https://doi.org/10.1016/j.apmr.2012.06.004

1 **TITLE**

2

3 The influence of contraction type, speed, and joint angle on ankle muscle weakness in

4 Parkinson's disease: implications for rehabilitation

5

ABSTRACT

Objective: To compare the ankle muscle strength and torque-angle relationship between individuals with PD and participants without impairments.

Design: Cross-sectional, exploratory study.

Setting: Motor control laboratory in a University.

Participants: A convenience sample of 59 community-dwelling individuals with PD recruited from a PD self-help group and 37 age-matched participants without impairments recruited from community elderly centers.

Interventions: Not applicable.

Main outcome measure(s): The peak torque and angle-torque profile during concentric and eccentric contraction of ankle dorsiflexors and plantarflexors at two different angular speeds (45°/s and 90°/s).

Results: The PD group displayed lower muscle peak torque values than participants without impairments in all test conditions. Generally, concentric strength was more compromised, with a greater between-group difference (Cohen's d=1.29-1.60) than eccentric strength (Cohen's d=0.81-1.37). Significant group by angular speed interaction was observed in ankle plantarflexion concentric peak torque ($p<0.001$), indicating that muscle weakness was more pronounced when the angular speed was increased. The group by joint angle interaction in concentric contraction of ankle plantarflexors at 90°/s was also significant ($p<0.001$), revealing that the between-group difference in torque values became increasingly more pronounced when the joint was moving towards the end range of the ankle plantarflexion. This exaggerated

ankle plantarflexor muscle weakness at the end range was significantly correlated with clinical balance measures ($p<0.05$).

Conclusions: Muscle weakness in PD is influenced by contraction type, angular speed and joint range. Exaggerated weakness is found in concentric contraction of ankle plantarflexors, particularly when the angular speed is high and the muscle is in shortened lengths.

Key Words: Muscle strength; Parkinson's disease; Exercise; Rehabilitation

LIST OF ABBREVIATIONS

1.	PD	Parkinson’s Disease
2.	ANOVA	analysis of variance
3.	UPDRS	Unified Parkinson Disease Rating Scale
4.	MHY	Modified Hoehn & Yahr
5.	OLS	One-leg-stand
6.	LOS	Limit of stability
7.	COG	Center of gravity
8.	ANCOVA	analysis of covariance

Mounting evidence suggests that muscle weakness is a primary feature of Parkinson's disease (PD).[1] Muscle weakness may have important functional implications for people with PD. For example, Schilling et al.[2] have shown that more impaired leg extensor strength is significantly related to a longer time taken to complete the Timed-up-and-go-test. It is thus important to address lower extremity muscle weakness in the PD population.

Weakness in major muscle groups that control the ankle joint, namely the ankle dorsiflexors and plantarflexors, warrants particular attention because these muscles play an important role in regulating important bodily function such as balance and gait.[3,4] The push-off phase of the gait cycle is also highly dependent upon the ability to generate power in the ankle plantarflexors.[3,4] Ankle muscle weakness in individuals with PD has been demonstrated by a few studies.[5-7] However, some of these studies used a small sample size (<30 individuals with PD).[5,6] Discrepancies in results were also found, probably due to the differences in testing protocol and use of medications.[5,7] Moreover, previous studies did not systematically investigate the relationship between muscle strength and joint angle. While it is known that force production in normal muscle is influenced by muscle length,[8] the torque-angle profile in different types of contraction (i.e. eccentric Vs concentric) and its relationship to contraction speed has not been systematically studied in PD. Examining the torque-angle relationship is clinically relevant, as it helps to identify the joint range at which the torque production is the most deficient, and thus provides important information for the design of optimal muscle strengthening protocol for individuals with PD.

The objectives of this study were to compare the isokinetic ankle muscle strength and torque-angle relationship between people with PD and participants without impairments, and to assess the relationship between muscle weakness and balance ability. Several research hypotheses were generated based on previous findings in PD and other patient populations. First, some research evidence has suggested that torque generation may be more compromised at higher contraction speeds in individuals with PD.[6] Second, studies in older adults and individuals with neurological pathologies have shown that eccentric muscle strength is better preserved than concentric muscle strength.[9-11] Finally, research in stroke has shown that muscle weakness is more prominent at shorter muscle lengths.[12-14] The present study was thus designed to the test the following hypotheses: (1) there would be a significant group × speed interaction, with the PD group showing more strength deficits at higher movement velocities, (2) eccentric muscle strength would be more compromised than concentric muscle strength in individuals with PD, (3) there would be a significant group × joint angle interaction, with the PD group showing more strength deficits in the inner range of the muscle.

METHODS

Participants

In Pedersen et al.[5], the comparison of peak torque values of isokinetic ankle dorsiflexion between individuals with PD and participants without impairments yielded effect sizes varying from 0.8-2.4 (t-test) (i.e., large effect sizes), depending on

the type of contraction and angular speed. This study involved 2 factors (group and angular speed) for each type of contraction (concentric and eccentric). Based on 2-way analysis of variance (ANOVA), with a large effect size of 0.50, power of 0.90, the minimum sample size required would be 64 (32 people with PD and 32 participants without impairments).

A convenience sample of people with PD and participants without impairments was recruited from a local PD patient self-help group and community centers respectively. The inclusion criteria for participants with PD were: diagnosis by a neurologist using the United Kingdom PD Society Brain Bank Criteria[15], disease duration of >1 year, aged 50 years or older, community-dwelling, able to ambulate for at least 10 meters with or without walking aids independently, and able to follow simple verbal commands. The exclusion criteria were: significant musculoskeletal conditions that would interfere with testing, diagnosis of other neurological diseases, and other serious illnesses that precluded participation. The eligibility criteria for the control group were identical as stated above, except that participants without impairments did not have any history of PD. The study was approved by the University Human Research Ethics Review Committee. All participants gave written informed consent. All experimental procedures were conducted in accordance with the Declaration of Helsinki.

A total of 59 individuals with PD and 37 participants without impairments were enrolled in the study. Participant characteristics are listed in table 1. Relevant demographic information (e.g., medical history) was obtained by interview. The Motor Examination of the Unified Parkinson Disease Rating Scale (UPDRS-III) was

administered by an experienced clinical researcher to assess the degree of motor impairment.[16] The Modified Hoehn & Yahr (MHY) staging was used to evaluate PD disease severity.[17] For individuals with PD, all experimental procedures were performed within 1 hour during the "ON" phase of their medication cycle.

Muscle strength assessment

Ankle muscle strength was quantified by an isokinetic dynamometer[a] (Lumex, Inc., Ronkonkoma, NY), which is capable of maintaining the movement speed constant during testing. Only the more affected side was assessed, as more exaggerated muscle weakness was typically found in the more impaired leg.[18] Laterality was defined as the items 20-23, 24 and 26 of the UPDRS differing between sides by one or more score.[19] Participants were placed in a prone position and a footplate was attached to their feet while the thigh and lower leg were stabilized by straps. Participants performed isokinetic concentric and eccentric contraction of the ankle between a range of 10^{o} dorsiflexion and 25^{o} plantarflexion at two discrete angular velocities of 45^{o}/s and 90^{o}/s. Previous studies employed angular velocities between 30^{o}/s and 180^{o}/s.].[1,5-7,18] We chose the angular velocity of 45^{o}/s because this velocity is close to that is used during functional walking.[20] To assess the influence of angular speed on torque generation, a higher angular velocity of 90°/s was also chosen. Both velocities were tolerated by individuals with PD as determined by our pilot study. The order of the testing conditions was randomized. Participants were allowed to practice each type of contraction at their sub-maximal effort twice, followed by the test trial where 3 maximum concentric or eccentric contractions were performed. Each participant was closely monitored by the researcher during the data collection process, to ensure that the participants performed the required movements as instructed. Participants

were given a 3-minute rest period between each mode of contraction. The torque (Nm) profiles of the 3 test trials were averaged for further analysis. The variables of interest included peak concentric and eccentric joint torques, and the torques recorded at different joint angles (in 5^{o} intervals of ankle dorsiflexion/plantarflexion).

Balance measurements

Stance stability was evaluated by the one-leg-stand (OLS) test.[21] Each participant was instructed to stand on their more affected leg with eyes open, and hands placed on the hips, and maintain this position for as long as possible. The OLS time was measured using a stopwatch. A practice trial was given before the actual recording.

The limit of stability (LOS) test was performed to assess dynamic postural control, using the Smart Balance System[b] (NeuroCom International, Inc., Clackamas, OR).[7] This test quantifies the individual's ability to voluntarily sway to various locations in space.[22] The system consists of dual force plates that are connected to a computer system and a screen display placed in front of the participant. Each participant stood on the forceplates while wearing a harness to prevent falls. The theoretical LOS of each participant (i.e., the maximum range in which the center of gravity (COG) can be moved safely without changing the base of support) was automatically computed by the Smart Balance System, based on the assumption that movement about the ankle while standing on a fixed surface behaves as an inverted pendulum.[22,23] The limits are defined as extending 6.25°, 4.45°, and 8.00° in anterior, posterior and mediolateral directions, respectively.[22,23] On the screen display, there were eight target boxes placed at the 100% of the theoretical LOS (forward, backward, left, right, forward right, forward left, backward right, and backward left), a center box, and a cursor

representing participant's COG.[22] In the starting position, the participant was required to maintain the COG cursor within the center box. During the LOS test, the participant was instructed to move the COG cursor towards a highlighted target box as quickly and accurately as possible, and maintain the cursor within the target box. The participants were given a maximum of 8 seconds to complete the movement toward the target. The endpoint excursion is the distance traveled by the COG on the primary attempt to reach the designated target and is expressed as a percentage of the LOS. The endpoint is defined as the point at which the initial movement towards the target stops and subsequent corrective movement begins (Figure 1).[22]

Data analysis

All analyses were conducted using SPSS 17.0[c] (SPSS Inc., Chicago, IL). Two-way analysis of covariance (ANCOVA) models with mixed design (within-subject factor: angular speed, between-subject factor: patient group, covariate: age) were used to compare the peak torque values in each of the following types of muscle contraction at 45°/s and 90°/s: concentric ankle dorsiflexion, eccentric ankle dorsiflexion, concentric ankle plantarflexion, and eccentric ankle plantarflexion. Post-hoc analysis was performed when appropriate. For analysis of the torque-angle profiles, two-way ANCOVA models with mixed design (within-subject factor: joint angle, between-subject factor: group, covariate: age) were then used, followed by post-hoc analysis as necessary. In isokinetic testing, acceleration and deceleration phases may occur.[24] Our pilot data indeed showed the existence of the acceleration and deceleration phases within the first and last 2-5 degrees of movement respectively. Therefore, we discarded the data in the first and final 5 degrees of each movement. Only the torque profiles between the range of 5° dorsiflexion and 20° plantarflexion were analyzed.

For ANCOVA, effect sizes were expressed in partial eta-squared (large=0.14, medium=0.06, small=0.01).[25] Finally, Pearson's correlation analysis was performed to determine the degree of association of muscle torques with balance parameters measured. A significance level of $p<0.05$ was set.

RESULTS

There were no significant differences in any demographic variables between the two groups (Table 1).

Peak torque

The peak torque data are displayed in Table 2. ANCOVA revealed a significant group × speed interaction in ankle plantarflexion concentric strength only ($F=11.201$, $p=0.001$, effect size in partial eta squared $\eta^2=0.11$), indicating exaggerated muscle weakness in concentric ankle plantarflexion among the PD patients when the angular speed was increased from 45°/s to 90°/s. Significant main effect of group for all muscle torque variables measured was found ($p<0.001$), with the PD group consistently showing lower peak torque values than the participants without impairments (partial $\eta^2=0.16$-0.38). Significant main effect of speed was detected in concentric ankle plantarflexion only ($F=6.665$, $p=0.011$, partial $\eta^2=0.07$). In addition, the effect sizes for the concentric conditions were substantially greater than those for the corresponding eccentric conditions, except for ankle dorsiflexion at 45°/s.

Angle-torque profiles using absolute torque values

When the absolute torque values were used to generate the angle-torque profiles, it was found that the group × angle interaction effect was statistically significant for all test conditions (partial η^2=0.04-0.19) (not shown). The main effects of angle and group were also significant for all test conditions ($p<0.05$). The significant group × angle interaction indicated that the relationship between torque production and joint range demonstrated in the PD group is different from that in participants without impairments.

Angle-torque profiles using relative torque values

To further explore the relationship between muscle weakness and joint angle, the torque value attained at a particular joint angle was expressed as a percentage of the peak torque and the angle-torque profiles were then compared. For ankle dorsiflexion strength (Fig. 2), the group × angle interaction was not significant for all test conditions (Fig. 2A-D)($p>0.05$). The main effect of group was significant for concentric contraction at 90°/s (Fig. 2B) and eccentric contraction (Fig. 2C and D) at both speeds ($p<0.05$, partial η^2=0.06-0.09), but was not statistically significant for concentric contraction at 45°/s (Fig. 2A) ($p>0.05$). The main effect of angle was only significant for eccentric contraction ($p<0.001$, partial η^2=0.05-0.06) (Fig. 2C and D), but not for concentric contraction ($p>0.05$) (Fig. 2A and B), regardless of angular speed.

For ankle plantarflexion (Fig. 3), the group × angle interaction was significant for concentric contraction at both angular speeds (Fig. 3A and B) and eccentric contraction at 90°/s (Fig. 3D) ($p<0.05$, partial η^2=0.03-0.19). The group × angle

interaction for eccentric plantarflexion at 45°/s did not quite reach statistical significance (p=0.080). The main effect of group was significant for all test conditions ($p<0.05$, partial $\eta^2=0.07$-0.34) (Fig. 3A-D). The main effect of angle was significant for eccentric ankle plantarflexion ($p<0.05$, partial $\eta^2=0.49$-0.56) (Fig. 3C and D), but not for concentric ankle plantarflexion (Fig. 3A and B) ($p>0.05$), regardless of angular speed.

Correlation with balance measures

Concentric ankle plantarflexion strength at 90°/s at 20° of ankle plantarflexion was selected for subsequent correlation analysis, as it showed the most pronounced deficit (Fig. 3B). The results showed that it was significantly correlated with OLS time ($r=0.306$, $p=0.022$), and end point excursion (LOS test) ($r=0.388$, $p=0.003$).

DISCUSSION

This novel study found that the PD group displayed significant weakness in both ankle dorsiflexors and plantarflexors. The weakness is particularly apparent during concentric contraction of the plantarflexors, when the angular speed is high and the joint is moving toward the end range of ankle plantarflexion.

Ankle muscle weakness in PD

Our findings of muscle weakness in both concentric ankle dorsiflexion and plantarflexion in the PD group are consistent with those reported earlier by previous

studies.[5-7,26,27] Pedersen et al. were the only investigators who demonstrated eccentric ankle dorsiflexors weakness in individuals with PD.[5,6,26] Our study extended their finding by showing that both eccentric ankle dorsiflexion and plantarflexion joint torques were compromised in PD.

Influence of speed

Our results proved our hypothesis that individuals with PD have more muscle strength deficits at higher movement speeds. With increase in angular speed, the difference in peak torque generated during concentric ankle plantarflexion contraction between the patients and participants without impairments became more conspicuous. Pedersen and Oberg reported that the deficits in peak torque generation were more apparent at angular speed of 180°/s than 30°/s for concentric ankle dorsiflexion.[6] Our lack of group × speed interaction effect in concentric ankle dorsiflexion could be due to the narrower range of angular speeds used in our study (i.e. from of 45°/s to 90°/s), compared with those used by Pederson and Oberg.[6] Other studies also demonstrated no relationship between concentric ankle joint torque and movement velocity.[7,25] However, these investigators compared the joint torque at much higher velocities, at 90°/s and 150°/s.

The impairment in torque generation at higher velocities is considered to represent bradykinesia, a symptom that reflects the dysfunction of central mechanisms. Hallett and Khoshbin[28] demonstrated that bradykinesia resulted from the inability to activate the appropriate muscle to generate force at a sufficient rate. Individuals with PD may have selective decrease in number of and atrophy of fast-twitch type II muscle fibers, and hence more weakness during movements at faster speeds.[29]

Influence of contraction type

Our results support our hypothesis that concentric strength is more impaired than eccentric strength in individuals with PD. By comparing the effect sizes (Table 2), we found that eccentric strength was better preserved than concentric strength, particularly for ankle plantarflexors. Pedersen and colleagues reported a similar phenomenon in ankle dorsiflexors.[5,6] The more pronounced deficits in concentric strength in the PD population appear to be consistent with those reported in older people and individuals with other neurological conditions (e.g., cerebral palsy, and stroke).[9-11] The mechanisms underlying this phenomenon are not entirely clear. Presumably, physical inactivity may lead to reduction in both concentric and eccentric strength. However, a sedentary lifestyle may also contribute to a decrease in contractile element and increase in connective tissue content in the muscle, which may alter the mechanical stiffness of the muscle. During eccentric contraction, the stiffness of the lengthening muscle would contribute to the tension development. The rigidity may also contribute to the force development as the muscle is lengthening during eccentric contraction. It is possible that the reduction in strength due to eccentric contractile inactivity may be partially compensated by gains in mechanical stiffness.[9] More study is required to examine the muscle structural changes in PD.

Influence of joint angle

In analyzing the angle-torque profiles, we used the relative torque values in conjunction with the absolute values. The results revealed substantial difference in peak torque values between the PD and control groups (Table 2). The peak torque value for a particular type of contraction also varied across the subjects within each of

the two groups, as reflected by the standard deviations (Table 2). Additionally, the peak torque values also differed considerably among the different types of contraction (Table 2). By expressing each individual's torque values generated at various joint angles as a percentage of his or her own peak torque value, the variability of the peak torque arising from different sources (i.e., between-group, within-group, between-contractions) could be taken into account when analyzing the angle-torque profiles. Using the relative torque values can thus facilitate the comparison of angle-torque profiles between the PD and control groups across the different test conditions. Previous studies in other patient populations have also used a similar approach in analysis of muscle torque data.[12,14]

In analyzing the absolute torque values, the group × angle interaction was statistically significant for all test conditions, clearly indicating that the relationship between torque generation and joint angle demonstrated in the PD group is distinct from that in the control group. In subsequent analysis using relative torque values, the significant group × angle interaction was found only in ankle plantarflexion. In particular, the PD group exhibit exaggerated muscle weakness in the inner range of concentric ankle plantarflexion, which is more apparent at higher speeds (Fig. 3B). At 90^{o}/s, the relative ankle plantarflexion joint torque in the PD group is at its maximum at the outer range of the muscle (i.e. when the ankle is in a dorsiflexed position) but is substantially reduced and reached its minimum at the inner range (i.e. when the ankle is in a plantarflexed position). Our findings thus support our hypothesis that muscle weakness is more pronounced in the inner range of the muscle. Our results are also consistent with the exaggerated weakness found in shorter elbow flexors and extensors[12,13] and knee extensors[14] among patients with stroke. The length-dependent

deficits might be due to impaired motor unit rate coding at shorter muscle lengths. It has been shown in neurologically intact participants that the twitch duration is reduced during voluntary isometric contraction at a shorter muscle length.[30] Participants without impairments could increase the motor unit firing rates to maintain the joint torque. People with PD may have central deficits in maintaining or sustaining the motor units firing rate for long period of time[31], inefficiency of muscle contraction[27] and/or depression in the rate of force production[32], resulting in exaggerated weakness ankle plantarflexors at shorter lengths.

We note that the length-dependent deficit was only present in ankle plantarflexors, but not ankle dorsiflexors. Individuals with PD are known to walk with shuffling gait with toes touching the ground first instead of the heel. Kinematic and kinetic analysis found that these patients had decreased ankle plantarflexion excursion[3] and reduced push-off ankle power.[3,4] The disuse of the ankle plantarflexors, especially in the inner range, during daily functional activities might result in more deficits in this range.

The length-dependent weakness found here could not be explained by the reduced effort in anticipation of reaching the end of movement. First, this finding is not observed in the participants without impairments (i.e. significant group × angle interaction)(Fig. 3B). Second, this pattern of muscle weakness is not found in the ankle dorsiflexors (Fig. 2) or eccentric contraction of ankle plantarflexors (Fig. 3D).

Clinical implications

Muscle weakness in individuals with PD may have important functional implications. Indeed, we found a significant relationship between ankle plantarflexor muscle

weakness and balance ability. Addressing muscle weakness is thus an important area in fall management of these patients. The findings in this study would be useful in guiding the design of muscle strengthening program for the PD population. For example, as muscle strength is more compromised at higher velocities and inner range of movement, strength training may focus on higher-velocity movements that forced the patients to use the inner range of the target muscle group. It has been reported that following speed-specific and angle-specific isometric and concentric training in unimpaired individuals, the strength gain was more apparent at the trained speeds and joint range.[33]

People with PD have more impairment in concentric than eccentric ankle muscle strength. On one hand, it is important to target this deficit in concentric contraction in the strength training program. On the other hand, the strengthening program may exploit their better-preserved eccentric strength to maximize functional capacity. Indeed, Dibble et al.[34] have shown that high-force eccentric training resulted in better outcomes in muscle volume, walking endurance, and stair descent in people with PD. Further study is required to investigate the effects of different exercise protocols on the torque-angle relationship and function in people with PD.

Study Limitations

This was a cross-sectional study that compared the muscle strength between people with PD and age-matched participants without impairments. It could not provide information on the temporal changes of muscle strength in people with PD. A prospective study would be required to examine the degree of muscle strength after PD as time progresses. All assessments were performed within 1 hour during the

"ON" phase of the medication cycle. Further study should investigate the muscle strength profiles during the OFF phase. Additionally, our participants in the PD group were all community-dwelling, ambulatory individuals. The results cannot be generalized to those who are institutionalized or wheelchair bound. Finally, all participants in the PD group were recruited from a local PD patient self-help group, which held regular meetings for its members. The PD group in our study may thus be more physically and socially active than their non-member counterparts.

CONCLUSIONS

To conclude, individuals with PD demonstrate significant ankle muscle weakness. The deficit in ankle muscle force production is influenced by contraction type, angular speed and joint range. The results are useful in guiding the design of muscle strength training program for individuals with PD.

REFERENCES

1. Falvo MJ, Schilling BK, Earhart GM. Parkinson's disease and resistive exercise: rationale, review, and recommendations. Mov Disord 2008;23:1-11.
2. Schilling BK, Karlage RE, LeDoux MS, Pfeiffer RF, Weiss LW, Falvo MJ.Impaired leg extensor strength in individuals with Parkinson disease and relatedness to functional mobility. Parkinsonism Relat Disord 2009;15:776-80.
3. Sofuwa O, Nieuwboer A, Desloovere K, Willems AM, Chavret F, Jonkers I.Quantitative gait analysis in Parkinson's disease: comparison with a healthy control group. Arch Phys Med Rehabil 2005;86:1007-13.
4. Svehlík M, Zwick EB, Steinwender G, Linhart WE, Schwingenschuh P, Katschnig P, et al. Gait analysis in patients with Parkinson's disease off dopaminergic therapy. Arch Phys Med Rehabil 2009;90:1880-6.
5. Pedersen SW, Oberg B, Larsson LE, Lindval B. Gait analysis, isokinetic muscle strength measurement in patients with Parkinson’s disease. Scand J Rehabil Med 1997;29:67-74.
6. Pedersen SW, Oberg B. Stretch-shortening contraction in Parkinson patients: evidence of normal muscle contraction execution with low efficiency. Scand J Rehabil Med 1997;29:251-5.
7. Nallegowda M, Singh U, Handa G, Khanna M, Wadhwa S, Yadav SL, et al. Role of sensory input and muscle strength in maintenance of balance, gait and posture in Parkinson’s disease. Am J Phys Med Rehabil Med 2004;83:898-908.
8. Rassier DE, MacIntosh BR, Herzog W. Length dependence of active force production in skeletal muscle. J Appl Physiol 1999;86:1445-.57

9. Eng JJ, Lomaglio MJ, MacIntyre DL.Muscle torque preservation and physical activity in individuals with stroke. Med Sci Sports Exerc 2009;41:1353-60.
10. Damiano DL, Martellotta TL, Quinlivan JM, Abel MF. Deficits in eccentric versus concentric torque in children with spastic cerebral palsy. Med Sci Sports Exerc 2001;33:117-22.
11. Porter MM, Vandervoort AA, Kramer JF. Eccentric peak torque of the plantar and dorsiflexors is maintained in older women. J Gerontol A Biol Sci Med Sci 1997;52:B125-31.
12. Ada L, Canning CG, Low S-L. Patients have selective muscle weakness in shortened range. Brain 2003;126:724-31.
13. Lum PS, Patten C, Kothari D, Yap R. Effects of velocity on maximal torque production in poststroke hemiparesis. Muscle Nerve 2004;30:732–42.
14. Lomaglio MJ, Eng JJ. Nonuniform weakness in the paretic knee and compensatory strength gains in the nonparetic knee occurs after stroke. Cerebrovasc Dis 2008;26:584-91.
15. Hughes AJ, Daniel SE, Kilford L, Lees AJ. (Accuracy of clinical diagnosis of idiopathic Parkinson's disease: A clinico-pathological study of 100 cases. J Neurol Neurosurg Psychiatry 1992;55:181-4.
16. Martinez-Martin P, Forjaz MJ. Metric attributes of the unified Parkinson's disease rating scale 3.0 battery: Part I, feasibility, scaling assumptions, reliability, and precision. Mov Disord 2006;21:1182-8.
17. Fahn S, Elton RL, and members of the UPDRS Development Committee. Unified Parkinson's disease rating scale. In: Fahn S, Marsden CD, Calne DB, Goldstein Meditors. Recent developments in Parkinson's disease. Vol. 2. Florham Park, NJ: Macmillan Health Care Information; 1987. p 153-63.

18. Kakinuma S, Nogaki H, Pramanik B, Morimatsu M. Muscle weakness in Parkinson's disease: isokinetic study of the lower limbs. Eur Neurol 1998;39:218-2.2
19. Baba Y, Markopoulou K, Putzke JD, Whaley NR, Farrer MJ, Wszolek ZK, et al. Phenotypic commonalities in familial and sporadic Parkinson disease. Arch Neurol 2006;63:579-83.
20. Grey MJ, Nielsen JB, Mazzaro N, Sinkjaer T. Positive force feedback in human walking. J Physiol 2007;15:99-105.
21. Jacobs JV, Horak FB, Tran VK, Nutt JG. Multiple balance tests improve the assessment of postural stability in subjects with Parkinson's disease. J Neurol Neurosurg Psychiatry 2006;77:322-6.
22. NeuroCom International, Inc. Balance Master operators manual. Clackamas: NeuroCom International, Inc., 2002.
23. Liston RAL, Brouwer BJ. Reliability and validity of measures obtained from stroke patients using the Balance Master. Arch Phys Med Rehabil 1996;77:425-30.
24. Osternig LR. Isokinetic dynamometry: implications for muscle testing and rehabilitation. Exerc Sport Sci Rev 1986;14:45-80.
25. Pallant JF. SPSS Survival Manual: A step by step guide to data analysis using SPSS for Windows (version 15). 3rd ed. Crows Nest, Australia: Allen & Unwin; 2007.
26. Durmus B, Baysal O, Altinayar S, Altay Z, Ersoy Y, Ozcan C. Lower extremity isokinetic muscle strength in patients with Parkinson's disease. J Clin Neurosci 2010;17:893-6.

27. Pedersen SW, Oberg B. Dynamic strength in Parkinson's disease. Eur Neurol 1993;33:97-102.
28. Hallett M, Khoshbin S. A physiological mechanism of bradykinesia. Brain 1980;103:301-14.
29. Valls-Sole J, Valldeoriola F. Neurophysiological correlate of clinical signs in Parkinson's disease. Clin Neurophysiol 2002;113:792–805.
30. Gandevia SC, McKenzie DK. Activation of human muscles at short muscle lengths during maximal static efforts. J Physiol 1988;407:599–613.
31. Glendinning DS, Enoka RM. Motor units behaviour in Parkinson's disease. Phys Ther 1994;74:61-70.
32. Godaux E, Koulischer D, Jacquy J. Parkinsonian bradykinesia is due to depression in the rate of rise of muscle activity. Ann Neurol 1992;31:93-100.
33. Morrissey MC, Harman EA, Johnson MJ. Resistance training modes: specificity and effectiveness. Med Sci Sports Exerc. 1995;27:648-60.
34. Dibble LE, Hale TF, Marcus RL, Droge J, Gerber JP, LaStayo PC. High-intensity resistance training amplifies muscle hypertrophy and functional gains in persons with Parkinson's disease. Mov Disord 2006;21:1444-52.

SUPPLIERS

a Lumex, Inc., Ronkonkoma, NY

Cybex International, Inc.

10 Trotter Drive

Medway, MA 02053

USA

b NeuroCom International, Inc., Clackamas, OR

NeuroCom, a division of Natus

9570 SE Lawnfield Road

Clackamas, OR 97015

USA

c SPSS Inc., Chicago, IL

IBM Corporation

1 New Orchard Road

Armonk, New York 10504-1722

United States

914-499-1900

FIGURE LEGENDS

Fig. 1 Limit of stability (LOS) test. Each participant was asked to move the center of gravity (COG) as quickly and accurately as possible towards a second target located at the perimeter of LOS. The endpoint excursion refers to the distance traveled by the COG on the initial attempt to reach the target.

Fig. 2 Ankle dorsiflexion angle-torque profiles. On the horizontal axis, negative values represent ankle dorsiflexion whereas positive represent ankle plantarflexion. The error bars represent one standard error of the mean. Between-group difference: *$p<0.05$.

Fig. 3 Ankle plantarflexion angle-torque profiles. The same convention was used as in figure 1. Between-group difference:*$p<0.05$, †$p<0.001$.