ELSEVIER

Gait and Posture 8 (1998) 8–14

GAIT & POSTURE

The initiation of gait in young, elderly, and Parkinson's disease subjects

Suzanne E. Halliday [a,*], David A. Winter [b], James S. Frank [b], Aftab E. Patla [b], François Prince [c]

[a] *Texas Scottish Rite Hospital for Children, 2222 Welborn Street, Dallas, TX 75219, USA*
[b] *Department of Kinesiology, University of Waterloo, Waterloo, Ontario N2L-3G1, Canada*
[c] *Centre de Recherche en Gérontologie et Gériatrie, Sherbrooke, Québec J1H-4C4, Canada*

Accepted 23 April 1998

Abstract

Gait initiation is a transient movement between upright posture and steady-state gait. Kinematic, kinetic and electromyographic data have been documented in healthy young subjects. However, there is little published data on the elderly and on Parkinson's disease (PD) subjects who are known to 'freeze' when initiating gait. It was the purpose of this project to measure gait initiation in young, healthy elderly and PD subjects. The results showed many differences between the young and elderly and the elderly and PD subjects. However, if all dependent variables were normalized to gait velocity there were no differences at the $P < 0.05$ level. The results showed a progressive slowing from the young to the elderly to the PD subjects. There does not appear to be a relationship between the PD subject's steady-state velocity and their age, number of years diagnosed, number of hours off medication, or the rating on the Hoehn and Yahr scale.

Keywords: Gait initiation; Elderly; Parkinson's disease

1. Introduction

1.1. Gait initiation

Both ageing and disease cause changes to the nervous system, muscular system, skeletal system, and every other system in the body. These changes affect the control of balance. The initiation of gait is a task that challenges the balance control system as it moves from stable static balance to continuously unstable gait. The initiation of gait has been well documented in young healthy subjects. There are studies that describe gait initiation with displacement, velocity, and acceleration of the centre of mass (COM), displacement of the centre of pressure (COP), angular displacement and velocity of the limbs, and electromyographic activity of the lower limbs and back. These are summarized in Table 1a. Kinematic, kinetic and electromyographic (EMG) variables of gait initiation have also been reported in the elderly and in Parkinson's disease patients and they are summarized in Table 1b.

1.2. Parkinson's disease

The diagnosis of Parkinson's disease (PD) is based on the presence of two or more of the major symptoms: tremor, rigidity, postural instability, and akinesia. Not all patients exhibit all symptoms. For the purposes of this project it is important to review akinesia.

Akinesia is defined as a lack or poverty of movement. Akinesia in Parkinsonism can be divided into three different parts: (1) slowness and unskilfulness of movement secondary to rigidity, (2) lack or poverty of

* Corresponding author. Tel.: +1 214 5597582; fax: +1 214 5597439; e-mail:tsrhgait@ix.netcom.com

0966-6362/98/$19.00
PII S0966-6362(98)00020-4

Table 1
Previous data collected on the initiation of gait in (a) young healthy subjects and (b) in elderly subjects and patients with Parkinson's disease

Ref.	Data collection
(a) Young healthy subjects	
[1]	Eight subjects age range 21–38. Three channels of EMG analyzed for onset and cessation, with respect to ground reaction forces measured from one force plate.
[2]	Seventeen subjects age range 18–28. Nine channels of EMG analyzed for onset and cessation. Kinematics and kinetics of the ankle, knee and hip measured with electrogoniometers. All variables analyzed with respect to initiation events chosen from two force plates, one under each foot to start. Five subjects were evaluated under conditions with tibial nerve suppression with xylocaine, following peroneal dantrolene sodium and while wearing an ankle foot orthosis designed to restrain ankle motion.
[3]	Ten subjects, age range 23–52. Five channels of EMG analyzed for onset and cessation. Velocity of a tachometer approximating the COM. Kinematics of the ankle, knee and hip measured with electrogoniometers. All variables analyzed with respect to initiation events chosen from one force plate.
[4]	Four subjects, age range 23–34. Documented ground reaction forces from one force plate, while wearing foot switches during gait initiation.
[5]	Ten subjects, age range 19–43. Eight channels of EMG analyzed for onset and cessation. 3D kinematics and kinetics collected of the ankle, knee and hip analyzed with respect to ground reaction forces and COP measured from one force plate.
[6]	Seven subjects. Fifteen channels of EMG analyzed for timing of postural responses. EMG of the tibialis anterior and soleus analyzed with respect to ground reaction forces measured from one force plate.
[7]	Eight subjects initiated gait from an upright position and from a 5° and a 35° forward inclination position. Ground reaction force data were collected from one force plate.
[8]	Six subjects age range 25–35 initiated gait at three different speeds. Data were collected from one force plate and processed to estimate the displacement of the COP and COM.
[9]	Five subjects, age range 25–35 initiated gait at three different speeds. Data were collected from one force plate and processed to estimate the displacement of the COP and COM.
[10]	One subject initiated gait at three different speeds. Data were collected from five triaxial accelerometers and one force plate to calculate the displacement of the COP, the displacement, velocity and acceleration of the COM.
[11]	Fifteen subjects initiated gait while 3D kinematic data were collected with reflective markers of lower limbs and kinetic data were collected from two force plates, one under each foot to start. Angular displacements were documented with respect to ground reaction force data.
[12]	Five subjects initiated gait with three different step lengths. Data were collected from one force plate and processed to estimate the displacement of the COP and COM.
[13]	Nine subjects initiated gait at three speeds. EMG of the tibialis anterior and gastrocnemius were analyzed for timing of onset and cessation, with respect to ground reaction forces measured from one force plate. Five subjects had reflective markers on the ASIS bilaterally and on the sacrum and velocity of the markers was calculated and analyzed with respect to the events from two force plates.
[14]	Six subjects. EMG collected from the tibialis anterior and soleus analyzed for onset and cessation. Kinematic data collected in the sagittal plane with reflective markers. Initiation of gait, rising on toes, stand from sit, throw, catch, and forward bend of trunk events analyzed from one force plate.
[15]	Five subjects initiated gait while data were collected from four triaxial accelerometers. Data were analyzed to estimate the velocity of the COM with respect to ground reaction forces collected from one force plate.
[16]	Four subjects, age range 25–31. 3D kinematic data collected using reflective markers on the whole body. Data analyzed to estimate the COM displacement, velocity and acceleration. Gait initiated from two force plates, and data used to calculate displacement of the COP.
[17]	Eight subjects initiated gait with electrogoniometers at both knees. Angular displacement and velocity were analyzed.
[18]	Seven subjects average age 24 years. 3D kinematic data collected using reflective markers on the whole body. Gait initiated from two force plates, all data used to calculate total energy of the body.
(b) Elderly subjects and patients with Parkinson's disease	
[19]	Fourteen PD patients age range 40–75. Hoehn and Yahr 1–3, off medication and not suffering from akinesia. 3D kinematic data collected using reflective markers on the whole body. EMG of the tibialis anterior and soleus analyzed with respect to ground reaction forces measured from one force plate.
[20]	Five subjects aged 22–47 and six subjects aged 64–82. Eight channels of EMG were analyzed for onset and cessation. 3D kinematics of the body were collected using reflective markers. Data were analyzed to estimate the COM displacement. The COP displacement was calculated from two force plates.
[21]	Five patients with deep cerebral infarcts exhibiting PD-like symptoms (age range, 76–92) and 12 age-matched control subjects. 3D kinematic data collected using reflective markers on the whole body. EMG of the tibialis anterior, soleus, rectus femoris, biceps femoris and gluteus medius analyzed with respect to ground reaction forces measured from two force plates.
[22]	Seven PD patients age range 55–74 and five age-matched controls. PD patients were Hoehn and Yahr 2–3 and suffered from akinesia. Initiation of normal walking, initiation of a single step and placement of one foot behind a mark on the ground performed. Eight channels of EMG were analyzed for onset and cessation with respect to ground reaction forces measured from a force plate. COM estimated from one reflective marker placed on the iliac crest.

Table 2
PD subject characteristics

	WQ									
	26	27	28	29	30	32	34	35	36	37
Sex	M	M	M	M	F	F	F	M	M	M
Age	54	74	65	55	53	75	75	74	68	62
Years diagnosed	10	10	4.5	6	4.5	4	1	7	1	2
Hours off medication	18	17	4.5	4	41	5	6	4	14	7
Hoehn and Yahr	3.0	2.5	2.5	2.5	2.5	3.0	3.0	2.5	2.5	3.0

movement even after complete abolition of rigidity and absence of muscular weakness, and (3) difficulty in initiation of movement also known as 'freezing'. These separations have been made on responsiveness to treatment. 'Freezing', the third type of akinesia, remains one of the most debilitating aspects of PD, and can appear even under complete relief of all other symptoms of PD.

Giladi et al. [23] studied 990 PD patients and found 318 of them had motor blocks. Longer disease duration, longer duration of L-dopa treatment, and higher Hoehn and Yahr rating were associated with the presence of motor blocks. The three motor tasks that are affected by motor blocks are speech, hand-writing, and gait. In gait, 86% of the 318 had blocks in initiation, 45% had blocks in turning, 25% had blocks in narrow spaces or doorways, and 23% had blocks on open runways.

The pathological process behind the motor disabilities of PD is a progressive degeneration of dopaminergic neurons of the substantia nigra, that results in dopamine depletion in the striatum. The majority of input to the basal ganglia is via the striatum. In general, brain dopamine deficiency is sufficient to explain all of the major symptoms of PD [24].

Dopamine-producing cells in a 65-year-old number only between 30–50% of those in a 20-year-old [25], therefore the PD subjects should be compared to age-matched controls in the initiation of gait. Some of the dependent measures used in the present study have not been defined previously, therefore a young control group should be compared to the healthy elderly. The purpose of this study was to identify any atypical biomechanical measures and patterns in the initiation of gait that result from age alone or result from PD.

2. Methodology

Ten healthy young students, 10 healthy elderly subjects with no history of neurological illness or degenerative condition, and 10 PD subjects participated in the study. The young subjects were a mean of 27.1 years old (range 22–37), the healthy elderly were a mean of 60.9 (range 56–65) and the PD subjects were a mean of 65.5 (range 53–75).

PD subjects were specifically chosen, by their treating neurologist, if they were rated either 2.5 or 3.0 on a modified Hoehn and Yahr scale and if they experienced akinesia. Specific characteristics of the PD subjects are detailed in Table 2. A rating of 2.5 indicates that the patient has mild bilateral disease and very mild postural instability. A rating of 3.0 indicates that the patient has mild to moderate bilateral disease and moderate postural instability but, however, remains physically independent; a more detailed description can be found in Fahn [26]. Table 2 shows considerable variation in the number of hours that patients were off medication because patients were tested after being off medication for at least one medication period.

An OPTOTRAK 3D sensor detected the 14 infrared emitting diodes (IREDs) that defined a 10-segment model. Bipolar surface electrodes were used to examine the EMG activity bilaterally of the tibialis anterior (TA) and medial gastrocnemius (MG). Subjects were asked to stand with their feet side by side and parallel at pelvic width, with each foot on a separate force platform five times. Each trial was 16 s in length, the first 10 s being quiet stance. Subjects initiated gait with a self selected foot at their own pace after an auditory cue.

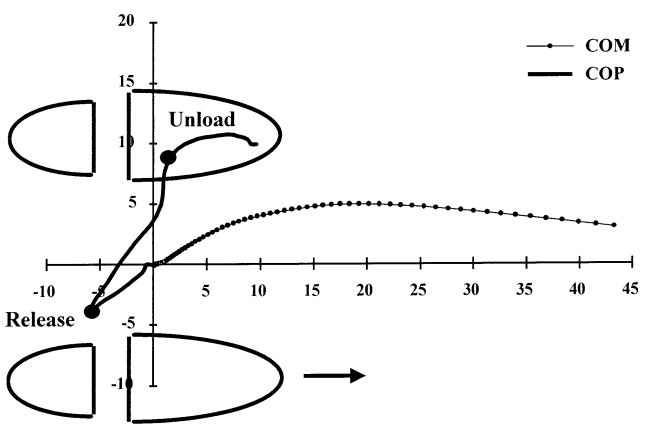

Fig. 1. Previously reported COP and COM displacements in gait initiation.

The 10 s of quiet stance prior to initiation were analyzed only for base line values for the purposes of this paper. As described in many of the papers in Table 1, and as seen in Fig. 1, the COP has a very characteristic pattern in initiation. The COP_{net} from two force plates, in either the A/P or M/L directions is calculated as in Winter et al. [27].

In Fig. 1 the point of maximum backward and medial, toward the first swing foot, displacement of the COP is labelled *release*, this corresponds to the first swing limb heel off. The point of maximum medial shift of the COP toward the first stance limb is labelled *unload*, this corresponds to toe-off of the first swing limb. The displacement to and time of both of these points were calculated in each trial. The horizontal impulse normalized to body weight was also calculated in each trial.

The total body COM location in the A/P, M/L or vertical directions was calculated as the weighted sum of the COM of every segment of the body in the same directions. The segmental COM locations and masses of the feet, legs, thighs, arms, trunk and the head and neck were determined from anthropometric tables [28]. The velocity of the COM at unload was calculated in each trial. Steady-state velocity was considered the velocity of the COM at the end of the second toe off.

EMG data were sampled at 250 Hz after being full-wave rectified and low pass filtered at 100 Hz. After performing a residual analysis, EMG data were low pass filtered at 3 Hz, time normalized to start, toe-off 1, heel contact 1 and toe-off 2 and amplitude normalized to the peak in between start and toe-off 1. EMG data were only analyzed to determine if the TA and MG were active or not.

All dependent measures were analyzed in a two-way ANOVA, and post hoc tested using Tukey's method. The level of significance was chosen at $P < 0.05$. Pearson correlations were calculated between all PD subject characteristics and all dependent variables. Only correlations with an $r > 0.8$ were interpreted.

3. Results

3.1. Young and elderly

In the 10-s quiet stance period prior to initiation young and elderly subjects stood with their COP the same distance ahead of their ankle joint centre. When the COP and COM separate, the young subject's COP moved further backward than the elderly at release. As seen in Fig. 2 the COP starts at the same location under the foot; however, the young subjects allow it to translate back an average of 1.2 cm posterior to the ankle joint centre, whereas the elderly subjects only allow the COP to move back close to the ankle joint. Table 3 summarizes all kinematic and kinetic results. The young subjects took the same amount of time to release, and shift the same amount of body weight as the elderly.

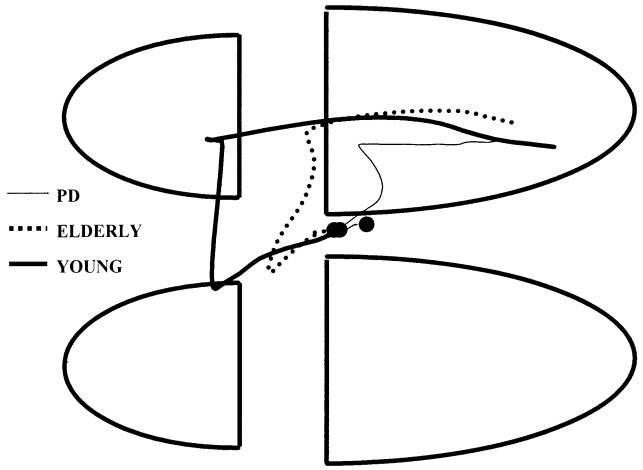

Fig. 2. COP displacement in the A/P and M/L directions in young, elderly and PD subjects.

Young and elderly subjects took approximately the same amount of time to unload; however, the forward impulse from initiation until the first swing foot toe off was significantly larger in the young than in the elderly. The velocity of the total body COM at that point in time was significantly higher in the young than in the elderly. The time to the first heel contact did not differ significantly, but the length of the first step was significantly greater in the young than in the elderly. The time to the second toe off was not different; however, young subjects were significantly faster at the end of the second step than the elderly.

3.2. Elderly and PD

Prior to initiation PD subjects stood with their COP significantly further ahead of their ankle joint than the elderly. The A/P and M/L displacements of the COP at release moved a significantly greater distance in the elderly. The PD subjects took significantly longer to release and at this point the first swing foot had only 56% of total body weight in the PD, significantly less than the elderly (64%).

PD subjects took significantly longer to unload, and the forward impulse that the first swing foot left the ground with was significantly smaller than the elderly. The times to the first heel contact did not differ, but the step length was significantly longer in the elderly compared to the PD. The time to the second toe off was significantly longer in the PD subjects and their velocity at this time was significantly slower.

In summary, the dependent variables measured showed some significant differences during the initiation of gait. All variables in Table 3 were normalized to

Table 3
Dependent variables measured during the initiation of gait

Dependent variable	Young		Elderly		PD
COP distance ahead of ankle (R&L) (cm)	3.57 ± 1.5		3.60 ± 1.4	$P<0.05$	4.51 ± 1.6
Body weight on swing foot at start (% BW)	49 ± 3		49 ± 4		47 ± 6
A/P displacement of COP at release (cm)	4.70 ± 1.5	$P<0.05$	3.54 ± 1.4	$P<0.05$	2.94 ± 1.6
M/L displacement of COP at release (cm)	3.63 ± 0.9		2.91 ± 1.1	$P<0.04$	2.02 ± 0.9
Time to release (s)	0.63 ± 0.12		0.63 ± 0.16	$P<0.002$	0.86 ± 0.28
Body weight on swing foot at release (% BW)	68 ± 5		64 ± 7	$P<0.02$	56 ± 8
Time to unload (s)	0.32 ± 0.06		0.34 ± 0.10	$P<0.001$	0.47 ± 0.14
Velocity of COM at unload (m/s)	0.63 ± 0.14	$P<0.02$	0.50 ± 0.13	$P<0.05$	0.43 ± 0.13
Horizontal impulse/BW (swing limb) (N s/kg)	0.18 ± 0.04	$P<0.05$	0.14 ± 0.06	$P<0.04$	0.07 ± 0.03
Horizontal impulse/BW (stance limb) (N s/kg)	0.19 ± 0.06	$P<0.05$	0.17 ± 0.08	$P<0.02$	0.13 ± 0.06
Time to heel contact 1	1.46 ± 0.20 s		1.52 ± 0.38		1.70 ± 0.42
First step length/height	0.40 ± 0.04	$P<0.03$	0.35 ± 0.04	$P<0.002$	0.25 ± 0.08
First step width/height	0.12 ± 0.01		0.12 ± 0.01		0.13 ± 0.02
Time to toe off 2 (s)	1.60 ± 0.20		1.66 ± 0.38	$P<0.05$	1.82 ± 0.34
Velocity of COM at toe off 2 (m/s)	1.68 ± 0.23	$P<0.02$	1.44 ± 0.17	$P<0.04$	1.14 ± 0.30

velocity at toe-off 2 and can be seen in Table 4. There were no significant differences at the $P < 0.05$ level in any of these normalized dependent variables between the young and elderly or between the elderly and PD.

The mean velocity, over the five trials, of the COM at heel contact of the second step was calculated to indicate steady-state velocity. In the PD subjects the steady-state velocity calculated showed no correlation with age, number of years diagnosed with PD, number of hours off medication prior to testing, or with any of the results on the Hoehn and Yahr rating scale.

The EMG profile of the MG showed cessation of all activity on both the stance and swing limbs in all young, elderly and PD patients prior to *release* in gait initiation. The EMG profile of the TA showed a burst of activity bilaterally after the MG cessation in all young subjects, in eight of 10 elderly subjects bilaterally, and in only three PD subjects on the stance limb and two PD subjects on the swing limb. All TA activations occurred prior to *release*; however, the auditory cue could not be located accurately enough to determine onset and cessation times.

Table 4
Dependent variables normalized to steady-state velocity

Dependent variable	Young	Elderly	PD
A/P displacement of COP at release	2.80 ± 1.9	2.45 ± 1.6	2.56 ± 1.6
M/L displacement of COP at release	2.16 ± 1.7	2.02 ± 1.2	1.82 ± 0.9
Time to release	0.38 ± 0.14	0.43 ± 0.14	0.53 ± 0.25
Body weight on swing foot at release	40 ± 5	44 ± 6	46 ± 7
Time to unload	0.21 ± 0.06	0.24 ± 0.10	0.36 ± 0.14
Velocity of COM at unload	0.38 ± 0.14	0.35 ± 0.15	0.38 ± 0.13
Horizontal impulse/BW (swing limb)	0.11 ± 0.05	0.10 ± 0.06	0.06 ± 0.04
Horizontal impulse/BW (stance limb)	0.11 ± 0.06	0.12 ± 0.05	0.12 ± 0.06
Time to heel contact 1	0.88 ± 0.20	1.02 ± 0.38	1.30 ± 0.42
First step length/height	0.07 ± 0.04	0.08 ± 0.03	0.19 ± 0.08
First step width/height	0.07 ± 0.02	0.08 ± 0.01	0.10 ± 0.02
Time to toe off 2	1.00 ± 0.27	1.14 ± 0.39	1.40 ± 0.34
Velocity of COM at toe off 2	1	1	1

4. Discussion

The PD patients were chosen for the present study because they were reported to have difficulty initiating movement. The patients did 'freeze' in the corridor on the way to the lab, in the doorway entering the lab, crossing over coloured tiles in the floor of the lab, and when turning at the completion of a trial; however, not when initiating gait. What can be said about the PD subjects is that they lean significantly further forward when standing still and that all measures of gait initiation are consistent with a slower gait velocity.

The Parkinsonian posture is well documented and described in general as 'stooped posture.' In detail the neck and head are inclined forward and the trunk is flexed forward [29] and the dorsal spine shows kyphosis [30]. The arms are slightly abducted, the elbows are flexed and the hands are carried in front of the body with the fingers partially flexed [31,32]. The hips and knees are flexed [30], and the ankle dorsiflexion angle decreases as the disability increases which causes the PD subjects to stand more on their toes [30]. This description is consistent with the subjects of the present study, and can explain why the COP is significantly further ahead of the ankle joint center in PD patients.

The forward lean may contribute to gait initiation with a smaller step and at a lower velocity. The slouch forward reduces the vertical height of the COM when

standing still. The energy that causes the initiation of gait comes from the conversion of potential energy, from height above the ground, to kinetic energy, in the velocity of the fall forward of the body, if height is reduced then velocity will be reduced as well. Inactivity of the tibialis anterior prior to *release* in PD subjects may also contribute to gait initiation at a lower velocity.

The gait initiation of young healthy subjects results reported by Brenière et al. [9] show the COP shift backward to covary with the progression velocity at the end of the first step ($r = 0.85$). Cook and Cozzens [3] show the horizontal impulse generated in both the swing limb and the total impulse to increase as the progression velocity increases.

Elble et al. [21] were the first to report that PD patients who had a greater mediolateral and anteroposterior displacement of their COP, and who had a greater shifting of weight between the two limbs took a longer step. Gantchev et al. [22] also showed PD patients to have a decreased acceleration of the COM, a decreased maximum velocity of the estimated COM and a shorter step length than age-matched controls initiating gait. All of these results agree with the findings of the present study.

Cook and Cozzens [3] showed the TA activity to increase as the speed of gait initiation increased in young healthy subjects. Crenna and Frigo [14], in their results of gait initiated from a forward-leaning posture in healthy adults, showed that there is a bilateral decrease in the amplitude of the excitatory TA activity at release.

Crenna et al. [19] found that the 14 PD patients they studied during gait initiation modified their tibialis anterior onset and their soleus inhibition according to the maximum velocity of the COM in the first step. The PD patients, who were considered to have mild to moderate motor impairment, showed three different abnormalities that may have affected the anticipatory postural synergy that promotes the initial forward lead of the body. The tibialis anterior onset and soleus inhibition could have been interacting with abnormal postural muscle activity, the onset and inhibition of the EMG could have been downscaled in time, or the onset and inhibition of the EMG could have been disorganized. Gantchev et al. [22] showed that PD patients had only unilateral activation of the tibialis anterior when initiating gait.

Elble et al. [21] found that all five patients diagnosed with 'lower limb Parkinsonism' showed activation of the tibialis anterior and inactivation of the triceps surae preceding the first complete step. Only three of the 10 patients of the current study showed onset of the TA after cessation in the MG activity, and tend to support the findings of Crenna et al. [19] and Gantchev et al. [22] more than those of Elble et al. [21].

The results of the present study show the elderly to control gait initiation slower but with the same muscle activation patterns and kinematic and kinetic patterns as the young. This statement agrees with Elble et al. [21] who reported that older people initiate gait in the same manner as young adults. Both Nigg and Skleryk [33] and Winter et al. [34], in studies of gait, have reported the elderly to move slower that the young as it was 'safer.'

What has been shown is that the temporal and spatial patterns of gait initiation are preserved in the elderly and in PD. There is a trend for the variables to be smaller, slower and less forceful when comparing the young to the elderly and the elderly to the PD. However, it appears that the 'gain' of the motor patterns in the elderly and PD subjects has been reduced. In PD the 'gain' may reduce to zero in the case of 'freezing.' The location of this tonic control is not known, but because the site of PD neural disruption is in the basal ganglia may suggest this as a possible site. There appear to be none of these trends among the PD subjects as they age, stay off medication, or progress in PD severity.

Acknowledgements

The authors acknowledge the financial support of the Medical Research Council of Canada (Grant MT4343) and the Parkinson's Foundation of Canada and the technical support of Paul Guy.

References

[1] Carlsöö S. The initiation of walking. Acta Anat 1966;65:1–9.
[2] Herman R, Cook T, Cozzens B, Freedman W. Control of postural reactions in man: the initiation of gait. In Stein RB, Pearson KG, Redford JB, editors. Control of Posture and Locomotion. New York: Plenum Press, 1973:363–388.
[3] Cook T, Cozzens B. Human solutions for locomotion: the initiation of gait. In Herman RM, Grillner S, Stein PSG, Stuart DG, editors. Neural Control of Locomotion. New York: Plenum Press, 1976:65–76.
[4] Yamashita T, Katoh R. Moving pattern of point of application of vertical resultant force during level walking. J Biomech 1976;9:93–9.
[5] Mann RA, Hagy JL, White V, Liddell D. The initiation of gait. J Bone Joint Surg 1979;61a:232–9.
[6] Brenière Y, Do MC, Sanchez J. A biomechanical study of the gait initiation process. J Biophys Méd Nucléaire 1981;5:197–205.
[7] Do MC, Brenière Y, Brenguier P. A biomechanical study of balance recovery during the fall forward. J Biomech 1982;15:933–9.
[8] Brenière Y, Do MC. When and how does steady state gait movement induced from upright posture begin? J Biomech 1986;19:1035–40.
[9] Brenière Y, Do MC, Bouisset S. Are dynamic phenomena prior to stepping essential to walking? J Motor Behav 1987;19:62–76.

[10] Brenière Y, Dietrich G, Do MC. Analytical expression of anticipatory movements in gait initiation. In: de Groot G, Hollander P, Huijing PA, Van Ingen Schenau GJ, editors. Biomechanics XI-A. Amsterdam, The Netherlands: Free University Press, 1988:371–376.
[11] Nissan M, Whittle MW. Initiation of gait in normal subjects: a preliminary study. J Biomed Eng 1990;12:165–70.
[12] Brenière Y, Do MC. Control of gait initiation. J Motor Behav 1991;23:235–40.
[13] Brunt D, Lafferty MJ, McKeon A, Goode B, Mulhausen C, Polk P. Invariant characteristics of gait initiation. Am J Phys Med Rehab 1991;70:206–11.
[14] Crenna P, Frigo C. A motor programme for the initiation of forward orientation movements in humans. J Physiol 1991;437:635–53.
[15] Brenière Y, Dietrich G. Heel-off perturbation during gait initiation: biomechanical analysis using triaxial accelerometry and force plate. J Biomech 1992;25:121–7.
[16] Jian Y, Winter DA, Ishac MG, Gilchrist L. Trajectory of the body COG and COP during initiation and termination of gait. Gait Posture 1993;1:9–22.
[17] Gormley JP, Barr DA, Bell AJ, Ravey J, Mollan RAB. Examination of the duration of gait initiation by use of an electrogoniometer. Gait Posture 1993;1:85–91.
[18] Miller CA, Verstraete MC. Determination of the step duration of gait initiation using a mechanical energy analysis. J Biomech 1996;29:1195–9.
[19] Crenna P, Frigo C, Giovannini P, Piccolo I. The initiation of gait in Parkinson's disease. Motor Disturbances 1990;II:161–73.
[20] Elble RJ, Moody C, Leffler K, Sinha R. The initiation of normal walking. In: Harsden CD, editor. Motor Disturbances II. New York: Academic Press, 1990:101-173.
[21] Elble RJ, Cousins R, Leffler K, Hughes L. Gait initiation by patients with lower-half parkinsonism. Brain 1996;119:1705–16.
[22] Gantchev N, Viallet F, Aurenty R, Massion J. Impairment of posturo-kinetic co-ordintion during initiation of forward oriented stepping movements in parkinsonian patients. Electroencephalogr Clin Neurophysiol 1996;101:110–20.
[23] Giladi N, McMahon D, Przedborski S, Flaster E, Guillory S, Kostic V, Fahn S. Motor blocks in Parkinson's disease. Neurology 1992;42:333–9.
[24] Marsden CD. The mysterious motor function of the basal ganglia. The Robert Wartenberg Lecture. Neurology 1982;32:514–39.
[25] Guttman M, Calne DB. In vivo characterization of cerebral dopamine systems in human Parkinsonism. In: Jankovic J, Tolosa E, editors. Parkinson's Disease and Movement Disorders. Baltinire: Urban and Schwarzenberg, 1988:49–58.
[26] Fahn S and members of the UPDRS Development Committee. In: Fahn S, Marsden CD, Goldstein M, Calne DB, editors. Recent Developments in Parkinson's Disease, vol II. New York: MacMillan, 1987:153–163.
[27] Winter DA, Prince F, Stergiou P, Powell C. Medial-lateral and anterior-posterior motor responses associated with centre of pressure changes in quiet standing. Neurosci Res Commun 1993;12:141–8.
[28] Winter DA, Biomechanics and Motor Control of Human Movement, 2nd ed. New York: Wiley, 1990.
[29] Knuttson E. Analysis of Parkinsonian gait. Brain 1972;9:475–86.
[30] Andrews K. Parkinson's disease. In: Rehabilitation of the Older Adult, ch 16. London: Edward Arnold, 1987.
[31] Murray MP, Sepic SB, Gardner GM, Downs WJ. Walking patterns of men with Parkinsonism. Am J Phys Med 1978;7:278–94.
[32] Barbeau A. Parkinson's disease: clinical features and etiopathy. In: Vinken PJ, Bruyn GW, Klawans HL, editors. Handbook of Clinical Neurology, ch 6. Amsterdam: Elsevier, 1986.
[33] Nigg BM, Skleryk BN. Gait characteristics of the elderly. Clin Biomech 1988;3:79–87.
[34] Winter DA, Patla AE, Frank JS, Walt SE. Biomechanical walking pattern changes in the fit and healthy elderly. Phys Ther 1990;70:340–7.