaged over trials to produce for each subject and condition a mean absolute difference between the RTs of the left and right arms, which we call the RT difference. The results are shown in fig 3, in which it can be seen that the Parkinson's disease group had almost exactly twice the RT difference of controls.

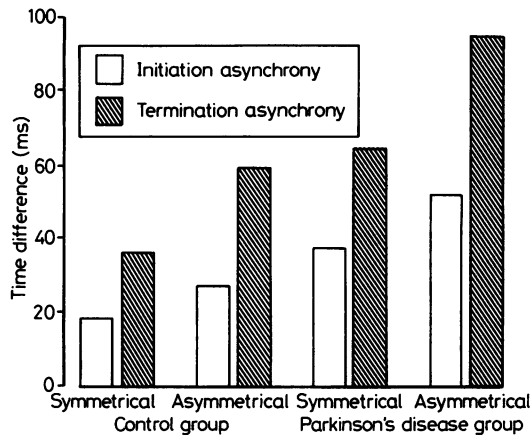


Fig 3 Group mean movement initiation and termination asynchrony.

This group difference was statistically significant ($p < 0.01$), as was the effect of condition ($p < 0.001$), in which the asymmetrical movement pairs were initiated more asynchronously than were symmetrical pairs. This increased asynchrony for asymmetrical movement pairs was not different for the two groups, however ($p > 0.5$).

Using a procedure identical to that for movement initiation, average absolute differences between the hands were obtained for total times (RT + MT). These showed that the control and Parkinson's disease subjects had greater movement termination asynchrony in asymmetrical than in symmetrical movements ($p < 0.001$), as depicted in fig 3. However, the movement termination asynchronies were not significantly larger in the Parkinson's disease group ($p > 0.1$), and there was no Group by Movement Type interaction.

Another perspective on movement initiation and termination asynchrony is provided by a correlational analysis of the relationships between RT and MT hand differences. The data already presented, showing a significantly greater degree of initiation asynchrony and a somewhat higher degree of termination asynchrony in Parkinson's disease, are based on data averaged across trials. A subject may also tend to compensate for any asynchrony in initiation by modifying the speed of execution. Within a trial, this could occur as follows: the left hand is inadvertently moved before the right by 50 ms, but is compensated for by the MT of the left hand being 50 ms longer than that of the right, ensuring synchronous termination. For perfect compensation with a gain of 1, RT differences would be offset by exactly equivalent MT differences, yielding an across-trial correlation between the two of -1.0 , and a regression line having a slope of 1.0 and an intercept of 0 . This would produce, for each sub-

ject, a series of points satisfying the equation $(RT_l - RT_r) = -(MT_l - MT_r)$ where the lower case letters denote the relevant times for the left and right hands in a bimanual movement pair.

To determine whether such a compensation occurred, the correlations described above were determined for all subject and bimanual conditions, with error trials excluded. After averaging (using Fischer's r to z transformation), the mean values were found to be -0.295 (controls), and -0.353 (Parkinson's disease group). Only one of the 20 subjects, a control, failed to have a negative correlation significant at the 0.01 level or better. While these correlations appear to provide evidence for a compensation mechanism for RT differences (which is not impaired in Parkinson's disease) the movement termination asynchrony data argue against this. If compensation were occurring, termination should be less asynchronous than initiation, and not the reverse. To probe this question further, the correlations were recomputed with the restriction that trials in which the total times for each hand differed by more than 200 ms would be excluded, so that infrequent but atypically large MT differences would not distort the data. The proportions of trials remaining in the analysis with this restriction were 89.1% (Parkinson's disease group) and 96.2% (controls). This had the effect of nearly doubling the correlations, to average values of -0.578 (controls) and -0.649 (Parkinson's disease group), which did not differ significantly from one another as assessed by analysis of variance ($p > 0.1$).

The slope of the regression lines, however, was on average less than 0.5 (0.45 for the Parkinson's disease group, 0.36 for controls, also not significantly different ($p > 0.1$)). This means that although the hand which moved first had a longer movement time, for most subjects there was *over-compensation*, with gains of more than 2 (for example, a 50 ms RT advantage for the left hand being matched by a > 100 ms MT advantage for the right). It also explains why termination asynchrony was greater than initiation asynchrony. Thus the consistently high correlations show a systematic effect for the hand which started second to catch up and "overtake" the other hand. There was great individual variation in this tendency, since in the control and Parkinson's disease groups, respectively, slopes ranged from 0.06 to 0.77 , and from 0.21 to 0.85 , representing extreme over-compensation to nearly perfect compensation. The number of Parkinson's disease and control subjects with gains greater than 2 were, respectively seven and eight (out of 10) (symmetrical, long movements), six and eight (left long, right short), seven and seven (left short, right long), and six and six (symmetrical, short movements). What this analysis revealed was the imperfect and highly idiosyncratic nature of the gain

Table 3 Group error rates for the three types of movement (percentage of all trials). (See text for definition of error types.)

	Anticipation	Slow response	Both hands moved	Incorrect response	Total
<i>Unimanual</i>					
Controls	0.8	3.1	0.6	2.2	6.7
Parkinson's disease group	2.0	3.6	0.6	3.1	9.3
<i>Symmetrical</i>					
Controls	1.1	1.8	—	0.6	3.5
Parkinson's disease group	1.6	2.9	—	2.2	6.7
<i>Asymmetrical</i>					
Controls	2.0	6.9	—	1.8	10.7
Parkinson's disease group	2.3	6.2	—	4.6	13.1

in this apparent compensation mechanism, as well as the fact that it was quite unaffected in Parkinson's disease.

Error patterns

The patterns of errors made by each group are shown in table 3. Parkinson's disease subjects made a slightly higher proportion of errors overall, but these were not distributed very differently from controls. It is notable that both groups made more errors on asymmetrical trials, most commonly trying to make an inappropriate symmetrical movement.

Discussion

Given the previous reports of impaired simultaneous movements in Parkinson's disease^{1 9 17 20 21} the overall similarity of performance between the Parkinson's disease subjects and controls is striking. In general, the requirement to perform a pair of movements did not differentially affect the Parkinson's disease subjects. Both groups took longer to prepare movements when a second symmetrical movement was introduced, with a greater increase if the movement pair was asymmetrical. Similarly, both groups executed the movements more slowly under these conditions. The planning and execution of bimanual movements of this type was therefore more demanding for both controls and Parkinson's disease subjects than were unimanual movements, but with no clear differential slowing for Parkinson's disease subjects.

The time course of unimanual movements was modified by both Parkinson's disease and control subjects when it was combined with a movement of the other limb, in the same way as had previously been described for young adults,¹⁰⁻¹² so that the strong association between MT and target size and distance no longer applied in the bimanual situation, that is, both groups appeared to control the bimanual movements as a single unit. These results are also in agreement with those of Marteniuk *et al.*,¹⁵ in that asynchronies still persist despite the tendency towards synchronisation of bimanual movements.

Another mechanism which was unaffected by Parkinson's disease was the consistent "catch-up" of the

hand which started second. Despite the variety of gains shown by individuals, with a majority in each group "over-compensating", this tendency was unchanged in the Parkinson's disease group. That such a subtle effect was equally present in both groups argues strongly against any general deficit of bimanual simultaneous movements in Parkinson's disease, although, as we shall argue below, certain types of simultaneous tasks may be far more susceptible to disruption than that used here.

Only in movement initiation asynchrony were the Parkinson's disease subjects impaired to a degree which achieved statistical significance. Even here the impairment was not large: in the order of an additional 25 ms difference between the hands. This effect may not necessarily reflect any impairment in the central planning of the movement: for example, the presence of tremor could alter the timing of switch openings as each hand left the home key if it were out of phase in the two limbs or if it were present in only one.

There have recently been several reports of functions which (contrary to earlier views) are relatively intact in Parkinson's disease. It has been shown, for example, that the use of advance information in planning discrete movements is slow but remains intact in Parkinson's disease,¹⁸ although the control of movement sequences shows some abnormality.¹⁹ In a related area, earlier speculation about abnormal visuospatial functioning in Parkinson's disease appears to have been discounted.²⁶ Our understanding of Parkinson's disease is furthered by the careful delineation of both what is normal and what is abnormal, and in the case of simultaneous movement, we will argue that deficits will not be seen in all simultaneous movement, rather they may be present in the simultaneous performance of *different* actions, but not present when a common timing element is shared by two similar movements. Before further outlining this distinction, we will first consider some individual results.

We can offer no clear explanation for the atypical slowness in the bimanual MTs of Parkinson's disease subjects 8 and 4, but emphasise two points. First, their MT patterns are dissimilar and hint at different

types of problems in these individuals. Note that, as shown in fig 1, subject 8, the most severely affected patient in our sample, was mostly slowed in the transition from unimanual to bimanual movements. Subject 4, who was not as profoundly affected by the disease, showed an unusually large decrement in asymmetrical movements as well. Secondly, we draw attention to the fact that even these subjects were not selectively impaired in other aspects of bimanual movement control. Both had converging MTs for asymmetrical movements of different extents, and converging MTs for symmetrical movements of the same extent for the "fast" and "slow" arms, compared with the corresponding unimanual values. The phenomenon of temporal linkage was therefore present in these cases. In addition, they both showed significant negative RT difference/MT difference correlations.

Like previous investigators, we used decreasing speed of movement as a principal test for the relative effects of bimanual movements on the two groups. It is therefore important to note that our Parkinson's disease group did show evidence of bradykinesia, suggesting that the absence of differential slowing effects was not due to the absence of this symptom. In both the bradykinesia test and the MT data, the Parkinson's disease subjects were reliably slower than controls, by 27% in the bradykinesia test, by 50% in short unimanual movements, and by 57% in long unimanual movements. As noted by Warabi, *et al.*,²² short hand movements have the same durations as those of controls in mildly bradykinetic patients, and are longer only in those with more prominent bradykinesia. Our data indicate that the presence of bradykinesia may not be associated with difficulties in simultaneous movements, at least of the type studied here.

How may the disagreement between our findings and those of previous investigators best be accounted for? In large measure it may reflect important differences between the task used in the present study and those used previously.^{1 9 17 20 26} If two tasks to be executed simultaneously are dissimilar and repetitive, performance will be affected by other than purely motor processes. Their similarity to real-life actions make these useful tasks for study, but their interpretation is not unambiguous. As Benecke *et al.*¹ have pointed out, in each of these earlier studies one task probably required more attention than the other. For example, Schwab *et al.*¹⁷ reported on the performance of repetitive ergograph bulb-squeezing with one hand while the subject traced triangles with the other hand. The Parkinson's disease subjects decreased the frequency and amplitude of the squeezing movement, and also tended to perform the two tasks sequentially rather than simultaneously. If Parkinson's disease

subjects have more difficulty with "automatic" movements¹⁴ then the differential attention demands of the tasks could have been the underlying cause of the deficit. The same process may also have occurred with bead-transfer and counter-pressing,^{20 21} and bead-transfer and tapping.⁹

A related difficulty in interpreting data from such disparate and repetitive movements, some of which were performed for as long as 60 seconds, is that Parkinson's disease subjects may have attempted to "time-share" between the tasks, and were slowed because of a difficulty in shifting from one set of task criteria to another, and then back again. This deficit is not necessarily confined to motor processes: on several cognitive tasks Parkinson's disease subjects have been shown to have significantly greater difficulty in shifting cognitive sets.^{4 8 13}

Discrete movements of the limbs do not present the same difficulties, so it is still necessary to reconcile our findings with those of Benecke *et al.*¹ who also used rapid and discrete movements and report significant deficits in their Parkinson's disease subjects. The apparent disparity is much reduced when our data is compared with their bimanual simultaneous tasks. Although still present, the reported deficit was far smaller when different limbs were used for simultaneous movements. We suggest that simultaneous bimanual aiming movements are particularly amenable to common temporal regulation: the two limbs are constrained to act as a single unit, as suggested by Kelso.^{10 11} This common regulation may take the form of the two limbs being governed by a single motor programme, or by distinct motor programmes which are readily integrated. Since the term "motor programme" is given differing interpretations, however, the distinction between these alternatives may be largely a semantic issue. Nevertheless, following the logic of Benecke *et al.*,¹ there would be no impairment if one programme does not have to be superimposed on another. In this instance, however, there is some evidence that the movements were not simply directed by a motor programme and executed in an open-loop fashion. Despite the fact that most subjects over-compensated for initiation asynchrony, the consistent "catch-up" of the limb which moved second suggests that some form of closed-loop control may have been in effect during execution, delaying the first hand to move and/or speeding up the second. It seems improbable that such effects could be fully programmed before movement initiation.

Data from other studies also tend to support the idea that the potential for common temporal regulation may be a factor in determining whether individuals with Parkinson's disease find simultaneous movements problematic. Perret¹⁶ observed that both controls and Parkinson's disease subjects performed

bimanual reciprocal tapping more slowly than unimanual tapping, but the extent of this slowing was only marginally greater for the Parkinson's disease subjects. In this task a common timing mechanism might have permitted the two limbs to be controlled as a single unit. While there are many possible criteria of task similarity which could be used to predict the degree of simultaneous movement impairment in Parkinson's disease, the potential for imposing a common timing structure on the movements seems a plausible one. Support for this notion also comes from the work of Cohen,³ who showed that Parkinson's disease subjects, like normals, tended to spontaneously reorganise out-of-phase bimanual wrist pronation and supination into in-phase, symmetrical movements pairs using homologous muscles. Moreover, there is some evidence that bimanual movements involving non-homologous muscles may also be combined with a speed decrement little greater than that of controls, since Perret's¹⁶ Parkinson's disease subjects performed parallel reciprocal tapping with no more difficulty than when using homologous muscles in symmetrical reciprocal tapping.

We conclude that the phenomenon of impaired simultaneous movements in Parkinson's disease is not universal, but is task-dependent. Our data, and that of Benecke *et al*¹ are in agreement that the impairment is less in bimanual movements than when the two tasks are performed by the same limb. There must be other features of the tasks, however, which determine the extent of the deficit. Elucidating these crucial task characteristics may provide more insight not only into Parkinson's disease motor deficits but also into the role of the basal ganglia in organising movement.

This work was supported by funding from the National Institute of Neurological Diseases and Stroke, grant number NS17421—US Public Health Service, awarded to the first author. The authors are grateful to Paul Amrhein for assistance with statistical analyses and comments on an earlier draft.

References

- Benecke R, Rothwell JC, Dick JPR, Day BL, Marsden CD. Performance of simultaneous movements in patients with Parkinson's disease. *Brain* 1986;**109**:739–57.
- Brown RG, Marsden CD. Visuospatial function in Parkinson's disease. *Brain* 1986;**109**:987–1002.
- Cohen L. Interaction between limbs during bimanual voluntary activity. *Brain* 1970;**93**:259–72.
- Cohen L. Synchronous bimanual movements performed by homologous and non-homologous muscles. *Perceptual and Motor Skills* 1971;**32**:639–44.
- Cools AR, Van den Bercken JHL, Horstink MWI, Van Spaendonck KPM, Berger HJC. Cognitive and motor shifting aptitude disorder in Parkinson's disease. *J Neurol Neurosurg Psychiatry* 1984;**47**:443–53.
- Della Salla S, Dilenzo F, Giordano A, Spinnler H. Is there a specific visuo-spatial impairment in Parkinsonians? *J Neurol Neurosurg Psychiatry* 1986;**49**:1258–65.
- Fitts PM. The information capacity of the human motor system in controlling the amplitude of movement. *J Exp Psychol* 1954;**47**:381–891.
- Flowers KA, Robertson C. The effect of Parkinson's disease on the ability to maintain a mental set. *J Neurol Neurosurg Psychiatry* 1985;**36**:742–6.
- Horne DJ de L. Sensorimotor control in Parkinsonians. *J Neurol Neurosurg Psychiatry* 1973;**36**:742–6.
- Kelso JAS, Southard DL, Goodman D. On the nature of human interlimb coordination. *Science* 1979;**203**:1029–31.
- Kelso JAS, Southard DL, Goodman D. On the coordination of two-handed movements. *J Exp Psychol [Hum Percept]* 1979;**5**:229–38.
- Kelso JAS, Putnam CA, Goodman D. On the space-time structure of human inter-limb coordination. *Q J Exp Psychol* 1983;**35A**:347–75.
- Lees AJ, Smith E. Cognitive deficits in the early stages of Parkinson's disease. *Brain* 1983;**106**:257–70.
- Marsden CD. The mysterious motor function of the basal ganglia: The Robert Wartenburg Lecture. *Neurology* 1982;**32**:514–39.
- Marteniuk RG, Mackenzie CL, Baba DM. Bimanual movement control: Information processing and interaction effects. *Q J Exp Psychol* 1984;**36A**:335–65.
- Perret E. Simple motor performance of patients with Parkinson's disease before and after a surgical lesion of the thalamus. *J Neurol Neurosurg Psychiatry* 1968;**31**:284–90.
- Schwab RS, Chafetz ME, Walker S. Control of two simultaneous voluntary motor acts in normals and in Parkinsonians. *Arch Neurol Psychiatry* 1954;**72**:591–8.
- Stelmach GE, Worringham CJ, Strand EA. The planning and execution of movement sequences in Parkinsonians. *Int J Neurosci* 1987 (in press).
- Stelmach GE, Worringham CJ, Strand EA. Movement preparation in Parkinson's disease: The use of advance information. *Brain* 1986;**109**:1179–94.
- Talland GA. Manual skill in Parkinson's disease. *Geriatrics* 1963;**18**:613–20.
- Talland GA, Schwab RS. Performance with multiple sets in Parkinson's disease. *Neuropsychologia* 1964;**2**:45–53.
- Warabi T, Noda H, Yanigasawa N, Tashiro K, Shindo R. Changes in sensorimotor function associated with the degree of bradykinesia of Parkinson's disease. *Brain* 1986;**109**:1209–24.