ASSESSING LIMB PROPULSION AND GAIT KINETICS USING RESISTANCE IN EARLY-STAGE PARKINSONS DISEASE

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Introduction: People with Parkinson's Disease (PD) often experience deviations in gait including freezing of gait, decreased step lengths, and a shuffling step pattern that may lead to falls. Despite the known deficits in spatiotemporal aspects of gait, relatively little is known about limb and joint kinetics in people with PD during ambulation.

Purpose: We sought to understand limb and joint kinetics associated with gait in people with PD and examined whether deficits (and a concurrent reserve) in the push-off and swing phases of gait might contribute to the development of shortened step lengths. We hypothesized that participants with PD will have reduced hip flexor torque during swing phase and reduced ankle plantar flexor torque during push-off phase compared to unimpaired individuals yet be able to take longer steps through an increase in both hip flexor and ankle plantar flexor torque.

Methods: We recruited 11 older adults without a history of neurologic involvement and 9 participants with idiopathic PD with a Hoehn & Yahr stage of 1 - 3. Testing was completed in a single session. All participants walked on an instrumented treadmill for 1 minute each at their self-selected comfortable and fast gait speeds. Then, participants walked with posterior resistance applied to the pelvis by a long elastic tubing. The resistance force was systematically increased from 0 to 10% bodyweight resistance in 2.5% increments to challenge push-off magnitude (e.g., limb propulsion). Lastly, participants walked with ankle resistance at 2.5% body weight to challenge leg swing and limb advancement. Spatiotemporal, kinematic, and force data were collected and compared between groups and conditions.

Results: We did not observe differences between the Control and PD groups for most spatiotemporal and kinetic outcomes (step length, propulsive impulse, trailing limb angle (TLA), plantarflexion (PF) impulse, and hip flexor (HF) impulse). However, the PD group exhibited significantly decreased hip extension (HE) impulse ($p=0.005$) compared to controls. When challenging limb propulsion at the pelvis, participants in the PD group increased their propulsive impulse ($p<0.001$), PF impulse ($p<0.001$), and HE impulse ($p<0.001$), while they decreased the HF impulse ($p<0.001$). With 2.5%BW resistance at the ankles, participants with PD were able to increase step length ($p<0.001$), ankle PF ($p<0.001$), and HE impulse ($p<0.001$), but did not alter HF impulse.

Conclusions: These data suggest that in early-stage PD, gait kinetics are not as disrupted as originally thought. However, the presence of reduced HE torque might be an early indicator of gait degeneration in this neurodegenerative disease. Our participants with PD were able to increase HE torque in response to demands, but they exhibited difficulty increasing HF torque when challenged. Given the deficits in HE, it is encouraging that HE seemed to be the source of increased force against resistance, providing a possible target for interventions. Likewise, we found that challenging leg swing appeared easily surmountable by our participants with PD, possibly indicating that our resistance force was not large enough. Nevertheless, participants with PD were able to increase leg swing against resistance, suggesting a reserve in leg swing mechanics.

Introduction:

Parkinson's disease (PD) is a neurodegenerative disease[1] that disrupts motor processing and planning and elicits altered gait. Specific gait impairments include decreased step length and increased cadence, which lead to the hallmark "shuffling" pattern commonly termed Parkinsonian gait.[2] Additionally, the presence of rigidity and stiffness in people with PD suggests diminished joint movements, whereas the presence of a flexed trunk (i.e., stooped posture) implies a redistribution of joint moments.[3] Despite extensive evidence of spatiotemporal gait changes with Parkinson's disease, little is known about the mechanics that underlie these changes.

There is, however, ample evidence regarding the neurologic contributions to altered movement in PD. For example, evidence from primates suggests neurons in the globus pallidus discharge at the end of each movement within a sequence, thus initiating automatic sub movements during performance tasks like locomotion.[4] Damage to basal ganglia and globus pallidus neurons with PD can disrupt movement sequences, which affects the timing and amplitude of successive steps.[5,6] Moreover, due to reduced automaticity and rhythmicity, individuals with PD may also experience episodes of 'freezing', which are brief moments of absent or reduced forward progression despite the intention of movement.[7] Although observations of reduced forward progression are considered to be a problem of 'automaticity', there is likely a mechanical contribution. In fact, reduced limb propulsion is common in many other patient populations that present with loss of muscle strength, motor control, and/or central drive.[8,9] Indeed, reduced limb propulsion has been closely associated with reduced step lengths, which is a common occurrence in people with PD.[10,11] Understanding the joint and limb kinetics

associated with these known differences in spatiotemporal parameters for people with PD would allow for successful treatment targets.

Several authors and researchers have proposed a distal to proximal redistribution of joint torques in PD.[3,12] However, this redistribution is also an observed phenomenon in otherwise healthy older adults and may not be a consequence of PD.[13] During both level and inclined walking, older adults demonstrate a shift in joint torque to the hip from the ankle, which is thought to be caused by strength, power, and exercise capacity.[13,14] Those with greater exercise capacity and strength demonstrate higher levels of biomechanical plasticity, allowing them to better adapt to challenging conditions.[15] Despite similar biomechanical plasticity, when people with PD are walking OFF medication, ankle plantarflexion moments are reduced compared to age-matched controls which is why we ensured to test participants during ON periods.[16] Importantly, PD medication can impact spatiotemporal, kinematic, and kinetic aspects of gait in people with PD. Baudendistel and colleagues noted increased step lengths, gait speed, limb propulsion, and ankle and hip kinetics when walking ON medication compared to OFF medication.[17] Studies have also found an increase in the neuromuscular activity of distal musculature with the use of anti-Parkinson's medication such as Levodopa.[18] However, even when patients are on "optimal prescription levels," there is still a notable decrease in gastrocnemius activation with an increase in proximal muscle activation compared to healthy older adults.[19]

The potential combination of deficits in limb mechanics and amplitude of movement introduces safety concerns during gait and balance tasks for people with PD.[20] Due to the progressive nature of PD, symptoms worsen over time and the spatiotemporal changes in gait can increase the risk of falls.[21] Falls often come with debilitating consequences that not only

impact activity levels and quality of life but also increase the risk of mortality.[22,23] Although there is no cure for PD, dopaminergic agents such as Levodopa and Carbidopa are commonly used by people with PD to improve motor complications. However, due to the pharmacokinetics and sensitivity to dosages of the medications, there is an on/off phenomenon noted by worsening or fluctuating motor function as the medication wears off.[24,25] Furthermore, there is limited research that supports medication's efficacy in improving but not eliminating gait and balance deficits, indicating a need for additional focus on non-pharmacological treatment approaches.[26]

Physical therapy interventions can successfully mitigate the consequence of gait and balance deficits for people with PD.[27] Interventions targeting increased step lengths have been a particular focus of therapy to address balance and falls. Gait training using auditory and visual cues for individuals with PD can induce longer steps.[28] The relationship between step lengths and limb propulsion[17,29] may suggest that individuals with PD can increase propulsive forces during gait. This mutability of gait in people with PD provides an opportunity to examine the underlying kinetics associated with impaired gait, as well as identify potential targets for therapeutic intervention.

Given the known presence of spatiotemporal impairments, this study aims to determine the limb and joint mechanics associated with reduced step lengths in people with PD. Because shortened step lengths can be due to both reductions in push-off force (stance limb) or reductions in swing leg advancement, we carefully designed a series of experiments to assess each of these potential mechanical contributions. First, we hypothesized that people with PD would exhibit diminished step lengths and gait speeds compared to unimpaired control subjects due to diminished limb propulsion arising from reduced plantarflexion and hip extension moments. Additionally, we hypothesized that the reduced step lengths will also be due to decreased hip

flexion (swing) moments. Finally, we hypothesized that people with PD would have the capacity to take longer step lengths through an increase in hip flexor moment (swing) and ankle plantar flexor moments during push-off.

Methods

Recruitment of Participants

We recruited participants with PD through outpatient neurology and physical therapy clinics and local PD support groups. All participants had a clinical diagnosis of idiopathic PD characterized by the asymmetrical onset of at least 2 of the 3 cardinal signs (tremor, bradykinesia, or rigidity) as well as the presence of mild to moderate gait or balance impairment characterized by a rating of 1-2 on MDS Unified Parkinson's Disease Rating Scale (MDS-UPDRS) item 10. Additionally, participants' disease progression had to be classified as mild to moderate severity (i.e., Hoehn and Yahr 1 – 3). To determine how participants walk in their daily lives, we chose to perform all measurements ON medication. We also recruited a group of similarly aged older adults who met all additional criteria to serve as a control.

All participants were only included if they self-reported the ability to walk at least 5 minutes at their comfortable gait speed on a treadmill. Participants were excluded if they presented with any of the following: uncontrolled cardiorespiratory or metabolic disease, vestibular dysfunction that affects gait and/or balance, implanted deep brain stimulator, dementia (MOCA score <21), history of traumatic brain injury, difficulties with vision or hearing that would impair the ability to hear cues from researchers or ambulate safely, communication impairments which could impact understanding of experimental procedures, a recent orthopedic surgery (within the last 6 months), or other neurological or orthopedic issues that would affect

gait. All participants signed an informed consent form approved by the University of North Carolina at Chapel Hill IRB (# 21-1856) prior to participation.

Procedures

Testing began with the collection of outcome measures intended to describe the PD group: Montreal Cognitive Assessment (MoCA), Freezing of Gait Questionnaire (FOGQ), and Mini-Best test. The Movement Disorder Society -Unified Parkinson's Disease Rating Scale (MDS-UPDRS) was completed by a neurologist. All participants then performed walking passes on a 20' (6.1 m) Zeno mat (Protokinetics, Havertown, PA), during which they were cued to walk "at your comfortable speed" for two passes, and "as fast as possible, while still being safe" for two additional passes. We used ProtoKinetics Movement Analysis Software (PKMAS) (Havertown, PA) to measure comfortable gait speed (CGS), fast gait speed (FGS), and the respective cadence for each speed.

All gait testing was completed on a dual-belt instrumented treadmill (Bertec FIT, Columbus, OH). Participants wore an overhead harness that prevented falls but did not provide body weight support. Everyone was given a few minutes to acclimate to walking on the treadmill as we slowly increased the velocity to reach their comfortable overground speed. In cases where participants were unable or unwilling to complete the treadmill trials at their baseline speed, we adjusted the treadmill to 80% of their baseline speed. Four PD participants were unable to complete the sessions without limited use of handrails. In these instances, participants were instructed to use "fingertip assistance" if they were concerned about losing their balance. Rest breaks were provided as needed between test conditions.

To comprehensively assess potential biomechanical subcomponents of gait associated with shuffling steps (commonly observed as shortened steps and rapid cadence), we probed push-off forces and swing limb advancement with systematic experiments. Participants began testing by completing comfortable gait speed (CGS) on the treadmill for one minute. All participants with PD then walked at their fast gait speed (FGS) for 60 seconds. After a brief rest break, participants with PD then walked at their comfortable speed with a posterior impeding force applied to either the pelvis (to resist limb propulsion) or the ankles (to resist anterior leg swing). Such resistance during gait is intended to elicit greater internal joint moments and muscle activation magnitudes.[30] The pelvis and ankle resistance conditions were counterbalanced by alternating which condition subjects completed first with at least a two-minute washout period provided between conditions.

Propulsion Restraint

We used a manual posteriorly impeding force on the pelvis (i.e., center of mass) with black elastic tubing (Thera-Band, Akron, OH) fixed to a gait belt.[31] Force was measured in real-time using a tension load cell (MLP-100, Transducer Techniques, Temecula, CA) sampling at 960 Hz. Care was taken to ensure that force was directly posterior to the direction of the participant's gait. The magnitude of the resistance was set relative to the participant's body weight (%BW) and was applied for 15-second intervals in a stepwise pyramid (ascending 0 to 10%BW in 2.5%BW increments, followed by descending from 10 to 0%BW). Prior modeling work has suggested that such a posterior resistance at the pelvis results in increased hip extension moments during gait.[30] Participants were instructed to remain in the center of the treadmill to minimize changes in the impeding force that might occur by drifting forwards or backward on the treadmill.

Swing Limb Advancement Restraint

We attached black elastic tubing to the posterior aspect of each ankle via a padded ankle cuff. To achieve bilateral leg swing restraint the ankle resistance was set to peak at 2.5%BW for each leg (just prior to heel strike). A similar impeding force has been suggested to strategically increase hip flexion moments during gait.[30] Because we were concerned that this impeding force would create artificially shortened steps, we included a condition that required participants to step to a metronome set to 85% of their typical overground cadence.[32] To ensure that the metronome did not influence stepping, we also included a second control condition with participants stepping to a metronome set to 100% of overground cadence. Therefore, we had three randomized conditions that employed the posterior impeding force at the ankles (i.e., no metronome, metronome set to 85%, and metronome set to 100% of overground cadence).

Data Collection

During all conditions, we recorded segment kinematics by tracking 14-mm retroreflective markers placed on both limbs and pelvis. Namely, we placed anatomical markers on the second metatarsal head, 5th metatarsal head, center of the posterior calcaneus, medial and lateral malleoli, medial and lateral femoral condyles, greater trochanter, iliac crest, radial styloid, acromioclavicular joint, and the sternoclavicular joint. Clusters of four markers were adhered to the posterolateral thigh and shank, and a cluster of three markers was fixed to the posterior pelvis to serve as tracking markers. All medial lower extremity anatomical markers were removed after performing a standing calibration, and prior to recording walking trials. The 3-dimensional locations of the markers were collected using an 8-camera motion capture system (Vicon, Los Angeles, CA), recording at 120 Hz. Additionally, ground reaction forces, and the restraining force recorded via the load cell were recorded at 960 Hz. Marker trajectories and ground reaction

forces were filtered using a 6 Hz and 20 Hz Butterworth filter, respectively. Joint angles and joint moments were calculated through inverse dynamics in Visual3D (C-Motion, Germantown, MD). From these time series curves, we computed a series of spatiotemporal and kinetic outcome measures using custom code (Labview, National Instruments, Austin, TX). Step length was measured as the anteroposterior distance between heel markers at each heel strike. Cadence was computed from the inverse of step time. We measured the propulsive impulse as the integral of the anterior-directed GRF. Hip extension (early stance), hip flexion (late stance/swing), and ankle plantarflexion impulses were computed as the integral of the respective joint moment curves. All measures were computed bilaterally and averaged across limbs.

Data Analysis

We used SPSS (v27, IBM, Aramonk, NY) for all statistical analyses. First, we evaluated the difference between the PD group and the control group by performing paired samples t-tests. Next, we sought to determine the capacity to modulate limb propulsion in the PD group, as well as ascertain kinetic alterations associated with control of limb propulsion. Specifically, we used separate repeated-measures ANOVAs (repeated for impeding force level) for limb propulsion and joint impulses. In the presence of significant main effects, we performed post-hoc paired samples t-tests between each restraining force condition and the 0% control condition. Finally, we intended to evaluate the ability of people with PD to maintain or extend their step length against swing limb resistance. We used separate repeated-measures ANOVAs (repeated for condition) to assess differences in step length and related joint impulses. When significant main effects were observed, we performed paired samples t-tests with a Bonferroni correction as post-hoc analysis. We used effect sizes (η_p^2 and Cohen's d), as appropriate, and an α=0.05 to determine significance.

Results

We recruited a total of nine participants with idiopathic PD (4 female and 5 male) and 11 Control (9 female and 2 male) participants. None of the participants reported using assistive devices for household or community mobility. The distribution of the PD participants' Hoehn &Yahr Stages were 4, 2, and 3, in stages 1-3 respectively which indicated mild to moderate levels of disability. The average age of participants was 61.6 ± 14.5 years, and the average number of years since diagnosis was 5.4 ± 3.5 years in the PD participant group (see Table 1). Lastly, their baseline overground comfortable gait speed was 1.17 ± 0.17m/s and fast gait speed was 1.52 ± 0.24 m/s (See Table 2).

In terms of the control group, there were 11 participants (9 female and 2 male), with an average age of 62.5 ± 10.5. Their baseline overground gait speed was 1.44 ± 0.19 (See Table 2).

PD vs Control

We observed minimal differences in spatiotemporal and kinetic gait parameters between our participants with PD and unimpaired older adults (see Table 2). In particular, we noted comparable gait speeds, step lengths, and cadence between the groups (all $p \geq 0.454$). Additionally, there was no difference between groups for propulsive impulse, plantarflexion impulse, or hip flexion impulse (all $p \geq 0.302$). However, we did note a significantly smaller hip extension impulse in people with PD compared to the control group ($p=0.005$; $d=1.44$) (See Figure 1, 2, and 3).

Propulsion Restraint

In response to a posterior impeding force to the center of mass, intended to require greater limb propulsion, participants with PD significantly increased their propulsive forces with each

successive increase in resisting force ($p<0.001$; $\eta_p^2=0.97$) (see Table 3). Notably, when faced with 10% BW resistance, the participants with PD produced 204% of their typical propulsive impulse. Despite the increase in limb propulsion, we did not note any change in step length ($p=0.550$; $\eta_p^2=0.07$) or cadence ($p=0.546$; $\eta_p^2=0.07$) in response to the application of impeding force to the pelvis. Throughout the stepwise pyramid, as propulsion restraint increased, participants consistently increased their hip extension impulse (see Figure 4). There were limited significant changes seen in hip flexion and plantarflexion impulses, particularly at higher resistances.

Qualitatively, we observed that participants adopted a slight anterior trunk lean that increased with increasing force, and a more notable arm swing when walking with pelvis resistance.

Swing Leg Advancement Restraint

We observed a significant main effect of this condition for the influence of the ankle resistance on step length in our participants with PD ($p<0.001$; $\eta_p^2=0.74$, Figure 4). Specifically, we noted that participants took significantly longer steps when walking with swing leg resistance and the metronome set to 85% of typical cadence ($p=0.004$). However, the resistance itself did not appear to alter step lengths when walking with (i.e., 100%) or without the metronome (both $p=1.000$) (See Figure 5). In evaluating joint kinetics (see Table 4), we observed a main effect for the condition in hip flexion impulse ($p=0.043$; $\eta_p^2=0.28$). Nevertheless, the significant main effect did not reveal any differences in post-hoc evaluations (all $p>0.415$). In contrast, the plantar flexor impulse was also influenced by the swing leg resistance ($p<0.001$; $\eta_p^2=0.76$) but was only larger when paired with the 85% metronome (i.e., enforced long steps) ($p=0.002$). Finally, the swing leg restraint influenced the hip extension impulse ($p<0.001$; $\eta_p^2=0.82$), with greater values observed in all swing leg restraint conditions (all $p<0.002$) (see Table 4).

Discussion

Our hypothesis that people with PD would walk with short, rapid steps due to decreased limb propulsion and swing leg advancement compared to unimpaired older adults was not supported by our data. Although we hypothesized that the expected altered spatiotemporal parameters would be due to underlying joint kinetic changes (i.e., less hip flexor torque (during swing) and less ankle plantar flexor torque during push-off compared to unimpaired individuals), we only observed deficits in hip extension moments in participants with PD. Additionally, we found that individuals with PD relied on the hip extensors to increase limb propulsion (as the first strategy), rather than increase hip flexor or plantar flexor torques, except at higher resistance levels. This data provides valuable information about gait in people with PD and has implications for rehabilitation strategies.

The presence of altered joint kinetics in people with PD has been understudied, with a clear emphasis on spatiotemporal characteristics. Here, we report that people with mild to moderate PD (H&Y stage 1-3) do not exhibit characteristic spatiotemporal alterations yet exhibit robust reductions in hip extension torque at baseline. The fact that our participants with PD demonstrated no significant differences in spatiotemporal parameters and limited differences in joint kinetics suggests that gait kinetics in early-stage PD are perhaps not as disrupted as originally thought. Kuhman and colleagues noted that compared to older adults without neurological deficits, individuals with PD show little ability to alter joint-level kinetics with greater gait speed demands.[14] However, their results evaluated total joint work and did not separate out the flexion and extension components.

Our finding of reduced hip extension torque in the PD group during typical walking may serve as an early indicator of neurodegenerative disease. Our participants were early in the

progression of PD and were on medication during testing. In more advanced stages of PD, postural instability is common, regardless of medication, and often produces a forward trunk lean which subsequently increases the hip extension moment.[33] We postulate that this increase in forward trunk lean may serve as a compensatory strategy to overcome the decreased hip extension torque by shifting the ground reaction force anteriorly. Perhaps targeting the reduced hip extension torque early in the disease progression could minimize or eliminate the development of other impairments that commonly arise (e.g., forward trunk lean) as the disease progresses. Such an approach might also minimize any increased tone in the hip flexors from a stooped posture, ultimately improving the hip-to-trunk torque ratio.[34,35]

People with PD were capable of eliciting substantial increases in limb propulsion, driven primarily by increased hip extension torque. That participants opted to increase hip extension torque with each increase in resistance to the center of mass, suggests that there remains considerable capacity for increasing torque when challenged or required, and offers the possibility of a focus for rehabilitation. Prior work on people with stroke suggested that people alter propulsive limb forces by increasing the trailing limb angle to help direct the ground force reaction more anteriorly,[36,37] which may coincide with increased hip extension torque. Somewhat surprisingly, we only observed a change in plantarflexion torque when participants were challenged with a resistance force of 10%BW. This finding suggests that deficits in PF may not exist, and instead, participants responded by increasing torque across the joint (i.e., hip) that did show deficits. Interestingly, though, participants significantly reduced the hip flexion moment when faced with greater resistance ($\geq$ 7.5%BW) which can provide a basis for the optimal prescription of the resistance magnitude needed to improve hip extension torque without compromising the hip flexors. This is consistent with previous literature that supports an inverse

relationship between hip flexor and plantarflexion power generation.[38] It has been suggested that the concentric activation of hip flexors to initiate leg swing can inhibit the production of ankle power generation needed for limb propulsion, and thus as participants attempted to increase propulsion in response to resistance, they subsequently reduced the hip flexion impulse.[39]

The resistance at the ankle was intended to challenge swing limb advancement and demonstrated that individuals with mild-to-moderate symptoms of PD can increase step lengths. The resistance at the ankle is believed to require greater hip flexor torque during swing, [30] but our findings instead showed that participants decreased their hip flexion torque and instead increased their hip extension torque. Importantly, participants were able to increase their step length when challenged with a slow metronome. Previous evidence shows that a slower cadence metronome can enforce a longer step length, which was demonstrated in our participants.[28] In this condition, step length was challenged with both a resisting force to the leg, as well as the presence of the slow tempo metronome on a fixed speed treadmill.[32] Given the lack of increased step length in other conditions, it appears that the slow tempo metronome was required to increase step length. Importantly, our PD participants did not have a deficit in step length at baseline compared to healthy controls. This may account for some of the lack of changes when challenged without a metronome, and at the 100% cadence trial. Nevertheless, it is encouraging that participants could increase their step length with an additional resistance impeding their swinging leg. We propose that such a technique may provide the means to train for increased step lengths with resistance. Interestingly, participants increased step length through increased plantar flexor and hip extensor torque. This could indicate an inability to increase hip flexor torque to overcome challenges. Furthermore, it may also suggest that the increase in step length is achieved through enhanced limb propulsion, rather than increasing swing limb mechanics. Nevertheless, reduced hip flexion

kinetics are well known to accompany increased limb propulsion demands[40,38] and thus, may simply be a consequence of the task requirement, rather than a functionally limiting impairment. Furthermore, research shows that hip extension and ankle plantarflexion kinetics are primary factors in dictating step length in older adults.[29] These data do not support our hypothesis that participants with PD would increase the hip flexor moment to increase step length.

Treadmill and gait training are common tools used during PD rehabilitation.[41] Functionally relevant strengthening may be a valuable addition to rehabilitation for people with PD. Previously, this idea has been incorporated into gait training on an incline, such that gravity becomes the applied resistance during gait. For people with chronic stroke, walking on an inclined treadmill leads to improved paretic step lengths, and increased gait speed.[42] Direct gait resistance training has shown benefit in other neurological populations and can result in neuromuscular adaptations. Individuals post-incomplete spinal cord injury can increase the amplitude of their hip flexor muscle activity when assisted in swing phase.[43] Individuals following a stroke in their paretic limb, have demonstrated a propulsive reserve while walking against resistance at the waist, which continued after removal of the impeding force.[37] The data found during this study suggests that using an impeding force to gait train can elicit upregulation of joint torques that are reduced in PD participants compared to healthy controls (i.e., hip extension impulse). As an intervention, this could be both a functional activity and targeted strengthening.

Limitations

There were several limitations that need to be acknowledged. First, we recruited individuals with mild-moderate PD (Hoehn & Yahr stage 1-3) and performed all testing ON medication. As a result, these results cannot be extended to more advanced stages or

circumstances in which people with PD are OFF medication. Next, we did not have the control participants undergo trials in which we systematically challenged biomechanical subcomponents of gait, leaving us unable to interpret our findings in this regard. Additionally, it is possible that the resistance applied to the ankle to impede swing limb advancement was insufficient to adequately challenge some of the participants. We selected 2.5%BW based on our pilot testing but acknowledge that it is possible that a larger resistance could have resulted in different findings. Nevertheless, we chose a magnitude that was twice the value used previously in people with stroke.[44]

Tables/Figures

Table 1. Parkinson's Group Demographics

PD Demographics											
	CGS (m/s)	**FGS (m/s)**	**CGS Cadence (bpm)**	**Hoehn & Yahr**	**MDS UPDRS**	**MOCA**	**FOG**	**Minibest Test**	**Age**	**Disease Duration (years)**	**Levodopa Dose (mg/day)**
1	0.89	1.00	105	3	39	25	5	23	81	3	800
2	1.14	1.56	121	3	35	30	2	26	64	5	300
3	1.49	1.73	113	1	20	30	4	27	41	5	150
4	1.2	1.62	103	2	43	27	0	26	75	9	400
5	1.2	1.74	121.5	1	11	30	4	26	69	11	300
6	1.28	1.69	112.6	1	31	27	3	25	52	1	150
7	1.06	1.43	102.3	1	10	28	0	26	45	2	100
8	1.04	1.29	101	3	24	30	9	28	75	9	700
9	1.22	1.6	108.16	34		30	1	25	52	4	300
Average	1.17 ± .17	1.52±0.2	109±8	1.9±0.9	26.3±13.	28.6 ± 1.88	3.1± 2.85	25.8 ± 1.39	61.6±14.5	5.4±3.5	356±244

Table 2. PD vs. Control Group Results

	Control Group	**PD Group**	**p-value**
CGS (m/s)	1.20 ± 0.18	1.14 ± 0.19	.508
Propulsive Impulse (%BW*sec)	0.030 ± 0.005	0.028 ± 0.005	.444
Step Length (m)	0.58 ± 0.07	0.55 ± 0.10	.454
Cadence (steps/min)	117.2 ± 12.1	113.7 ± 8.1	.467
PF Impulse (%BW*sec)	-0.25 ± 0.07	-0.28 ± 0.05	.302
Hip Ext Impulse (%BW*sec)	**-0.11 ± 0.05**	**-0.05 ± 0.03**	**.005***
Hip Flex Impulse (%BW*sec)	0.06 ± 0.03	0.07 ± 0.04	.345

Key: *= significantly (P<.05) different

Table 3. PD Pelvic Restraint Condition Results

	0% BW	2.5% BW	5% BW	7.5% BW	10% BW
Hip Ext **(%BW*sec)**	-0.044± 0.027	**-0.052±0.028*† (p=0.001)**	**-0.063±0.036* (p=0.01)**	**-0.075±0.035*† (p=0.001)**	**-0.091±0.039*†** (p<0.001)
Hip Flex (%BW*sec)	0.080± 0.037	0.077±0.037	0.076±0.038	**0.070±0.035*†(p=0.046)**	**0.065±0.033* (p=0.011)**
PF (%BW*sec)	-0.272± 0.048	-0.269±0.049	-0.275±0.050	-0.281±0.050	**-0.294±0.047* (p=0.012)**

Key: *= significantly (P<.05) different from 0% condition; †= significantly different from preceding condition

Table 4. PD Ankle Restraint Condition Results

	CGS	**Ankle (no metronome)**	**Ankle 100**	**Ankle (85% metronome)**
Propulsive Impulse (%BW*sec)	0.281± 0.00535	**0.0302** ± **0.00535*** **(P=.020)**	**0.0336** ± **0.00495*** **(P=.001)**	**0.0429** ± **0.00949*** **(P=.002)**
HF Impulse (%BW*sec)	0.0746 ± 0.0415	0.0656 ± 0.0311	0.0629 ± 0.0343	0.0603 ± 0.0264
PF Impulse (%BW*sec)	-0.282 ± 0.051	-0.280 ± 0.0491	-0.293 ± 0.0389	**-0.341** ± **0.0634*** **(P=.002)**
HE Impulse (%BW*sec)	-0.0451 ± 0.0282	**-0.0659** ± **0.0327*** **(P=.002)**	**-0.0788** ± **0.0431** **(P=.002)**	**-0.0934** ± **0.0435** **(P=.000)**

Key: *= significantly (P<.05) different from CGS condition

Figure 1. PD vs. Control Hip Kinematics

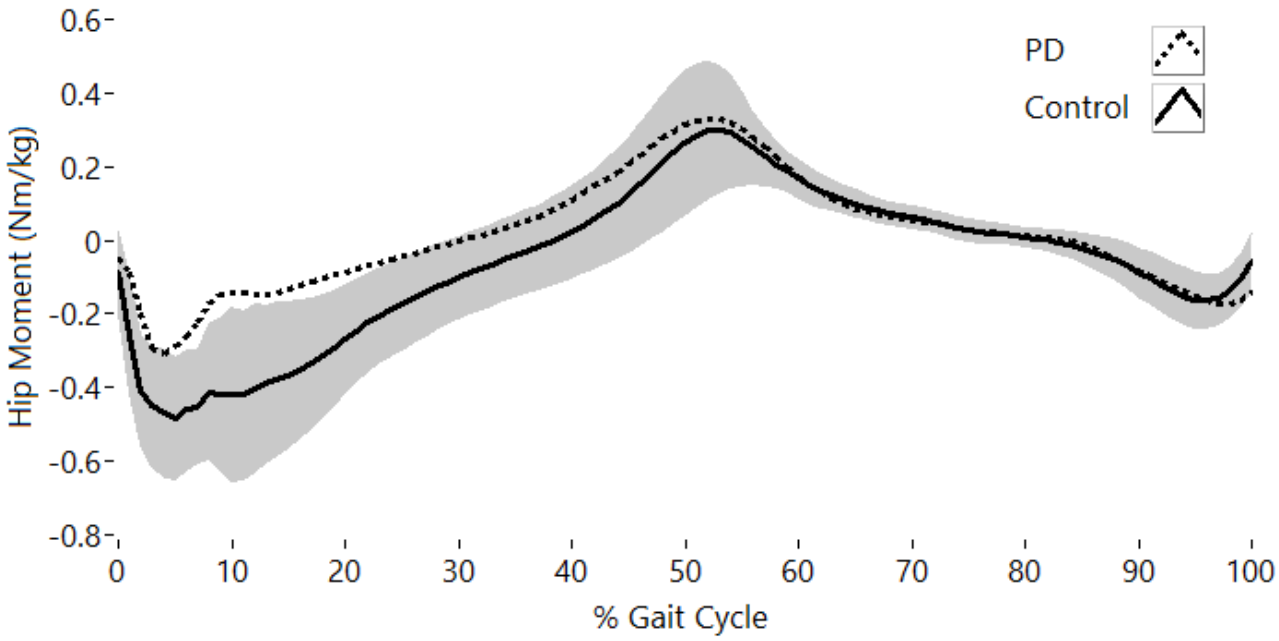


Figure 2. PD Vs. Control Ankle Kinematics

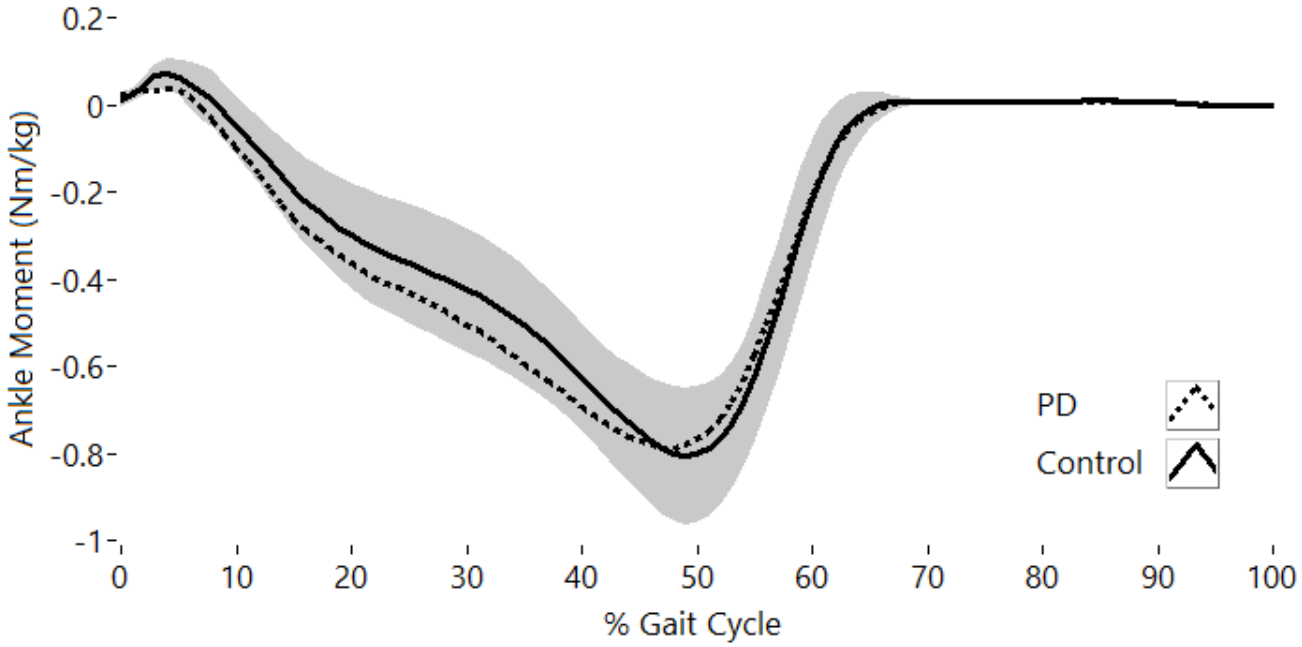


Figure 3. PD vs. Control GRF/Propulsion

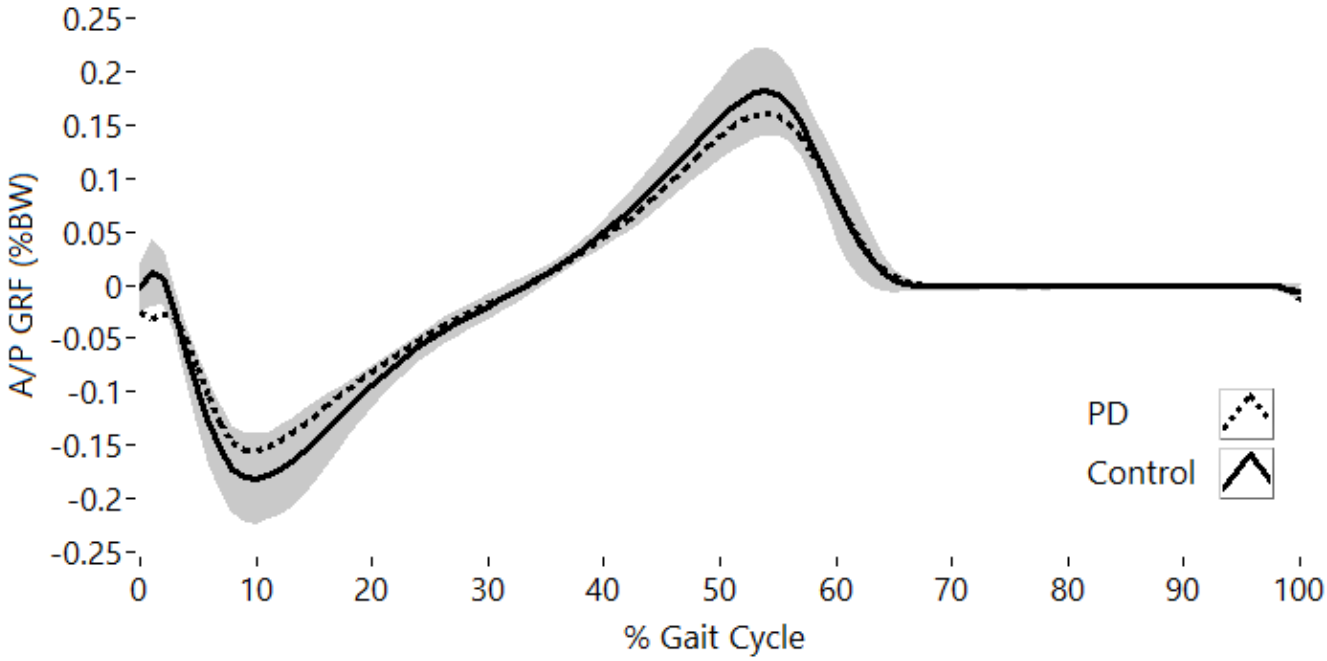


Figure 4. Propulsive Impulse with Pelvis Restraint

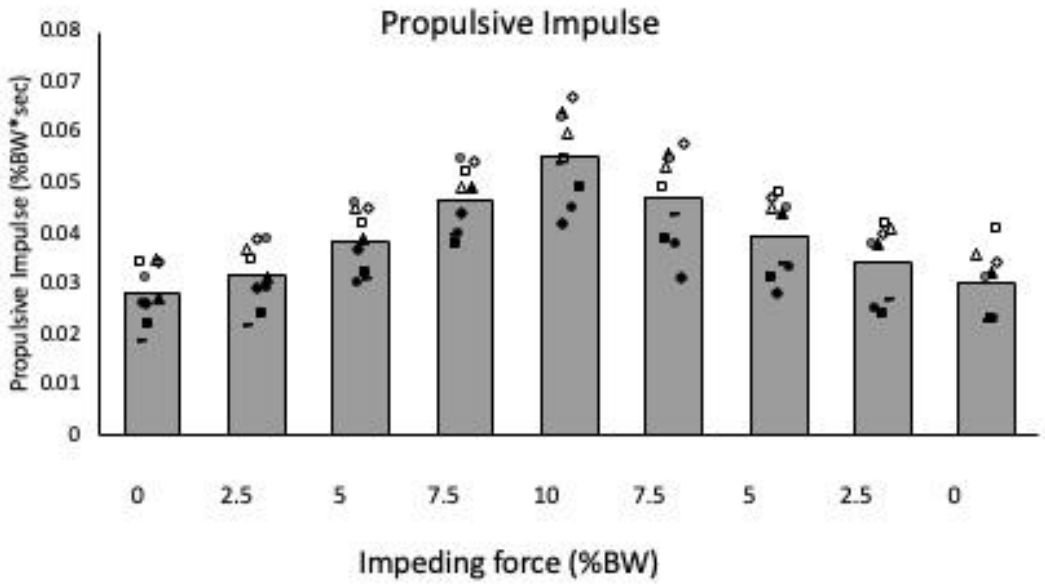
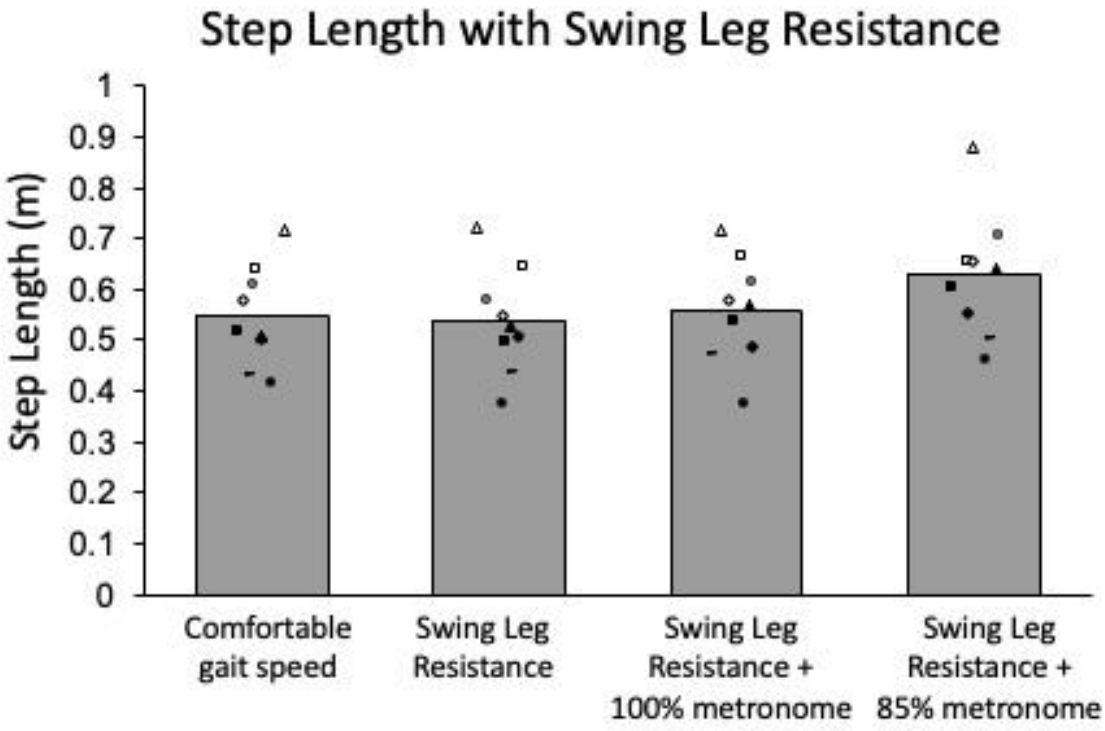


Figure 5. Step Length with Swing Resistance



Bibliography

1. Sveinbjornsdottir S. The clinical symptoms of Parkinson's disease. *J Neurochem*. 2016;139 Suppl 1:318-324. doi:10.1111/jnc.13691

2. Mazzoni P, Shabbott B, Cortés JC. Motor control abnormalities in Parkinson's disease. *Cold Spring Harb Perspect Med*. 2012;2(6):a009282. doi:10.1101/cshperspect.a009282

3. Skinner JW, Lee HK, Hass CJ. Redistribution of joint moments and dynamic balance control during sit to stand task in persons with Parkinson's disease. *Parkinsonism Relat Disord*. 2021;90:21-22. doi:10.1016/j.parkreldis.2021.07.027

4. Brotchie P, Iansek R, Horne MK. Motor function of the monkey globus pallidus. 1. Neuronal discharge and parameters of movement. *Brain*. 1991;114 (Pt 4):1667-1683. doi:10.1093/brain/114.4.1667

5. Patla AE. *Adaptability Of Human Gait*. Elsevier; 1991:453.

6. Morris ME, Iansek R, Matyas TA, Summers JJ. The pathogenesis of gait hypokinesia in Parkinson's disease. *Brain*. 1994;117 (Pt 5):1169-1181. doi:10.1093/brain/117.5.1169

7. Giladi N, Nieuwboer A. Understanding and treating freezing of gait in parkinsonism, proposed working definition, and setting the stage. *Mov Disord*. 2008;23 Suppl 2:S423-5. doi:10.1002/mds.21927

8. Ihira H, Shimada H, Suzukawa M, Furuna T, Matsuyama K, Ishiai S. Differences between Proximal and Distal Muscle Activity of the Lower Limbs of Community-dwelling Women during the 6-minute Walk Test. *J Phys Ther Sci*. 2012;24(2):205-209. doi:10.1589/jpts.24.205

9. Awad LN, Hsiao H, Binder-Macleod SA. Central drive to the paretic ankle plantarflexors affects the relationship between propulsion and walking speed after stroke. *J Neurol Phys Ther*. 2020;44(1):42-48. doi:10.1097/NPT.0000000000000299

10. Gait Analysis: Normal and Pathological Function. *Journal of Sports Science & Medicine*. Published online June 2010.

11. Shearin S, Medley A, Trudelle-Jackson E, Swank C, Querry R. Differences in predictors for gait speed and gait endurance in Parkinson's disease. *Gait Posture*. 2021;87:49-53. doi:10.1016/j.gaitpost.2021.04.019

12. Conway ZJ, Silburn PA, Blackmore T, Cole MH. Evidence of compensatory joint kinetics during stair ascent and descent in Parkinson's disease. *Gait Posture*. 2017;52:33-39. doi:10.1016/j.gaitpost.2016.11.017

13. DeVita P, Hortobagyi T. Age causes a redistribution of joint torques and powers during gait. *J Appl Physiol*. 2000;88(5):1804-1811. doi:10.1152/jappl.2000.88.5.1804

14. Kuhman D, Hammond KG, Hurt CP. Altered joint kinetic strategies of healthy older adults and individuals with Parkinson's disease to walk at faster speeds. *J Biomech*. 2018;79:112-118. doi:10.1016/j.jbiomech.2018.07.038

15. Kuhman D, Willson J, Mizelle JC, DeVita P. The relationships between physical capacity and biomechanical plasticity in old adults during level and incline walking. *J Biomech*. 2018;69:90-96. doi:10.1016/j.jbiomech.2018.01.006

16. Albani G, Cimolin V, Fasano A, Trotti C, Galli M, Mauro A. "Masters and servants" in parkinsonian gait: a three-dimensional analysis of biomechanical changes sensitive to disease progression. *Funct Neurol*. 2014;29(2):99-105.

17. Baudendistel ST, Schmitt AC, Roemmich RT, Harrison IL, Hass CJ. Levodopa facilitates improvements in gait kinetics at the hip, not the ankle, in individuals with Parkinson's disease. *J Biomech*. 2021;121:110366. doi:10.1016/j.jbiomech.2021.110366

18. Cioni M, Richards CL, Malouin F, Bedard PJ, Lemieux R. Characteristics of the electromyographic patterns of lower limb muscles during gait in patients with Parkinson's disease when OFF and ON L-Dopa treatment. *Ital J Neurol Sci*. 1997;18(4):195-208. doi:10.1007/BF02080464

19. Islam A, Alcock L, Nazarpour K, Rochester L, Pantall A. Effect of Parkinson's disease and two therapeutic interventions on muscle activity during walking: a systematic review. *npj Parkinsons Disease*. 2020;6(1):22. doi:10.1038/s41531-020-00119-w

20. Oh J, Eltoukhy M, Kuenze C, Andersen MS, Signorile JF. Comparison of predicted kinetic variables between Parkinson's disease patients and healthy age-matched control using a depth sensor-driven full-body musculoskeletal model. *Gait Posture*. 2020;76:151-156. doi:10.1016/j.gaitpost.2019.11.011

21. Canning CG, Paul SS, Nieuwboer A. Prevention of falls in Parkinson's disease: a review of fall risk factors and the role of physical interventions. *Neurodegener Dis Manag*. 2014;4(3):203-221. doi:10.2217/nmt.14.22

22. Nilsson MH, Drake A-M, Hagell P. Assessment of fall-related self-efficacy and activity avoidance in people with Parkinson's disease. *BMC Geriatr*. 2010;10:78. doi:10.1186/1471-2318-10-78

23. Farombi TH, Owolabi MO, Ogunniyi A. Falls and their associated risks in parkinson's disease patients in nigeria. *J Mov Disord*. 2016;9(3):160-165. doi:10.14802/jmd.16011

24. DeMaagd G, Philip A. Parkinson's disease and its management: part 4: treatment of motor complications. *P T*. 2015;40(11):747-773.

25. Bhidayasiri R, Tarsy D. Parkinson's Disease: "On-Off" Phenomenon. In: *Movement Disorders: A Video Atlas*. Humana Press; 2012:14-15. doi:10.1007/978-1-60327-426-5_7

26. Non-pharmacological management of motor and non-motor symptoms. July 2017. Accessed February 2, 2022. https://www.ncbi.nlm.nih.gov/books/NBK535845/

27. Radder DLM, Sturkenboom IH, van Nimwegen M, Keus SH, Bloem BR, de Vries NM. Physical therapy and occupational therapy in Parkinson's disease. *Int J Neurosci*. 2017;127(10):930-943. doi:10.1080/00207454.2016.1275617

28. Ni M, Hazzard JB, Signorile JF, Luca C. Exercise Guidelines for Gait Function in Parkinson's Disease: A Systematic Review and Meta-analysis. *Neurorehabil Neural Repair*. 2018;32(10):872-886. doi:10.1177/1545968318801558

29. Judge JO, Davis RB, Ounpuu S. Step length reductions in advanced age: the role of ankle and hip kinetics. *J Gerontol A Biol Sci Med Sci*. 1996;51(6):M303-12. doi:10.1093/gerona/51a.6.m303

30. Washabaugh EP, Augenstein TE, Krishnan C. Functional resistance training during walking: Mode of application differentially affects gait biomechanics and muscle activation patterns. *Gait Posture*. 2020;75:129-136. doi:10.1016/j.gaitpost.2019.10.024

31. Gottschall JS, Kram R. Energy cost and muscular activity required for propulsion during walking. *J Appl Physiol*. 2003;94(5):1766-1772. doi:10.1152/japplphysiol.00670.2002

32. Hoppe M, Chawla G, Browner N, Lewek MD. The effects of metronome frequency differentially affects gait on a treadmill and overground in people with Parkinson disease. *Gait Posture*. 2020;79:41-45. doi:10.1016/j.gaitpost.2020.04.003

33. Alghamdi W, Preece SJ. How does normal variability in trunk flexion affect lower limb muscle activity during walking? *Hum Mov Sci*. 2020;72:102630. doi:10.1016/j.humov.2020.102630

34. Wright WG, Gurfinkel VS, Nutt J, Horak FB, Cordo PJ. Axial hypertonicity in Parkinson's disease: direct measurements of trunk and hip torque. *Exp Neurol*. 2007;208(1):38-46. doi:10.1016/j.expneurol.2007.07.002

35. AminiAghdam S, Karamanidis K, Rode C. Uneven running: How does trunk-leaning affect the lower-limb joint mechanics and energetics? *Eur J Sport Sci*. Published online June 17, 2021:1-8. doi:10.1080/17461391.2021.1938691

36. Penke K, Scott K, Sinskey Y, Lewek MD. Propulsive forces applied to the body's center of mass affect metabolic energetics poststroke. *Arch Phys Med Rehabil*. 2019;100(6):1068-1075. doi:10.1016/j.apmr.2018.10.010

37. Lewek MD, Raiti C, Doty A. The presence of a paretic propulsion reserve during gait in individuals following stroke. *Neurorehabil Neural Repair*. 2018;32(12):1011-1019. doi:10.1177/1545968318809920

38. Browne MG, Franz JR. More push from your push-off: Joint-level modifications to modulate propulsive forces in old age. *PLoS ONE*. 2018;13(8):e0201407. doi:10.1371/journal.pone.0201407

39. Siegel KL, Kepple TM, Stanhope SJ. Joint moment control of mechanical energy flow during normal gait. *Gait Posture*. 2004;19(1):69-75. doi:10.1016/s0966-6362(03)00010-9

40. Lewis CL, Ferris DP. Walking with increased ankle pushoff decreases hip muscle moments. *J Biomech*. 2008;41(10):2082-2089. doi:10.1016/j.jbiomech.2008.05.013

41. Mehrholz J, Kugler J, Storch A, Pohl M, Elsner B, Hirsch K. Treadmill training for patients with Parkinson's disease. *Cochrane Database Syst Rev*. 2015;(8):CD007830. doi:10.1002/14651858.CD007830.pub3

42. Gama GL, de Lucena Trigueiro LC, Simão CR, et al. Effects of treadmill inclination on hemiparetic gait: controlled and randomized clinical trial. *Am J Phys Med Rehabil*. 2015;94(9):718-727. doi:10.1097/PHM.0000000000000240

43. Lam T, Wirz M, Lünenburger L, Dietz V. Swing phase resistance enhances flexor muscle activity during treadmill locomotion in incomplete spinal cord injury. *Neurorehabil Neural Repair*. 2008;22(5):438-446. doi:10.1177/1545968308315595

44. Savin DN, Tseng S-C, Whitall J, Morton SM. Poststroke hemiparesis impairs the rate but not magnitude of adaptation of spatial and temporal locomotor features. *Neurorehabil Neural Repair*. 2013;27(1):24-34. doi:10.1177/1545968311434552