Published in final edited form as:

Gait Posture. 2012 March ; 35(3): 373–377. doi:10.1016/j.gaitpost.2011.10.180.

Both Coordination and Symmetry of Arm Swing Are Reduced in Parkinson's Disease

Xuemei Huang, MD, PhD^{1,4,6}, Joseph M. Mahoney, MS⁸, Mechelle M. Lewis, PhD^{1,2}, Guangwei Du, MD, PhD¹, Stephen J. Piazza, PhD^{5,7}, and Joseph P. Cusumano, PhD⁸

¹Department of Neurology, The Pennsylvania State University-Milton S. Hershey Medical Center, Hershey PA 17033

²Department of Pharmacology, The Pennsylvania State University-Milton S. Hershey Medical Center, Hershey PA 17033

³Department of Radiology, The Pennsylvania State University-Milton S. Hershey Medical Center, Hershey PA 17033

⁴Department of Neurosurgery, The Pennsylvania State University-Milton S. Hershey Medical Center, Hershey PA 17033

⁵Department of Orthopaedics & Rehabilitation, The Pennsylvania State University-Milton S. Hershey Medical Center, Hershey PA 17033

⁶Department of Kinesiology, The Pennsylvania State University, University Park, PA 16802

⁷Department of Mechanical Engineering, The Pennsylvania State University, University Park, PA 16802

⁸Department of Engineering Science and Mechanics, The Pennsylvania State University, University Park, PA 16802

Abstract

Objective—A recent study reporting significantly reduced symmetry in arm swing amplitude in early Parkinson's disease (PD), as measured during single strides in a gait laboratory, led to this investigation of arm swing symmetry and coordination over many strides using wearable accelerometers in PD.

Methods—Forearm accelerations were recorded while eight early PD subjects and eight Controls performed eight-minute walking trials. Arm swing asymmetry (ASA), maximal cross-correlation (MXC), and instantaneous relative phase (IRP) of bilateral arm swing were compared between PD and Controls. Correlations between arm swing measurements (ASA and MXC) and Unified PD Rating Scale (UPDRS) scores were estimated.

Results—PD subjects demonstrated significantly higher ASA ($p = 0.002$) and lower MXC ($p < 0.001$) than Controls. The IRP probability distribution for PD was significantly different than

© 2011 Elsevier B.V. All rights reserved.

Corresponding Author: Xuemei Huang, M.D., Ph.D., Penn State University-Milton S. Hershey Medical Center, H037, Department of Neurology, 500 University Drive, Hershey, PA 17033-0850, Phone: 717-531-1803, Fax: 717-531-0226, xuemei@psu.edu.

Publisher's Disclaimer: This is a PDF file of an unedited manuscript that has been accepted for publication. As a service to our customers we are providing this early version of the manuscript. The manuscript will undergo copyediting, typesetting, and review of the resulting proof before it is published in its final citable form. Please note that during the production process errors may be discovered which could affect the content, and all legal disclaimers that apply to the journal pertain.

Conflict of interest statement

The authors report no conflicts of interest.

Controls ($p < 0.001$), with an angular standard deviation of 67.2° for PD and 50.6° for Controls. Among PD subjects, ASA was significantly correlated with the UPDRS score for the limbs ($R^2 = 0.58$, $p = 0.049$), whereas MXC was significantly correlated with the tremor subscore of the limbs ($R^2 = 0.64$, $p = 0.031$).

Discussion—The study confirms previously reported higher arm swing asymmetry in PD but also shows there is significantly *lower* MXC and *greater* IRP variability, suggesting that reduction in bilateral arm coordination may contribute to clinically observed asymmetry in PD. The differential correlation of clinical measures of motor disability with measurements of arm swing during gait is intriguing and deserves further investigation.

Keywords

Parkinson's disease; Arm swing asymmetry; Maximum cross correlation; Relative Phase; Synchronization; Gait; Accelerometers

Introduction

Tremor, rigidity, bradykinesia, and postural instability are the hallmarks of the diagnosis of Parkinson's disease (PD) [1,2] with abnormal gait (*e.g.*, small “shuffling” steps and falls) more common in the later stages of the disease. Most comprehensive assessments of gait in PD have focused on the lower extremities, noting reduced walking velocity, stride length, proportion of swing time to stance time, and cadence [3-5]. Dynamical analysis of walking has demonstrated irregular timing of strides [6,7] and reduced bilateral coordination of heel strike timing in PD [8], implying impairment of a central locomotor pattern generator and altered basal ganglia regulation of walking.

Although decreased arm swing is the most frequently reported motor dysfunction in individuals with PD [6], and arm swing changes in PD are associated with elevated fall risk [7], there have been only few attempts to describe the changes in the motions of the upper extremities of PD patients during walking. Recently, Lewek et al. [8] demonstrated a significant difference in the asymmetry of arm swing amplitude measured during single strides recorded in a gait laboratory between early PD and healthy control subjects (hereafter termed *Controls*), but left unresolved whether arm swing asymmetry was due to asymmetrical differences in rigidity and bradykinesia, the breakdown of the central synchronization between the arms during walking, or possibly to a combination of these factors.

The goal of the current study was to characterize the dynamics of arm swing coordination during walking in PD subjects and healthy Controls using forearm angular accelerations collected during extended walking trials. We hypothesized that we would see differences between PD patients and Controls not only in the amplitude of the arm swing accelerations but also in their coordination. To make it possible to determine how coordination may be affected by conditions such as PD, we attached accelerometers to each forearm and used dynamical analysis techniques focused on the relative timing of the accelerations as well as their magnitudes. The use of wearable accelerometers facilitated the collection of data over a large number of consecutive strides and may form the basis for future clinical and population-based studies for which motion analysis in the gait laboratory is not practical.

Methods

Participants

Eight PD patients within three years of clinical diagnosis of PD were recruited through the Penn State Hershey Medical Center Movement Disorder Clinic. The Controls group was made up of eight individuals age- and sex-matched to the PD group. PD diagnosis was confirmed by a movement disorder specialist (XH) according to published guidelines [1]. The mean age of the PD subjects ($\pm$ SD) was 63.2 ± 8.4 years versus 62.1 ± 7.3 years for Controls. For the PD group the UPDRS III motor score was 10.5 ± 4.5 , and the Hoehn-Yahr score 1.3 ± 0.5 . The mean disease duration ($\pm$ SD) was 15.5 ± 13.1 months, and the levodopa equivalent dose (LED) [9] was 262 ± 179 mg for PD subjects. Each group had one male and seven females. No participants had a history of stroke, pathology of or surgery to the upper or lower extremities (including joint replacements), or major medical or neurological illness other than PD. The study protocol followed the Helsinki principles, and was reviewed and approved by The Pennsylvania State University Hershey Medical Center Institutional Review Board (IRB # 31265). Written informed consent was obtained from each participant prior to testing.

Accelerometry of Arm Swing During Walking Trials

Accelerometer assemblies were affixed to the right and left forearms of each subject. Each assembly (mass=152 g) consisted of two triaxial G-Link accelerometers (MicroStrain, Inc.; Williston, VT) mounted on a $160 \times 25 \times 3$ mm aluminum bar (Figure 1A). The most distal accelerometer was positioned approximately 30 mm from the wrist. The entire assembly was secured to the forearm using elastic wrap such that the bar was aligned with the long axis of the forearm and parallel to the dorsum of the hand when the wrist was placed in an anatomically neutral position. Each accelerometer was calibrated using the known acceleration due to gravity (9.81 m/s^2), and readings made during two static trials made with each accelerometer placed right side up and upside down on a surface known to be horizontal within 0.4° . A linear fit to these two data points was used to determine the conversion between the raw output (in A/D units) and acceleration (in m/s^2).

The use of pairs of accelerometers separated by a known distance permitted calculation of the angular acceleration of each forearm [10,11], in that the accelerations due to gravity and translation of the elbow from each accelerometer are the same and thus may be canceled out. The tangential acceleration components from the two sensors in each assembly were combined to obtain the angular acceleration α of each forearm in its plane of rotation according to

$$\alpha = (a_{2T} - a_{1T}) / L \quad (1)$$

where a_{1T} and a_{2T} are the tangential components of the acceleration (perpendicular to the long axis of the bar, as shown in Figure 1A) measured by each accelerometer and L is the separation between the accelerometers (100 mm in this study). Data logging at 512 Hz was begun on all four accelerometers simultaneously to ensure synchronization of all four time series. Subjects were instructed to walk continuously for about eight minutes at a comfortable pace around a rectangular circuit in an indoor hallway approximately 400 m long, after which all acceleration data were downloaded to a personal computer.

Data Processing

To obtain data representative of steady walking, the first and last minute were trimmed from each trial to remove transients related to starting and stopping. Angular accelerations were computed using Eq. (1) and low-pass filtered at 50 Hz using a 3rd order Butterworth filter.

Deviations from planarity resulted in small nonzero mean angular accelerations that did not exceed 5% of the total peak-to-peak amplitude for any subject. Because the focus of this study was the oscillatory dynamics of the arm swings, this mean was subtracted. Scatter plots displaying right versus left forearm angular acceleration for typical Control and PD subjects are presented in Figures 1B and 1C, respectively.

For each trial, the root mean square (RMS) amplitude of each angular acceleration was calculated by computing the standard deviation of each mean-subtracted time series. The smaller and larger standard deviations were denoted by $A_{\min}$, and $A_{\max}$, and the *arm swing asymmetry* (ASA) was defined using a variant of the general symmetry angle of Zifchock and colleagues [12]:

$$ASA = \frac{45^\circ - \arctan(A_{\min}/A_{\max})}{45^\circ} \times 100\%. \quad (2)$$

The above definition of ASA is similar to that used by Lewek et al. [8], but differs in that the quantities $A_{\max}$ and $A_{\min}$ are the RMS values of the forearm angular acceleration of approximately 360 strides, whereas Lewek et al. [8] used the maximum excursion of the wrist relative to the pelvis during a *single* stride. In addition, the argument of the arctan function here is the reciprocal of that used by Lewek et al. [8], so the ASA measure used here ranges from 0% to 100%, with values near 0% indicating perfect symmetry of the angular acceleration amplitudes and values near 100% indicating very poor symmetry (*i.e.*, higher values of ASA indicate a *greater* degree of asymmetry).

Next, we let L_n and R_n denote the n^{th} sample of the mean-subtracted, filtered angular acceleration time series from the left and right arms, respectively. The normalized sample cross-correlation [13] at lag k , $R_{LR}(k)$, was computed from

$$R_{LR}(k) = \frac{1}{\sigma_L \sigma_R} \left[\frac{1}{N-1} \sum_{n=0}^{N-k-1} L_{n+k} R_n \right] \quad (3)$$

where N is the total number of samples, σ_L is the standard deviation (RMS amplitude) of the left time series, and σ_R the standard deviation of the right time series. The maximum magnitude of $R_{LR}(k)$ defined the maximum cross correlation (MXC). High values of MXC indicate a high degree of statistical dependence between the arms and thus suggest a greater degree of interlimb coordination during gait. For all subjects in both groups, the MXC occurred when $R_{LR}(k) > 0$, for a lag that was equal to about half of the arm swing period. This is consistent with what one would expect for two similar periodic arm swings that are nominally 180° out of phase with each other.

Finally, the following complex signals were constructed:

$$\begin{aligned} Z_L &= L + iL_H \\ Z_R &= R + iR_H \end{aligned} \quad (4)$$

where L_H and R_H are the Hilbert transforms [14] of original time series L and R , and i is the square root of -1 . We then write each complex signal in amplitude and phase form:

$$\begin{aligned} Z_L &= A_L e^{i\phi_L} \\ Z_R &= A_R e^{i\phi_R} \end{aligned} \quad (5)$$

which defines the *instantaneous phase* time series ϕ_L and ϕ_R . Using these, we computed the instantaneous *relative phase* (IRP) by $\Delta\phi_{LR} = \phi_L - \phi_R$. The IRP was computed to lie in the

interval $[0^\circ, 360^\circ)$. We expected the IRP to be approximately equal to 180° *on average* since that is the nominal phase relationship observed between bilateral limb pairs during gait. The probability distribution of IRP gives insight into the regulation of this phase relationship: a distribution with a sharp peak near 180° indicates strong synchronization and greater bilateral coordination between the right and left arms.

Statistical Analysis

Statistical analyses were performed using MATLAB (Version 7.10.0, The MathWorks, Inc.; Natick, MA). One-way ANOVAs were used to test for group differences in the ASA and MXC values, with two-tailed $\alpha = 0.05$. MATLAB also was used to quantify the relationship between MXC and ASA values using linear regression, and to test the difference between IRP empirical probability distributions for both groups using a two-sample Kuiper test on the unbinned data, with $p < 0.05$ considered significant. Associations between arm swing measurements and clinical scores were quantified by multiple linear regression analysis performed using the REG procedure in SAS 9.2 (SAS Institute Inc., Cary, NC, USA), with UPDRS score or subscore of limbs used as an independent variable and age included as a covariate. The coefficient of determination (R^2) and the t-statistic for the significance of the slope were used to examine the association between arm swing measurements and clinical scores. R values > 0.6 (or $R^2 > 0.36$) and p values < 0.05 were defined as indicating significant correlations.

Results

Clear differences were observed between the forearm accelerations of PD subjects and Controls during the walking trials. Compared to PD subjects, Controls had time series that tended to be symmetrically distributed about a 45° line in a characteristic shape resembling a wing (Figure 1B). For Controls, the typical maximum angular acceleration on one side occurred near the typical minimum angular acceleration on the other side. In contrast, PD patients display less symmetry with respect to the 45° line, and have a more disordered appearance (Figure 1C).

For the arm that swung less, the RMS amplitude ($A_{\min}$) was significantly lower for PD subjects than for Controls ($p = 0.003$, Figure 2A), whereas this measure failed to show a significant difference for the arm that swung more ($A_{\max}$, $p = 0.061$, Figure 2A). PD patients had significantly increased ASA compared to Controls ($p = 0.002$, Figure 2B). In addition, MXC for PD subjects was highly significantly less than for Controls ($p < 0.001$, Figure 2C). Across all subjects, MXC was highly significantly correlated with ASA ($R^2 = 0.69$, $p < 0.001$; see Figure 3). As illustrated, however, analysis within groups showed that the correlation between MXC and ASA lost significance if only PD subjects were included ($R^2 = 0.26$, $p = 0.190$), whereas among Controls the relationship was again highly significant ($R^2 = 0.73$, $p = 0.007$).

The Kuiper test showed that the difference between the IRP distributions was highly significant ($p < 0.001$) (Figure 4). While the EPDF was unimodal for both groups with peaks near 180° (angular mean: 178° for PD patients, 177° for Controls), PD subjects showed substantially greater variability in IRP (angular standard deviation: 67° for PD patients and 51° for Controls).

In PD subjects, ASA was significantly correlated with the UPDRS scores of limbs (equal to the sum of UPDRS akinetic/rigidity and tremor scores of upper and lower extremities; $R^2 = 0.58$, $p = 0.049$ for all limbs, and $R^2 = 0.69$, $p = 0.021$ for most affected limbs, Table 1). The correlations of ASA with either UPDRS akinetic/rigidity or tremor subscores, however, did not reach statistical significance (Table 1). The MXC was not significantly correlated with

either the UPDRS total score or akinetic/rigidity subscore of all limbs (Table 1). MXC was, however, significantly correlated with the UPDRS tremor subscore of all limbs ($R^2 = 0.64$, $p = 0.031$).

Discussion

This study demonstrated for the first time that PD subjects have reduced bilateral coordination of arm swing during walking (Figures 2C and 4). The MXC measurements of coordination were found to correlate with reduced arm swing symmetry across all subjects (Figure 3). The correlation between MXC and ASA was stronger and more significant among Controls, perhaps indicating that this is a sign of healthy motor function. This correlation, however, is apparently not obligatory, as it was much weaker among PD subjects. The alterations in coordination and ASA were associated with different clinical motor signs of PD. Specifically, ASA was correlated with the total UPDRS score of all limbs, representing the sum of tremor, rigidity, and bradykinesia, whereas the MXC was strongly correlated with the total tremor subscore (Table 1). The differential clinical correlations of arm swing asymmetry and arm swing coordination suggest that these measurements may be used complementarily to identify PD-related changes, as well as to understand the control of arm swing during normal walking.

Lewek et al. [8] demonstrated that wrist excursions relative to the trunk recorded during single strides in a gait laboratory were more asymmetrical in early PD subjects. Zampieri et al. [15] reported similar asymmetries in peak arm swing velocity measured using wearable sensors during timed up-and-go tests. In our study, the forearm angular accelerations were measured and substantially higher asymmetry in the amplitude of arm swing angular accelerations were found in PD gait as expected [8,15] given that motor dysfunction is known to asymmetrically affect patients with PD [16].

Reduced synchronization or coordination between both legs has been reported in PD [17-20], and has been associated with the occurrence of “freezing” during gait in the later stages [21]. The reduced MXC values constitute, to our knowledge, the first report of reduced bilateral coordination of arm swing during PD gait. In addition, the present study investigated the nature of this reduced bilateral coordination in PD gait by examining IRP properties. Whereas subjects in both groups maintained a phase difference of approximately 180° between arms, PD patients exhibited much greater variability in the phase angle than did Controls (Figure 4). This suggests that the synchronization between arms is not as well regulated in PD and further reinforces the interpretation of lower values of MXC indicating reduced coordination. Together, these data support the hypothesis that PD negatively affects locomotor central pattern generation and basal ganglia regulation during walking.

Interestingly, MXC was shown to be strongly correlated with ASA in Controls, but not in PD subjects. If a central pattern generator regulates the motions of the legs and arms and their asymmetry of movements in Controls [22], it may be that disruption of its function leads to increased asymmetry and reduced coordination. Our findings for PD subjects, however, suggested additional determinants of the observed arm swing asymmetry. For example, a moderate correlation ($R^2=0.33$, Table 1) was found between ASA and bradykinesia/rigidity of the more affected limbs in this study, suggesting that increased tone or slowness in initiating movement may contribute in part to the observed asymmetry. This correlation, however, was not statistically significant ($p = 0.184$), and further study with bigger sample size may be useful.

The significant correlation between MXC and tremor in PD is intriguing and may suggest that diminished bilateral arm swing coordination and tremor genesis share the same central

mechanisms. Our finding that the correlation between MXC and ASA is strong in Controls but weaker in PD subjects (Figure 3) suggests that reduced MXC is not attributable to tremor alone, but is an indicator of the overall quality of regulation and bilateral coordination of arm swing. It has been suggested that tremor in PD has an underlying pathophysiology that differs from that of bradykinesia and rigidity [23,24], and a role for the cerebellum in tremorgenesis has been suggested [25]. Thus, further studies of the role of the cerebellum in bilateral arm coordination are warranted.

There are several limitations to this study. Forearm angular acceleration represents just one measure that could be used to characterize arm swing. Other potentially useful parameters include measures of position, such as wrist excursion [8] and shoulder [5,26-28] and elbow joint angles [29]. We chose acceleration based on the hypothesis that it would be more sensitive to motor force outputs than positions (the latter represent accelerations that have been integrated twice with respect to time). The paired-accelerometer assemblies we used permitted forearm angular accelerations to be measured only in the quasi-sagittal plane fixed to the forearm. Simultaneous three-dimensional tracking of the forearm segments using a motion analysis system would help determine how much arm swing actually occurred that was not in this plane. Methods other than MXC, such as mutual information, synchronization measures, and cross prediction, also may prove useful for quantifying coordination. In this study, PD patients were taken off anti-PD medications overnight. Although commonly used as a “practically defined” off state for PD patients [30], residual drug effects may be present. It would be of interest to characterize arm swing asymmetry in newly diagnosed, drug naïve patients.

This study made use of wearable inertial sensors that have great potential for measurement of arm swing and other motions in PD patients. Such sensors are relatively inexpensive and easier to implement in a clinical setting than video-based motion analysis typical of gait analysis laboratories. The large amount of data that can be collected enables the application of new dynamical analysis techniques for diagnosing and tracking the progress of neuromotor disorders. Inertial sensors may be especially useful in clinical or population studies aimed at early and differential diagnosis of PD, and tracking its progression.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

This work was partially supported by NS060722. We also would like to thank all the participants in the study, as well as the support of the study coordinator Ms. Eleanore Hernandez and technical support from Nori Okita, Noam Shaham and Josh Baxter. We also would like to thank Dr. Richard Mailman for his helpful comments on the manuscript. The sponsor had no involvement in the study design; collection, analysis, and interpretation of the data; or in the writing of the manuscript or decision to submit the manuscript for publication.

Reference List

- (1). Gelb DJ, Oliver E, Gilman S. Diagnostic criteria for Parkinson disease. Arch Neurol. Jan; 1999 56(1):33–9. [PubMed: 9923759]
- (2). Calne DB, Snow BJ, Lee C. Criteria for diagnosing Parkinson's disease. Ann Neurol. 1992; 32(Suppl):S125–S127. [PubMed: 1510370]
- (3). Alice N, Fabienne C, Anne-Marie W, Kaat D. Does freezing in Parkinson's disease change limb coordination? A kinematic analysis. J Neurol. Sep; 2007 254(9):1268–77. [PubMed: 17401738]

- (4). Ferrarin M, Rizzone M, Bergamasco B, Lanotte M, Recalcati M, Pedotti A, et al. Effects of bilateral subthalamic stimulation on gait kinematics and kinetics in Parkinson's disease. *Exp Brain Res*. Jan; 2005 160(4):517–27. [PubMed: 15502989]
- (5). Knutsson E. An analysis of Parkinsonian gait. *Brain*. 1972; 95(3):475–86. [PubMed: 4655275]
- (6). Nieuwboer A, De WW, Dom R, Lesaffre E. A frequency and correlation analysis of motor deficits in Parkinson patients. *Disabil Rehabil*. Apr; 1998 20(4):142–50. [PubMed: 9571381]
- (7). Wood BH, Bilclough JA, Bowron A, Walker RW. Incidence and prediction of falls in Parkinson's disease: a prospective multidisciplinary study. *J Neurol Neurosurg Psychiatry*. Jun; 2002 72(6): 721–5. [PubMed: 12023412]
- (8). Lewek MD, Poole R, Johnson J, Halawa O, Huang X. Arm swing magnitude and asymmetry during gait in the early stages of Parkinson's disease. *Gait Posture*. Feb; 2010 31(2):256–60. [PubMed: 19945285]
- (9). Herzog J, Volkmann J, Krack P, Kopper F, Potter M, Lorenz D, et al. Two-year follow-up of subthalamic deep brain stimulation in Parkinson's disease. *Mov Disord*. Nov; 2003 18(11):1332–7. [PubMed: 14639676]
- (10). Elble RJ. Gravitational artifact in accelerometric measurements of tremor. *Clin Neurophysiol*. Jul; 2005 116(7):1638–43. [PubMed: 15905122]
- (11). Liu K, Liu T, Shibata K, Inoue Y, Zheng R. Novel approach to ambulatory assessment of human segmental orientation on a wearable sensor system. *J Biomech*. Dec 11; 2009 42(16):2747–52. [PubMed: 19748624]
- (12). Zifchock RA, Davis I, Higginson J, Royer T. The symmetry angle: a novel, robust method of quantifying asymmetry. *Gait Posture*. May; 2008 27(4):622–7. [PubMed: 17913499]
- (13). Bendat, JS.; Piersol, AG. *Random Data: Analysis and Measurement Procedures*. 4th ed.. Wiley-Blackwell; Hoboken, NJ: 2010.
- (14). Bracewell, R. *The Fourier Transform and Its Applications*. 3rd ed.. McGraw-Hill; New York: 1999.
- (15). Zampieri C, Salarian A, Carlson-Kuhta P, Aminian K, Nutt JG, Horak FB. An Instrumented Timed Up and Go Test Characterizes Gait and Postural Transitions in Untreated Parkinson's Disease. *J Neurol Neurosurg Psychiatry*. Sep 2.2009
- (16). Lee CS, Schulzer M, Mak E, Hammerstad JP, Calne S, Calne DB. Patterns of asymmetry do not change over the course of idiopathic parkinsonism: implications for pathogenesis. *Neurology*. Mar; 1995 45(3 Pt 1):435–9. [PubMed: 7898691]
- (17). Hausdorff JM, Cudkowicz ME, Firtion R, Wei JY, Goldberger AL. Gait variability and basal ganglia disorders: stride-to-stride variations of gait cycle timing in Parkinson's disease and Huntington's disease. *Mov Disord*. May; 1998 13(3):428–37. [PubMed: 9613733]
- (18). Plotnik M, Giladi N, Hausdorff JM. A new measure for quantifying the bilateral coordination of human gait: effects of aging and Parkinson's disease. *Exp Brain Res*. Aug; 2007 181(4):561–70. [PubMed: 17503027]
- (19). Ebersbach G, Heijmenberg M, Kindermann L, Trottenberg T, Wissel J, Poewe W. Interference of rhythmic constraint on gait in healthy subjects and patients with early Parkinson's disease: evidence for impaired locomotor pattern generation in early Parkinson's disease. *Mov Disord*. Jul; 1999 14(4):619–25. [PubMed: 10435499]
- (20). Bartsch R, Plotnik M, Kantelhardt JW, Havlin S, Giladi N, Hausdorff JM. Fluctuation and synchronization of gait intervals and gait force profiles distinguish stages of Parkinson's disease. *Physica A*. Sep 15; 2007 383(2):455–65. [PubMed: 18163154]
- (21). Plotnik M, Giladi N, Balash Y, Peretz C, Hausdorff JM. Is freezing of gait in Parkinson's disease related to asymmetric motor function? *Ann Neurol*. May; 2005 57(5):656–63. [PubMed: 15852404]
- (22). Shapkova EY, Terekhov AV, Latash ML. Arm motion coupling during locomotion-like actions: An experimental study and a dynamic model. *Motor Control*. 2010 In press.
- (23). Marjama-Lyons J, Koller W. Tremor-predominant Parkinson's disease. Approaches to treatment. *Drugs Aging*. Apr; 2000 16(4):273–8. [PubMed: 10874522]
- (24). Jankovic J, Kapadia AS. Functional decline in Parkinson disease. *Arch Neurol*. Oct; 2001 58(10): 1611–5. [PubMed: 11594919]

- (25). Deiber MP, Pollak P, Passingham R, Landais P, Gervason C, Cinotti L, et al. Thalamic stimulation and suppression of parkinsonian tremor. Evidence of a cerebellar deactivation using positron emission tomography. *Brain*. Feb; 1993 116(Pt 1):267–79. [PubMed: 8453462]
- (26). Carpinella I, Crenna P, Marzegan A, Rabuffetti M, Rizzone M, Lopiano L, et al. Effect of L-dopa and subthalamic nucleus stimulation on arm and leg swing during gait in Parkinson's Disease. *Conf Proc IEEE Eng Med Biol Soc*. 2007; 2007:6665–8. [PubMed: 18003554]
- (27). Zijlmans JC, Poels PJ, Duysens J, van der Straaten J, Thien T, van't Hof MA, et al. Quantitative gait analysis in patients with vascular parkinsonism. *Mov Disord*. Sep; 1996 11(5):501–8. [PubMed: 8866491]
- (28). Behrman AL, Teitelbaum P, Cauraugh JH. Verbal instructional sets to normalise the temporal and spatial gait variables in Parkinson's disease. *J Neurol Neurosurg Psychiatry*. Oct; 1998 65(4): 580–2. [PubMed: 9771792]
- (29). Kuhtz-Buschbeck JP, Brockmann K, Gilster R, Koch A, Stolze H. Asymmetry of arm-swing not related to handedness. *Gait Posture*. Apr; 2008 27(3):447–54. [PubMed: 17616462]
- (30). Langston JW, Widner H, Goetz CG, Brooks D, Fahn S, Freeman T, et al. Core assessment program for intracerebral transplantations (CAPIT). *Mov Disord*. 1992; 7(1):2–13. [PubMed: 1557062]

Figure 1.

(A) Schematic of the wireless, portable apparatus used during walking to obtain forearm angular acceleration time series data. One such device was placed on each arm. (B) Typical scatter plot of recorded right (vertical axis) vs. left (horizontal axis) arm angular acceleration for a Control and (C) PD subject. Both axes in the scatter plots have the same scale. The dashed 45° line is included to aid in visualizing the symmetry properties of the arm swing dynamics. For the Control (B), the angular acceleration of the arms is similar for each arm and almost perfectly straddles the symmetry line. For the PD subject (C), the overall angular acceleration of the arms is notably reduced compared to the Control. In addition, the right arm angular acceleration is significantly reduced relative to the left arm, resulting in marked asymmetry.

Figure 2.

Comparison of statistics for bilateral arm swing angular acceleration between PD and Control subjects: (A) smaller and larger RMS amplitudes (standard deviations), A_{min} and A_{max} ; (B) arm swing asymmetry angle, ASA; and (C) maximum cross correlation, MXC. The A_{min} , ASA, and MXC statistics provide very strong discrimination between groups (A_{min} , $p = 0.003$; ASA, $p = 0.002$; MXC, $p < 0.001$). The error bars indicate standard error.

Figure 3.

Scatter plot demonstrating the relationship between arm swing asymmetry angle (ASA) and maximum cross-correlations (MXC) of bilateral forearm angular acceleration during gait. Whereas there is a good overall fit across all subjects (thick gray line), there is a pronounced difference between the groups: the Controls (solid black line) display a highly significant correlation, whereas the weaker correlation among PD subjects (black dashed line) fails to be significant.

Figure 4.

Plot of the empirical probability density function for the instantaneous relative phase (IRP) between the left and right arms, estimated for PD and Control subjects using 100 bins. The Kuiper test applied to the unbinned data shows a highly significant difference between the distributions ($p < 0.001$). While the mean value of the IRP is near 180° for both groups, the PD subjects show substantially greater variability in the phase angle: for PD subjects the angular standard deviation is 67° , whereas for Controls it is 51° .

Table 1
Clinical correlations of arm swing asymmetry and maximal cross-correlations after adjusting for age effects

Clinical characteristics	Arm swing asymmetry (ASA)		Maximal Cross-correlations (MXC)	
	R ²	P values	R ²	P values
UPDRS scores of all limbs	0.58	0.049 *	0.01	0.869
Akinetic rigidity scores of all limbs	0.16	0.380	0.26	0.246
Tremor scores of all limbs	0.08	0.560	0.64	0.031 *
UPDRS of more affected side	0.69	0.021 *	0.05	0.630
Akinetic rigidity score of more affected side	0.33	0.184	0.06	0.586
Tremor scores of more affected side	0.16	0.379	0.42	0.113

* Indicates statistical significance ($p < 0.05$).