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THE PREPARATION AND PRODUCTION OF ISOMETRIC FORCE IN PARKINSON'S DISEASE

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Abstract—Subjects with Parkinson's disease (PD) and age-matched controls performed an isometric force production task, aiming at different target force levels without concurrent force feedback. Overall, PD subjects were as accurate as controls in attaining the target force levels, but executed the task differently. They had longer times to peak force and contraction durations, larger impulses and lower rates of force development, and force–time profiles with many more irregularities. They also initiated lower force contractions with longer latencies, unlike controls. The data suggest that PD subjects are deficient in the regulation of force and time parameters, rather than simply in force production. The ability to produce peak forces accurately limits the generality of previous assertions that PD subjects are heavily dependent on concurrent visual feedback.

INTRODUCTION

STUDIES OF motor abnormalities in Parkinson's disease have generally used tasks requiring overt, isotonic movements, whether they have focused on higher level processes (e.g., motor planning and co-ordination of movement [4, 9, 21, 25, 26]), or on peripheral manifestations of the disease (e.g., EMG characteristics [2, 15]). In this paper we describe the performance of PD subjects and controls in producing target peak forces by isometric contraction of the elbow flexors. An isometric task has some interesting properties relevant to the study of motor function in Parkinson's disease. First, since the goal of the task is to achieve a specific *peak* force level, there is some independence of force and time parameters. The isotonic analogue of this task would be to produce a given peak acceleration. The only constraint on the forces preceding this peak is that they do not exceed the target force level specified for that particular trial. In isotronic movements, on the other hand, the position and velocity of the limb at any given time are not the products of the force being generated at that moment, rather, they reflect the net effect of the forces applied to the limb up to that point. In Parkinson's disease, an impaired ability to generate a desired level of force could explain the tendency for those with the disease to undershoot targets with the initial movement [2, 3], or to do so with insufficient velocity, which BERARDELLI *et al.* [2] have described as a "breakdown in the link between the perceptual appreciation of what is needed and the delivery of the appropriate instructions to the motor cortex". On the other hand, the same phenomena could result from impaired regulation of force *over time*, rather than from force levels *per se*, so that an inappropriate impulse is generated. An isometric task allows these

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alternatives to be evaluated, since a pure force production deficit should be manifest in decreased accuracy relative to a control group.

A second distinctive aspect of isometric force production is that it cannot be controlled using concurrent visual feedback, since there is no movement, and hence no error signal related to position or velocity as is present in isotonic movements. Several authors have argued that an increased reliance on visual feedback is a principal characteristic of Parkinson's disease. FLOWERS [11, 12, 13] showed, for example, that PD subjects are deficient in the conduct of visual "open-loop" movements—both discrete and continuous, thereby implying that one aspect of basal ganglia function is in the regulation of movements which are either preprogrammed, and therefore independent of feedback, or which may be guided by an internal representation of the target. COOKE *et al.* [7] also argued that there is an increased dependence on visual information for the control of movement in Parkinson's disease based on results from a tracking task using movements about the elbow. STERN *et al.* [28, 29] found that PD subjects were impaired in completing the missing segments of regular (e.g., "saw-tooth") patterns, and suggest that they either cannot generate an appropriate motor plan, or that they cannot execute it correctly, and that this deficit is particularly evident when external guidance from the environment is removed. In eye movement research BRONSTEIN and KENNARD [5] cite a reliance on visual feedback as a partial explanation for the decreased frequency of anticipatory saccadic eye movements to predictable targets in Parkinson's disease, in contrast to the relative normality of (closed-loop) smooth pursuit movements. In addition, FRITH *et al.* [14] conclude that PD subjects will be excessively reliant on feedback in the early stages of performance on a novel task. If this visual feedback dependence is a general phenomenon, the PD subjects should be at an additional disadvantage when confronted with a novel task in which visual feedback is neither available nor informative.

Another motive for studying an isometric task was that some previous studies had suggested that there may be abnormal force production in PD. In one, PD subjects showed greater instability in maintaining a given force with lip, tongue or jaw muscles, even though the attained and target forces were displayed on an oscilloscope [1]. Two experiments in our laboratory had also hinted at difficulties in producing appropriate force in finger-tapping sequences. In the first, PD subjects showed an abnormal prolongation of the first inter-tap interval in the repetitive tapping of a single finger. In conjunction with this, response latency increased linearly with sequence length in the PD group but not in controls [27]. One possible explanation for these effects is that the requirement to initiate the sequence rapidly caused PD subjects to produce inappropriate excessive force on the force tap, thereby extending the "dwell" time on the response key and elongating the initial inter-tap interval. In the second experiment, PD subjects were slower in initiating five-tap sequences which included a stress on one of the taps, than sequences of the same length which did not require a stress [24]. The introduction to the task of a differential force component apparently slowed preparation. Since the force produced on the stressed tap had only to be greater than that of the other taps rather than to a specified level, no direct assessment of force production characteristics was possible.

The experiment described here was therefore designed to determine if PD subjects have a "pure" deficit in force production (i.e., the production of a peak force rather than the production of a force–time pattern), to test the generalizability of the assertion that they are excessively dependent on concurrent visual feedback, and to permit some description of the preparation and execution characteristics of this isometric task. Based on observations made

in previous studies, we hypothesized that PD subjects would be slower in the preparation of low force contractions, since PD patients often report difficulty in initiating movements unless there is a large enough external stimulus. We also expected that PD subjects would have particular difficulty when high precision was required, since it has been previously shown that they are impaired in the production of high precision isotonic movements [e.g., 11, 23].

METHODS

Subjects

Seven PD subjects and seven control subjects were used. The Parkinson's disease group had a mean age of 62.4 yr (SD: 7.4), while controls averaged 63.5 yr (SD: 7.9). There were four women and three men in the PD group, and three men and four women in the control group. PD subjects had been diagnosed as having Parkinson's disease, but no other neurological disease, and all were taking medication at the time of the study. A profile of the PD subjects is given in Table 1, including HOEHN and YAHR scores [16]. Subjects followed their normal schedule of medication during testing, but times of testing were chosen to represent the end-of-dose period as much as possible. Control subjects were free from any signs or symptoms of neurological disease. Subjects were paid for their participation.

Table 1. Profile of Parkinson's disease subjects

Subject number	Age (yr)	Duration of disease	Hoehn and Yahr	Predominant symptoms	Medication
1	65	8	III	Mild rigidity Moderate tremor	Sinemet
2	61	27	IV	Severe rigidity	Sinemet Artane Bromocriptine
3	63	9	II	Moderate tremor	Sinemet
4	73	22	IV	Severe tremor	Sinemet Amantadine
5	67	6	III	Moderate rigidity	Sinemet Bromocriptine
6	59	17	III	Moderate rigidity Mild tremor	Sinemet Pergolide
7	49	20	II	Moderate tremor	Sinemet Amantadine Bromocriptine

Apparatus and subject position

The apparatus consisted of a strain gauge force transducer (Interface SSM 500) attached to a rigid, wall-mounted shelf at shoulder level on the PD subjects' more affected side, or the control subject's non-dominant side. A vertically aligned plastic plate was bolted to the strain gauge, making contact with the palmar surface of the subject's wrist (at the level of the carpal bones). The subject rested on the upper arm and forearm on the shelf on a padded surface, with the arm in abduction at shoulder height. The subject's elbow was at approximately ninety degrees of flexion, and approached full supination so that the thumb was uppermost. Isometic elbow flexion in the horizontal plane, with an attempt to bring the palm in toward the trunk, led to the development of force in a direction along the recording axis of the force transducer. Chair position and height was adjusted to ensure correct positioning of the subject.

The force transducer output was directed via an amplifying circuit and A/D conversion board to a PDP 11-73 mini-computer, which controlled the experiment and recorded force data at 500 Hz. The force output was linear throughout the range of interest as determined from a calibration procedure in which known-masses were suspended from the transducer via a pulley. The following measures were recorded from each trial: reaction time (msec), duration of contraction (msec), absolute value of peak force (N), relative value of peak force (%), impulse, in Newton-seconds (N.s), time to peak force (msec), and average rate of force development from initial force increase until peak force—in Newtons per second (N/s). In front of the subject was a CRT, which provided the subject with stimulus information prior to each trial and knowledge of results (KR) following it.

Procedure

Following a description of the procedures, each subject gave written informed consent, and was then familiarized with the task by means of a demonstration. After subjects were appropriately seated, the experiment commenced. Each trial began with the subject's wrist lightly touching the force transducer. Blocks of 39 trials commenced with an assessment of maximal flexor force, in which the subject was required to develop maximal force against the transducer in a contraction lasting between 2–4 sec. This procedures was repeated for a total of three trials, with the highest value serving as an estimate of maximal force for use in the remaining 36 trials.

The subject's maximal force was scaled to 100%, and on all subsequent trials targets were presented as a percentage of maximum. On average, the PD group produced lower forces during maximal force contractions. Group means for maximal force were 71.9 N and 96.6 N for Parkinson's disease and control groups respectively, a difference which was not statistically significant [$F(1, 12) = 1.34$, $P > 0.3$], but was similar to the difference between controls and PD subjects previously reported [18].

The procedure for the remaining "aiming" trials was as follows: a graph was displayed on the CRT in front of the subject, with the ordinate ranging from 0 to 100%. An auditory warning signal (two "beeps") coincided with the display of a target, which comprised two horizontal lines intercepting the ordinate, and between which the subject was required to aim the peak force of the subsequent contraction. The target was centered on one of three force levels (25, 50, 75% of maximum), and had one of the following widths: 10, 20, or 30%. After a random interval of between 1.7 and 2.3 sec, a high-pitch auditory response signal was given. On hearing this, the subject had to generate a rapid contraction of sufficient magnitude so that the peak force would lie between the two horizontal target lines which defined the target force width. Subjects were told to be accurate, and that responses which fell in the center of the target were not considered better than those just within it. They were also instructed to perform the contraction as a single "pulse" of force as rapidly as possible following the response signal, following which forces were samples for 6 sec. Quantitative knowledge of results was then presented to the subject in the form of a histogram bar representing the attained peak force superimposed on the target display. Subjects could then readily see whether the force was within the target, or whether too much or too little force had been generated.

Reaction time was measured by the first detectable increase in force above the threshold, which was determined as the maximum force recorded during a 200 msec period when the subject's arm was at rest before the beginning of the trial. This procedure proved sensitive to force increases, yet allowed for minimal force fluctuations produced by physiological or resting tremor (the latter was largely damped by friction between the arm and support surface). Reaction times of less than 130 msec or of more than 1200 msec were designated anticipation errors and late responses, respectively, and led to an error message being generated at the terminal so that subjects would be aware of the nature of the error.

Design

The 36 aiming trials on each block comprised two sets of 18 conditions, structured in factorial design. The factors were: target force (three levels), target force width (three levels) and repetition (two levels). The last factor was produced by repeating each combination of target force and width on the next trial, before randomly switching to another combination.

Each subject undertook a total of 10 blocks spread over 2 days, for a total of approx. 4.5 hr of testing, including rest breaks after each block. Data were therefore gathered for a total of 16 trials in each of 18 experimental conditions for each subject. The first block for each day was designated a practice, and was not analysed.

Data were analysed in the following manner: summary statistics were obtained for each of the 18 conditions for each subject. These mean values were then used in a four-factor split-plot factorial analysis of variance. One-tailed tests were used to evaluate hypotheses with directional predictions.

RESULTS

Response initiation

The hypothesis that PD subjects would initiate responses more slowly for low target forces than for high target forces received support, and contrasted with the performance of controls. The decreasing mean RT for PD subjects is evident for both first and second repetitions (Fig. 1). The group by force level interaction was significant [$F(2, 24) = 3.00$, $P < 0.05$], being especially evident in repetition one trials [$F(2, 24) = 4.2$, $P < 0.03$]. The slight tendency towards an opposite trend in the control group did not achieve statistical significance.

While there was a slightly decreasing mean RT for PD subjects when the target force width was high (397, 386 and 384 msec for 10, 20 and 30% target force widths), and a marginally increasing value for controls (360, 360 and 365 msec) the greater accuracy constraints did not

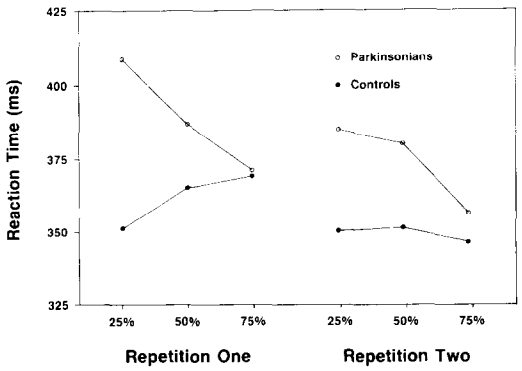

FIG. 1. Reaction time by group and force level for (a) repetition 1, (b) repetition 2.

slow PD subjects' response preparation disproportionately, as shown by the absence of any interaction between group and target width for RT [$F(2, 24)=0.11$, $P>0.8$]. In one other respect the groups were also similar, the second repetition of each target pair was initiated more quickly than the first by both groups: 16 msec faster by the PD group, 13 msec faster by controls [$F(1, 12)=13.8$, $P<0.005$].

Accuracy

There was no overall difference between the accuracy of both groups, measured either by mean hit rate (proportion of response inside the target) or by the mean force levels attained. PD subjects hit the target on 59.3% of trials, controls on 60.3%. Lower hit rates were recorded for both groups for narrower target, as expected. For 10, 20 and 30% target widths the proportions of hits were: 34.9, 62.2, 83.9% (controls) and 33.3, 63.0, 81.4% (PD group). These results are shown in Fig. 2. The proportion of hits increased significantly as the target

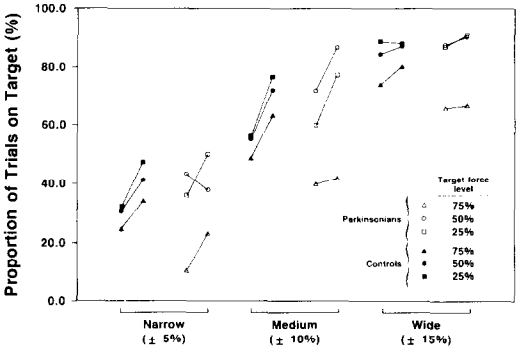

FIG. 2. Hit rates by group, target force level, target width, and repetition (connected points are first and second repetitions of force level target width combinations).

force decreased, for both groups [$F(2,24)=16.1$, $P<0.001$] as is apparent in Fig. 2. This was expected, since any given target force with decreases relative to the peak force produced as the target force level increases. In addition to this effect, a group by target force level interaction is present, indicating a lower PD group hit rate for the 75% target, but not for

the 25% target [$F(2, 24) = 4.2$, $P < 0.03$]. The lack of a significant group by force level by target width interaction, for the peak relative force measure [$F(4, 48) = 0.77$, $P > 0.5$] also showed that both groups were affected similarly by target width. Overall, PD subjects were able to achieve the appropriate peak forces no less accurately than controls.

Both groups produced peak relative forces which tended towards the mean, a classic range effect in which high targets were undershot and low targets were overshot, as is clear in Fig. 2 and was somewhat stronger for the Parkinson's disease group. The latter undershot the high force targets by nearly 6% more than did the controls, but overshot the low force targets by an overage of 1% *less* than controls. The significant main effect for group on the peak relative force measure [$F(1, 12) = 9.2$, $P < 0.02$] showed that, overall, PD subjects produced slightly lower forces relative to their maxima, averaging 3.7% less. The lack of significant group by force level by target force width interaction for the peak relative force measure [$F(4, 48) = 0.77$, $P > 0.5$] also showed that both groups were affected similarly by target width. Overall, PD subjects were able to achieve the appropriate peak forces no less accurately than controls.

Both groups showed evidence of a range effect, with peak relative forces tending to approach the mean. This effect was moderated by target force width for both groups. For the 75% force level, subjects tended to undershoot less for the wider targets. For the 25% force level, they overshot less for the wider target, as shown in Fig. 3—by statistically significant interaction [$F(4, 48) = 10.71$, $P < 0.001$].

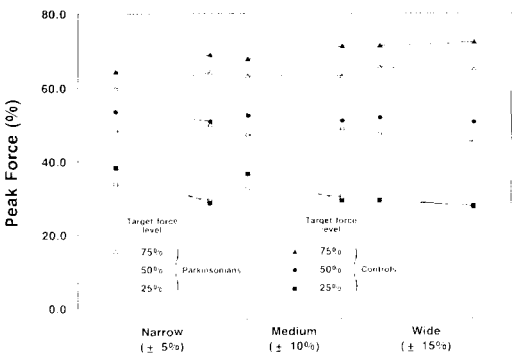

FIG. 3. Peak relative force by target force level, target width and repetition (connected points are first and second repetitions of force level target width combinations).

Execution characteristics

The average time to peak force was longer in the PD group, as was the average duration of each contraction. The mean values for the Parkinson's disease and control groups was 577 msec and 255 msec (time to peak force) [$F(1, 12) = 4.52$, $P < 0.06$]; and 1160 msec and 561 msec (duration) [$F(1, 12) = 594$, $P < 0.05$]. PDs had significantly lower average rates of force development: 77.5 N/s as opposed to 222.5 N/s for the control group [$F(1, 12) = 9.87$, $P < 0.01$]. Impulses were larger for the Parkinson's disease group, but the difference did not attain statistical significance, since their increased durations were partially offset by lower average forces. The mean impulses were 23.86 N.s and 15.85 N.s for PD and control groups, respectively [$F(1, 12) = 1.01$, $P > 0.3$].

Representative force–time curves from each of the groups are shown in Fig. 4. In addition to the characteristics described above, two additional features may be noted. The first is the heterogeneous shapes of the PD subjects' curves, which vary from the near-normal (Subject 7)* to profoundly impaired (Subject 1), a range which is especially obvious in the increased durations. A second point is the presence of irregularities in the PD data. Some of these may be ascribed to action tremor [10, 30], which tends to be apparent as the force reaches its maximum for any given trial. Others may represent a difficulty in performing the task as a single movement, such problems are absent in the control group's curves.

DISCUSSION

The requirements of this task were twofold: (1) to attain the target peak forces; and (2) to do so with rapid and discrete contractions. Judged by the criterion of peak force accuracy, the PD group was not impaired relative to age-matched controls. The results suggest that the difficulties manifest in overt movement are not directly attributable to the inability of PD subjects to produce a desired force level. Thus the hypothesized breakdown in the link between "perceptual appreciation" of the goal and "delivery of the appropriate instructions" [3] or the "difficulty . . . in coordinating the motor and perceptual activity" [29] seem not to apply in the case of achieving a given peak force level.

The second task requirement, that the peak force be attained with rapid and discrete contractions, was fulfilled by controls far more satisfactorily than by PD subjects. Indeed, the execution characteristics of the groups differed substantially, primarily in the time domain. Longer times to peak force and overall durations were apparent, with a consequent increase in the size of impulses for equivalent absolute force levels. Subjects were carefully instructed to make the responses as rapidly as possible and to do so with smooth increases and decreases in force, but most subjects in the PD group were not able to accomplish this. There are two possible causes for this slowness. First, there may be an inherent limitation in the rate at which PD subjects can develop force—the isometric equivalent of bradykinesia. Indeed, rates of force development were substantially slower than in controls. A second possibility, which we will discuss later, is that PD subjects employed a different speed–accuracy trade-off strategy.

A major difference between overt and isometric aiming is that reaching a peak force is independent of timing parameters such as duration and impulse, while the attainment of a given displacement is not: force and timing parameters must be co-regulated to achieve the requisite impulse. A deficit in regulating force and time parameters is consistent with our earlier finding that sequences with a differential stress take more preparation than unstressed sequences, with a loss of rhythm following the stressed tap [25] and with previous reports of an inability for some PD subjects to maintain a specific rhythm in tapping [20]. The deficit in the time domain is also consistent with the suggestion that PD subjects are deficient in timing, especially at the level of an internal timekeeper [32]. Our data suggest that the basal ganglia may be involved in the co-regulation of timing and force development, but not with the accuracy of peak force attainment. While our data suggest that the PD subjects may achieve the appropriate peak force required by a task accurately, many patients would not be able to regulate the time course of the force production well enough to stop the movement where intended. That is because there are very few real-life acts which can be characterized by

*PD Subject 7 also had irregular force time profiles on a minority of trials.

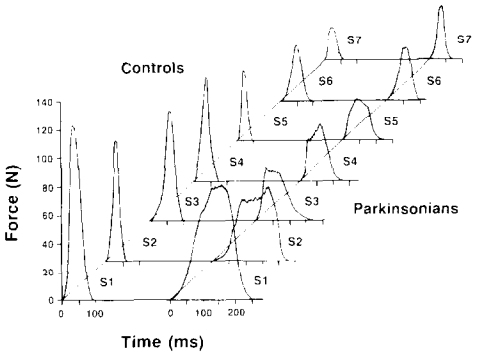

FIG. 4. Representative force time curves for members of Parkinsonian and control group.

force attainment criteria independent of timing. There is an important timing element even in those tasks which have an objective peak force criterion, such as sliding a heavy object from one location to another on a table, in which the force necessary to overcome friction is an objective task characteristic. Most tasks demand the production of an appropriate force–time *pattern*.

The Parkinson's disease group accuracy decrement was greater than that of controls for the 75% force level (the isometric equivalent of an increased movement extent), but there was no target width effect. In contrast, SANES [23], found that PD subjects were disproportionately less accurate when *either* a given target width was combines with a longer movement extent, *or* when a given movement extent was made to a narrower target. Since, in addition to the differences already noted between pure force production and actual movement, Sanes used a reciprocal task. We have frequently noted that reciprocal tapping movements made by our subjects tend to become disrupted after several seconds, while discrete movements are not affected as severely.

The findings that SD subjects produced accurate peak forces in the absence of visual feedback argues against the generality of previous assertions that they are critically dependent on such feedback [7, 11, 14]. It seems that in some circumstances, PD subjects can indeed form and utilized "an internal model" of the task, although this has been put forward as a specific deficit in certain perceptual-motor tasks [28, 29]. The accuracy achieved by the PD group suggests either than an alternative, non-visual form of force feedback was available, or that they could guide the contractions on the basis of some form of efference copy. CORDO [8] has shown that the torque generated in the first 100 msec of a similar task performed by young normals is highly correlated with the target torque, and is unaffected by visual feedback if the target was known in advance. Early adjustments made to torque production in trials where the target was unknown before the response appear to have been mediated by corollary discharge on kinesthetic feedback from the limbs, since visual based connections were not seen before 200 msec. In our experiment, the unavailability of visual feedback suggests that any connections must have been based on corollary discharge and/or kinesthetic input. It is possible that PD subjects used such kinesthetic information more than controls, and therefore required more time to complete the contractions. Preferential use of such afference may thus have required a different speed-accuracy trade-off for the PD group, as alluded to earlier. Logically, however, it seems that an accurate "internal model" of the required forced must have been available to the PD subjects, since, even if such force-related

afference were used, the appropriate force level had to be inferred from a purely visual stimulus.

Both groups were able to use the knowledge of results provided at the end of each trial to approach the target more closely on the second repetition. This is evident from the increase in hit rates from repetition one to repetition two (Fig. 1) [$F(1, 12) = 19.71, P < 0.001$]. Controls increased from an average of 55.0% hits on repetition one (average across target force levels and widths) to one of 65.7% on repetition two. The corresponding increase for the PD subjects was from 53.3 to 62.4%. There was no interaction between group and repetition [$F(1, 12) = 0.8, P > 0.3$] for error rate.

Little is known about motor learning in Parkinson's disease, partly because most patients are of an age at which new skills are rarely acquired. Our data suggest that PD subjects can use knowledge of results at the end of the preceding trial to improve performance on the same target, and also to initiate the contraction with a shorter latency. This latter finding is reminiscent of the sequential effects described by THEIOS [31] and may indicate that PD subjects, like normals, retain the commands for the previous response in a "memory buffer", taking less time for their retrieval on the next trial, even though the commands are modified. The improvement between the first and second repetitions does not necessarily reflect learning, but rather the type of improvement discussed by SALMONI *et al.* [22], in which KR acts as guidance for short-term improvement in performance. As such, these results are at odds with those of FRITH *et al.* [14], who concluded that PD subjects "are markedly impaired in the temporary component (of learning)". Their tasks were more complex, however, including the use of a spatially incompatible tracking task.

The Parkinson's disease group showed a target-force RT relationship quite different from that of the controls. While the latter had stable or slightly increasing latencies for high forces, the former had higher latencies for *lower* forces. The significance of this interaction depends on when the delay occurs. If it were exclusively in motor time (i.e., the interval between onset of the EMG agonist activity and the first increase in force), the effect would be less interesting than if it were manifest in the premotor time (i.e., the interval between the response signal and EMG onset). In the former case, it would suggest a slowing in central processing associated with low level force preparation in Parkinson's disease. This delayed movement initiation would be compatible with certain clinical observations noted in Parkinsonism, such as kinesia paradoxica, a phenomenon in which suitable external stimuli can allow the PD subjects to make rapid gross movements when the same movement can be self-generated only with great difficulty (particularly in locomotion [19]). A common explanation for these phenomena might be in a deficit of an activation mechanism associated with movement [33]. By "activation" we mean the attainment of a threshold level of neural activity below which no movement is actually elicited. The obtained rection time data are compatible with the notion of defective activation, since the higher is the required force level, the greater is the likelihood that sufficient neural activity will occur to initiate movement rapidly.

If the delay is largely or exclusively in motor time, no differential central processing in Parkinsonism can be inferred. CARLTON *et al.* [6] have recently proposed that rate of force development in normals is the fundamental parameter related to response latency, with an exponential drop in RT as rate of force development increases, until an asymptote is reached from moderate to high force development rates. If PD subjects are limited to low rates of force development by their disease, their decreased RTs for higher forces may reflect a trend which would also be present in normals at equally low rates. Thus, our control and PD groups may simply be operating at different ends of a curve which describes normal

physiological functioning: the PD group, with their lower force development rates, have decreasing RTs, while the controls show minimal differences in RT for different force levels, in agreement with earlier observations [17]. While the interaction of their RT effect must await determination of whether it is primarily a pre-motor or a motor-time effect, the remaining results suggest that PD subjects are deficient in producing the desired force–time *pattern* rather than the desired peak force, and that their dependence on visual feedback does not extend to all tasks.

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