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GAIT ANALYSIS, ISOKINETIC MUSCLE STRENGTH MEASUREMENT IN PATIENTS WITH PARKINSON'S DISEASE

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ABSTRACT. The aim of this study was to describe motor performance in Parkinson patients in relation to controls. Gait, concentric isometric and eccentric strength in the ankle dorsiflexors were investigated in 25 patients with Parkinson's disease and in 37 control subjects of the same age. In patients concentric torque was significantly lowered but, eccentric torque was significantly lowered in male patients only. Among controls a significant difference in strength was found between sexes. This was not found in the patient group. No correlation between strength tests and clinical ratings was found in the patient group. Patients walked with significantly lower maximum and ordinary velocity, compared with controls. At constant velocity, stride length was shorter, single support had a shorter duration. The duration for heel on to ball on in males was shorter, reflecting the flat foot strike of Parkinson patients. Single support and the duration of heel on to ball on correlated significantly to the clinical ratings in male but not in female patients. In the male group of patients there was a relation between concentric strength on the one hand and gait velocity, stride length and stride frequency on the other. Female patients generally had fewer symptoms and less deviation from their respective controls in the measurements. The comparatively simple neurophysiological tests presented here may be suitable for evaluation of patients with Parkinson's disease.

Key words: gait, isokinetic, measurement, muscle, Parkinson's disease, strength.

INTRODUCTION

In Parkinson's disease (PD) the principal clinical symptoms are those of rigidity, bradykinaesia and tremor, which reflect basic motor disturbance. Even though the clinical impression when tested at bedside may be that of normal muscle strength, recent studies show that muscle strength is reduced in PD (3, 24–26). The traditionally used methods are clinical scoring (9, 28), isometric strength measurement (14, 25, 26) or resistance measurement in passive movement (2, 7, 28). To evaluate motor function, muscle strength and rigidity, several pieces of equipment and procedures have been elaborated (5, 17, 20, 26, 30). Cinematographic and computer-based optoelectronic devices have been used to describe a complex set of movements (11–13). The above-mentioned technically advanced procedures are in general not readily applicable in clinical practice apart from isometric strength measurement, which can readily be performed at the bedside.

The introduction of methods for quantification of disability are important in the evaluation of different therapeutic and pharmacological interventions as a supplement to the clinical impression. We studied motor performance in two movement test procedures, gait and isokinetic strength testing. In these two procedures quantification methods are readily performed and have good reproducibility. Different kinds of movements put different demands on the motor system. The evaluation of a single joint movement (isokinetic contraction) in combination with the evaluation of a complex movement (gait) can be of advantage when studying movement disorders. In previous work (21, 22) we have also shown that these measurements are strongly influenced by the patient's current medication.

The aim of this study was to describe the different motor performances in the Parkinson patients and to illustrate their mutual relations and their relation to the degree of disability. Our intention was to obtain a better background for objective measurements of different therapeutic interventions. To our knowledge this has previously been done only to a limited extent.

PATIENTS AND METHODS

Subjects

Twenty-five patients were investigated, 14 males, mean age 63.4 years, range 50–69 years, and 11 females, mean age 64

years, range 60–69 years. They were estimated to have a mild-to-moderate idiopathic parkinsonism (Webster rating scale 4–16, Hoehn & Yahr I–III). All were treated with ordinary Parkinson drugs and all were in a clinical steady state. The duration of disease varied from 1 to 8 years. None of the patients had any other major medical, orthopaedic or neurological disease.

The control group consisted of 37 healthy, active senior citizens, 19 males, mean age 59.6 years, range 50–70 years, and 18 females, mean age 61.3 years, range 50–70 years. They included members of a senior citizens' organization, the hospital staff or were relatives of the hospital staff. None had a history of any major medical, orthopaedic or neurological disease. All patients volunteered and the study was approved by the local ethical committee.

Methods

Patients and controls were tested with isokinetic strength measurement and computerized gait analysis according to the following schedule.

Gait analysis. This was done according to a standardized method (21, 22). Patients wore plastic-soled stockings and walked a 10 m distance on a metal net with metal tape foot switches placed over the heel and ball of the foot. They walked at five different self-selected paces, ranging from the slowest possible to the fastest possible. Velocity (vel.), stride frequency (SF) and stride length (SL) and the phases of the stride were calculated on-line by a computer. A stride is a whole gait cycle (a double step). The relation between gait velocity (Vel.), stride length (SL) and stride frequency (SF) is shown by the following equation.

$$\text{Vel. (m/sec)} = \text{SL (m/stride)} \times \text{SF (strides/sec.)}. \quad (1)$$

The correlation between SL and SF is usually very strong ($r > 0.90$) (22).

The relations between phases are shown by the following equations

$$S = ST + SW \quad (2)$$

$$ST = HOBO + FF + HUBU \quad (3)$$

$$ST = SS + DS \quad (4)$$

The abbreviations indicate the duration of the following phases. S = Stride duration, ST = Stance, SW = Swing, SS = Single support, DS = Double support, HOBO = Heel on to ball on, FF = Foot flat, HUBU = Heel up to ball up. Note that the duration of swing equals the duration of contralateral single support.

A ratio between right and left was calculated for each and was called the symmetry factor. The quotient was always set to be below 1. Since there were no systematic differences between left and right, the duration of phases will be presented as mean values of both sides.

Most gait parameters are strongly dependent on velocity. Without normalization the possibility to compare gait results between patients and controls is limited. The usual way to normalize measurements of stride phases is to calculate their percentage relation to stride duration. This method assumes that the relations between phases are constant and independent of velocity, an obviously erroneous assumption. We here present a method where individuals can be compared with each other regardless of the individual variations.

In the present paper the results of actual measurements have been recalculated in relation to constant velocity or to constant stride duration (15, 16). This can be done by using the linear regression, $y = B0 + B1 \times M$, where B0 is the intercept and B1 is the slope of the curve, y the dependent variable and M the independent variable. The regression coefficients can be calculated from actual measurements and used to calculate stride phases and stride length under constant conditions. We chose two different velocities, 0.5 m/sec and 1.1 m/sec, and the approximate corresponding stride durations of 1800 and 1100 ms. These values are within the limits of what all patients could achieve.

Strength testing. Prior to strength testing 10 minutes of warming up was performed on a bicycle ergometer with a load of 1 W/kg. Dorsi- and plantarflexors are important for performance of normal gait. Dorsiflexors were tested because this is a single joint muscle and because the test procedure is simple. Plantarflexors were not tested. Strength of ankle dorsiflexion was tested according to a standardized method (32) with a Cybex II dynamometer (Lumex Inc. New York). Both legs were tested at different angular velocities 0, 15, 30, 120, 180 deg/sec in eccentric and concentric contractions. Two to four measurements were made at each velocity and the best measurement was chosen by the computer. Intervals between tests were 1–2 minutes. Peak torque, torque area, and range of motion (ROM) were recorded.

Electromyography. Integrated EMG (iEMG) from the tibialis anterior muscle and the triceps surae were recorded in the patient group. The intent was to control maximal effort and to record possible antagonistic activity. Silver/silver chloride electrodes were taped on the skin. Impedance was kept below 5 KOhm. The EMG was displayed on a screen by the computer. Area calculations of integrated EMGs were done on-line by the computer. Equal amounts of EMG were recorded when doing the contractions, indicating that they were performed at maximal effort. No antagonistic activity was found. The size of musculus gastronemius was not measured, but no severe atrophy was noted in any of the patients tested.

Clinical scoring. After strength testing, clinical ratings were assessed using two rating systems. The Webster rating scale (28) and the Hoehn & Yahr rating scale (9). The clinical evaluations were made by the same observer (SP). These tests are comparatively easy to perform, with low expenditure of time, and give reliable information on the actual clinical status and symptoms. On the other hand, the ratings give no detailed information on symptoms and activities of daily living.

The complete test session (gait analysis, strength measurement and clinical scoring) lasted about 2 1/2 hours and was done at the same time of the day, preferentially before lunch for the patient group.

Statistics

Correlation analyses between clinical scoring and strength/gait results were carried out using Spearman's correlation coefficient. For comparison between groups, the Student's t-test (two-sample t-test) was used. Non-parametric statistics (Mann-Whitney) were used for comparison of clinical scores between sexes. A two-tailed p value below 0.05 was considered significant. Data are given as means and standard errors of the mean.

RESULTS

Patients and controls did not differ in age, weight, length or distribution of sex.

Clinical ratings did not differ between men and women using the Hoehn & Yahr scales, but on the Webster rating scale men were found to be somewhat more disabled ($p < 0.04$) by the disease.

In the patient group there were no significant differences between the sexes in age or duration of the disease.

Gait

The regression coefficients. The intercept, (B0), and the slope, (B1), for the relations between stride frequency and stride length and for the relations between phases and stride duration are presented in Table I. For stance and for single and double support there were significant differences between patients and controls, especially in the male group. The coefficients given will allow the calculation of expected stride lengths, stride frequencies and phase durations at given velocities and stride durations.

Velocity. Ordinary and maximal velocity were lower in patients, both men and women, than in the corresponding controls (see Table II).

Stride length. Stride length at ordinary and maximum velocity was shorter in male and female patients compared with the stride length of normal men and women (Table I).

Stride length at constant velocity. Changes in stride length at the calculated constant velocity of 0.5 and 1.1 m/s, using the correlation coefficients calculated from the relation between stride length and stride frequency, are shown in Table II. The results show that stride length in patients at these velocities is significantly lower in both men and women in comparison with the control group.

Stride frequency. Stride frequency was significantly lower only in Parkinson women, and then only at maximum velocity (Table II).

Stride frequency at constant velocity. Changes in stride frequency at the calculated constant velocity of 0.5 and 1.1 m/s, using the correlation coefficients, are shown in Table II. The results show that stride frequency at constant velocity increased significantly in both men and women in comparison with the control group. These results are the obvious consequence of stride shortening at constant velocity (see equ. 1).

Stance and swing phase

The phases of the stride will be presented in relation to constant stride durations at 1800 and 1100 ms, which were the approximate means of the real stride durations recorded at the velocities 0.5 and 1.1 m/sec. The results are shown in Table III. There were obvious differences between men and women. Men generally displayed larger differences when compared to controls than did women.

The most striking features found were lengthening of stance, a shortening of single support and a shortening of HOBO, leaving HUBU, unchanged as compared to the

Table I. *Gait parameter regression analysis with gait velocity as the independent variable. The regression coefficients presented as means and standard errors ( ). Coefficients presented as calculated from the equation of linear regression* $Y = B0 + B1 * M$ *will be sufficient for calculation of all other stride parameters according to the equations in Methods. Stride frequency (SF), Stride length (SL), Stance (ST), Single support (SS), Double stance (DS), Heel On-Ball On (HOBO) and Heel-Up-Ball Up (HUBU), for further explanation, see text. Significant differences between patient and control groups indicated by asterix*. * indicates* $p < 0.05$ *and ** indicates* $0 < 0.01$

	Patients $n = 14$		Controls $n = 14$	
Men	B0	B1	B0	B1
SF/SL	−0.014 (0.09)*	0.826 (0.08)ns	−0.361 (0.14)	0.837 (0.10)
ST	−218 (22.2)***	0.797 (0.22)***	−108 (19.8)	0.679 (0.01)
SS	208 (19.0)***	0.204 (0.02)***	133 (11.7)	0.308 (0.01)
DS	−211 (19.8)**	0.296 (0.02)***	−107 (16.6)	0.187 (0.01)
HOBO	18 (8.5) ns	0.022 (0.01)*	10 (3.9)	0.042 (0.003)
HUBU	187 (26.3) ns	0.007 (0.02) ns	145 (10.3)	0.05 (0.01)
	Patients $n = 11$		Controls $n = 15$	
Women	B0	B1	B0	B1
SF/SL	−0.373 (0.13) n	1.192 (0.12) ns	−0.547 (0.09)	1.18 (0.08)
ST	−174 (29.3)*	0.745 (0.03)*	−103 (11.7)	0.677 (0.01)
SS	96 (21.8)**	0.231 (0.02)**	126 (11.7)	0.298 (0.01)
DS	−177 (29.8)*	0.261 (0.02)**	−113 (51.9)	0.191 (0.01)
HOBO	12 (5.8) ns	0.035 (0.007) n	6 (3.5)	0.04 (0.004)
HUBU	62 (16.4) ns	0.093 (0.02) ns	101 (11.0)	0.077 (0.009)

Table II. *Gait results. Data given as means and standard errors (). Velocity given in metres per second (m/s), distance in meters (m) and time in seconds (s), Stride frequency (SF), Stride length (SL), Stance (ST), Single support (SS), Double stance (DS), Heel On-Ball On (HOBO) and Heel-Up-Ball Up (HUBU). For further explanation, see text. The symmetry factor is calculated from the results from the left and the right leg, always given as a quotient below one. Significant differences between patient and control groups indicated by asterix*. * indicates $p < 0.05$ and ** indicates $p < 0.01$*

	Men		Women	
	Patients ($n = 14$)	Controls ($n = 19$)	Patients ($n = 11$)	Controls ($n = 18$)
Maximum velocity				
Velocity m/s	1.47 (0.06)**	2.00 (0.05)	1.47 (0.05)**	1.90 (0.05)
Stride length m/stride	1.34 (0.05)**	1.77 (0.04)	1.27 (0.04)**	1.52 (0.03)
Stride frequency stride/sec	1.07 (0.03)	1.13 (0.02)	1.14 (0.03)*	1.25 (0.03)
Single support symmetry %	0.92 (1.4)**	0.97 (0.4)	0.94 (0.6)**	0.97 (0.4)
HOBO symmetry %	0.72 (2.6)*	0.79 (2.3)	0.74 (3.5)	0.78 (2.6)
HUBU symmetry %	0.80 (2.6)**	0.90 (1.0)	0.84 (1.7)	0.88 (1.7)
Ordinary velocity				
Velocity m/s	0.97 (0.05)**	1.35 (0.04)	1.05 (0.06)*	1.30 (0.05)
Stride length m/stride	1.06 (0.05)**	1.50 (0.04)	1.09 (0.05)**	1.30 (0.04)
Stride frequency m/stride	0.87 (0.02)	0.90 (0.02)	0.94 (0.03)	0.99 (0.02)
Single support symmetry %	0.92 (1.1)**	0.97 (0.3)	0.93 (0.5)	0.97 (0.04)
HOBO symmetry %	0.67 (3.5)*	0.77 (2.5)	0.73 (4.6)	0.81 (1.8)
HUBU symmetry %	0.78 (2.5)**	0.86 (1.6)	0.79 (2.3)*	0.86 (1.5)
Stride length (m) At 0.5 m/s	0.78 (0.02)**	1.00 (0.4)	0.82 (0.02)**	0.92 (0.02)
Stride length (m) At 1.1 m/s	1.17 (0.02)**	1.38 (0.03)	1.14 (0.02)**	1.23 (0.02)

controls. In women the results usually followed the same trend, but differences were not as distinct as in men. Owing to the lowered gait velocity double support thereby increased.

Symmetries in gait. Asymmetries were found for all phases in both groups. The means ranged from 55 to 92% in the patient group and from 69 to 97% in the control group. They were generally more pronounced among the male patients. With one exception, the patient group had a significantly lower value when compared with the corresponding value in the control group. There was no side dominance for left or right.

Strength testing

Active range of motion. ROM for the ankle dorsiflexors was of the same range in both groups (male and female).

Isometric contraction. In isometric contraction the torque output in the left and right ankle dorsiflexors was found to be lower in the Parkinson group (male and female) than in the control group (Table IV A and B).

Concentric contraction. In the patient group concentric torque in men and women was found to be significantly lower at all velocities compared with the values in normal controls (Table IV).

Eccentric contraction. Eccentric contraction in Parkinson males showed a significantly lower torque output at all velocities compared with results from normals. This was in contrast to the findings in the female patients where torque output was normal compared with the controls (Table V).

Torque velocity relation. In both groups the eccentric torque was significantly above, and the concentric torque significantly below, the torque level in the isometric contraction. A typical curvilinear relation is seen (Fig. 1). The slope of this curve as calculated from the isometric

Table III. *Means of left and right stride phases were calculated and presented as means and standard errors ( ). Heel On-Ball On (HOBO) and Heel-Up-Ball Up (HUBU), for further explanation see text. All values are given in milliseconds. Significant differences between patient and control groups indicated by asterix*. * indicates $p < 0.05$ and ** indicates $p < 0.01$*

	Men		Women	
	Patients	Controls	Patients	Controls
1800 ms				
Stance	1217 (20)***	1114 (15)	1166 (24)*	1115 (11)
Single Support	575 (16)***	688 (9)	611 (21)*	663 (11)
HOBO	57 (8)**	86 (6)	75 (10)	77 (5)
HUBU	199 (18)	234 (11)	229 (14)	239 (9)
1100 ms				
Stance	659 (8)	639 (12)	645 (10)	641 (4)
Single Support	432 (7)***	472 (3)	449 (10)	454 (4)
HOBO	42 (4)**	56 (4)	51 (5)	49 (3)
HUBU	194 (11)	199 (6)	164 (6)**	185 (5)

value to the concentric value at the highest velocity is an expression of the decrease of torque with increasing concentric angular velocity. Its value is typically negative, a high negative value indicating that the slope is steep. It was slightly more negative in patients than in controls, but the difference was not significant.

Inter sex differences. No difference was found between sexes in range of motion.

In torque output the difference between sexes was evident in the control group ($p < 0.001$), where the men were much stronger than the women at all angular velocities. The difference was found in both eccentric and concentric contractions.

In the patient group no measurable difference in concentric contraction was found between male and female patients at any angular velocity. In isometric and eccentric contractions there was a significant difference. Males were stronger ($p < 0.03$), but not to the same degree as in the control group. The slope of the concentric torque velocity curve was significantly different between men and women, both in patients and in controls. Men had a higher negative value than women.

Table IV. *Peak torque concentric contraction. Torque values (Nm) given as means and standard errors ( ). Significant differences between patient and control groups indicated by asterix*. * indicates $p < 0.05$ and ** indicates $p < 0.01$. Angular velocity is given as degrees per second (deg/sec). Range of motion (ROM)*

	Right		Left	
	Patients	Controls	Patients	Controls
A. Peak torque *males* (14 patients, 19 controls) concentric contraction (Nm).				
ROM (deg)	65.8 (4.0)	72.7 (2.1)	67.1 (3.2)	74.9 (3.2)
Velocity				
0 deg/sec	29.8 (2.6)**	43.8 (1.7)	31.0 (2.8)**	45.9 (1.4)
15 deg/sec	24.7 (2.1)**	38.8 (1..4)	24.5 (2.8)**	40.8 (1.5)
30 deg/sec	22.5 (2.2)**	36.4 (1.4)	23.4 (2.7)**	39.2 (1.5)
120 deg/sec	12.8 (1.9)**	24.9 (1.6)	12.6 (1.9)**	26.1 (1.8)
180 deg/sec	9.5 (1.2)**	20/9 (1.0)	9.4 (1.3)**	20.7 (1.6)
B. Peak torque *females* (11 patients, 18 controls) concentric contraction (Nm).				
ROM	72.0 (5.4)	72.6 (2.9)	78.0 (3.4)	70.9 (2.5)
Velocity				
0 deg/sec	22.3 (2.0)*	28.6 (1.6)	24.0 (1.9)*	30.2 (1.4)
15 deg/sec	20.6 (1.4)*	25.2 (1.3)	19.5 (2.1)**	28.0 (1.3)
30 deg/sec	19.4 (1.0)**	23.3 (1.0)	18.7 (2.4)**	26.0 (1.3)
120 deg/sec	9.7 (0.9)**	16.8 (0.7)	9.9 (1.1)**	16.9 (1.0)
180 deg/sec	8.0 (0.8)	13.4 (0.8)	7.7 (1.0)**	13.3 (0.9)

Table V. *Peak torque eccentric contraction. Torque values (Nm) given as means and standard errors (). Significant differences between patient and control groups indicated by asterisk*. * indicates $p < 0.05$ and ** indicates $p < 0.01$. Angular velocity is given as degrees per second (deg/sec)*

	Right		Left	
	Patients	Controls	Patients	Controls
A. Peak torque *males* (14 patients, 19 controls) eccentric contraction (Nm).				
Velocity				
30 deg/sec	39.4 (1.8)**	45.5 (1.5)	39.7 (2.8)*	46.9 (1.6)
120 deg/sec	41.0 (2.0)**	48.0 (1.5)	41.6 (2.8)**	52.8 (1.7)
180 deg/sec	41.0 (1.6)*	46.3 (1.6)	40.4 (2.7)**	51.5 (2.0)
B. Peak torque *females* (11 patients, 18 controls) eccentric contraction (Nm).				
Velocity				
30 deg/sec	30.6 (1.3)	36.1 (1.6)	31.5 (1.7)	32.4 (1.4)
120 deg/sec	32.1 (1.4)	34.3 (1.4)	33.4 (2.1)	36.5 (1.4)
180 deg/sec	31.9 (1.3)	33.0 (1.6)	32.4 (1.6)	34.8 (1.5)

Inter extremity difference. In both groups no difference was found in range of motion of dorsiflexors.

No difference in torque between left and right dorsiflexors was found in the group of Parkinson patients, whereas in controls some differences were found.

Correlations

Correlations were found between the Webster rating scale and the following gait parameters for men: stance ($p < 0.05$), single support ($p < 0.02$) and HOBO ($p < 0.001$) calculated at the stride duration 1800 ms. At 1100 ms only HOBO was found to correlate with the Webster rating scale. In women no correlations were found between the Webster rating scale and gait parameters.

The regression coefficients B0 and B1 for the duration of stance, single support and double support relative to stride duration correlated to the Webster rating scale in the male but not in the female group of patients. Significant correlations to the Webster rating scale were also found in the male patient group for HOBO (B1) and for HUBU (B0).

In the male patients the correlation factors between concentric isokinetic strength and gait velocity, stride length and stride frequency increased with increasing gait velocity. There were no significant correlations for these parameters for the male controls at any gait velocity, nor for female patients or controls. These findings clearly disclose that in normal controls the strength plays no major part in gait, whereas in the disabled the muscular strength may be an increasingly important factor for adequate gait performance. No correlations were found between strength test and clinical evaluations in patients. The clinically most affected side did not correlate to the strength measured.

DISCUSSION

The aim of the present paper was to obtain quantitative values of strength and gait in Parkinson's disease (4, 12, 13, 17), and to investigate how they related to each other and to the clinical evaluation of the patient. We have shown previously (21, 22) that some of these values are clearly impaired if medication is withdrawn. The test results are therefore dependent upon the medication used, but to what extent do they also depend upon the stage of the disorder as such? Can they be used to describe the patient's current condition? According to clinical ratings (Webster rating scale), the female patients were not as affected as men, which is reflected in the weaker correlations. The difference in sensitivity between the two tests is probably responsible for the difference in results. Correlations between clinical ratings (Webster rating scale) and gait (stance, single support and HOBO) were found predominantly in men. Previous studies have shown stride length and velocity to be good indicators of the patient's current medication (21). It was therefore surprising that these parameters did not show good correlations to the clinical ratings. An explanation is that in the medication studies the patients were matched against themselves, and the large inter-individual variations which load the present correlations were therefore eliminated. On the other hand, it has been difficult for other authors to establish any correlation with clinical correlates of strength and gait (1, 6, 10, 20, 30).

Single support is a sensitive measure of gait function

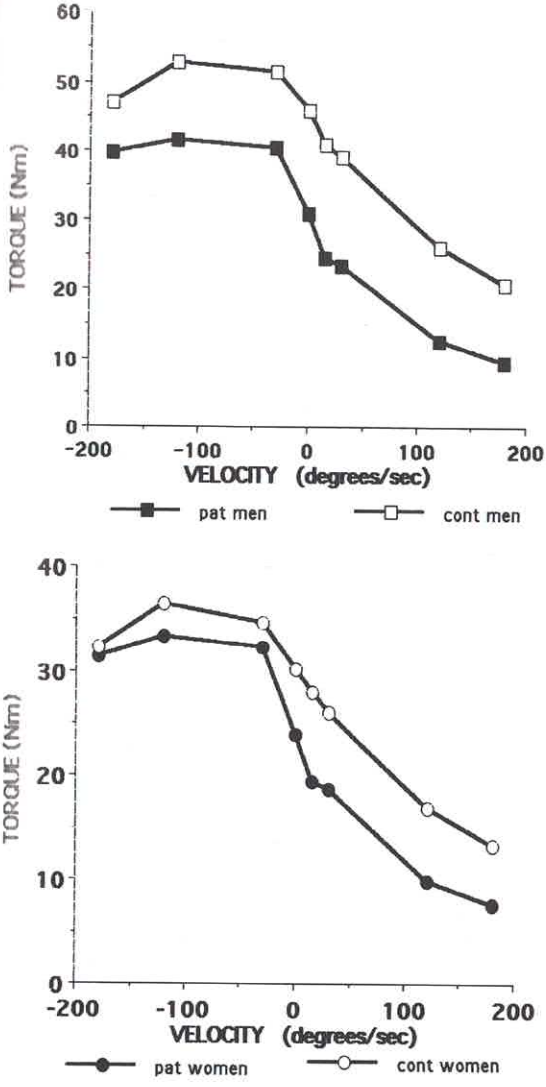

Fig. 1. Torque velocity relation shown for men and women.

in many gait disturbances (16, 23). The duration for single support and HOBO (for men) shows good correlation to the clinical rating, this is clinically reflected in the flat foot strike found in Parkinson patients (7, 10, 12, 13, 18).

The findings that the same relations were quite insignificant for women was unexpected and could not be explained. HUBU in women was significantly shorter for patients if calculated at a stride duration of 1100 ms. The difference compared to the male group may be a chance occurrence, since the male and female values go in the same direction.

The gait analysis may be useful both for individual follow-up of patients acting as their own controls, and for group comparisons.

The strength measurement showed that, in general, patients were weaker than a group of controls of the same age. Torque in isometric and concentric contractions, measured in both men and women, were lower than torque measured in normal controls. Differences were observed in torque production in relation to angular velocity. The torque velocity relation in concentric contraction exhibited the typical non-linear form (29, 32, 33). The torque velocity curves were seemingly parallel in all groups. Comparatively low eccentric torque was found only in Parkinson males, indicating that males were relatively more affected than women. The results supported the findings of clinically observed differences in disability between the sexes.

The eccentric torque production at higher angular velocity in relation to isometric contraction was well preserved. In the patient group the eccentric part of the torque velocity curve was found to increase relatively more compared with isometric torque than was found in controls, suggesting that in patients there may be an increase in muscular stiffness or an inadequate control of autogenetic or other inhibitory muscle-protecting mechanisms (29).

Previous isokinetic measurements have shown that in young adults the left ankle is stronger than the right one (31, 32). This was considered to depend upon the left leg's more frequent function as a supporting leg. In the present control group of old people a corresponding difference was observed, although it is much less obvious. It is not present in the patient group, which is in contrast to Nogaki et al. (20) where an interextremity difference was found. If this interpretation is correct, the decreasing difference in strength between the two legs could be due to a decreasing use of the left leg for support with increasing age and oncoming disease. The strength measurement may be used both for individual follow-up of patients, and for group comparisons.

An additional difference between male and female patients was also demonstrated, in that only the male patients showed an increasing correlation between isokinetic strength in the legs and increasing gait velocity, stride length and stride frequency. In a normal individual these parameters are influenced by several factors, only one of which is strength in the legs. Based on other criteria the male patient group is the one most affected, so it seems that the strength which remains becomes increasingly important as disease progresses.

In conclusion, using the above-described methods,

patients with Parkinson's disease could be monitored and quantitative measurements could be collected for clinical use.

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REFERENCES

1. Bowes, S. G., Charlett, A., Dobbs, R. J., Lubel, D. D., Mehta, R., O'Neill, C. J. A., Weller, C., Hughes, J. & Dobbs, S. M.: Gait in relation to ageing and idiopathic parkinsonism. Scand J Rehab Med *24:* 181–186, 1992.
2. Broberg, C. & Grimby, G.: Measurement of torque during passive and active ankle movement in patients with muscle hypertonia. A methodological study. Scand J Rehab Med *9:* 108–117, 1983.
3. Casanova, D. L., Montmayeur, A., Legallet, E., Trousset, A. & Trouche, E.: Etude biomecanique et electromyographique d'un mouvement de flexion isocinetique d'un coude chez le parkinsonien. Actes du 13eme Congres, Societe de biomecanique, Paris C 30–31, 1981.
4. Dietz, V., Quintern, J. & Berger, W.: Electrophysiological studies of gait in spasticity and rigidity. Evidence that altered mechanical properties of muscle contribute to hypertonia. Brain *104:* 431–449, 1981.
5. Duggan, T. C. & McLellan, D. L.: Measurement of muscle tone: A method suitable for clinical use. Electroencephalogr Clin Neurophysiol *35:* 654–658, 1975.
6. Fernandez, A. M. & Blin, O. A.: Comparison between the effect of intentional modulations and the action of L-Dopa on gait in Parkinson's disease. Behav Brain Res *45:* 177–183, 1995.
7. Forsberg, H., Johnels, B. & Steg, G.: Is parkinsonian gait caused by a regression to an immature walking pattern? Adv Neurol *40:* 375–379, 1984.
8. Halpern, D., Patterson, R. & Makie, R.: Muscular hypertonia: Quantitative analysis. Arch Phys Med Rehab *60:* 208–218, 1979.
9. Hoehn, M. M. & Yahr, M. D.: Parkinsonism: onset, progression and morbidity. Neurology *17:* 427–442, 1967.
10. Hughes, J. R., Bowes, S. G., Leeman, A. L., O'Neill, C. J. A., Deshmukh, A. A., Nicholson, P. W., Dobbs, S. M. & Dobbs, R. J.: Parkinsonian abnormality of foot strike: a phenomenon of ageing and/or one responsive to levodopa therapy. Br J Clin Pharmacol *29:* 179–186, 1990.
11. Johnels, B., Ingvarsson, P. E., Thorselius, M., Valls, M. & Steg, G.: Disability profiles and objective quantitative assessment in Parkinson's disease. Acta Neurol Scand *79:* 227–238, 1989.
12. Knutsson, E. & Mårtensson, A.: Quantitative effects of L-DOPA on different types of movements and muscle tone in Parkinsonian patients. Scand J Rehab Med *3:* 121–130, 1971.
13. Knutsson, E.: An analysis of parkinsonian gait. Brain *95:* 475–486, 1972.
14. Koller, W. & Kase, S.: Muscle strength testing in Parkinson's disease. Eur Neurol *25:* 130–133, 1986.
15. Larsson, L. E., Odenrick, P., Sandlund, B., Weitz, P. & Öberg, P. Å.: The phases of the stride and their interaction in human gait. Scand. J Rehab Med *12:* 107–12, 1980.
16. Larsson, L. E.: Gait analysis: an introduction. *In* Scientific Basis of Clinical Neurology (ed. M. Swash & J. Kennard), pp. 98–107. Churchill Livingstone, New York, 1985.
17. Larsson, L. E. & Olsson, J. E.: Measurement of gait. *In* Parkinson's disease (ed. F. C. Rose), pp. 107–115. Demos Publications, New York, 1990.
18. Martin, J. P.: The basal ganglia and posture. Pitman Medical, London, 1967.
19. Nistor, L., Markhede, G. & Grimby, G. A.: Technique for measurement of plantar flexion with Cybex II dynamometer. Scand. J Rehab Med *14:* 163–166, 1982.
20. Nogaki, H., Fukusaki, T., Sasabe, F., Negoro, K. & Morimatsu, H.: Muscle strength in early Parkinson's disease. Mov Disord *10:* 225–226, 1995.
21. Pedersen, S. W., Eriksson, T. & Öberg, B.: Effects of withdrawal of antiparkinson medication on gait and clinical score in the Parkinson patient. Acta Neurol Scand *84:* 7–13, 1991.
22. Pedersen, S. W. & Öberg, B.: Dynamic strength in Parkinson's disease. Quantitative measurements of withdrawal of medication. Eur Neurol *3:* 97–102, 1993.
23. Perry, J.: Clinical analyser. Bull Prosthet Res 188–192, 1974.
24. Saltin, B. & Landin, S.: Work capacity, muscle strength and SDH activity in both legs of hemiparetic patients and patients with Parkinson's disease. Scand J Clin Invest *5:* 531–538, 1978.
25. Stelmach, G. H. & Worringham, C. J.: The preparation and production of isometric force in Parkinson's disease. Neurophysiologia *26:* 93–103, 1988.
26. Stelmach, G. E., Teasdale, N., Philips, J. & Worringham, C. J.: Force production characteristics in Parkinson's disease. Exp Brain Res *76:* 165–172, 1989.
27. Webster, D. D.: Rigidity in extrapyramidal diseases. J Neurosurg 24(supp 11): 299–307, 1966.
28. Webster, D. D.: Critical analysis of the disability in Parkinson's disease. Med Treat *5:* 257–282, 1968.
29. Westing, S.: Skeletal muscle strength in man with special reference to eccentric torque-velocity characteristics. Medical dissertation. Karolinska Institute Stockholm Sweden, pp. 20–25, 1990.
30. Yanagawa, S., Shindo, M. & Yanagisawa, N.: Muscular weakness in Parkinson's disease. *In* Parkinson's Disease: Anatomy, Pathology and Therapy (ed. M. B. Streifler, A. D. Korczyn, E. Melamed & M. B. H. Youdim), pp. 259–269. Raven Press, New York, 1990.
31. Öberg, B.: Effect of vertical jump training on muscle strength and jump performance in soccer players. Int J Sports Med *5:* 213–216, 1984.
32. Öberg, B., Bergman, T. H. & Tropp, H.: Testing of isokinetic muscle strength in the ankle. Med Sci Sport Exercise *19:* 318–322, 1987.
33. Öberg, B., Larsson, L. E.: Eccentric and concentric characteristics of ankle dorsiflexors in patients with neuromuscular disorders and in healthy subjects. *In* International Series on Biomechanics, Biomechanics XI-A (ed. G. de Groot, A. P. Hollander & G. van Ingen Schenau), Vol. *7A* pp. 237–242. Amsterdam, The Netherlands, 1988.

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