

Review

The biomechanics and motor control of gait in Parkinson disease

Meg E. Morris^{a,*}, Frances Huxham^{a,b}, Jennifer McGinley^{a,b}, Karen Dodd^a,
Robert Ianseck^b^a School of Physiotherapy, La Trobe University, Bundoora 3086, Australia^b Kingston Centre, Warrigal Rd., Cheltenham 3192, Australia

Received 5 April 2001; accepted 5 April 2001

Abstract

Parkinson disease is a progressive neurological condition characterised by hypokinesia (reduced movement), akinesia (absent movement), tremor, rigidity and postural instability. These movement disorders are associated with a slow short-stepped, shuffling gait pattern. Analysis of the biomechanics of gait in response to medication, visual cues, attentional strategies and neurosurgery provides insight into the nature of the motor control deficit in Parkinson disease and the efficacy of current therapeutic interventions. In this article we supplement a critical evaluation of the Parkinson disease gait literature with two case examples. The first case describes the kinematic gait response of an individual with Parkinson disease to visual cues in the “off” phase of the levodopa medication cycle. The second case investigates the biomechanics and motor control of turning during walking in a patient with Parkinson disease compared with elderly and young control subjects. The results are interpreted in light of the need for gait analysis to investigate complex functional walking tasks rather than confining assessment to straight line walking, which has been the trend to date. © 2001 Elsevier Science Ltd. All rights reserved.

Keywords: Parkinson disease; Levodopa; Mobility; Gait; Turning; Locomotion; Physical therapy

1. Introduction

The biomechanics and motor control of gait in people with Parkinson disease (PD) is a topic of growing interest for researchers and clinicians, given the rapid population ageing that is currently occurring throughout the world. Parkinson disease mainly affects older people and leads to difficulty in the performance of skilled motor tasks, such as walking, writing and speaking. Although people with PD can perform simple straight line walking tasks relatively easily, they experience considerable difficulty when walking and turning, performing simultaneous motor or cognitive tasks, crossing obstacles or when they attempt to walk in complex community settings [1–3]. This is because the basal ganglia are dysfunctional in PD and their role in the motor control of skilled voluntary movements is compromised [4]. When imbalance in neurotransmitters such as levodopa, GABA and acetylcholine occurs in the basal ganglia, patients find that sequential [5] and complex [6] motor skills are difficult to perform and

movements become excessively slow and underscaled in size. Balance disturbance is also common in the advanced stages of the disease [7] and further compromises the ability to walk independently and safely.

In this article we discuss the ways in which the biomechanics and motor control of gait are affected by hypokinesia, which is the most common movement disorder in PD. The term hypokinesia refers to reduced movement speed and size. Akinesia (absent movement) and dyskinesia (involuntary choreiform movements) are less common causes of gait disturbance. For information on akinetic motor blocks and freezing, readers are referred to Giladi et al. [8], Burleigh-Jacobs et al. [9] and Vidailhet et al. [10]. For discussion of the clinical manifestations of gait dyskinesia in PD, the articles by Morris [11] and Hagell and Widner [12] are recommended. Information on how gait disturbance can occur secondary to reduced fitness and aerobic capacity is found in Canning et al. [13].

2. Straight line walking in PD

Although PD affects the control of complex motor skills, the vast majority of experiments on gait distur-

* Corresponding author.

E-mail address: m.morris@latrobe.edu.au (M.E. Morris).

bance in this condition have investigated straight line walking tasks, examining the spatiotemporal, kinematic or kinetic parameters of gait. Most of these experiments have been conducted in gait laboratory or hospital ward settings. They can be grouped into: (i) those which have evaluated gait characteristics in the “off” phase of medication, when PD medication has either been withdrawn or is at very low levels; (ii) those evaluating gait in the “on” phase and at peak-dose within the medication cycle, when medication is most effective; (iii) studies quantifying the effects of neurosurgery. The research can be further divided into gait analyses with or without the use of external cues or attentional strategies.

External cues are visual, auditory or somatosensory stimuli that enable the person with PD to bypass the defective basal ganglia [11]. For hypokinesia, visual cues are most frequently used, in the form of white strips of cardboard placed on the floor at the appropriate step length for the person’s age, sex and height [3]. Attentional strategies refer to cognitive processes used by the person to compensate for their movement disorders, such as thinking about walking with large steps [3], visualisation or mental rehearsal. It can be hypothesised that external cues and attentional strategies enable the pre-motor cortex (PMC) and supplementary motor area (SMA) to better compensate for defective motor control mechanisms in the basal ganglia, therefore enabling the person to move more easily. Cerebral blood flow studies show that activity in the PMC and SMA is elevated in people with PD when they perform sequential motor skills, and is increased when external cues or attentional strategies are available to drive motor performance [14].

2.1. Straight line walking OFF medication, at end of dose or in de novo patients

Only a handful of studies have measured walking in people with PD after the withdrawal of medication, at the end of dose or in those who have never taken medication (*de novo*) (refer to Table 1 for a summary of the main articles). Although these studies provide insight into the specific effects of PD on gait whilst controlling for the effects of medication, it is acknowledged that it can take up to 3–4 weeks for the effects of levodopa to completely subside after it is withdrawn [15]. In addition, some investigations were performed on patients who had received deep brain stimulation (e.g., [16]) or other types of neurosurgery (e.g., [17]). The earliest gait analyses in patients not documented as being on PD medication were by Martin [18] and Murray [19]. Purdon Martin [18] reported in detail his clinical observations of the characteristics of parkinsonian gait, noting the hallmark features of reduced or absent arm swing, reduced trunk rotation, a forward stooped posture, reduced amplitude of motion at the hips, knees and ankles, slowness, reduced footstep size and decreased

ground clearance. Murray [19] subsequently confirmed these observations using interrupted light photography. She noted in particular that transverse plane motion of the pelvis and thorax was rigidly coupled in people with PD, compared to the out-of-phase motion of the pelvis and thorax observed in control subjects. In addition, there was excessive flexion at the hips and knees throughout the gait cycle and the total excursions of movement for the lower limb joints were reduced.

The vast majority of the investigations in this group have focussed on the spatiotemporal (time and distance) parameters of the footstep pattern, showing that people with PD walk more slowly than usual, typically in the range 40–60 m/min [20,21] rather than 75–90 m/min, which is more typical for age-matched control subjects [22,23]. As shown by O’Sullivan et al. [21] cadence values are within normal range at around 100–110 steps/min, whereas stride length is much shorter than normal. Stride length values range from 1.2 to 1.5 m for healthy older people [22,23] compared to only 0.4–0.9 m for people with PD after withdrawal of medication [17] or 0.8–1.0 m for those at the end-of-dose [20]. The percentage of the gait cycle spent in double limb support is also markedly increased when levodopa levels are low, increasing from the usual range of 20–30% gait cycle to over 35% gait cycle [20]. As a consequence, the person shuffles, not only with slow short steps, but also with reduced ground clearance, which increases the risk of tripping.

Only one study has reported the kinematics and kinetics of gait after withdrawal of medication in patients who have not undergone neurosurgery [24]. In a single case study of a 71 year old woman with a 20 year history of PD, we found a marked reduction in the movement excursion (amplitude) across the hip, knee and ankle joints. These kinematic movement profiles were shifted upwards towards flexion, with limited hip extension range and reduced ankle plantar-flexion range. The associated kinetic profiles were abnormal, revealing an elevated and prolonged hip extensor moment in the early stance phase of gait, and markedly decreased ankle power generation at push-off. This study did not examine the effects of visual cues or attentional strategies on PD gait after withdrawal of medication. A case example will therefore be presented to demonstrate typical changes in “off” phase gait kinematics in response to visual cues.

Mr C was a 51 year old man diagnosed with PD 9 years previously. He was 172 cm tall and weighed 63 kg and his most troublesome movement disorders were gait hypokinesia and motor fluctuations. Gait data were obtained using a three-dimensional motion capture system (Vicon, Oxford Metrics, UK) and biomechanical software (Vicon Clinical Manager) according to a previously described protocol [24]. Walking was firstly measured without levodopa medication, (UPDRS motor examination score of 17) [25] and then as Mr C

Table 1
Studies on straight line walking after withdrawal of medication, at the end of dose or in de novo patients

Authors	Subjects	Medication status	Main findings
Murray et al. [19]	44 men with Parkinsonism	Not stated; possibly de novo	Reduced arm swing and trunk rotation, forward stooped posture, slow, shuffling gait.
Koozekanani et al. [48]	Two male subjects with PD	De novo	Decreased ground reaction forces and sagittal plane kinematics at the knee.
Blin et al. [34]	21 PD patients: 12 females	12 h withdrawal of medication	Decreased stride length and speed; cadence within normal range.
Bowes et al. [32]	14 PD patients: gender not specified	Withdrawal of medication for 12, 14, 16, 18 h	Stride length and speed decreased; double support phase duration within normal values.
Blin et al. [33]	20 PD patients: 11 females	Withdrawal of medication for 12 h	Walking speed and stride length decreased; rhythmicity remained intact; increased variability in all parameters in the “off” phase.
Pedersen et al. [76]	12 mildly affected PD patients; five females	24 h withdrawal of medication	Maximum gait speed, stride length at maximal speed and stride length at constant speed were significantly decreased without medication; stride frequency did not change.
Defebvre et al. [16]	7 PD patients with long term monopolar stimulation of the ventral intermediate thalamic nucleus; gender not stated	12 h withdrawal of medication before deep brain stimulation	Stride and step times and double limb support times were longer in the “off” condition than the “on” condition.
Meyer, [29]	26 PD patients; gender not specified	Withdrawal of medication for 12 h, prior to surgery and tested again after surgery in “on” and “off” states	In “off” state prior to surgery nine patients could not walk and walking speed was reduced for remainder; in many cases pallidotomy restored independent mobility and speed increased.
Cioni et al. [77]	15 PD patients; two females	Most patients refrained from taking their morning medication	When “off” qualitative disturbances in muscle activation patterns seen on EMG.
McIntosh et al. [45]	10 PD patients: four females	Withdrawal of medication for 24 h	Compared with the control group or the “on” experimental group, PD subjects showed decreased walking speed and stride length and moderate stride asymmetry. Cadence was also decreased.
O’Sullivan et al. [21]	15 PD patients: five females; 14 of these had levodopa-induced dyskinesias	Overnight withdrawal of PD medication	Reduced velocity and stride length; cadence within normal range.
McKay-Lyons, [44]	5 PD patients: two females	Measured at 10% intervals throughout the levodopa cycle	Unpredictable variability in spatiotemporal parameters of gait throughout the levodopa cycle.
Van Emmerik et al. [69]	27 PD patients: 10 female	De novo	Marked axial (trunk) rigidity in PD patients.
Azulay et al. [78]	16 PD patients: seven females	Withdrawal of medication for 12 h	Walking speed and stride length reduced; cadence increased.
Hanakawa et al. [58]	10 PD patients: four females	Withdrawal of medication for 12 h	Reduced speed, stride length and cadence, which increased towards normal values with the introduction of visual cues. NB: this was a treadmill study.
Ebersbach et al. [50]	30 PD patients: 11 females	12 subjects de novo 18 “on” medication	Increased step-to-step variability of all gait parameters and reduced stride length and speed.
Morris et al. [20]	1 elderly female with PD	Withdrawal of medication for 12 h	Reduced speed, stride length, cadence, angular displacement; reduced plantar-flexor power.
Siegel et al. [17]	11 PD patients: three females	Withdrawal of medication for 8 h, measured before and after thalamic stimulation	Decreased walking speed, cadence, stride length, despite thalamic stimulation, for both “on” and “off” conditions.

walked with visual cues. The cues comprised white strips of cardboard (50 × 600 mm) placed over the 8 m walkway at a step-size distance obtained from an age-matched healthy control subject. Fig. 1 illustrates the

changes in lower limb kinematics in typical single trials for each condition. In the “off” medication condition, gait was a little slow for his age (70.1 m/min), with short stride length (1.0 m) and a rapid stepping rate (139 steps/

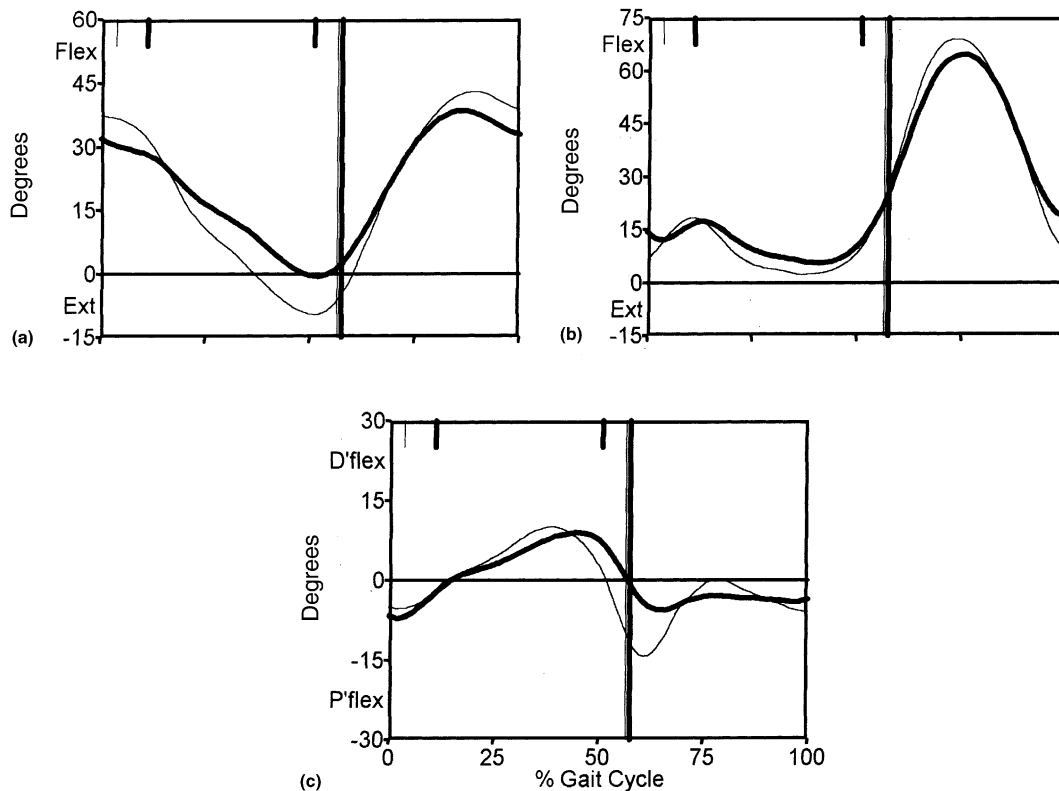


Fig. 1. Sagittal plane kinematic profiles for the hip, knee and ankle in the ‘off’ medication condition (thick line) and the ‘off medication with visual cues’ condition (thin line): (a) hip flexion/extension, (b) knee flexion/extension, (c) ankle dorsiflexion/plantarflexion.

min). Hip extension range was mildly reduced (around 2° of extension), as was ankle range of movement (only 15°). With the introduction of visual cues, both gait velocity and stride length increased markedly, to 83 m/min and 1.47 m, respectively, with an associated reduction in cadence to 113 steps/min. Hip extension range increased to around 10 degrees of extension, and ankle range of movement normalised at around 24° . Interestingly, the flexion range of the hip and knee further increased and remained relatively large compared to age-matched controls, suggesting that the patient used a ‘‘high stepping’’ gait, possibly to ensure clearance of the cues. These findings indicate that, even when levodopa levels are low, some patients with PD can use visual cues to generate more normal step length and walking speed.

2.2. Studies on PD gait following deep brain stimulation, thalamotomy or pallidotomy

Research is accumulating analysing gait in patients with PD who have received deep brain stimulation, thalamotomy or pallidotomy, with divergent results. Whilst some investigations have shown increases in walking speed after neurosurgery (e.g., [26–29]), others show that neurosurgery has little effect on hypokinesia, even though it may be effective for reducing dyskinesia,

dystonia and tremor in a proportion of patients with severe motor fluctuations (e.g., [16]). Before the beneficial effects of levodopa medication on movement disorders had been reported [30] and adopted routinely, neurosurgery was one of the few options available for the management of severe motor fluctuations. For example, Knutsson’s 1972 [31] landmark paper on parkinsonian gait included analyses of five patients who had undergone unilateral thalamotomy and one bilateral thalamotomy amongst the sample of 21 patients with idiopathic and postencephalitic parkinsonism. Unfortunately the data were pooled, hence the specific characteristics of gait following thalamotomy could not be separated from the general finding of a slow, shuffling footstep pattern with poverty of movement in the trunk, reduced arm swing and underscaling of angular displacement at the hips, knees and ankles. Recently Defebvre et al. [16] measured the effects of ventral intermediate thalamic nucleus stimulation on gait kinematics in PD using three-dimensional motion analysis (Table 1). They found that stimulation did not affect the spatiotemporal parameters of the footstep pattern and that lower limb joint positions remained the same from the pre-test to the stimulation condition, despite a reduction in large amplitude tremor. In contrast, Siegel and Metman [17] conducted a three dimensional motion analysis study before and after unilateral posteroventral

pallidotomy, finding that it produced a twofold increase in walking speed and stride length. Almost all the improvement (96%) in stride length could be accounted for by changes in foot-floor angle, and knee and hip angular excursion. Further studies are needed to clarify the effects of specific surgical interventions, taking into account the status of PD medication, disease duration and methods of analysing gait disturbance.

2.3. Straight line walking in the ON phase of the PD medication cycle

There is now considerable information on the characteristics of PD gait in the “on” phase of the PD medication cycle (refer to [32–58] for the most relevant articles containing data on PD gait for steady-state locomotion in the “on” phase of medication). The most common theme to emerge from this literature is that the *speed* of walking is reduced in the majority of people with PD, despite the best attempts to control movement using levodopa medication and other forms of pharmacotherapy [32–58]. For example, Bowes et al. [32] conducted a randomised placebo controlled study of the effects of levodopa carbidopa in people with moderately severe PD. Objective measures of the spatio-temporal parameters of gait obtained from a gait assessment trolley showed that walking speed was on average 23.9 m/min after 12 h withdrawal of medication and increased by 9.6 m/min for the placebo medication condition and only 27 m/min for the levodopa condition to only around two-thirds of normal value. The low walking speeds were the result of reduced *stride length*, which was on average just over two-thirds of that of age-matched control subjects whereas cadence values were within normal range and did not change markedly in response to medication. These findings echoed the results of Blin et al. [33,34] and our own research [35–37] demonstrating that peak speed and stride length in patients with hypokinesia were dopa-sensitive whereas temporal variables such as cadence and swing and stance duration were dopa-resistant. Moreover Forssberg and colleagues [38] noted that in addition to gait changes associated with slowness, timing disorders were not responsive to medication. Rhythmicity of walking may well be controlled by brainstem, cerebellar and spinal regions with overriding control by the frontal motor cortices [59], rather than the basal ganglia-SMA-PMC-cortex motor control loop, which may play a greater role in the scaling of movement size. Supporting this hypothesis, our research team has shown a disorder in the scaling of step length in hypokinetics, despite normal ability to modulate footstep timing [36,37], for both the “on” and “off” phases of the levodopa cycle [35]. Although some studies have documented reduced cadence values for people with PD [45,46] these appeared to include

patients with akinesia in addition to hypokinesia. It would seem likely that the pathogenesis of gait hypokinesia, akinesia and dyskinesia are different, requiring careful control when studying motor performance in people with PD.

Another emerging theme in the literature on straight line walking in the “on” phase of medication is that *force regulation* is abnormal in parkinsonian hypokinesia. Using a pedodynograph system, Nieuwboer et al. [47] showed that, after correcting for speed of walking, patients with PD walked with considerably lower relative peak forces at the forefoot and heel and increased load at the mid-foot compared with control subjects. This means that patients were walking with a flat-footed gait pattern with reduced roll-off. This type of gait pattern would be predicted to increase the risk of trips and may require greater energy expenditure than normal. In addition, a force platform analysis of PD gait by Koozekanani et al. [48] showed underscaling in the vertical and frontal ground reaction forces in patients with PD, with the push-off peaks being considerably underscaled. In agreement with these findings, evidence is now accumulating showing that ankle plantar-flexor power generation at push-off is underscaled [24], and that hip flexor “pull off” at early stance is increased [24], possibly to compensate for the reduced force generation at push-off. Electromyographical (EMG) studies confirm that after gait speed is controlled for, the amplitude of gastrocnemius EMG is reduced in PD, even though tibialis anterior activity remains within normal range [49]. Ebersbach et al. [50] speculated that the resultant decreased stride length and force gain in PD hypokinesia may help to minimise energy expenditure in people with this disabling disease.

Excessive variability in gait parameters in the “on” phase of the PD medication cycle is another characteristic of gait in PD hypokinesia. In one study comparing walking patterns in people with PD, cerebellar ataxia and subcortical arteriosclerotic encephalopathy, Ebersbach et al. [50] found that step-to-step variability was similar for medicated patients with PD and control subjects and was much greater for ataxics and those with encephalopathy. Similarly our results [24,51] and those of Schenkman et al. [52], MacKay-Lyons [53] and Hausdorff et al. [54] showed that gait performance was relatively consistent from one trial to the next when patients were tested at peak dose of the medication cycle. However, there was marked variability from one trial to the next when trials were performed after withdrawal of PD medication. There was also marked variability when end of dose performance was compared with peak dose performance. Thus, although patients with PD show fluctuations in their walking performance, this appears to be due to the variable effects of medication rather than an inherent feature of hypokinesia.

2.4. Straight line walking ON with visual cues and attentional strategies

One of the interesting findings of recent research is that most patients with PD retain the ability to overcome their gait disorders using external cues and attentional strategies. People with PD hypokinesia can increase the size and speed of their movements by using frontal cortical areas of the brain (and perhaps the cerebellum) to bypass the defective basal ganglia. For example, Behrman et al. [55] showed that patients with PD were responsive to verbal instructions that specified movement size or speed. Simply asking a patient with hypokinesia to walk with long strides can lead to an immediate increase in walking speed which can last for up to 2 h [3]. Many other researchers have demonstrated that people with PD can use visual [35–37,60,61] and auditory [45,46,56] cues to enhance motor performance. One of the more notable of these studies was conducted by Hanakawa and associates [57,58]. They explored the physiological mechanisms underlying response to visual cues by measuring regional cerebral blood flow during gait on a treadmill whilst patients stepped over white lines placed either transverse to the direction of walking or parallel to the direction of walking. Whereas the parallel lines had little effect, the transverse lines led to a marked improvement in the temporal and spatial parameters of gait, and a reduction in cadence, which was unusually high in their sample. This was associated with increased activation in the posterior parietal cortex and cerebellar hemisphere and particularly in the right lateral premotor cortex, suggesting that patients might have compensated for basal ganglia dysfunction by utilising non-affected areas of the brain to activate movement. Thus the ability to generate normal patterns of movement is not lost in people with PD, rather there is an activation problem. Physical therapists and other health professionals play a major role in teaching patients with PD how to bypass defective basal ganglia circuitry in order to move more quickly and easily (refer to [11] for a review).

3. Walking and turning

Even though the vast majority of PD gait research is on straight line walking, the ability to turn during walking is an integral part of functional locomotion. At least two turns every 10 steps are used to perform common daily activities such as going to the bathroom or making a cup of tea [62]. Turning during walking is a potent source of akinetic blocks, triggering freezing episodes in almost half of akinetics [8]. Importantly, turning is associated with increased fall risk in people with PD, with the reported incidence of falling in the disease ranging from 38% [63] to 62% [64] annually. In

people with PD falls carry a higher risk of serious injury than corresponding falls in the elderly, presumably because protective postural responses are impaired by the disease [7].

Despite the importance of turning during walking, these “online” turns have only recently been studied in normal young adults [65–67]. We therefore commenced an investigation into the spatio-temporal and kinematic parameters of gait for online turns in people with PD compared with elderly and young adult controls. Subjects were evaluated turning whilst walking at preferred speed, using turn magnitudes in the range commonly used during household activities (refer to [62]). The main aim was to gain information on the changes occurring in the frontal (y) plane as subjects walked along the laboratory then turned towards positive y (Fig. 2).

Light reflective VICON markers were attached to each subject to delineate the right forehead (RFHD), right shoulder (RSHO), right anterior superior iliac spine (RASI) and the toes and heels of both feet for a PD subject, an elderly age-matched control subject and a young adult of similar height and weight. Table 2 provides a summary of the subject characteristics. The PD subject had a modified Hoehn and Yahr disease severity rating of III, signifying bilateral disease with postural involvement yet independent ambulation. He was assessed whilst “on” medication at which time his UPDRS [25] motor examination score was 14. For each of the gait trials, subjects were instructed to walk at a comfortable speed along the walkway, turning at a retro-reflective corner on the force platform to one of five coloured targets (Fig. 2). The targets were placed to evoke 30°, 60°, 90° and 120° turns. Six turns of each magnitude were performed, counterbalanced to avoid series effects. After visual examination of the data, one representative trial was selected from each subject for each turn. The number of frames corresponding to two steps in and two steps out of the corner was clipped and used for further analysis. These will be referred to as the first and second “approach” steps and the first and second “turn” steps (T1 and T2, Fig. 2). Because online turning is not a steady-state activity, it was not possible to normalise data to the percentage of the gait cycle.

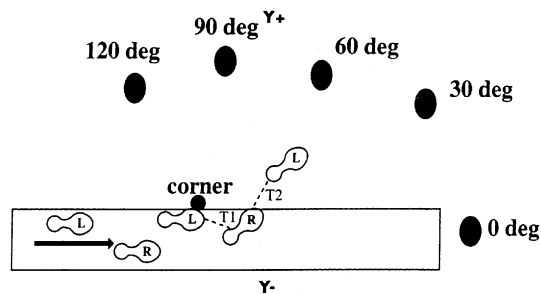


Fig. 2. Experimental set-up showing laboratory coordinates.

Table 2
Subject characteristics for turning study

	Young control	Elderly control	Parkinson disease
Age	22.5 yr	76.1 yr	77.5 yr
Height	166.5 cm	166 cm	169 cm
Weight	66.4 kg	74.0 kg	60.8 kg
Velocity	1.30 m/s	1.16 m/s	0.99 m/s
Stride length	1.25 m	1.21 m	1.06 m
Cadence	112 steps/min	115 steps/min	125 steps/min
Stride width	73.31 mm	98.88 mm	137.97 mm

Table 3
Number of steps taken to turn (in 1500 mm square – averaged over six trials)

Turn (°)	Young control (mean (SD))	Elderly control (mean (SD))	Parkinson disease (mean (SD))
0	1.76 (0.86)	2.35 (0.01)	2.95 (0.24)
30	2.37 (0.12)	2.51 (0.24)	3.25 (0.25)
60	2.54 (0.22)	2.89 (0.20)	4.36 (0.73)
90	3.15 (0.22)	4.03 (0.16)	5.18 (0.61)
120	4.07 (0.23)	4.68 (0.31)	6.75 (0.50)

It can be seen that the PD subject required more steps for each turn, and his step number increased more steeply as turn magnitude increased, suggesting greater difficulty with larger turns (Table 3).

The spatiotemporal parameters of gait showed the predicted decrement in stride length with age and disease (Table 2). Stride width was derived from the mean inter-heel distances in the *y* (frontal plane) over the straight trials and found to increase with age (young control 73.31 mm; elderly control 98.88 mm) and again with PD (137.97 mm). This conformed well with a large study of elderly and PD subjects in which stride width was noted to increase early in PD, presumably to compensate for altered posture and balance, later narrowing in advanced disease [68]. At Hoehn and Yahr stage III, our PD subject carried a moderate disease burden, with markedly increased stride width that had not yet narrowed with severe disease.

Examination of marker *y* trajectories showed clear differences between subjects. In the young and control subjects, T1 toe trajectories increased from 30° to 60° turns, reducing for the more destabilising 90° and still further for the 120° turns, in both of which an extra step was added for stability (Fig. 3). The PD turns showed reduced direction change with maximum achieved in the 90° turn. The different subjects also showed different responses to the potential destabilisation inherent in the 90° and 120° turns. The young subject used a remarkably consistent approach path for the five trial types (0°, 30°, 60°, 90° and 120° turns) and was the only one to pivot on the turn (circled), although even he included an extra step around the two largest turns. In contrast, the elderly control deviated in his

approach path to take the corner wide (particularly obvious in the 120° trial), but then made large and effective direction changes after the corner. The PD subject also used a variable approach path. However, closer inspection revealed that this variability did not serve a safety purpose as the 90 and 120 paths resulted in a sharper corner rather than a wider one. All PD direction changes after the corner were underscaled and comparatively stereotypical. The subject with PD did not demonstrate the pattern of increasing size seen in the others. Although this probably represents the characteristic underscaling of hypokinesia, it may also act to improve stability around the corner at the cost of decreased efficiency in turning.

The results further suggested that the 60° turn might actually present the greatest challenge to stability because it was accomplished without the additional step/s used for larger turns. It would therefore require more control of trunk momentum during a large swing phase of T2 (Fig. 4). In the typical trials chosen for analysis, it can be seen that both the young and elderly subjects had a much bigger distance between T1 and T2 (long arrows). This represented a wider-based double support during the critical turn phase, and can be seen to be considerably reduced in the PD subject. A similar reduction in lateral T1–T2 distance for all turns in the PD subject suggested that, despite his increased stride width during straight walking, his gait narrowed during turning. Furthermore, the T1 foot stepped outside the forehead, shoulder and pelvis markers in young and elderly subjects (small arrows), but not in the PD, suggesting that the centre of mass of this person was likely to be closer to his limits of stability.

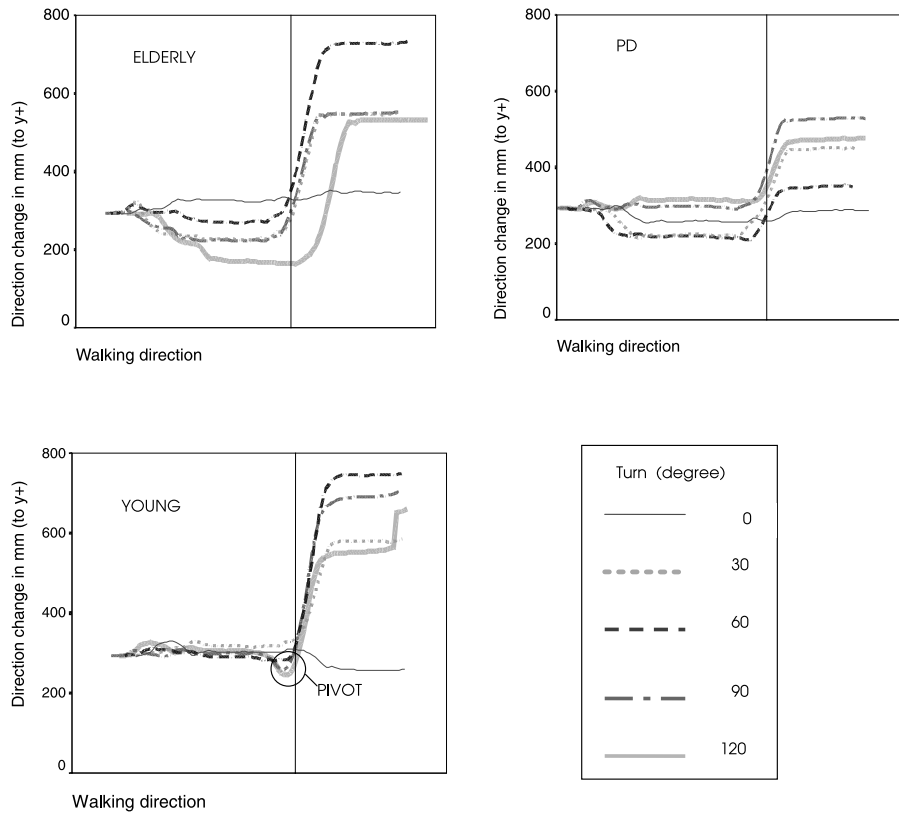


Fig. 3. T1 toe trajectories. Thin vertical line represents corner position. Walking direction signifies walking forwards. Note that elderly 30° and 60° trajectories overlap.

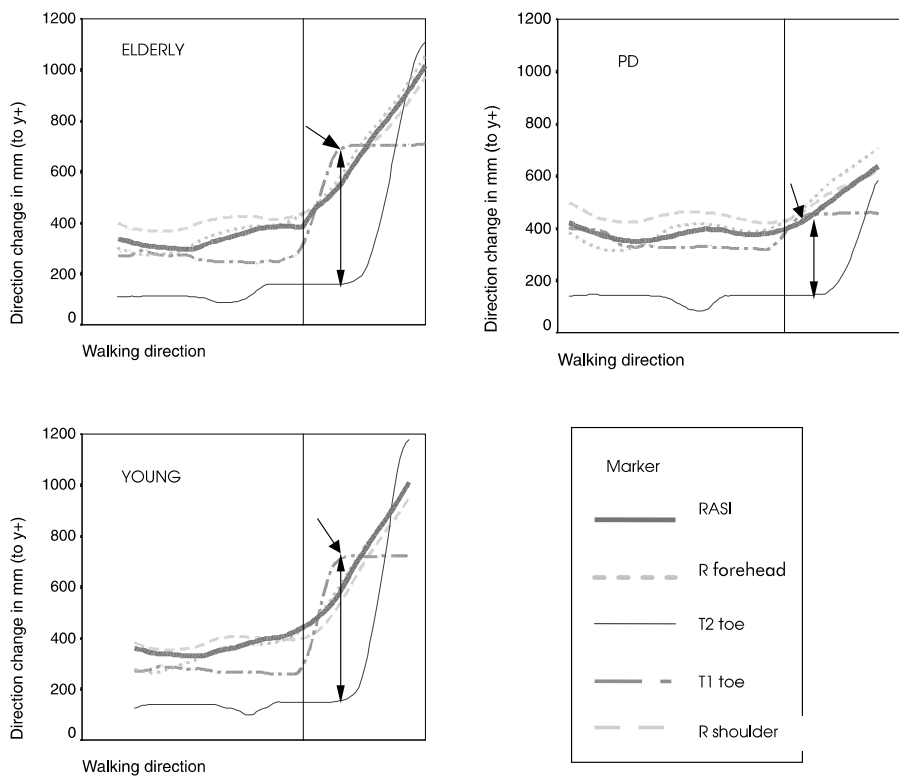


Fig. 4. Sixty degree trajectories. Thin vertical line represents corner position. Walking direction signifies walking forwards. Long vertical arrows highlight differences in width of double support. Short arrows highlight differences in relationship between T1 and upper body.

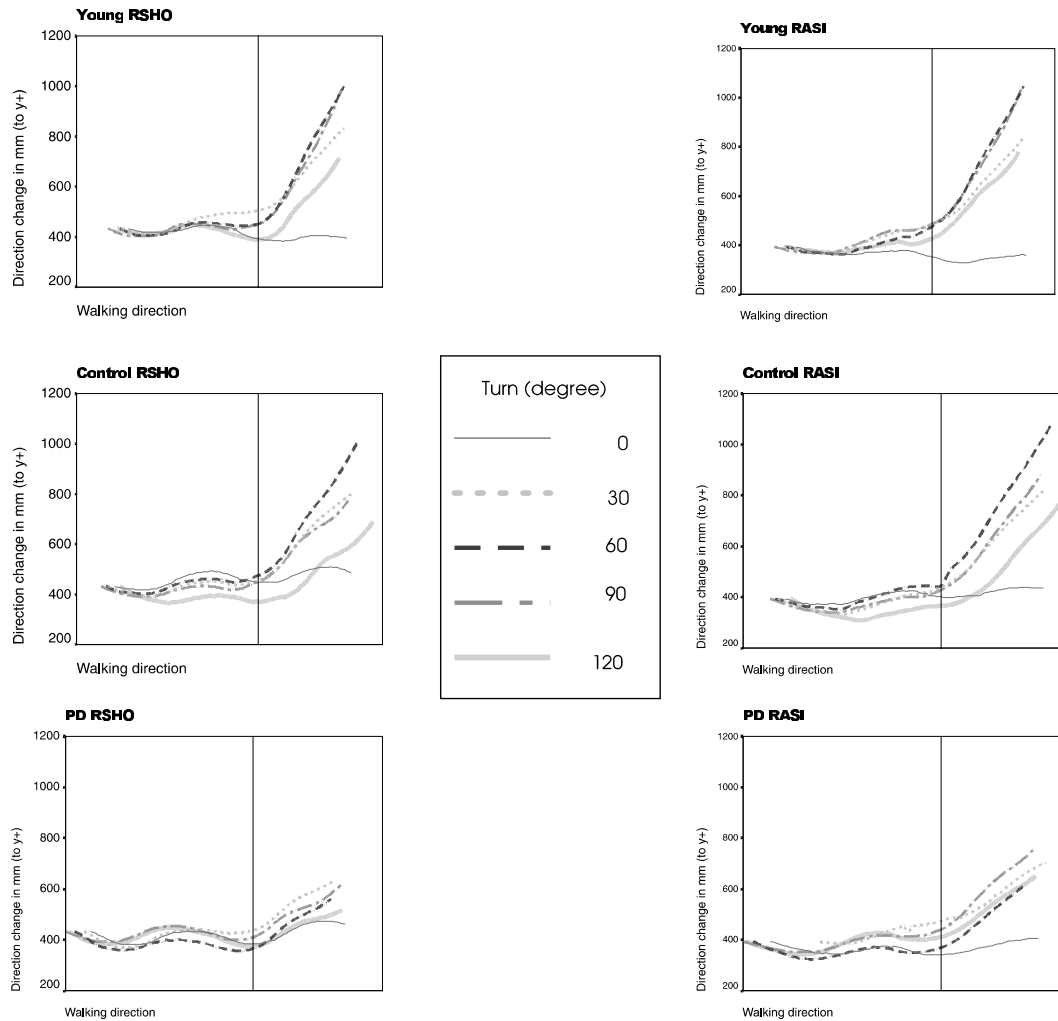


Fig. 5. Upper and lower trunk relationships. RSHO denotes R shoulder. RASI denotes right anterior superior iliac spine. Thin vertical line represents corner position. Walking direction signifies walking forwards.

Differences were also seen in the relative relationship between the upper trunk, as represented by the right shoulder marker (RSHO), and the lower trunk as defined by the right anterior superior iliac spine marker (RASI). As with the toe trajectories, the PD RASI trajectories were underscaled relative to the two unimpaired subjects, but this underscaling was much more marked in the PD RSHO trajectories (Fig. 5). This may represent the result of trunk rigidity, with the trunk tending to move en bloc, although that would seem more likely to lead to shoulder trajectories mirroring pelvic ones. Abnormalities in the harmonic relationship of upper and lower trunks develop early in PD [69] and are believed to impact on the ability to decrease forward momentum during walking in a straight line [70]. Such changes might be particularly relevant in turning, where arrest and re-direction of forward trunk momentum is vital to success. Decreased ability to control trunk momentum has been noted in older unimpaired subjects [71,72]. The relative down-

sizing of shoulder trajectories may therefore represent an attempt to constrain movement for increased stability. Controlled clinical trials utilising large groups of subjects are required to verify the generalisability of these findings.

4. Community ambulation in people with PD

As well as studying online turns during walking, there is a need for future research to investigate gait disorders and disability during community ambulation tasks. In view of the prevalence and diversity of gait disorders in people with PD, it is not surprising that many patients report that community mobility is markedly impaired. Characteristic features of PD include difficulty attaining and then sustaining adequate walking speed [3,50,51] and reduced gait endurance [50]. Moreover balance and postural control are usually impaired [7,73], increasing the risk of slips, trips and falls. It can be further

suggested that maintaining balance over varied surfaces, negotiating obstacles, and successfully monitoring changes in the surrounding environment are particularly demanding for these individuals, although this has never been studied. The attentional demand of monitoring open environments such as crowded shopping centres, stations or roadways is also likely to be troublesome for people with advanced PD, given that cognitive impairment is common in the latter stages of this condition. People with PD also experience difficulty carrying objects such as a tray with glasses [1] or talking fluently [2] whilst ambulating. In such tasks, diverting attention from the primary task of maintaining stable gait performance may compromise safety and increase the risk of falling. Community ambulation also requires the ability to turn at frequent intervals, which, as shown by preliminary results discussed above and the reports by Stack et al. [64] and Van Emmerik et al. [69], can be markedly compromised in people with this disabling neurological condition. In future studies it might be beneficial to incorporate more global measures of gait disability as provided by validated disability rating scales (e.g., [74,75]) in order to achieve a greater depth of understanding of the functional consequences of movement disorders on gait performance.

5. Conclusion

The ability to walk quickly and easily in a range of different environments is a highly developed motor skill that takes many years to master. Due to neurotransmitter imbalances in the brain, people with PD progressively lose flexibility and adaptability in their locomotor responses and walk with a stereotyped short-stepped, narrow based, shuffling gait for a range of different tasks. They also experience difficulty in modulating gait parameters according to changing task demands, despite the best attempts at pharmacotherapy or surgery. This limits their ability to ambulate in the home and community and to participate in work, social and leisure activities. Future PD research needs to shift from the current narrow focus on laboratory-based studies of straight line walking to gait analyses in the home and community, where more complex locomotor activities are severely compromised.

References

- [1] Bond JM, Morris M. Goal-directed secondary motor tasks: their effects on gait in subjects with Parkinson disease. *Arch Phys Med Rehabil* 2000;81:110–6.
- [2] Camicioli R, Oken BS, Sexton G, Kaye JA, Nutt JG. Verbal fluency task affects gait in Parkinson's disease with motor freezing. *J Geriatric Psychiatry Neurol* 1998;11:181–5.

- [3] Morris ME, Ianseck R, Matyas TA, Summers JJ. Stride length regulation in Parkinson's disease: normalisation strategies and underlying mechanisms. *Brain* 1996;119:551–68.
- [4] Marsden CD, Parkes JD. "On-off effects" in patients with Parkinson's disease on chronic levodopa therapy. *Lancet* 1976;1:292–6.
- [5] Benecke R, Rothwell JC, Dick JPR, Day BL, Marsden CD. Disturbance of sequential movements in patients with Parkinson's disease. *Brain* 1987;110:361–79.
- [6] Berardelli A, Dick JPR, Rothwell JC, Day BL, Marsden CD. Scaling of the size of the first agonist EMG burst during rapid wrist movements in patients with Parkinson's disease. *Brain* 1986;49:1273–9.
- [7] Bloem BR, Beckley DJ, Van Dijk JG, Zwiderman AH, Remler MP, Roos RAC. Influence of dopaminergic medication on automatic postural responses and balance impairment in Parkinson's disease. *Movement Disord* 1996;11:509–21.
- [8] 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.
- [9] Burleigh-Jacobs A, Horak FB, Nutt JG, Obeso JA. Step initiation in Parkinson's disease: influence of levodopa and external sensory triggers. *Movement Disord* 1997;12:206–15.
- [10] Vidailhet M, Stocchi F, Rothwell JC, Thompson PD, Day BL, Brooks DJ, Marsden CD. The Bereitschaftspotential preceding simple foot movement and initiation of gait in Parkinson's disease. *Neurology* 1993;43:1784–8.
- [11] Morris ME. Movement disorders in people with Parkinson disease: a model for physical therapy. *Phys Therapy* 2000;80:578–97.
- [12] Hagell P, Widner H. Clinical rating of dyskinesias in Parkinson's disease: use and reliability of a new rating scale. *Movement Disord* 1999;14:448–55.
- [13] Canning CG, Alison JA, Allen NE, Groeller H. Parkinson's disease: an investigation of exercise capacity, respiratory function, and gait. *Arch Phys Med Rehabil* 1997;78:199–207.
- [14] Jueptner M, Weiller C. A review of differences between basal ganglia and cerebellar control of movements as revealed by functional imaging studies. *Brain* 1998;121:1437–49.
- [15] Kempster PA, Frankel JP, Bovingdon M, Webster R, Lees AJ, Stern GM. Levodopa peripheral pharmacokinetics and duration of motor response in Parkinson's disease. *J Neurol, Neurosurg Psychiatry* 1989;52:718–23.
- [16] Defebvre L, Blatt J, Blond S, Bourriez J, Guieu J, Destee A. Effect of thalamic stimulation on gait in Parkinson disease. *Arch Neurol* 1996;53:898–903.
- [17] Siegel KL, Metman LV. Effects of bilateral posteroventral pallidotomy on gait of subjects with Parkinson disease. *Arch Neurol* 2000;57:198–204.
- [18] Martin P. The basal ganglia and posture. London: Pitman Medical; 1967.
- [19] Murray MP, Sepic SB, Gardner GM, Downes WJ. Walking patterns of men with Parkinsonism. *Amer J Phys Med* 1978;278–94.
- [20] Morris ME, Matyas TA, Ianseck R, Summers JJ. Temporal stability of gait in Parkinson's disease. *Phys Therapy* 1996;76:763–89.
- [21] O'Sullivan JD, Said CM, Dillon LC, Hoffman M, Hughes AJ. Gait analysis in patients with Parkinson's disease and motor fluctuations: influence of levodopa and comparison with other measures of motor function. *Movement Disord* 1998;13:900–6.
- [22] Kerrigan DC, Todd MK, Croce UD, Lipsitz LA, Collins JJ. Biomechanical gait alterations independent of speed in the healthy elderly: evidence for specific limiting impairments. *Arch Phys Med Rehabil* 1998;79:317–22.
- [23] Ostrosky KM, VanSwearingen JM, Burdett RG, Gee Z. A comparison of gait characteristics in young and old subjects. *Phys Therapy* 1994;74:637–46.

- [24] Morris ME, McGinley J, Huxham F, Collier J, Ianseck R. Constraints on the kinetic, kinematic and spatiotemporal parameters of gait in Parkinson's disease. *J Hum Movement Sci* 1999;18:461–83.
- [25] Fahn, S, Elton R. Members of the UPDRS development committee. Unified Parkinson's disease rating scale. Recent developments in Parkinson's Disease. In: Fahn S, Marsden CD, Calne DB, Goldstein M, editors, *Recent developments in Parkinson's disease*, vol II. New York: MacMillan; 1987. p. 153–63.
- [26] Dogali M, Fazzini E, Kolodny E, et al. Stereotactic ventral pallidotomy neurosurgery for Parkinson's disease. *Neurology* 1995;45:753–61.
- [27] Lozano AM, Lang E, Galvez-Jimenez N, et al. Effect of GPi pallidotomy on motor function in Parkinson's disease. *Lancet* 1995;346:1383–7.
- [28] Kishore A, Turnbull IM, Snow BJ, et al. Efficacy, stability and predictors of outcome of pallidotomy in patients with advanced Parkinson's disease. *Brain* 1997;120:729–37.
- [29] Meyer CHA. Unilateral pallidotomy for Parkinson's disease promptly improves a wide range of voluntary activities especially gait and trunk movements. *Acta Neurochir* 1997;68:37–41.
- [30] Cotzias GC, Papavasiliou PS, Gellene R. Modification of Parkinsonism – chronic treatment with L-dopa. *New England J Med* 1969;280:337–45.
- [31] Knutsson E. An analysis of parkinsonian gait. *Brain* 1972;95:475–86.
- [32] Bowes SG, Clark PK, Leeman AL, O'Neill CJA, Weller C, Nicholson PW, Deshmukh AA, Dobbs SM, Dobbs RJ. Determinants of gait in the elderly Parkinsonian on maintenance levodopa/carbidopa therapy. *Br J Clin Pharmacol* 1990;1:13–24.
- [33] Blin O, Ferrandez AM, Pailhous J, Serratrice G. Dopa-sensitive and Dopa resistant gait parameters in Parkinson's disease. *J Neurol Sci* 1991;103:1–54.
- [34] Blin O, Ferrandez AM, Serratrice G. Quantitative analysis of gait in Parkinson patients: increased variability of stride length. *J Neurol Sci* 1990;98:91–7.
- [35] Morris M, Ianseck R, Matyas T, Summers J. Abnormalities in the stride length-cadence relation in parkinsonian gait. *Movement Disord* 1998;13:61–9.
- [36] Morris ME, Ianseck R, Matyas TA, Summers JJ. The pathogenesis of gait hypokinesia in Parkinson's disease. *Brain* 1994;117:1169–81.
- [37] Morris ME, Ianseck R, Matyas TA, Summers JJ. Ability to modulate walking cadence remains intact in Parkinson's disease. *J Neurol, Neurosurg Psychiatry* 1994;57:1532–4.
- [38] Forssberg H, Johnels B, Steg G. Is parkinsonian gait caused by a regression to an immature walking pattern? *Adv Neurol* 1984;40:375–9.
- [39] 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. *Movement Disord* 1999;14:619–25.
- [40] Mesure S, Azulay JP, Pouget J, Amblard B. Strategies of segmental stabilization during gait in Parkinson's disease. *Exp Brain Res* 1999;129:573–81.
- [41] Sutton JP, Couldwell W, Lew MF, et al. Ventroposterior medial pallidotomy in patients with advanced Parkinson's disease. *Neurosurgery* 1995;36:1112–7.
- [42] Grasso R, Peppe A, Stratta F, Angelini D, Zago M, Stanzione P, Lacquaniti F. Basal ganglia and gait control: apomorphine administration and internal pallidum stimulation in Parkinson's disease. *Exp Brain Res* 1999;126:139–48.
- [43] Nieuwboer A, DeWeerd W, Dom R, Nuttin B, Peeraer L, Pattyn A. Walking ability after implantation of a pallidal stimulator: analysis of plantar force distribution in patients with Parkinson's disease. *Parkinsonism Related Disord* 1998;4:189–99.
- [44] MacKay-Lyons M. Variability in spatiotemporal gait characteristics over the course of the L-dopa cycle in people with advanced Parkinson disease. *Phys Therapy* 1998;78:1083–94.
- [45] McIntosh GC, Brown SH, Rice RR, Thaut MH. Rhythmic auditory-motor facilitation of gait patterns in patients with Parkinson's disease. *J Neurol, Neurosurg Psychiatry* 1997;62:22–6.
- [46] Miller RA, Thaut MH, McIntosh GC, Rice RR. Components of EMG symmetry and variability in parkinsonian and healthy elderly gait. *Electroencephalography Clin Neurophysiol: Electromyography Motor Control* 1996;101:1–7.
- [47] Nieuwboer A, De Weerd W, Dom R, Peeraer L, Lesaffre E, Hilde F, Baunach B. Plantar force distribution in Parkinsonian gait: a comparison between patients and age-matched control subjects. *Scand J Rehabil Med* 1999;31:185–92.
- [48] Koozekanani SH, Balmaseda MT, Mohammad TF, Lowney ED. Ground reaction forces during ambulation in Parkinsonism: pilot study. *Arch Phys Med Rehabil* 1987;68:28–30.
- [49] Dietz V, Zijlstra W, Prokop T, Berger W. Leg muscle activation during gait in Parkinson's disease: adaptation and interlimb coordination. *Electroencephalography Clin Neurophysiol: Electromyography Motor Control* 1995;97:408–15.
- [50] Ebersbach G, Sojer M, Valdeoriola F, Wissel J, Muller J, Tolosa E, Poewe W. Comparative analysis of gait in Parkinson's disease, cerebellar ataxia and subcortical arteriosclerotic encephalopathy. *Brain* 1999;122:1349–55.
- [51] Urquhart DM, Morris ME, Ianseck R. Gait consistency over a 7-day interval in people with Parkinson's disease. *Arch Phys Med Rehabil* 1999;80:696–701.
- [52] Schenkman ML, Cutson TM, et al. Reliability of impairment and physical performance measures for persons with Parkinson's disease. *Phys Therapy* 1997;77:19–27.
- [53] MacKay-Lyons M. Variability in spatiotemporal gait characteristics over the course of the L-dopa cycle in people with advanced Parkinson's disease. *Phys Therapy* 1998;78:1083–94.
- [54] 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. *Movement Disord* 1998;13:428–37.
- [55] 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* 1998;65:580–2.
- [56] Thaut MH, McIntosh GC, Rice RR, Miller RA, Rathbun J, Brault JM. Rhythmic auditory stimulation in gait training for Parkinson's disease patients. *Movement Disord* 1996;11:193–200.
- [57] Hanakawa T, Katsumi Y, Fukuyama H, Honda M, Hayashi T, Kimura J, Shibasaki H. Mechanisms underlying gait disturbance in Parkinson's disease. A single photon emission computed tomography study. *Brain* 1999;122:1271–82.
- [58] Hanakawa T, Fukuyama H, Katsumi Y, Honda M, Shibasaki H. Enhanced lateral premotor activity during paradoxical gait in Parkinson's disease. *Ann Neurol* 1999;45:329–36.
- [59] Mori S. Integration of posture and locomotion in acute decerebrate cats and in awake, freely moving cats. *Prog Neurobiol* 1987;28:161–95.
- [60] Weissenborn S. The effect of using a two-step verbal cue to a visual target above eye-level on the Parkinsonian gait: a case study. *Physiotherapy* 1993;79:26–31.
- [61] Bagley S, Kelly B, Tunnicliffe N, Turnbull GI, Walker JM. The effect of visual cues on the gait of independently mobile Parkinson's disease patients. *Physiotherapy* 1991;77:415–20.
- [62] Sedgman R, Goldie P. Development of a measure of turning during walking. In: *Advancing rehabilitation: Proceedings inaugural conference of Faculty of Health Sciences, La Trobe University, 1994; Melbourne, Australia.*

- [63] Smithson F. Predisposing factors for falls in Parkinson's disease. In: Older people in our society: Conference proceedings, 1994; Perth, Australia.
- [64] Stack E, Ashburn A. Fall events described by people with Parkinson's disease: implications for clinical interviewing and the research agenda. *Physiotherapy Res Internat* 1999;4:190–200.
- [65] Hase K, Stein RB. Turning strategies during human walking. *J Neurophysiol* 1999;81:2914–22.
- [66] Patla AE, Adkin A, Ballard T. Online steering: coordination and control of body centre of mass, head and body reorientation. *Exp Brain Res* 1999;129:629–34.
- [67] Patla AE, Prentice SD, Robinson C, Neufeld J. Visual control of locomotion: strategies for changing direction and for going over obstacles. *J Exp Psychol, Hum Percep Perform* 1991;17:603–34.
- [68] Charlett A, Weller C, et al. Breadth of base whilst walking: effect of ageing and parkinsonism. *Age Ageing* 1998;27:49–54.
- [69] Van Emmerik REA, Wagenaar RC, Winogradzka A, Wolters EC. Identification of axial rigidity during locomotion in Parkinson disease. *Arch Phys Med Rehabil* 1999;80:186–91.
- [70] Van Emmerick REA, Wagenaar RC. Effects of walking velocity on relative phase dynamics in the trunk in human walking. *J Biomech* 1996;29:1175–84.
- [71] Kaya BK, Krebs DE, O'Riley PO. Dynamic stability in elders: momentum control in locomotor ADL. *J Gerontol: Med Sci* 1998;53A:M126–134.
- [72] Cao C, Schultz AB, Ashton-Miller JA, Alexander NB. Sudden turns and stops while walking: kinematic sources of age and gender differences. *Gait Posture* 1998;7:45–52.
- [73] Horak FB, Nutt JG, Nashner LM. Postural inflexibility in Parkinsonian subjects. *J Neurol Sci* 1992;111:46–58.
- [74] Vieregge P, Stolze H, Klein C, Heberlein I. Gait quantification in Parkinson's disease – locomotor disability and correlation to clinical rating scales. *J Neural Trans* 1997;104:237–48.
- [75] Ginanneschi A, Degl'Innocenti F, Magnolfi S, Maurello MT, Marini P, Amaducci P. Evaluation of Parkinson's disease: reliability of three rating scales. *Neuroepidemiology* 1988;7:38–41.
- [76] Pedersen SW, Eriksson T, Oberg B. Effects of withdrawal of antiparkinsonian medication on gait and clinical score in the Parkinson patient. *Acta Neurol Scand* 1991;84:7–13.
- [77] Cioni M, Richards CL, Malouin F, Bedard PJ, Lemieux R. Characteristics of the electromyographic patterns of lower limb muscles during gait in patients with Parkinson's disease when OFF and ON L-Dopa treatment. *Ital J Neurol Sci* 1997;18:195–208.
- [78] Azulay J-P, Mesure S, Amblard B, Blin O, Sangla I, Pouget J. Visual control of locomotion in Parkinson's disease. *Brain* 1999;122:111–20.