Leg-Extension Strength and Chair-Rise Performance in Elderly Women With Parkinson's Disease

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The lower extremity performance in elderly female patients with mild to moderate Parkinson's disease (PD; $n = 12$) and controls ($n = 16$) was compared. Isometric dynamometry and force-plate measurements were used. PD patients had lower ($p < .05$) bilateral (BL) maximal isometric leg-extension force (MF), BL isometric MF relative to body mass, and maximal rate of isometric force development than control participants. BL strength deficit was greater ($p < .05$) in PD patients than in controls. A significantly longer chair-rise time and lower maximal rate of vertical-ground-reaction-force development while rising from a chair was found in PD patients than in controls. These findings suggest that elderly women with PD have lowered voluntary isometric force-generation capacity of the leg-extensor muscles. Reduced BL leg-extension strength might contribute to the difficulty of individuals with PD to rise from a chair.

Key Words: aging, muscle strength, rising from a chair, lower limbs

Leg extension is an important movement involved in many activities of daily life, one of them being rising from a chair. Many elderly people, in particular those with Parkinson's disease (PD), often have difficulty rising from a chair (Brod, Mendelsohn, & Roberts, 1998; Inkster, Eng, MacIntyre, & Stoessl, 2003). The ability to rise from a sitting position is important in maintaining an independent life. The inability to perform chair rising, especially quickly, has been associated with increased risk of falling (Nevitt, Cummings, Kidd, & Black, 1989). It is important to understand what factors limit chair-rise performance in patients with PD so that effective remedial approaches can be developed.

Rising from a chair requires greater leg-extensor strength and joint ranges of motion than other activities of daily living, such as walking or stair climbing (Gross, Stevenson, Charette, Pyka, & Marcus, 1998). In particular, large hip- and knee-extension forces are required at the point when the buttocks are lifted from the chair (Kotake et al., 1993; Schenkman, Berger, Riley, Mann, & Hodge, 1990). This

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physical task also requires relatively high balance control (Gross et al.). Several investigations stress the importance of lower extremity strength in older adults for successful chair-rise performance (Corrigan & Bohannon, 2001; Gross et al.; Hughes, Myes, & Schenkman, 1996; Schenkman, Hughes, Samsa, & Studenski, 1996).

Patients with PD are typically elderly and demonstrate slowness in initiating and performing movement. Lower hip- and knee-extension isokinetic torque (Inkster et al., 2003) and maximal isometric knee-extension force and rate of force development (Pääsuke, Mõttus, Ereline, Gapeyeva, & Taba, 2002) have been documented in PD patients than in age- and sex-matched controls. The relationship, however, between force-production capacity of the leg-extensor muscles and chair-rise performance in patients with PD is not clear. Only a few studies have examined the determinants of chair-rise performance among elderly individuals with PD. One relevant study (Inkster et al.) found that reduced hip- and knee-extension isokinetic strength might contribute to the difficulty of people with PD to rise from a chair. Our previous study (Pääsuke et al.) suggested that chair-rise performance reduction was most obvious in PD patients with considerable decreased unilateral (UL) knee-extension isometric strength.

Maximal voluntary strength of simultaneous bilateral (BL) exertion is known to be smaller than the sum of the UL exertions of the right and left limbs. This phenomenon is designated as BL strength deficit (Jakobi & Chilibeck, 2001; Oda & Moritani, 1995; Ohtsuki, 1983; Secher, Rube, & Elers, 1988; Vandervoort, Sale, & Moroz, 1984). No clear explanation for this phenomenon has emerged, but BL strength deficit would indicate a significant limitation of motor control. It is suggested that this deficit is a result of neural inhibition (Ferbert et al., 1992; Oda, 1997; Oda & Moritani, 1995). It remains unclear, however, whether such inhibition is mediated by supraspinal mechanisms or by reflex pathways at the level of spinal cord. Little is known about BL strength deficit of the leg-extensor muscles in patients with PD and its relation to chair-rise performance.

The aim of this study was to compare the isometric force-production capacity of the leg-extensor muscles (hip, knee, and ankle extensors) and chair-rise performance in elderly sedentary female patients with mild to moderate idiopathic PD and in age- and sex-matched nondisabled controls. More specifically, BL and UL maximal isometric leg-extension force (MF), BL strength deficit, and BL maximal rate of isometric leg-extension force development (RFD) were correlated with chair-rise time, maximal vertical ground-reaction force, and rate of vertical-ground-reaction-force development measured during rising from a chair at essential (self-paced) speed without the use of the hands. We hypothesized that PD patients have a reduced maximal voluntary isometric force-generation capacity of the leg-extensor muscles during BL contraction and greater BL strength deficit than their age- and sex-matched controls, and this is an important factor of reduced chair-rise performance in PD patients.

Methods

PARTICIPANTS

Twelve female patients with idiopathic PD and 16 age- and sex-matched nondisabled control participants took part in this study. The ages and physical characteristics of the participants are presented in Table 1. All were medically stable, had adequate comprehension of instructions, and were able to rise from a chair independently without using their upper limbs. Based on the analysis of complaints and objective status examination, they had no arthritis in the lower limbs. The patients were recruited from the Department of Neurology at Tartu University Hospital. Inclusion criteria were a diagnosis of idiopathic PD confirmed by the individual's neurologist, with a disease-severity rating of Stage I to III (mild to moderate PD) on the Hoehn and Yahr's scale (Hoehn & Yahr, 1967); a score of more than 20 points on the Short Test of Mental Status (Kokmen & Offort, 1991); and a score less than 20 points on the modified Webster rating scale (Kempster et al., 1989). The duration of Parkinson's disease ranged from 4 to 18 years, with a mean of 10.7 ± 4.5 years. All patients were medicated with ordinary anti-PD drugs, and they were measured during the stable "on" period, 1–2 hr after taking their morning or afternoon dose. The control participants had also no Ménière's syndrome, cerebellar signs, cognitive deficits, or peripheral neuropathy under standard neuropsychological assessment. Participants answered a questionnaire detailing their major movement problems and their regular exercise habits. All participants were then categorized according to physical activity as sedentary—that is, only daily chores were performed. Before testing, each participant was familiarized with the testing procedures. The more affected and less affected leg in PD patients was identified clinically, and dominant and nondominant leg in controls was identified by self-report. The participants performed all testing procedures on the same day. Written informed consent was obtained from all participants. The study was approved by the university ethics committee.

EXPERIMENTAL PROCEDURES

Isometric Dynamometry. During the measurement of isometric leg-extension force, the participants were seated comfortably in a specially designed chair

Table 1   Age and Physical Characteristics of the Participants ($M \pm SD$)

Group	n	Age (years)	Height (cm)	Body mass (kg)	Body-mass index (kg/m^2)
Parkinson's disease patients	12	74.3 ± 6.9	157.4 ± 5.9	64.8 ± 6.9	26.2 ± 2.4
Controls	16	71.7 ± 4.4	157.6 ± 6.0	65.3 ± 8.0	26.3 ± 2.8

with the trunk upright and the hip, knee, and ankle angles equal to 110°, 120°, and 60°, respectively (Raudsepp & Pääsuke, 1995). The body of each participant was secured to the chair by a seat belt at the hip and straps from the seat belt over the shoulders. The feet were placed on an adjustable footplate mounted on the frame of the chair. The isometric leg-extension force production was recorded by standard strain-gauge transducer (1778 DST-2, Moscow, Russia) connected with a footplate and amplified via a high-gain strain-gauge amplifier. The strain-gauge transducer proved to be linear within the measurement range of 150–15,000 N. The electrical signals from the strain-gauge transducer were digitized online (sampling frequency 1 kHz) using a personal computer. The output from the strain-gauge amplifier was also displayed on an analogue voltmeter, which the participants could observe during their efforts in order to obtain immediate feedback.

The participants performed two tests. During the first test they were instructed to push the footplate as forcefully as possible for 2–3 s in three conditions: UL isometric extension of the right leg, UL isometric extension of the left leg, and BL isometric leg extension. Three trials were performed for each case, and the best result was taken as the isometric MF. Verbal encouragement to motivate producing maximal effort was provided throughout each trial. A rest period of 2 min was allowed between the trials. During UL exertions the contralateral leg was allowed to rest on the floor. BL isometric leg-extension MF relative to body mass was calculated. Bilateral index (BI) was calculated by the formula described by Howard and Enoka (1991):

$$BI\ (\%) = 100[BL/(UL_R + UL_L)] - 100\%$$

where BL = maximal isometric bilateral leg-extension force, UL_R = maximal isometric unilateral leg-extension force of the right leg, and UL_L = maximal isometric unilateral leg-extension force of the left leg. A negative BI indicated a BL strength deficit, and a positive BI indicated a BL strength facilitation.

During the second test, the participants were instructed to react to the light signal (ignition of the signal lamp, placed 1.5 m from the participant) as quickly and as forcefully as possible by extending both legs against a footplate. Maximal BL isometric contraction was held for approximately 2–3 s. The force–time curve was analyzed by personal computer, and BL maximal isometric leg-extension RFD as the first derivative of the development of force (dF/dt) was calculated. The best result out of three trials was taken for the final analysis. A rest period of 2 min was allowed between the trials.

Rising From a Chair.   A force plate (VISTI, Russia) with dimensions of 75 × 75 cm and with a sampling rate 150 Hz was used to record the vertical ground-reaction forces exerted by lower limbs as the participants stood up from the seated position. The participants were barefoot and dressed in shorts. They were seated on an armless, backless chair, which was adjusted to the height of each participant's knee, determined as the distance from the lateral knee-joint line to the floor. The

feet were placed parallel on a force plate, with the medial border of the feet 10–15 cm apart, according to the participant's body size. Each participant's ankle was placed in about 10° of dorsiflexion, and the knee angle was at about 100° of flexion. The arms were folded, with hands grasping the elbows and kept against the chest. The movement amplitude of the knee joint while rising from a chair was recorded with an electrogoniometer (XM180, Penny and Giles Biometrics, Gwent, England) attached to the lateral side of the participant's right knee. The participants were instructed not to use their arms or move their feet from the initial position as they stood up from a chair. The participants performed rising from a chair as they "usually do," that is, at a self-paced, comfortable speed. One of the investigators gave the command "ready, start." The task was initiated with the word *start,* and the rising phase ended when the vertical force equated to body weight. Three trials were performed, and the trial with the shortest chair-rise time was used for further analysis. A rest period of 2 min was allowed between trials. The following force–time characteristics were calculated while rising from a chair: Maximal vertical ground-reaction force (VGRF) = the highest value of vertical-force production, and maximal rate of vertical-ground-reaction-force development (VGRFD) = the first derivative of development of force (dF/dt). Chair-rise time (T_{rise}) was calculated by electrogoniogram from initial knee flexion to the time at which full extension of the knee was first reached.

STATISTICAL ANALYSIS

Data are means plus or minus standard deviations (*SD*). One-way analysis of variance (ANOVA) followed by Scheffé post hoc comparisons was used to test for differences between groups and for each leg. Linear correlations were calculated to assess the relationship between selected characteristics. A level of significance of $p < .05$ was selected to indicate statistical significance.

Results

No significant differences ($p > .05$) in age, height, body mass, and body-mass index were observed between the measured groups (Table 1). PD patients had lower BL isometric leg-extension MF, $F(1, 27) = 4.16, p < .05$; isometric leg-extension MF relative to body mass, $F(1, 27) = 3.81, p < .05$; and maximal isometric leg-extension RFD, $F(1, 27) = 3.96, p < .05$, than did control participants (Figure 1). No significant differences ($p > .05$) were found in isometric leg-extension MF between the more and less affected legs of PD patients or between the dominant and nondominant legs for the nondisabled controls (Figure 2[a]). PD patients had significantly higher BL strength deficits, $F(1, 27) = 4.21, p < .05$, than did control participants (Figure 2[b]). PD patients had significantly lower maximal VGRFD, $F(1, 27) = 4.24, p < .05$, while rising from a chair and longer chair-rise time, $F(1, 27) = 12.07, p < .001$, than those of controls (Figure 3). No significant differences ($p > .05$) in maximal VGRF while rising from a chair were observed between the groups.
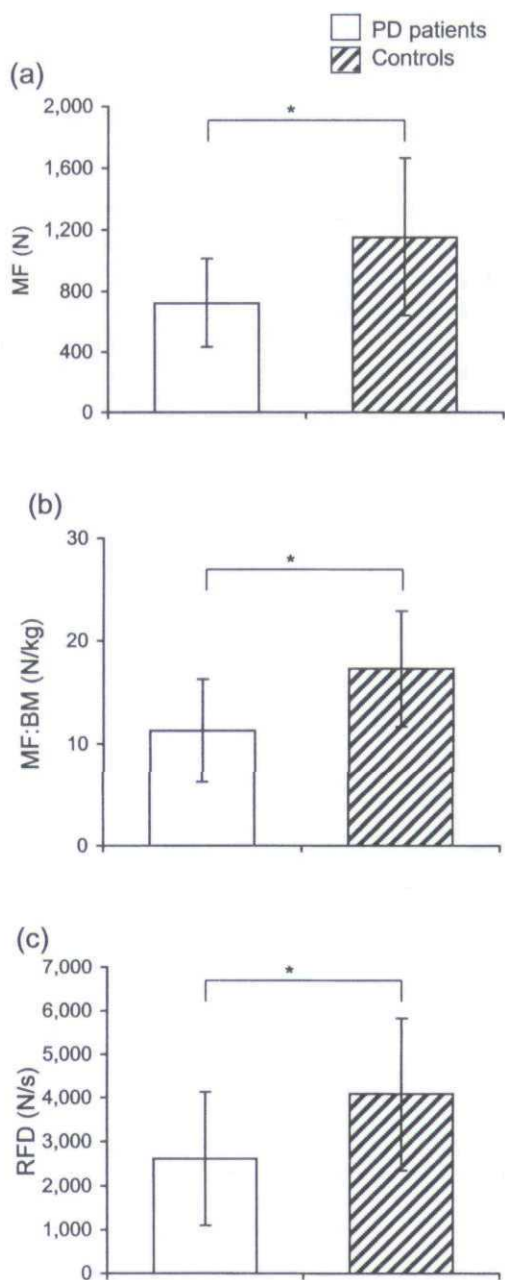
Figure 1. Mean (± *SD*) values of (a) maximal isometric bilateral leg-extension force (MF), (b) MF relative to body mass (MF:BM), and (c) maximal rate of isometric bilateral leg-extension-force development (RFD) in elderly female Parkinson's (PD) patients (*n* = 12) and controls (*n* = 16). *$p < .05$.
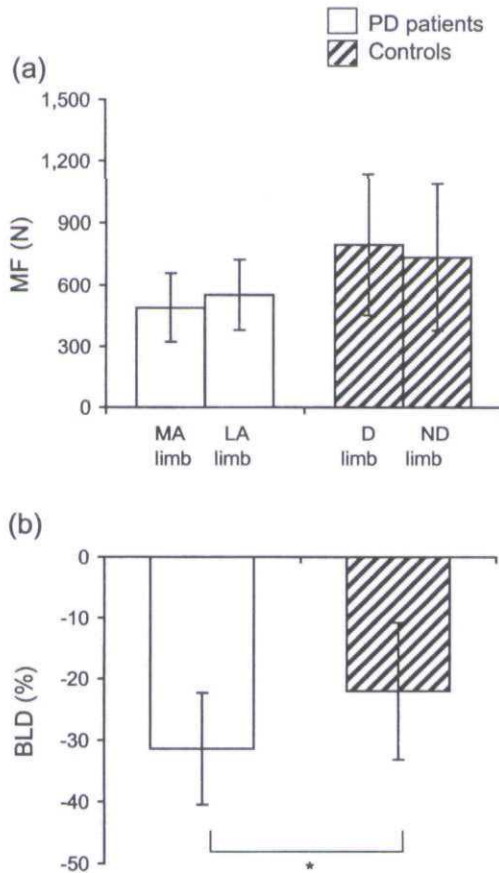
Figure 2. Mean (± *SD*) values of (a) maximal isometric unilateral leg-extension force (MF) and (b) bilateral strength deficit (BLD) in elderly female Parkinson's (PD) patients (*n* = 12) and controls (*n* = 16). MA = more affected; LA = less affected; D = dominant; ND = nondominant. *$p < .05$.

Table 2 provides the correlation coefficients between various indices of chair-rise performance and the leg-extension isometric force in elderly female PD patients and nondisabled control participants. In PD patients chair-rise time correlated moderately negatively with BL isometric leg-extension MF ($r = -.63$) and BL isometric leg-extension MF relative to body mass ($r = -.47$) and moderately positively with BL strength deficit ($r = .60$). A moderate negative correlation was found between maximal VGRFD while rising from a chair and BL strength deficit ($r = -.41$) in PD patients. In control participants, maximal VGRF while rising from a chair correlated moderately positively with BL isometric leg-extension MF ($r = .42$) and UL isometric MF of the dominant leg ($r = .43$).
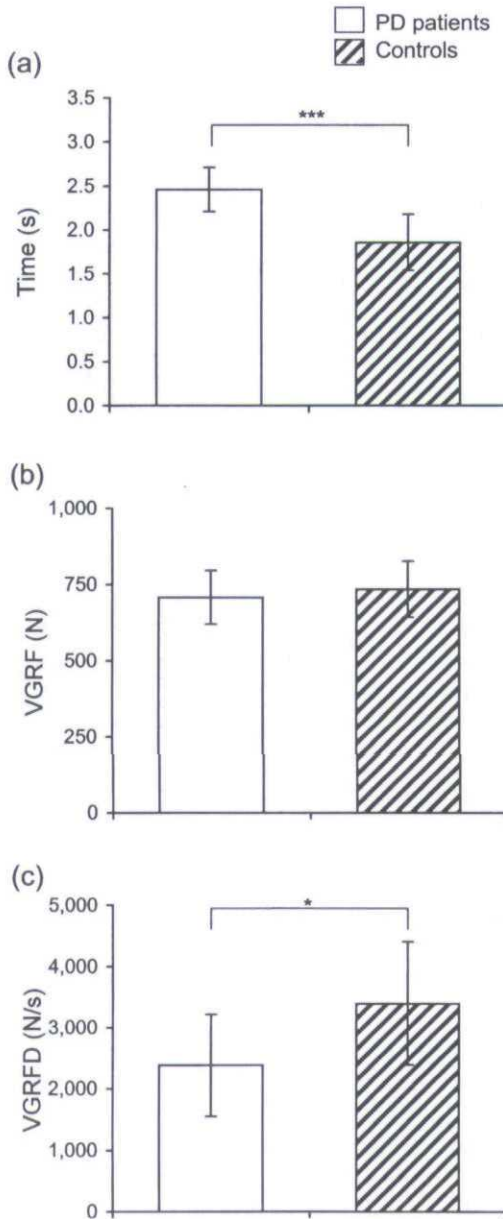
Figure 3. Mean (± *SD*) values of (a) chair-rise time, (b) maximal vertical ground-reaction force (VGRF), and (c) maximal rate of VGRF development (VGRFD) while rising from a chair in elderly female Parkinson's (PD) patients (*n* = 12) and controls (*n* = 16). *$p < .05$; ***$p < .001$.

Table 2   Correlation Coefficients Between the Indices of Isometric Leg-Extension Force and Chair-Rise Performance in Female Parkinson's Patients and Controls

	Patients ($n = 12$)			Controls ($n = 16$)		
	VGRF	VGRFD	T_{rise}	VGRF	VGRFD	T_{rise}
BL	−.16	.08	−.63*	.42	.24	−.17
BL/BM	−.36	.10	−.47	.20	.30	−.28
RFD	.02	−.11	−.30	−.02	.16	−.11
UL_{MA}	−.02	−.08	−.17	.43	.25	−.12
UL_{LA}	−.24	−.10	−.08	−.23	.12	−.02
BLD	.08	−.41	.60*	−.10	−.05	.21

Note. VGRF = maximal vertical ground-reaction force while rising from a chair; VGRFD = VGRF development while rising from a chair; T_{rise} = chair-rise time; BL = maximal isometric bilateral leg-extension force; BL/BM = BL relative to body mass; RFD = maximal rate of isometric bilateral leg-extension force development; UL = maximal isometric unilateral leg-extension force; BLD = bilateral strength deficit. Subscripts MA and LA refer to the more and less affected leg in Parkinson's patients, respectively.
*$p < .05$.

Discussion

The major findings of this study were that (a) female PD patients had lower maximal voluntary isometric force-generating capacity of the leg-extensor muscles during bilateral contractions and greater bilateral strength deficit than those of age- and sex-matched controls and (b) chair-rise performance reduction was most obvious in PD patients with decreased bilateral isometric force-generating capacity of the leg-extensor muscles.

Maximal isometric bilateral leg-extension force, maximal isometric bilateral leg-extension force relative to body mass, and maximal rate of isometric bilateral leg-extension-force development in PD patients were 37.3%, 34.7%, and 36.0% lower than in control participants, respectively. The present results are in agreement with several previous findings (Kakinuma, Nogaki, Pramanik, & Morimatsu, 1998; Pedersen, Oberg, Larsson, & Lindval, 1997; Saltin & Landin, 1975).

The specific cause of muscle weakness during PD is not known, and questions such as whether it is of central (Yanagawa, Shindo, & Yanagisawa, 1990) or peripheral origin (Pedersen, Backman, & Oberg, 1991) and intrinsic to the disease (Nogaki, Fukusako, Sasabe, Negoro, & Morimatsu, 1995) or a secondary phenomenon remain a matter of debate. Muscle voluntary isometric force production might be limited by the central nervous system's ability to activate maximally all agonist muscles or to control antagonist muscles. Patients with PD often demonstrate excessive coactivation of antagonist muscles in the lower extremities in activities of daily living such as standing or sitting (Horak, Nutt, & Nashner, 1992) and

during strength testing (Glendinning & Enoka, 1994). Therefore, increased levels of coactivation of antagonist muscles might restrain the action of the agonist muscles and reduce isometric force production in PD patients. Weakness and slowness in generating force during maximal voluntary isometric contraction might be an expression of the general bradykinetic features and more variable motor-unit firing. It has been suggested that in patients with PD, motor-unit behavior is altered so that the discharge patterns of motor units are irregular and intermittent, and a greater number of motor units are recruited at low thresholds than in age-matched controls (Dengler et al., 1990; Glendinning & Enoka). Possible reasons for these changes include imbalance in excitatory and inhibitory inputs to motor neurons, adaptation in motor neurons secondary to disuse, or deviation in the normal aging process (Glendinning & Enoka). In addition, peripheral changes have been reported in PD. Muscle biopsies taken from biceps brachii (Edstrom, 1970) and tibialis anterior (Rossi et al., 1996) from patients with PD have shown an increased number of slow-twitch (Type I) fibers and decreased number of fast-twitch (Type II) fibers. It is not known, however, whether these changes are attributed to the disease process or are secondary to reduced mobility. In the present study, patients had only mild to moderate PD; hence, reduced mobility was not an important factor.

Several investigators have reported differences in strength between more and less affected sides in patients with PD (Kakinuma et al., 1998; Nogaki et al., 1995), indicating that decreased strength is caused by the effects of the disease process. The present study, however, demonstrated no significant differences in isometric strength between the more affected and less affected legs in elderly female patients with mild to moderate PD. The aforementioned studies measured isokinetic muscle strength of lower extremities at different angular velocities in PD patients who showed marked laterality in symptoms (tremor, rigidity). We measured isometric strength of the lower extremities without special selection of PD patients by marked laterality, and, therefore, this might be the reason for these conflicting data.

Corcos, Chen, Quinn, McAuley, and Rothwell (1996), who measured elbow-flexor and -extensor muscle strength in PD patients, showed that the reduced maximal muscle strength is related to a reduced rate of force development in some patients. The present study indicated a moderate positive correlation ($r = .53$) between maximal isometric force and rate of isometric force development of the leg-extensor muscles during bilateral exertion in PD patients.

This study showed a marked negative bilateral index, that is, a bilateral strength deficit of the leg-extensor muscles in elderly female PD patients and non-disabled controls. The mean values of bilateral index in PD patients and age- and sex-matched controls were –31.4% and –22.0%, respectively, and significant differences between the groups were found. It was one of the most important finding of the present study. There were 2 nondisabled women, however, who had positive bilateral indexes, that is, bilateral facilitation. Less information is available on bilateral strength deficit in participants with PD. Several investigators, however, have observed bilateral strength deficit of the leg-extensor muscles in healthy adult participants. Taniguchi (1997) has published data on bilateral index

ranging from –19% to –7% in male students. Secher et al. (1988) reported bilateral indexes of –20% in untrained, –14% in weight lifters, and –24% in cyclists. The bilateral index described in the study of Schantz, Moritani, Karlson, Johansson, and Lundh (1989) was –14% in the untrained male group and –8% in a heavy-resistance-trained male group.

Neural mechanism seems to be the possible cause of the bilateral strength deficit. The bilateral strength deficit can be caused by reduced activation of either low-threshold (slow) motor units (Secher et al.) or high-threshold (fast) motor units (Owings & Grabiner, 1998; Vandervoort et al., 1984). Some investigations have suggested that bilateral strength deficit is the consequence of a disproportionate increase in antagonist coactivation (Howard & Enoka, 1991; Koh, Grabiner, & Clough, 1993), which is typical for those with PD. The mechanisms of bilateral strength deficit themselves, however, have been discussed and are still unclear (Jakobi & Chilibeck, 2001). Therefore, one explanation for the bilateral strength deficit is that there could be reduced neural interaction between the two hemispheres connected by commissural nerve fibers (Oda, 1997; Oda & Moritani, 1996). It has been shown that bilateral strength deficit was associated with reduced movement-related cortical potentials caused by a mechanism of interhemispherical inhibition (Ferbert et al., 1992; Oda & Moritani, 1995).

Rising from a chair is one of the motor tasks essential for an elderly adult to maintain an independent life. An understanding of chair-rise dynamics as performed by healthy elderly individuals gives guidance regarding how the task is organized and provides a basis for interpreting abnormalities when they occur. The results of the present study indicated that the elderly female PD patients performed rising from a chair more slowly, with a lower rate of vertical-ground-reaction-force development, than age-matched nondisabled controls. In PD patients the chair-rise time was 24.4% longer and maximal rate of vertical-ground-reaction-force development while rising from a chair was 29.8% lower than in controls. No significant differences in maximal vertical ground-reaction force produced while rising from a chair were observed between the measured groups. Thus, these data suggest that elderly participants with mild to moderate PD seem to be more deficient in the regulation of force–time parameters, rather than simply in force production of the leg-extensor muscles, during BL exertion while rising from a chair. It has been suggested that preprogramming movement requires more effort from PD patients than from nondisabled controls (Praamstra, Meyer, Cools, Horstink, & Stegeman, 1996). It was suggested, however, that in participants with PD, translating intention into movement did not differ significantly from healthy controls (Bloxam, Mindel, & Frith, 1984; Sheridan, Flowers, & Hurrell, 1987).

Correlation analysis indicated a moderately negative relationship between chair-rise time and bilateral maximal isometric leg-extension force ($r = -.63$) and a positive relationship between chair-rise time and bilateral strength deficit ($r = .60$) in PD patients. In PD patients, a moderate negative correlation was observed between maximal VGRFD while rising from a chair and BL strength deficit ($r = -.41$). These results suggest that reduced chair-rise performance was most obvious

in PD patients with considerably decreased voluntary bilateral isometric force-generating capacity of the leg-extensor muscles.

It has been suggested that in elderly individuals with PD the levels of physical activity decrease significantly more rapidly over time than in control participants (Fertl, Doppelbauer, & Auff, 1993; Toth, Fishman, & Poehlman, 1997). Recent studies indicated that exercise training produces greater improvement in activities of daily living and motor performance in patients with mild to moderate PD (Miyai et al., 2000; Reuter, Engelhardt, Stecker, & Baas, 1999). Limitations of the present investigation, however, include a modest sample size and using only female participants. We measured sedentary elderly female participants, and it is not possible to say with any certainty whether the differences in isometric leg-extension force found between PD patients and nondisabled controls were the result of their declining levels of physical activity. The results of the present study indicated, however, that PD patients with lower maximal isometric bilateral leg-extension force and greater bilateral strength deficit typically performed chair rising more slowly. These findings suggest that clinicians caring for elderly patients with mild to moderate PD should monitor leg-extensor muscle strength and recommend to them that they perform strengthening exercises for their leg extensors in order to maintain chair-rise ability.

Acknowledgments

References

Bloxam, C.A., Mindel, T.A., & Frith, C.D. (1984). Initiation and execution of predictable and unpredictable movements in Parkinson's disease. *Brain,* **107**, 371-384.

Brod, M., Mendelsohn, G.A., & Roberts, B. (1998). Patients' experiences of Parkinson's disease. *Journal of Gerontology,* **53**, 213-222.

Corcos, D.M., Chen, C.M., Quinn, N.P., McAuley, J., & Rothwell, J.C. (1996). Strength in Parkinson's disease: Relationship to rate of force generation and clinical status. *Annals of Neurology,* **36**, 79-88.

Corrigan, D., & Bohannon, R.W. (2001). Relationship between knee extension force and sit-up performance in community-dwelling elderly women. *Archives of Physical Medicine and Rehabilitation,* **82**, 1666-1672.

Dengler, R., Konstanzer, A., Gillespie, J., Argenta, M., Wolf, W., & Struppler, A. (1990). Behavior of motor units in Parkinsonism. *Advances in Neurology,* **53**, 167-173.

Edstrom, L. (1970). Selective changes in the size of red and white muscle fibers in upper motor lesion and Parkinsonism. *Journal of Neurological Science,* **11**, 537-550.

Ferbert, A., Priori, A., Rothwell, J.C., Day, B.L., Colebach, J.G., & Marsden, C.D. (1992). Interhemispheric inhibition of the human motor cortex. *Journal of Physiology (London),* **453**, 525-545.

Fertl, E., Doppelbauer, A., & Auff, E. (1993). Physical activity and sport in patients suffering from Parkinson's disease in comparison with healthy seniors. *Journal of Neural Transmission,* **5**, 157-161.

Glendinning, D.S., & Enoka, R.M. (1994). Motor unit behavior in Parkinson's disease. *Physical Therapy,* **74**, 61-70.

Gross, M.M., Stevenson, P.J., Charette, S.L., Pyka, G., & Marcus, R. (1998). Effect of muscle strength and movement speed on the biomechanics of rising from a chair in healthy elderly and young women. *Gait and Posture*, **8**, 175-185.

Hoehn, M.M., & Yahr, M.D. (1967). Parkinsonism: Onset, progression and mortality. *Neurology*, **5**, 427-442.

Horak, F.B., Nutt, J., & Nashner, L.M. (1992). Postural inflexibility in Parkinsonian subjects. *Journal of Neurological Sciences*, **111**, 46-58.

Howard, J.D., & Enoka, R.M. (1991). Maximum bilateral contractions are modified by neurally mediated interlimb effects. *Journal of Applied Physiology*, **70**, 306-316.

Hughes, M.A., Myes, B.S., & Schenkman, M.L. (1996). The role of strength in rising from a chair in the functionally impaired elderly. *Journal of Biomechanics*, **29**, 1509-1513.

Inkster, L.M., Eng, J.J., MacIntyre, D.L., & Stoessl, A.J. (2003). Leg muscle strength is reduced in Parkinson's disease and relates to the ability to rise from a chair. *Movement Disorders*, **18**, 157-162.

Jakobi, J., & Chilibeck, P. (2001). Bilateral and unilateral contractions: Possible differences in maximal voluntary force. *Canadian Journal of Applied Physiology*, **26**, 12-33.

Kakinuma, S., Nogaki, H., Pramanik, B., & Morimatsu, M. (1998). Muscle weakness in Parkinson's disease: Isokinetic study of the lower limbs. *European Neurology*, **39**, 218-222.

Kempster, P.A., Frankel, J.P., Bovingdon, M., Webster, R., Lees, A.J., & Stern, G.M. (1989). Levadopa peripheral pharmacokinetics and duration of motor response in Parkinson's disease. *Journal of Neurology, Neurosurgery and Psychiatry*, **52**, 718-723.

Koh, T.J., Grabiner, M.D., & Clough, C.A. (1993). Bilateral deficits are larger for step than ramp isometric contraction. *Journal of Applied Physiology*, **74**, 1206-1205.

Kokmen, E., & Offort, K.P. (1991). The short test of mental status: Correlations with standardized psychometric testing. *Archives of Neurology*, **48**, 725-728.

Kotake, T., Nobuyuki, D., Toshio, K., Sumi, N., Koyama, Y., & Miura, T. (1993). An analysis of sit-to-stand movements. *Archives of Physical Medicine and Rehabilitation*, **74**, 1095-1099.

Miyai, I., Fujimoto, Y., Ueda, Y., Yamamoto, H., Nozaki, S., Saito, T., & Kang, J. (2000). Treadmill training with body weight support: Its effect on Parkinson's disease. *Archives of Physical Medicine and Rehabilitation*, **81**, 849-852.

Nevitt, M.C., Cummings, S.R., Kidd, S., & Black, D. (1989). Risk factors for recurrent nonsyncopal falls. A prospective study. *Journal of the American Medical Association*, **261**, 2663-2668.

Nogaki, H., Fukusako, T., Sasabe, F., Negoro, K., & Morimatsu, M. (1995). Muscle strength in early Parkinson's disease. *Movement Disorders*, **10**, 225-226.

Oda, S. (1997). Motor control for bilateral muscular contractions in humans. *Japanese Journal of Physiology*, **47**, 487-498.

Oda, S., & Moritani, T. (1995). Movement-related cortical potentials during hand grip contractions with special reference to force and electromyogram bilateral deficit. *European Journal of Applied Physiology*, **72**, 1-5.

Oda, S., & Moritani, T. (1996). Interlimb co-ordination on force and movement-related cortical potentials. *European Journal of Applied Physiology*, **74**, 8-12.

Ohtsuki, T. (1983). Decrease in human voluntary isometric arm strength induced by simultaneous bilateral exertion. *Behavior and Brain Research*, **7**, 165-178.

Owings, T.M., & Grabiner, M.D. (1998). Fatigue effects on the bilateral deficit are speed dependent. *Medicine and Science in Sports and Exercise*, **30**, 1257-1262.

Pääsuke, M., Mõttus, K., Ereline, J., Gapeyeva, H., & Taba, P. (2002). Lower limb performance in older female patients with Parkinson's disease. *Aging: Clinical and Experimental Research*, **14**, 185-191.

Pedersen, S.W., Backman, E., & Oberg, B. (1991). Characteristics of tetanic muscle contraction in Parkinson patients. *Acta Neurologica Scandinavica*, **84**, 250-255.

Pedersen, S.W., Oberg, B., Larsson, L.E., & Lindval, B. (1997). Gait analysis, isokinetic muscle strength measurement in patients with Parkinson's disease. *Scandinavian Journal of Rehabilitation Medicine*, **29**, 67-74.

Praamstra, P., Meyer, A.S., Cools, A.R., Horstink, M.W.I.M., & Stegeman, D.F. (1996). Movement preparation in Parkinson's disease: Time course and distribution of movement-related potentials in movement precueing task. *Brain*, **119**, 1689-1704.

Raudsepp, L., & Pääsuke, M. (1995). Gender differences in fundamental movement patterns, motor performances and strength measurements of prepubertal children. *Pediatric Exercise Science*, **7**, 294-304.

Reuter, I., Engelhardt, M., Stecker, K., & Baas, H. (1999). Therapeutic value of exercise training in Parkinson's disease. *Medicine and Science in Sports and Exercise*, **31**, 1544-1549.

Rossi, B., Siciliano, G., Carboncini, M.C., Manca, M.L., Massetani, R., Viacava, P., & Muratorio, A. (1996). Muscle modifications in Parkinson's disease: Myoelectric manifestations. *Electroencephalography and Clinical Neurophysiology*, **101**, 211-218.

Saltin, B., & Landin, S. (1975). Work capacity, muscle strength and SDH activity in both legs of hemiparetic patients and patients with Parkinson's disease. *Scandinavian Journal of Clinical Investigation*, **35**, 531-538.

Schantz, P.G., Moritani, T., Karlson, E., Johansson, E., & Lundh, A. (1989). Maximal voluntary force of bilateral and unilateral leg extension. *Acta Physiologica Scandinavica*, **136**, 185-192.

Schenkman, M., Berger, R.A., Riley, P.O., Mann, R.W., & Hodge, W.A. (1990). Whole-body movements during rising to standing from sitting. *Physical Therapy*, **70**, 638-651.

Schenkman, M., Hughes, M.A., Samsa, G., & Studenski, S. (1996). The relative importance of strength and balance in chair-rise by functionally impaired older individuals. *Journal of American Geriatrics Society*, **44**, 1441-1446.

Secher, N.H., Rube, N., & Elers, J. (1988). Strength of two- and one-leg extension in man. *Acta Physiologica Scandinavica*, **134**, 333-339.

Sheridan, M.R., Flowers, K.A., & Hurrell, J. (1987). Programming and execution of movement in Parkinson's disease. *Brain*, **110**, 1247-1271.

Taniguchi, Y. (1997). Lateral specificity in resistant training: The effect of bilateral and unilateral training. *European Journal of Applied Physiology*, **75**, 144-150.

Toth, M.J., Fishman, P.S., & Poehlman, E.T. (1997). Free-living daily energy expenditure in patients with Parkinson's disease. *Neurology*, **48**, 88-91.

Vandervoort, A.A., Sale, D.G., & Moroz, J.R. (1984). Comparison of motor unit activation during unilateral and bilateral leg extension. *Journal of Applied Physiology*, **56**, 46-51.

Yanagawa, S., Shindo, M., & Yanagisawa, N. (1990). Muscular weakness in Parkinson's disease. In M.B. Streifler, A.D. Korczyn, E. Mclamed, & M.B.H. Youdim (Eds.), *Parkinson's disease: Anatomy, pathology and therapy* (pp. 256-285). New York: Raven.