

Scaling of Rate of Force Development with Force Amplitude is Impaired by Motor Segmentation in Parkinson's Disease

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Abstract

Healthy nervous systems scale peak rates of force development (RFD) to peak force in rapid isometric contractions, with low variability. In people with Parkinson's disease (PwPD), scaling of RFD is reduced and more variable compared to healthy adults. Some PwPD also have transient reductions in RFD (motor segmentation) within individual force pulses, but how this affects scaling is unclear. Thus, we aimed to determine how the magnitude and variability of rate-scaling differs between PwPD with (PD_{Seg}) and without (PD_{NoSeg}) motor segmentation and compared to healthy young (YA) and older adults (OA). Twenty YA, 22 OA, and 58 PwPD performed 60 rapid isometric finger abduction force pulses to 20–80% maximal voluntary contraction force (MVC). The slope (RFD-SF) and variability (R^2) of the relationship between peak force and peak RFD were computed per individual. PwPD with a median of $\geq$ two force segments were classified as PD_{Seg}. The Kinesia ONE system quantified bradykinesia and total motor symptom scores. In PwPD, the 42 PD_{Seg} had reduced RFD-SF and R^2 compared to PD_{NoSeg} ($p \leq 0.004$), OA ($p < 0.001$) and YA ($p < 0.001$). In PD_{Seg}, peak RFD was lower in segmented force pulses compared to pulses without motor segmentation ($p < 0.001$). Total symptom scores ($p = 0.002$) and bradykinesia scores ($p = 0.004$) related to R^2 but not RFD-SF ($p \geq 0.154$) in PwPD. Motor segmentation reduces overall RFD and impairs rate-scaling in PwPD, but with remarkable preservation of rate scaling in the first segment. Rate-scaling and motor segmentation measures provide specific information about Parkinson's disease impairments that is not visible in other assessments.

Introduction

Rate-based function is a key area of interest in aging and clinical populations such as people with Parkinson's disease (PwPD), for whom bradykinesia is a defining symptom. Whether rapid isometric contractions or movements are small or large, the healthy nervous system delivers discrete bursts of neural excitation to muscle that result in force pulses (Gordon and Ghez 1987; Freund and Büdingen 1978) or velocity trajectories (Corcos et al. 1989) that are smooth and surprisingly consistent in duration across the range of submaximal amplitudes. In isometric conditions, the time to reach peak force is relatively invariant because the rate of force development (RFD) increases linearly with the peak force (PF) of the contraction (Freund and Büdingen 1978). This scaling of rate with amplitude, which is quantified with linear regression parameters, is remarkably linear and invariant (high R^2), and has been observed by multiple labs and in multiple muscle groups, indicating robust control strategy in healthy adults (see Kozinc et al. (2022) for review). In early work on this topic, Freund and Büdingen (1978) postulated that the scaling of rate with amplitude exists to simplify the neural control of movements involving many synergistic muscles, so that all muscles achieve their peak contraction simultaneously regardless of their level of contribution.

The mechanisms that contribute to this scaling are those that also determine the quality of the discrete bursts of neural excitation. While efferent commands are less accessible where they exit the brain, surface electromyograms (EMG) from peripheral musculature reflect the sum of these commands,

segmental contributions, and the intrinsic properties of the alpha motor neuron. In rapid contractions across the range of force amplitudes, good quality rectified EMG bursts from the agonist muscle have a steep rate of rise, are correctly scaled to the amplitude of the contraction, and are of the correct duration to reach the intended peak force without interruption (Park and Stelmach 2006). The bursts are described as discrete, meaning they have a clear start and finish without prolonging muscle relaxation. These unimodal bursts often terminate slightly before PF. Accordingly, at the level of the motor unit, a high initial spike density is expected due to recruitment, high initial firing rates, and an appropriate number of spikes which terminate prior to peak force (Desmedt and Godaux 1977). Indeed the first three interspike intervals are known to be modifiable with exercise training (Van Cutsem et al. 1998).

While maximal RFD (isometric surrogate for power), are important and are known to decline with normal aging (Bellumori et al. 2013) and chronic conditions (Uygur and Barone 2023; Pelicioni et al. 2021; Lomborg et al. 2022), the focus of the present work is on the scaling of rate across a range of submaximal force levels. We are interested in this approach because it is congruent with the forces expected in many activities of daily living, and it provides a comprehensive description of rate-control mechanisms and their decline or impairment. In the present cross-sectional study, three groups of subjects were recruited to examine three potential contributors to changes in rate scaling in PwPD. Young (YA) and older adults (OA) are compared to describe effects of normal aging on rate-scaling. OA and PwPD are compared to broadly characterize the effects of PD on rate scaling. The third comparison is between two subgroups of PwPD who either have (PD_{Seg}) or do not have (PD_{NoSeg}) disruptions in their motor output that are described as motor segmentation.

The clinical rationale for splitting PwPD into these subgroups lies in the heterogeneous nature of Parkinson's disease presentation and the absence of good specific markers of the disease. While all patients are typically required to have evidence of bradykinesia for a diagnosis (Postuma et al. 2015), there may be different presentations of slowing. Bradykinesia is broadly defined as slow and/or small movements, and clinical ratings take into account the speed, amplitude, and rhythm of rapid movements (Postuma et al. 2015). In this framework, bradykinesia can be reported due to a general slowness that resembles accelerated aging (still smooth but slow), or it can be reported due to poor performance that is expressed more as decrements in amplitude or freezes during movement. In studies that used isometric conditions to examine bradykinesia and rate control in PwPD, others have reported rate impairments that parallel both types of slowing. Theoretically, motor segmentation represents the latter type of slowing in which the neural excitation to muscle is small and incomplete, resulting in isometric force-pulse profiles that have a bumpy or stepwise appearance during the progression to peak force (Wierzbicka et al. 1991). In a previous study, we quantified motor segmentation with multiple measures that have good test-retest reliability and large effect sizes in comparisons with older adults (Howard et al. 2022). We then reported that 68% of PwPD exhibit motor segmentation while on their medications (Daniels and Knight 2025). Lastly, we demonstrated that these force-based measures of motor segmentation correlate with the fragmentation of EMG bursts, where the initial bursts are small and incomplete with respect to achieving the intended peak force (Daniels and Knight 2026). What is not yet

known is the extent to which motor segmentation further exacerbates rate control in PwPD, compared to a general decline in rate.

Our purpose was to address two questions. First, how does PD affect rate scaling compared to typical aging? Second, in what manner does motor segmentation, as a specific marker of impaired control, affect rate scaling mechanisms in PwPD? We hypothesized that PwPD would have reduced RFD-SF and R^2 compared to healthy adults, and that PD_{Seg} would produce reduced and less consistent RFD-SF compared to PD_{NoSeg}. Additionally, we hypothesized that in PwPD, greater segmentation would negatively correlate with RFD-SF and R^2 . Due to the importance of the first segment in rapid force production or movements, we also quantified scaling in the first segment in PD_{Seg}, and compared it to scaling computed from the total force pulse. We hypothesized that PD_{Seg} would have higher RFD-SF and R^2 in the first segment compared to their overall scaling to peak force. In addition to comparing the effects of segmentation between the PD_{Seg} and PD_{NoSeg} groups, the fact that PwPD produce some force pulses with and without segmentation afforded within-subject comparisons. We hypothesized that PwPD would produce reduced peak RFD and have prolonged time to peak RFD and peak force when their force pulses have ≥ 2 segments compared to when they reach 90% of peak force with a single segment. Lastly, to understand associations between rate-scaling measures and clinical tests, we tested the hypothesis that RFD-SF and R^2 would have small to moderate relationships with total motor symptom scores and bradykinesia scores assessed with the Movement Disorder Society-Unified Parkinson's Disease Rating Scale part III (MDS-UPDRS-III).

Methods

Subjects

This study included 58 PwPD (46 males, aged 68.5 ± 7.9 years, 5.0 ± 4.0 years since diagnosis, 1.70 ± 0.10 m tall, mass: 80.5 ± 14.5 kg, Hoehn and Yahr stage: 2.0 ± 0.5 , levodopa equivalent daily dose: 594.9 ± 451.4 mg, and MDS-UPDRS-III: 34.9 ± 12.9) on their usual medications, 22 OA (11 males, aged 68.8 ± 9.7 years, 1.69 ± 0.11 m tall, 76.0 ± 15.8 kg, and MDS-UPDRS Score: 3.7 ± 3.5), and 20 YA (7 males, aged 25.5 ± 3.2 years, 1.71 ± 0.08 m tall, 74.8 ± 14.3 kg). PwPD were tested on their medication to test their ability to rate-scale in their normal functional state.

Clinical Ratings

PwPD performed all tasks in the MDS-UPDRS-III and were rated by a trained investigator. Additionally, the Kinesia ONE system quantified motor symptoms scores for bradykinesia tasks, tremor tasks, and gait. Total scores for the MDS-UPDRS-III and Kinesia ONE included the sum of scores from all tasks in each respective scale. The Kinesia ONE was also used to quantify total upper extremity bradykinesia subscores including sums of speed, amplitude and rhythm ratings from the finger taps, hand movements, and pronation-supination tasks for the side tested during the isometric force protocol.

Instruments

Isometric index finger abduction forces were measured using a custom device placed on a standard height office table in front of the subject. The tested hand grasped a wooden stabilization bar (45 x 4 x 3 cm) using a cylindrical grip with digits 2–5 above the bar and digit 1 beneath. The palmar surface was horizontal and the second digit was extended in an anterior direction with the second proximal interphalangeal joint fastened against a firm pad on a force transducer (MB-25, Interface Inc., Scottsdale, AZ, USA). Force was sampled at 200 Hz (NI cDAQ-9178, Module 9329, National Instruments, Austin, TX, USA), amplified, and low-pass filtered at 50 Hz (Model SGA, Interface Inc.). DASYlab software (Measurement Computing Corporation, Norton, MA, USA) was used to control digitization and provided real-time visual feedback of force.

Isometric Rapid Force Pulse Protocol

PwPD performed the force tasks with their self-reported more symptomatic hand, which corresponded to 31 PwPD using their dominant hand. Healthy adults used a randomly determined hand, resulting in 14 OA and 10 YA performing the task with their dominant hand. Subjects first performed three maximal voluntary contractions (MVC), and the greatest MVC was used to normalize force feedback on a vertical bar graph in %MVC units. Next, subjects were cued to perform rapid isometric finger abduction force pulses to small, medium, and large amplitudes spanning 20–80% MVC. A beep cued subjects to perform force pulses approximately 3-5s apart with variability in the period between beeps to prevent anticipation. Subjects were instructed to prioritize the speed of force production rather than reaching a specific peak force (Gordon and Ghez 1987). Subjects performed practice contractions with feedback prior to recorded trials. Each subject completed five 60s trials with adequate rest in between, to collect approximately 75 force pulses within the desired force range. An additional MVC was performed after the force pulse testing to evaluate fatigue.

Analysis of Force Pulses

Scripts written in Matlab software (Mathworks, Natick, MA, USA) were used to analyze force data. We calculated the first (RFD) and second time derivatives of force ($F''(t)$) using a moving slope (D'Errico 2025) with an overlapping 50 ms window (Fig. 1; Howard et al. 2022). The peak force of each pulse was recorded. A RFD threshold of 20% MVC/s determined the start and end of each force pulse, which was visually inspected and confirmed by an investigator for each force pulse. Peak RFD for each force pulse was the highest RFD value between force initiation and peak force and is reported in %MVC/s units.

Rate-scaling

To analyze the scaling of rate with amplitude in rapid force production, linear regression equations were calculated from the peak RFD and peak force from 60 randomly sampled pulses in each subject (Bellumori et al. 2011). Randomly selecting a subset of pulses was used to ensure that every subject had the same number of pulses used in analysis, as the number of pulses may affect RFD-SF values

(Bellumori et al. 2011). Considering the total of 75 pulses recorded, 60 allows for exclusions of errors like false starts and force pulses that were outside of the force range of interest. The same subset of pulses was used for segmentation analysis. The slope of the regression equation (RFD-SF) and the associated R^2 were the dependent measures used to describe rate-scaling and its consistency for each subject.

Motor Segmentation

Segmentation was quantified using the method described by Howard et al. (2022), visible in Fig. 1. For each pulse, the number of zero crossings in $F''(t)$ was counted between force initiation and 90% of peak force (F_{90}). We use 90% peak force in our segmentation analysis to separate this aspect of the disorder during excitation from other difficulties that PwPD exhibit near peak force and in the transition from excitation to relaxation (Howard et al. 2022). The number of force segments was determined by adding one to the number of zero crossings in $F''(t)$ and dividing the total in half. PwPD were considered to have segmentation if the median number of force segments to F_{90} was ≥ 2 . Additionally, the percent of pulses with ≥ 2 segments to F_{90} was calculated. The end of the first segment was defined at the minimum between the first two zero crossings in $F''(t)$. In PD_{Seg} , we also computed the peak RFD and peak force for the first force segment.

Analysis of the First Segment

Within the PD_{Seg} who produced at least 90% of their pulses with ≥ 2 segments, linear regression was used to calculate the RFD-SF and R^2 between peak RFD and peak force within the first force segment, as well as between peak RFD in the first segment and the maximum peak force of each pulse (see Wierzbicka et al. 1991). A visual depiction of force landmarks used in calculations is found in Fig. 1.

Comparisons of Segmented and Non-Segmented Pulses Within Subjects

A subset of 19 PwPD had at least 15 pulses with ≥ 2 segments and 15 pulses with 1 segment between 30 to 50% MVC. For these subjects, 15 segmented pulses and 15 non-segmented pulses were randomly selected between 30–50% MVC, and medians were computed for time to F_{90} , time to peak RFD, and peak RFD within segmented and non-segmented samples.

Statistical Analysis

All statistical tests were performed using SPSS version 31 (IBM, Armonk, NY) with the significance level set to $p < 0.05$. For all comparisons, Shapiro-Wilk's tests were used to assess normality. In cases of a significant Shapiro-Wilk's test, non-parametric statistical tests were used. Wilcoxon signed-rank tests were used to determine whether post-testing MVC differed from pre-testing MVC within each group. A one-way ANOVA was used to compare the initial MVC across the three groups. Independent t-tests were used to compare characteristics between PD_{Seg} and PD_{NoSeg} groups. Where Levene's test indicated unequal variances, the adjusted t-statistic and degrees of freedom for unequal variances were used.

Prior to comparisons of R^2 values, the Fisher's r-to-z transform was used. Effect sizes were expressed as Pearson's correlation coefficient (r), derived from the t-values and degrees of freedom or z scores as described by Field (2013). Effect sizes were categorized as small (0.10), moderate (0.30), or large (0.50) according to Cohen (1988).

Kruskall Wallis tests were used to compare RFD-SF and R^2 between YA, OA, PD_{Seg} and PD_{NoSeg}. Subsequent pairwise comparisons were made as needed. Within PwPD, Kendall's Tau-b was used to describe the relationships between the median number of segments and rate-scaling measures. Spearman's correlations were used to describe relationships between the percentage of pulses with segmentation and rate-scaling measures. Paired t-tests were used to compare overall RFD-SF and R^2 to the RFD-SF and R^2 calculated within the first segment, and the RFD-SF and R^2 calculated between the first segment peak RFD and overall peak force.

Results

Post-testing MVCs, measured in N•m, were greater than pre-testing MVCs in YA ($T = 154.0$, $p = 0.018$, $r = 0.38$), OA ($T = 207$, $p = 0.009$, $r = 0.39$), PD_{NoSeg} ($T = 115$, $p = 0.015$, $r = 0.43$), and PD_{Seg} ($T = 643$, $p = 0.006$, $r = 0.30$), indicating that the force pulse protocol was not fatiguing. There was a significant group effect on MVC ($F(3,96) = 3.47$, $p = 0.019$), where PD_{NoSeg} had greater MVC than YA ($p = 0.020$, $g = 1.04$), but otherwise groups were not different (all $p \geq 0.167$, all $g \leq 0.60$).

Prevalence of Segmentation

Forty-two (72%) in the present sample of PwPD were classified as having segmentation (PD_{Seg}) and sixteen PwPD did not (PD_{NoSeg}). Within PD_{Seg}, the percentage of pulses with 2 or more segments ranged from 55 to 100%. PD subgroups did not differ in age ($t(56) = 0.15$, $p = 0.883$, $r = 0.02$), height ($t(56) = 0.29$, $p = 0.773$, $r = 0.04$), weight ($t(56) = 0.26$, $p = 0.799$, $r = 0.03$), Hoehn and Yahr stage ($t(56) = 1.06$, $p = 0.293$, $r = 0.14$), or time since medication intake in medicated PwPD ($t(53) = 1.02$, $p = 0.312$, $r = 0.14$).

Suggesting a possible link to disease progression, compared to PD_{NoSeg}, PD_{Seg} had an average time since diagnosis that was 3.6 years longer ($t(44.8) = 4.15$, $p < 0.001$, $r = 0.53$), a 231.1 mg greater levodopa equivalent daily dose ($t(55.2) = 2.46$, $p = 0.017$, $r = 0.31$), and they scored 13.2 higher on the MDS-UPDRS-III ($t(56) = 3.90$, $p < 0.001$, $r = 0.46$).

Group Comparisons in Rate-scaling

Data from a representative subject from each group are presented in Fig. 2. Group box-and-whisker plots for RFD-SF and R^2 are depicted in Fig. 3. RFD-SF was significantly different across groups ($H(3) = 47.56$, $p < 0.001$). Young adults had significantly higher RFD-SF compared to PD_{Seg} ($z = 6.09$, $p < 0.001$, $r = 0.77$) and PD_{NoSeg} ($z = 2.43$, $p = 0.015$, $r = 0.40$), but not OA ($z = 1.05$, $p = 0.296$, $r = 0.16$). OA had significantly greater RFD-SF compared to PD_{Seg} ($z = 5.06$, $p < 0.001$, $r = 0.63$) but not PD_{NoSeg} ($z = 1.50$, $p = 0.135$, $r =$

0.24). PD_{NoSeg} had significantly greater RFD-SF compared to PD_{Seg} ($z = 2.86$, $p = 0.004$, $r = 0.38$). R^2 was also significantly different across groups ($H(3) = 43.19$, $p < 0.001$). YA had greater R^2 compared to OA ($z = 2.00$, $p = 0.046$, $r = 0.31$), PD_{NoSeg} ($z = 2.53$, $p = 0.011$, $r = 0.42$), and PD_{Seg} ($z = 6.20$, $p < 0.001$, $r = 0.79$). OA had greater R^2 than PD_{Seg} ($z = 4.05$, $p < 0.001$, $r = 0.51$) but not PD_{NoSeg} ($z = 0.71$, $p = 0.479$, $r = 0.11$). PD_{NoSeg} had greater R^2 than PD_{Seg} ($z = 2.84$, $p = 0.005$, $r = 0.37$).

Consolidating both PD groups, greater median segments to F90 correlated with poorer RFD-SF ($\tau_b = -0.55$, $p < 0.001$), and R^2 ($\tau_b = -0.44$, $p < 0.001$). Additionally, a high percent of segmented pulses was associated with low RFD-SF ($\rho = -0.67$, $p < 0.001$), and R^2 ($\rho = -0.53$, $p < 0.001$).

Analysis of the First Segment

In the 16 PD_{Seg} who had $\geq 90\%$ pulses with segmentation, there was better rate scaling in the first segment compared to overall scaling of the force pulses (RFD-SF: $t(15) = 9.44$, $p < 0.001$, $r = 0.93$, R^2 : $t(15) = 6.61$, $p < 0.001$, $r = 0.86$). Comparisons of these relationships within a representative subject are shown in Fig. 4. However, when scaling between the first segment peak RFD and peak force, RFD-SF ($t(15) = -2.92$, $p = 0.011$, $r = 0.60$) and R^2 ($t(15) = -5.58$, $p < 0.001$, $r = 0.82$) were worse compared to scaling between peak RFD and peak force.

Figure 5 illustrates the breakdown of scaling with segmentation, in this case with substantially less scaling after approximately 40% MVC. Figure 6 provides RFD-SF and R^2 results for the first segment within segmented pulses for comparison with overall RFD-SF and R^2 . Note that compared to the overall scaling and its variance, the rate-scaling in the first segment was better and its variability was less. The median duration of the first segment was 90 ms, while the median periods from rest to F90 and to peak force were 246 ms and 322 ms, respectively. The median $\pm$ interquartile range duration of the first segment for this subject was 74 ± 30 ms, while the median $\pm$ interquartile range for periods from rest to F90 and to peak force were 253 ± 110 ms and 329 ± 100 ms, respectively.

Comparisons of Segmented and Non-Segmented Pulses Within Subjects

Figure 7 shows box-and-whisker plots for rate and timing variables in force pulses with and without segmentation. Peak RFD ($t(18) = 5.02$, $p < 0.001$, $r = 0.76$) and average RFD ($t(18) = 4.54$, $p < 0.001$, $r = 0.73$) were lower in segmented pulses compared to non-segmented pulses. Additionally, time to F90 ($t(18) = 7.82$, $p < 0.001$, $r = 0.88$) and time to peak RFD ($t(18) = 3.82$, $p < 0.001$, $r = 0.67$) were longer in segmented pulses compared to non-segmented pulses.

Correlations with clinical ratings

Table 1 contains correlations between RFD-SF, R^2 , clinical motor symptom ratings, and the timed-up-and-go as a reference for comparison. RFD-SF had no significant relationships with clinical ratings, but R^2

had small to moderate relationships of comparable magnitude to relationships with time-up-and-go scores.

Table 1
Relationships between symptom ratings and rate-scaling metrics in people with Parkinson's disease (n = 58) and the timed-up-and-go times as a comparator.

Total UPDRS	R² (ρ, p)	RFD-SF (ρ, p)	Timed Up and Go (ρ, p)
	$\rho = -0.36$, 0.006	$\rho = -0.22$, 0.105	$\rho = 0.36$, 0.006
Total Kinesia One	$\rho = -0.40$, 0.002	$\rho = -0.25$, 0.063	$\rho = 0.35$, 0.007
Total Speed	$\rho = -0.31$, 0.017	$\rho = -0.14$, 0.300	$\rho = 0.34$, 0.009
Total Rhythm	$\rho = -0.31$, 0.017	$\rho = -0.17$, 0.194	$\rho = 0.25$, 0.059
Total Amplitude	$\rho = -0.28$, 0.034	$\rho = -0.18$, 0.176	$\rho = 0.31$, 0.019
Total Bradykinesia	$\rho = -0.37$, 0.005	$\rho = -0.20$, 0.133	$\rho = 0.36$, 0.006
Timed Up and Go	$\rho = -0.27$, 0.042	$\rho = -0.03$, 0.835	--

RFD-SF=rate of force development scaling factor; UPDRS=Movement Disorder Society-Unified Parkinson's Disease Rating Scale Part III (Motor) Score

Discussion

Rate-scaling with low variability (a high R^2) is a feature of healthy neuromuscular control that declines with age and central nervous system impairment. In this study, our overall goal was to evaluate how Parkinson's disease (PD) and segmentation affect rate-scaling in PwPD. Our results can be organized into four main findings. First, we identified that PD_{Seg} have reduced RFD-SF and R^2 compared to healthy young and older adults with large effect sizes. Within PwPD, we identified that the 72% who were classified as PD_{Seg} had poorer scaling of RFD across force amplitudes compared to PD_{NoSeg} with moderate effect sizes, and that PD_{NoSeg} 's performance did not significantly differ from OA (Fig. 3). We also identified that the amount of segmentation had moderate to strong negative correlations with RFD-SF and R^2 in PwPD. Second, we identified that in PD_{Seg} the first segment exhibits remarkably good scaling compared to measures of scaling for the overall force pulse. Third, we observed that when PwPD use multiple segments to reach 90% peak force, rather than a single segment, peak RFD is reduced, and the time to peak RFD and time to F90 are prolonged. The fourth finding was that R^2 but not RFD-SF had small negative relationships with MDS-UPDRS-III scores, indicating that PwPD with worse disease severity had less consistency in the scaling of rate with amplitude. Collectively, these results indicate that rate scaling is impaired in PwPD and that motor segmentation worsens this impairment.

Group Comparisons in Rate-scaling

As expected, most YA demonstrated scaling between peak RFD and peak force with little variability and RFD-SF values that mostly overlap with previous literature (Fig. 3, Bellumori et al. 2011; Corrêa et al. 2020). OA showed a non-significantly lower median RFD-SF and greater variability (Fig. 3). Though YA and OA did not have significantly different RFD-SF as previously demonstrated (Bellumori et al. 2013; Corrêa et al. 2020), they were also closer in average age than in the study by Bellumori et al. (2013). Additionally, both YA and OA demonstrated more variability than previously reported, and this is likely due to greater precision of the small window size used to calculate RFD (50 ms). Some variability in OA is also not surprising considering that the OA group incorporated a wide age range (53–85 years old), where the oldest adults had the lowest R^2 . Advanced age shares some physiological features of PD, such as mild neuronal loss in the substantia nigra and Lewy body accumulation (Franceschi et al. 2018), and very old adults can experience some symptoms without qualifying for a PD diagnosis (Algotsson et al. 2023). The range in OA age and function is a strength of this study, as PwPD ranged between 45 and 86 years old, and the compatible age range with OA subjects helps us to better evaluate the effects of PD versus advanced age on rate-scaling performance.

Meanwhile, OA outperformed PD_{Seg}, indicating altered central control with disease state, even at early disease stages and while medicated. Within PwPD, PD_{NoSeg} outperformed PD_{Seg}, and PD_{NoSeg} performed similarly to OA in both measures (Fig. 3). The high performance of PD_{NoSeg} likely reflects lower disease severity compared to PD_{Seg} in addition to the potential benefits of medication, which improve movement rate (Vaillancourt et al. 2004). At the group level, this indicates that segmentation may negatively impact a PwPD's ability to appropriately scale rate with movement size, which was supported by the significant negative correlations between the number of segments and rate scaling measures. Our previous study identified that PD_{Seg} produce lower peak RFD compared to PD_{NoSeg} (Daniels and Knight 2025), and Howard et al. (2022) found a strong relationship ($\rho = 0.93$) between median peak RFD and RFD-SF. Wierzbicka et al. (1991) originally identified subgroups of PwPD including a group with faster, less variable force production more similar to healthy adults, and a separate group with more visible disorder. Though both groups of PwPD had greater variability compared to healthy adults, the more impaired PD group required multiple bursts in the surface electromyogram (EMG) to reach peak force, which resulted in a segmented force-time curve, high variability, and prolonged time to peak force due to smaller increases in RFD with movement amplitude. Similar to findings by Wierzbicka et al. (1991), we identified larger increases in the time to peak force in PD_{Seg} compared to PD_{NoSeg} or OA (Fig. 2, Daniels and Knight 2025, c.f. Wierzbicka et al. 1991). This is the result of a reduced RFD-SF. With insufficient scaling, the RFD does not increase enough with the size of the muscle contraction produced.

Analysis of the First Segment

Despite the evidence that segmentation negatively affects overall rate-scaling, we found that scaling between peak RFD and peak force in the first force segment rivaled or even exceeded that of healthy

adults. Indeed, in all PD_{Seg} with segmentation in $\geq 90\%$ of force pulses, both RFD-SF and R^2 improved when measured from the first segment compared to overall peak RFD and peak force. Figure 5 shows a subject's performance that was representative of all PD_{Seg} subjects. Similarly, Wierzbicka et al. (1991) identified that a PD_{Seg} scaled initial EMG and peak RFD appropriately with the first segment peak but was insufficient to reach peak force. The first segment duration is highly consistent in PwPD (Fig. 5, Daniels and Knight 2025), which agrees with findings by Robichaud et al. (2009) that PwPD who require multiple EMG bursts to complete rapid contractions have short bursts with little variability. The tightly controlled first segment duration explains the high R^2 and demonstrates that within the first segment, PD_{Seg} appropriately scale movement rate with movement size. Meanwhile, the peak RFD they produce in the first segment is low compared to PD_{NoSeg} and OA (Daniels and Knight 2025). In PwPD, the duration and amplitude of the first EMG burst correlate with the duration and peak RFD of the first segment, respectively, and more force segments relate to a greater number of EMG bursts (Daniels and Knight 2026). Thus, similar to the conclusions by Wierzbicka et al. (1991), we find that while PD_{Seg} scale effectively in the first segment due to fairly invariable times to the first peak, they are not able to produce sufficient EMG amplitudes to reach a high peak RFD within that time. In this sample, the median greatest peak force PD_{Seg} achieved within the first segment of a segmented pulse was 47% MVC. Reaching peak RFD, which most often occurs in the second force segment (Daniels and Knight 2025), requires additional EMG bursts and is ultimately insufficient for the peak force level. Thus, time to peak force increases progressively with each additional segment required while peak RFD improves minimally, resulting in more variable force production and reduced scaling in peak RFD per increase in peak force. Considering our interests in improving rate dependent mobility, this suggests that interventions to increase the duration and/or amplitude of the first EMG burst would greatly improve rate-scaling.

Comparisons of Segmented and Non-Segmented Pulses Within Subjects

The present findings extend those of Howard et al. (2022), with a larger and more clinically well-characterized sample. We similarly found strong correlations between the number of segments to F90 and RFD-SF and R^2 across PwPD. In the present study we were also adequately powered to make comparisons between PD_{NoSeg} and PD_{Seg} , as well as comparisons within individuals of pulses with and without segmentation. When PwPD produced force pulses with segmentation, they also had reduced peak RFD, and prolonged time to peak RFD and time to F90 compared to when they produced force without segmentation. In this context, prolonged time to F90 is the result of poor rate-scaling.

Remarkably, while the timing of segmentation seems relatively invariant and the results on force production are severe (Daniels and Knight 2025), our findings also indicate that some PD_{Seg} can still scale overall peak RFD to peak force similarly to PD_{NoSeg} and healthy adults (see high performers in Fig. 3), and some subjects with poorer segmentation measures can still produce some high quality, single segment, pulses. For example, one subject who produced force in ≥ 2 segments in 90% of pulses had an RFD-SF of 7.66 and an R^2 of 0.78 (Fig. 4). Though far from the group's median, this indicates that normal scaling in PD_{Seg} is still possible, which was an encouraging and unique finding from previous

works (Howard et al. 2022; Wierzbicka et al. 1991). This subject achieved a higher median peak RFD in the first segment and overall compared to the PD_{Seg} group medians. In addition to performing testing on medication and being in earlier disease stages, many PwPD in our study reported regular participation in speed-based exercise. Considering the benefits of levodopa therapy (Vaillancourt et al. 2004, 2006) and exercise on movement rate and scaling (David et al. 2016; Van Cutsem et al. 1998; Bellumori et al. 2017), it is likely these factors contributed to high performance in some PwPD. Finding interventions to improve the quality of the first segment, with good consistency, can theoretically reduce the number of total segments needed to complete a fast movement. Previous research indicates some interventions can improve movement rate by increasing initial EMG size and decreasing the number of EMG bursts required to perform a rapid contraction, including medication (Vaillancourt et al. 2004, 2006), deep brain stimulation (Vaillancourt et al. 2004, 2006), resistance exercise (David et al. 2016), or movement practice (Flament et al. 2003). This suggests that even in PD_{Seg}, initial RFD may be improved to reduce segmentation and improve rate-scaling.

Correlations with clinical ratings

It is natural to question how the scaling factor and segmentation measures relate to clinical rating scores. We conducted this analysis with a tempered expectation of strong associations. Our rationale for this position is that while the scaling and segmentation measures have high neuromuscular and measurement precision, the movements observed in the ratings, whether instrumented (Kinesia ONE) or not, do not precisely measure specific symptoms, kinetics or kinematics. For example, the effects of rigidity and/or bradykinesia on finger tapping tests are hard to separate, if both are present. Second, while the segmentation measure have already demonstrated good sensitivity to distinguish between PD and normal aging, samples in our work are mild-to moderate in disease severity, potentially imposing a restricted range effect on the results (Janse et al. 2021). Nevertheless, in this study, we identified small to moderate negative relationships between clinical scores and R^2 (Table 1). These relationships were similar in size to the relationships identified between clinical measures and timed up and go time, which is a reliable measure of whole body mobility in PwPD (Morris et al. 2001). RFD-SF reflects how well neuromuscular mechanisms scale RFD with peak force, while R^2 measures the consistency of this scaling in rapid movements. Thus, we hypothesized that similar mechanisms might contribute to both altered rate control and clinical scores of disease severity. Small to moderate correlations were anticipated partly due to the small range of disease severity in our sample, and the effects of anti-Parkinson medication. Previous studies also found mostly small to moderate relationships between clinical scores and experimental measures, including torque rise time (Robichaud et al. 2005), RFD (Jordan et al. 1992), EMG duration variability (Robichaud et al. 2009), and the scaling of initial EMG with movement distance (Pfann et al. 2001). In this study, we found that RFD-SF did not significantly relate to clinical scores in PwPD. We found this surprising, considering that Pfann et al. (2001) found that EMG burst scaling with distance during rapid contractions had a moderate relationship with a similar range of UPDRS scores, though peak RFD has less variability across PwPD compared to initial RFD (Daniels and Knight 2025). It is also important to consider that strong relationships with clinical scores are not

required to identify an effective disease marker. For instance, measures of olfactory function (Tissingh et al. 2001) or free water in the substantia nigra (Ofori et al. 2015) had small or non-significant relationships with motor symptom scores within samples of PwPD, despite their utility in detecting neurodegeneration. A small relationship with clinical scores rather indicates that the measures performed by Tissingh et al. (2001) or Ofori et al. (2015) were far more specific. Similarly, clinical assessments of bradykinesia may indicate the speed or size of movements, but do not indicate how well a subject scales speed with movement amplitude, as measured by RFD-SF, or their consistency in this ability, as measured by R^2 . The Kinesia ONE also cannot distinguish between bradykinesia and other factors, such as small hand size or age-related slowing, as it identified non-zero ratings in YA and OA. Despite this, the large range of R^2 values measured in PwPD indicates its sensitivity to disease-related departures in normal control, and were present in a cohort of limited disease severity on medication.

Limitations and Delimitations

It is important to note that the results of this study are delimited to individuals who, on average, had mild-to-moderate disease severity and were tested on their usual medications (see characteristics in methods). While testing in the ON-meds state may limit the testing of certain hypotheses about the disease, we intentionally chose to test PwPD on their usual medications so that we could examine rate scaling and segmentation in PwPD in a state that represents their daily function. Indeed, part three of the UPDRS indicates that the rater should first record whether the patient is being tested ON or OFF their medications, and it is common for patients to visit their clinical specialists while taking their meds so that they can evaluate efficacy. Considering that large effect sizes were observed in individuals with mild-to-moderate disease severity, and that their medication likely improved task performance (Vaillancourt et al. 2004, 2006), testing in the ON-medication state did not interfere with the study objectives. We believe that future studies in the OFF-medication state would reveal greater segmentation and impairment in rate scaling.

Summary and implications

In sum, our findings demonstrate that segmentation directly impairs rate scaling in PwPD, which contributes to increased variability and reduced scaling in the peak force-RFD relationship. Furthermore, the greater variability in rate-scaling, measured by R^2 , is associated with poorer clinical measures and slowing. Considering the importance of the precise control of the rate and amplitude of muscle force production during activities of daily living, these rate-scaling measures provide valuable information relevant to impairments in PwPD. These findings support that segmentation contributes to a departure from normal central control of rapid movement, and that R^2 may provide a helpful metric of disease-related impairment in PD. One might begin to consider whether categorization of symptoms as bradykinetic with or without segmentation can add precision to tracking disease progression and the effects of interventions. With this paradigm, we demonstrated impaired control, but also that good scaling and pulse performance are still possible in PwPD. Thus, the quantity or frequency of

segmentation, performance variability, and the peak RFD within the first force segment present specific intervention targets that play a central role in rate-scaling capacity.

Declarations

Competing Interests

We report no competing interests.

Financial Interests

We declare we have no financial interests.

Ethics approval

All study procedures were approved by the University of Delaware Institutional Review Board (4/22/2022, approval number 1884124) and compliant with the Declaration of Helsinki.

Consent to participate

All subjects provided consent via institutionally-approved informed consent documents prior to participating.

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Author Contribution

Conceptualization, Methodology, Writing – review and editing, and Funding acquisition: R.D. and C.K.; Formal analysis, investigation, Writing - original draft preparation and Visualization: R.D.; Resources and Supervision: C.K.

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Data Availability

The data that support the findings of this study are available upon reasonable request.

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Figures

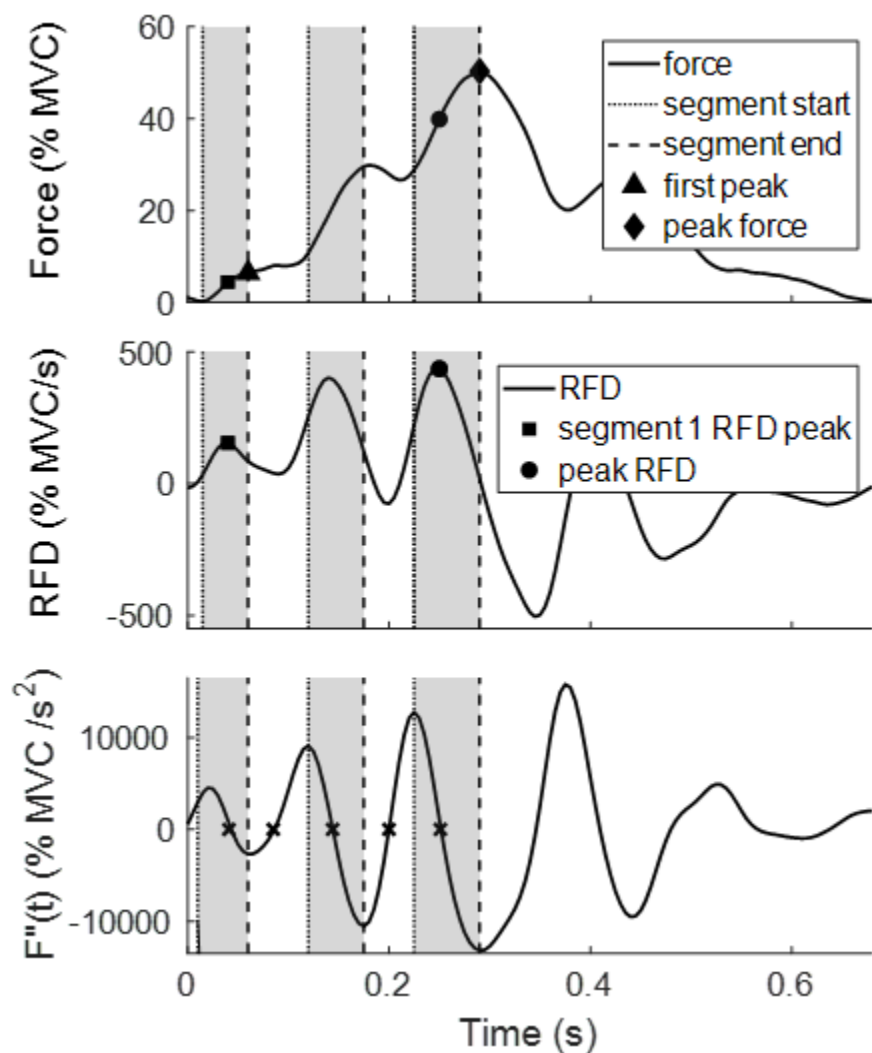


Figure 1

Visual depiction of segment and rate calculations. Top panel shows a force pulse with multiple segments to peak force. Middle panel shows the associated rate of force development (RFD) normalized to maximal voluntary contraction force (MVC). The bottom panel shows the second derivative of force ($F''(t)$). The first segment was determined from force initiation to the first minimum in $F''(t)$ following the first zero crossing (indicated with an x). Successive segments were determined from the maximum following a zero crossing and the following minimum, and are shaded in gray across all panels. The peak RFD in the first segment, and the maximum RFD in the entire pulse were determined and plotted in the top and middle panel. The magnitude of the first force peak and the maximum peak force were also determined and plotted on the first and second panel. In people with Parkinson's disease with segmentation, linear regression was used to determine scaling between the maximum RFD and force across all force pulses, as well as between the first peak in RFD and the first segment peak magnitude.

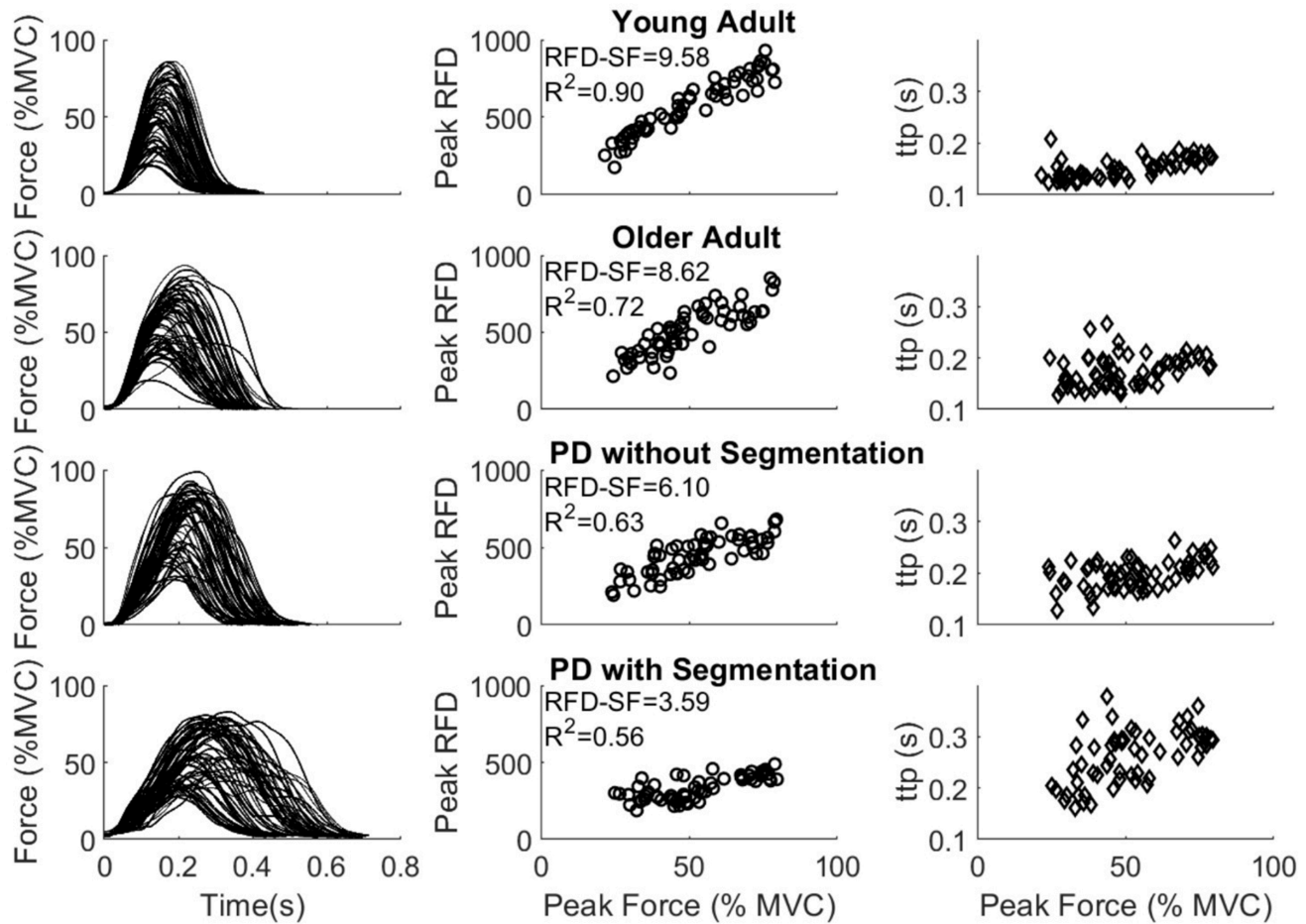


Figure 2

Overlaid force pulses (left panel), the relationship between peak force and peak rate of force development (RFD) normalized to maximal voluntary contraction force (MVC, middle panel) in %MVC/s units, and the time to peak force across 60 randomly selected pulses used in analysis (right panel) in a sample young adult (YA), older adult (OA), person with Parkinson's disease without motor segmentation (PD without segmentation) and a person with Parkinson's disease with motor segmentation (PD with segmentation).

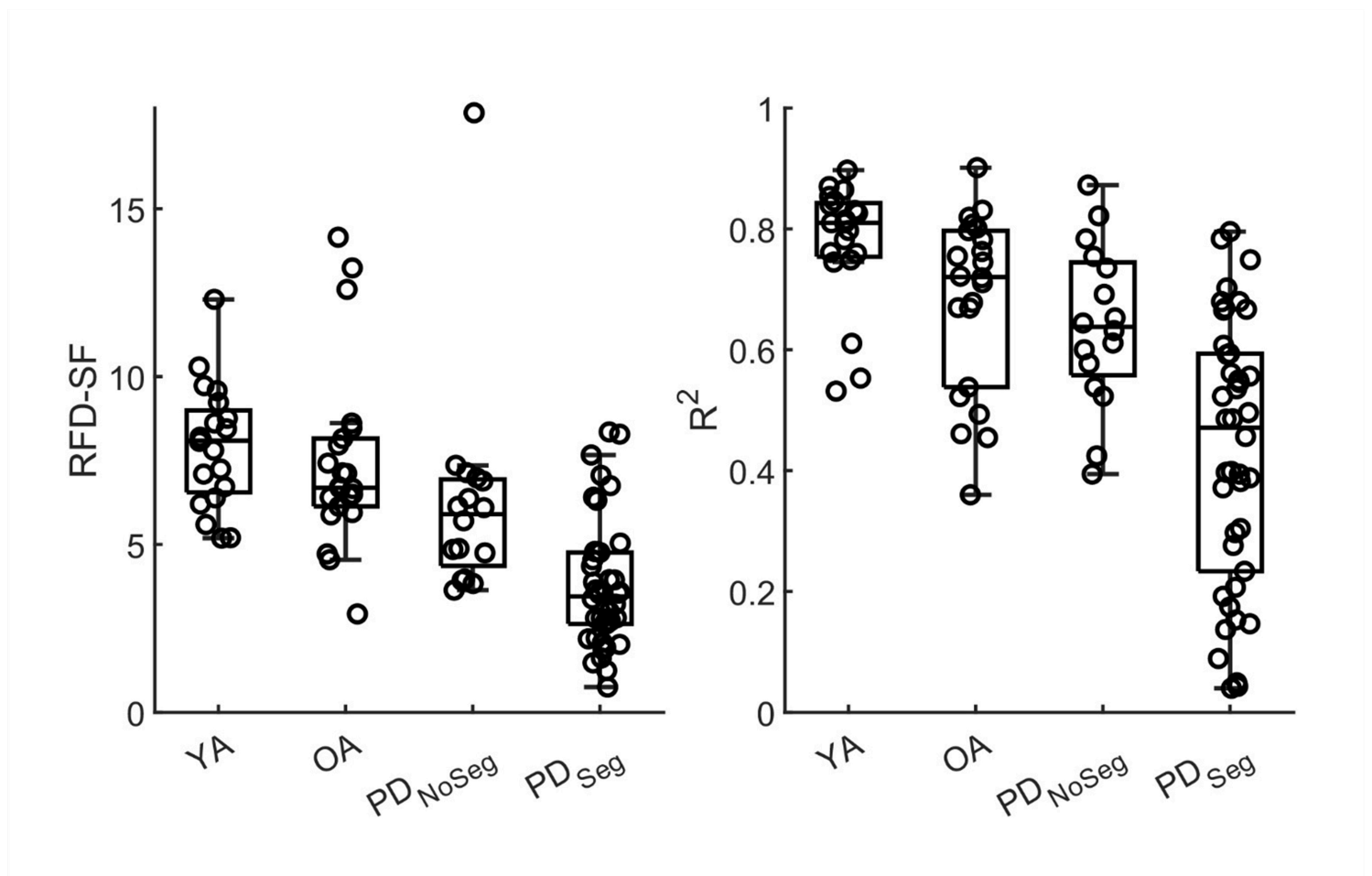


Figure 3

Box plots depict rate of force development scaling factor (RFD-SF) and its R^2 between peak force and peak rate of force development in young adults (YA), older adults (OA), people with Parkinson's disease without segmentation (PD_{NoSeg}) and people with Parkinson's disease with segmentation (PD_{Seg}). Individual subject data are represented by overlaid circles.

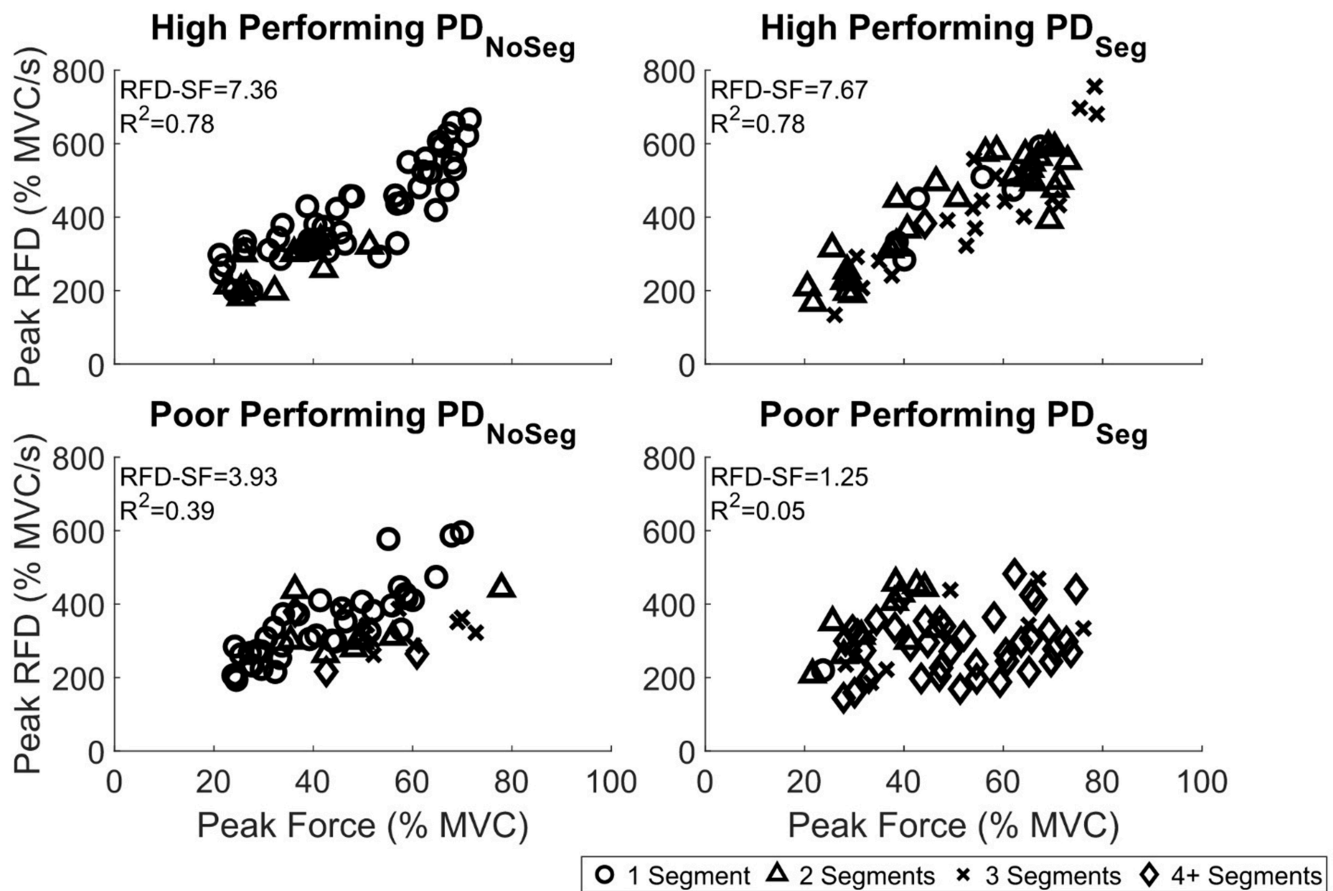


Figure 4

Example relationships between peak rate of force development (RFD) and peak force normalized to maximal voluntary contraction force (MVC) for representative best (high rate of force development scaling factor, RFD-SF, and R^2) and poorest (low RFD-SF and R^2) performers in the people with Parkinson's disease without motor segmentation group (PD_{NoSeg}) and the people with Parkinson's disease with motor segmentation group (PD_{Seg}). Each value plotted indicates the peak RFD and peak force for a single force pulse, and the number of segments required to reach 90% of peak force within that pulse is indicated by the shape.

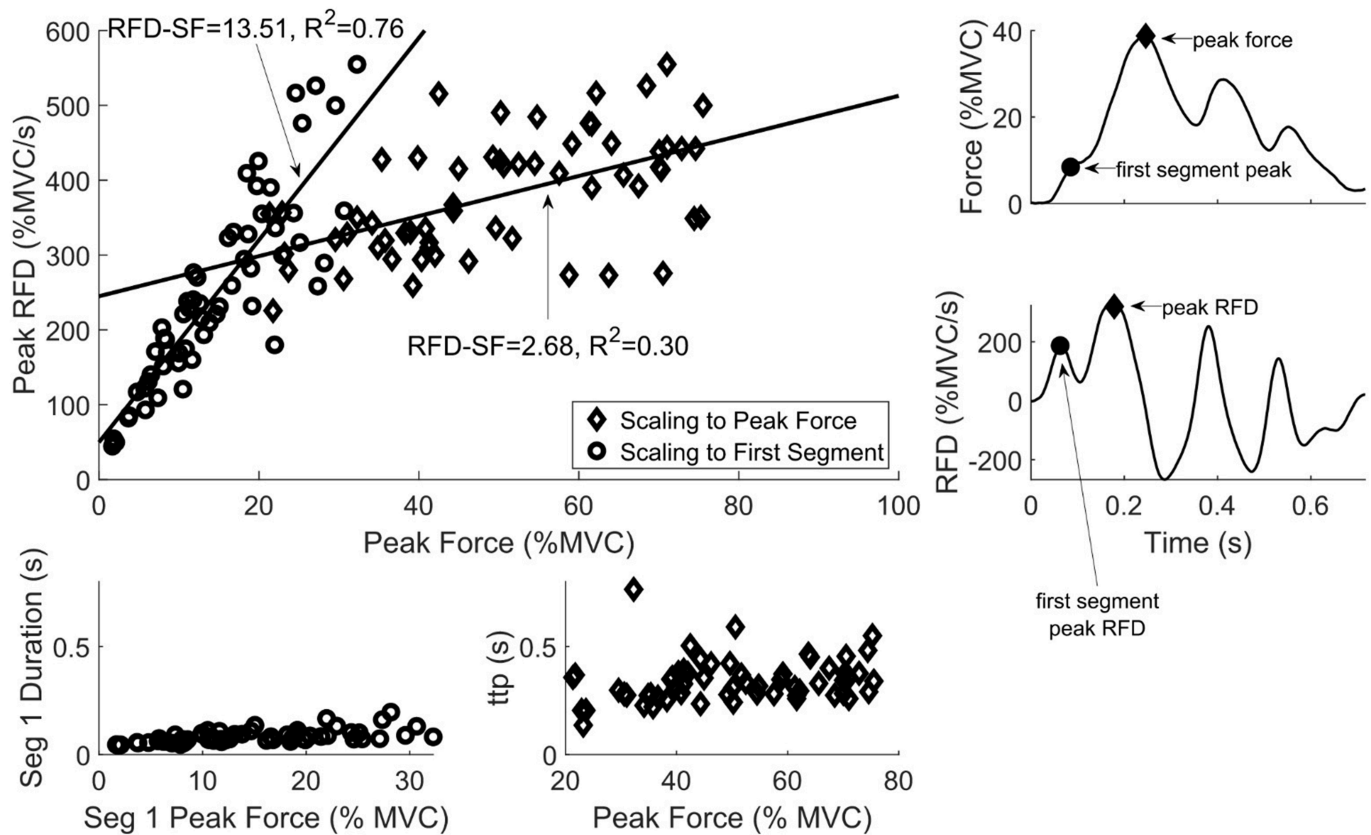


Figure 5

Left panel indicates relationships between the peak rate of force development (RFD) and peak force measured at the first segment (circles) and at maximum force (diamonds) for a person with Parkinson's disease with 90% of their pulses requiring multiple segments to reach peak force. The duration of the first segment (Seg 1) and the time to peak force (ttp) for each force pulse are also plotted in the top right and bottom right panels, respectively.

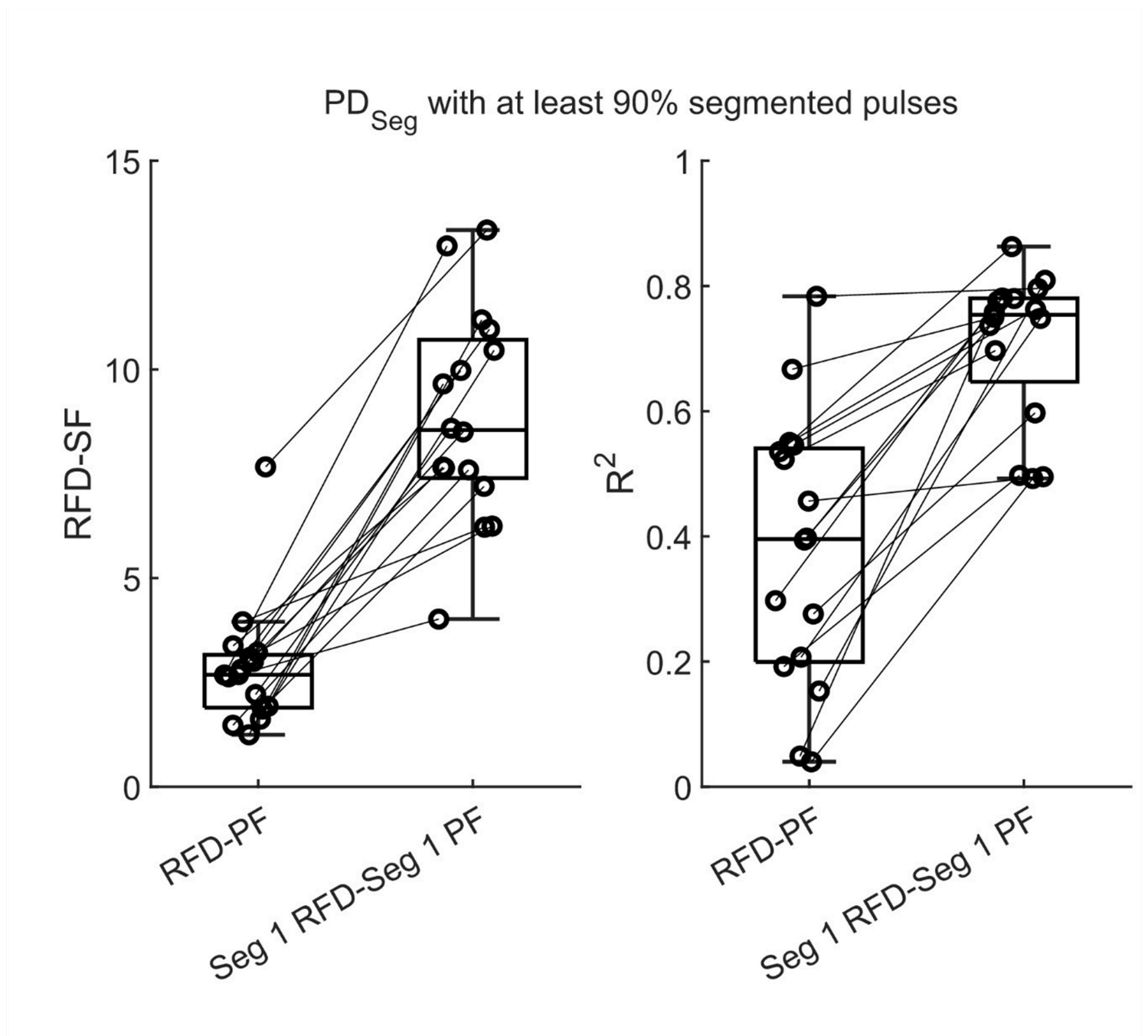


Figure 6

Box plots indicate the slope (RFD-SF) and R^2 of the relationship between the maximum rate of force development and maximum peak force (RFD-PF) and the relationship between the first segment peak rate of force development and peak force (Seg 1 RFD-Seg 1 PF) in people with Parkinson's disease who required multiple segments to reach peak force in at least 90% of their pulses. Each individual's data are represented by circles and connected with lines.

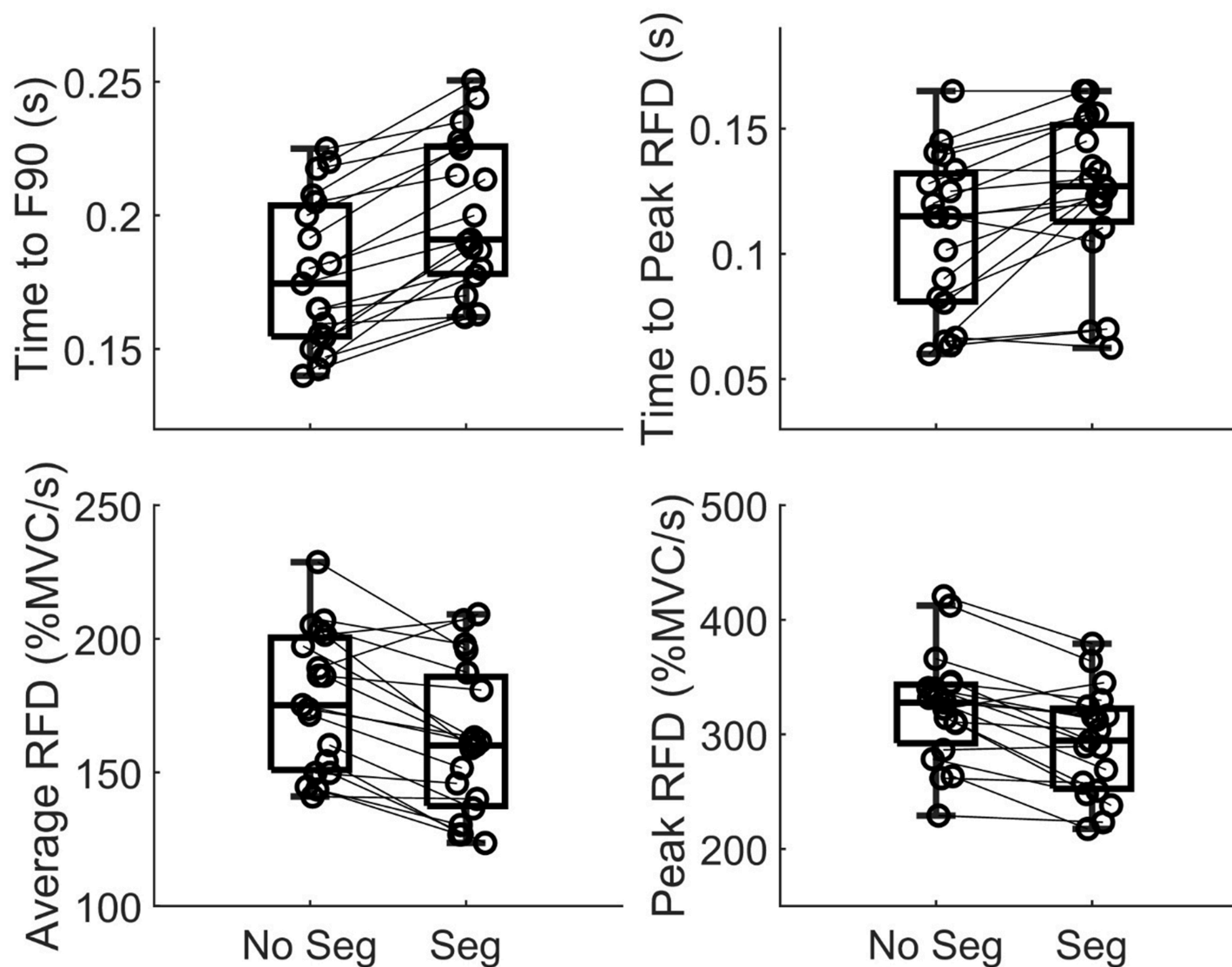


Figure 7

Box plots that indicate within-subject relationships for variables calculated from their pulses that reached peak force in one segment (No Seg) compared to variables calculated from their pulses that reached peak force in multiple segments (Seg). Individual subject data is represented by circles and connected with lines. RFD=rate of force development, F90=90% of peak force.