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HIP PULL-OFF AND ANKLE PUSH-OFF IN HEALTHY AND PARKINSON'S DISEASE'S GAIT

Master of Philosophy.

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ABSTRACT

FACULTY OF MEDICINE, HEALTH AND LIFE SCIENCES

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Master of Philosophy

HIP PULL OFF AND ANKLE PUSH-OFF IN HEALTHY AND

PARKINSON'S DISEASE'S GAIT

By Olumide Sofuwa

An emerging theme in the analysis of the gait of people with Parkinson's disease (PD) is that the kinetic features such as joint power generated during the events of ankle push-off and hip pull-off required for propulsion and progression of the lower limb is diminished in spite of PD medication. However, there is lack of sufficient evidence from the proponents of these PD gait deficiencies. Furthermore, it was thought in the present study that the cause of reduced joint power (if observed) could be attributed to their preferred slow walking speed. Therefore, the research presented in this thesis investigated the pull and push-off events at usual walking speed (in healthy and people with PD) and at fast walking speed (in PD) to examine and substantiate what the other studies suggested.

The first part of the investigation into pull and push-off was to characterise and describe these gait events in healthy people whilst the second part was carried out on people affected with PD. A laboratory protocol/setup was designed for the first part of the study that was modified for use in the second part.

Fourteen healthy adults (mean age 63.6 ± 7 years) participated in the first (laboratory based) study. Their gait was measured using a camera based motion analysis system (Coda mpx30) and a force platform that was situated on an 8m walkway. A gait laboratory test session was conducted per subject and the result of gait components of pull and push-off powers, ankle and hip joint angular excursion and corresponding gait velocity, stride length and cadence were recorded. Similar laboratory sessions with modification to the protocol/set up were also conducted (after an initial home clinical assessment and screening) for people with PD ($n=11$, mean age 66.4 ± 5.07 years, disease duration 6 ± 3.1 years, motor UPDRS score 24.3 ± 9.98) walking at their usual and fast speeds during their on phase of PD medication.

The results showed that the gait of healthy subjects and subjects with PD was not significantly different from each other. Some PD subjects had greater pull-off and push-off powers than healthy subjects whilst a relationship between hip pull-off power and ankle plantarflexion suggests a compensatory strategy being used by PD subjects. PD subjects were able to significantly walk fast when asked to do so, with increments in the gait components. The results suggest that reduced pull and push-off powers are not applicable to all cases of PD and in spite of any diminished gait features their capacity to walk fast was still preserved.

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DECLARATION OF AUTHORSHIP

I, Olumide Sofuwa,

declare that the thesis entitled

...Hip pull-off and ankle push-off in healthy and Parkinson's disease's gait....

and the work presented in the thesis are both my own, and have been generated by me as the result of my own original research. I confirm that:

- this work was done wholly or mainly while in candidature for a research degree at this University;
- where any part of this thesis has previously been submitted for a degree or any other qualification at this University or any other institution, this has been clearly stated;
- where I have consulted the published work of others, this is always clearly attributed;
- where I have quoted from the work of others, the source is always given. With the exception of such quotations, this thesis is entirely my own work;
- I have acknowledged all main sources of help;
- where the thesis is based on work done by myself jointly with others, I have made clear exactly what was done by others and what I have contributed myself;
- none of this work has been published before submission

Signed:

Date:.....

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O.A Sofuwa

Abbreviations

APOP	Ankle push-off power
BG	Basal ganglia
CNS	Central Nervous System
CODA	Cartesian Optoelectronic Dynamic Anthropometer
CPG	Central Pattern Generators
GPe	Globus Pallidus External Nucleus
GPi	Globus pallidus Internal Nuclues
HPOP	Hip pull-off power
KnAbP	Knee absorption Power
LED	Light Emitting Diode
Nm/kg	Newton metre per kilogram
PD	Parkinson's disease
ROM (h,k,a)	Range of motion (at the hip or knee or ankle)
SMA	Supplementary Motor Area
SNc	Subtanstia Nigra Pars Compacta
SNr	Substanstia Nigra Pars Reticulata
STN	Subthalamic Nucleus
W/kg	Watts per kilogram

Glossary

Cadence

Number of steps taken within a minute.

Force

Product of mass (e.g. body segment) and acceleration. It is measured in Newtons.

Gait

Style or manner of walking.

Gait cycle

An equivalent of a stride. Consists of two main phases i.e. stance and swing. These two are further divided into sub phases: initial contact, loading response, midstance, terminal stance, preswing, initial swing, midswing, and terminal swing. Measured from 0% i.e. the first initial contact (or heel strike) to the end of terminal swing and beginning of the next initial contact 100%.

Gait cycle velocity

The velocity in which a gait cycle is accomplished. It is measured in metres per second.

Initial contact

This is the start point (0%) of the gait cycle (100%). It sometimes called the heel strike and marks the beginning of the Loading response period.

Initial swing

This starts when the foot breaks contact with the ground (toe-off) until maximum knee flexion occurs and the

swinging limb is directly under the body opposite the stance limb. It represents 60-75% of the 100 gait cycle.

Kinematics

Relating to motion without reference to kinetics (e.g. joint displacement or range of motion ($^{\circ}$) and angular acceleration/ velocity (radians per second)).

Kinetics

Relating to forces generated across a joint when walking e.g. moment of force (Normalised to body weight i.e. Newton metre per second-Nm/kg) and power (Normalised to body weight i.e. Watts per second-W/kg).

Loading response

This is when the foot is lowered to the ground after the heel strike. It represents 0-10% of the 100 % gait cycle.

Mid stance

The is the period that the foot is flat on the ground after loading response. It is when the body weight travels along the length of the foot until it is aligned over the foot.

Mid swing

This begins from the point of maximum knee flexion in swing and ends when the leg is in vertical position. It represents 75-85% of the 100% gait cycle.

Phase (in gait)	Terminology for parts or sections of a gait cycle e.g. loading response, mid-stance & terminal swing.
Power	Work done across a joint per unit time. It is measured in Watts (normalised to body weight in this thesis i.e. Watts/kg).
Period (in gait)	Time interval or duration required to accomplish the a complete gait cycle or a phase of the cycle (measured in seconds or percentages).
Preswing	This begins when the contralateral foot contacts the ground and ends at ipsilateral toe-off. It represents 50-60% of the 100 gait cycle.
Pull-off	An event of gait occurring within the late stance/preswing phase due to the flexion action at the hip joint. The peak pull-off power generated at the hip joint is graphically recognised as A2.
Push-off	An event of gait occurring within the late stance/preswing phase due to the plantarflexion action at the ankle joint. The peak ankle push-off is graphically recognised as H3.

Spatiotemporal	Time-distance component of gait (e.g. stride length, gait velocity and cadence).
Stance	The period of the gait during which the foot/feet is on the ground.
Stride length	the distance between an initial contact to the next. It is measured in metres.
Swing phase	The period of gait cycle between the point the foot breaks contact with the ground to the point of initial contact of the next gait cycle. It is about 60-100% of the gait cycle.
Terminal stance	This begins with heel rise (after the midstance period) and ends when the contralateral foot contacts the ground. It represents about 30-50% of the 100% gait cycle.
Terminal swing	This begins from the vertical position of the leg in swing to full extension in preparation for initial contact of the next gait cycle. It represents 85-100% of the 100% gait cycle.

Chapter One

Introduction

1.0 Introduction to Thesis

The focus of this thesis is on the gait events of “pull-off” at the hip joints, and “push-off” at the ankle joints when people affected with Parkinson’s disease (PD) are walking. These two terminologies describe in healthy people what appear to be a propulsion (at the ankle joint) and a haulage (at the hip joint) due to the action of the muscles (and the power they generate) that primarily act on the joints to move the lower limbs when walking.

My particular interest in investigating these two gait events in PD grew out of a combination of events from previous clinical experience. Whilst working as a rehabilitation physiotherapist (in my home country) with patients having movement disorders due to neurological deficits, I observed that cases of PD referred for physiotherapy were extremely few. This was in great contrast to the number of referred patients with CVA. This may either be due to societal perception of PD (to dismiss it as early onset of old age) or low prevalence of the disease. Moreover, with the benefit of hindsight, I realised that on the occasions when PD cases were referred, the management strategies employed by therapists were not as informed as the management of patients with CVA especially in the area of clinical gait analysis and rehabilitation. With PD patients, the secondary health problems such as decreased flexibility, strength and endurance were often the focus of management. Thus, my clinical experience with this group of patients was limited. Fortunately, the opportunity arose for a postgraduate research study in Belgium on the gait of people with PD using a 3-D motion analysis system (Vicon) - a measurement technique that was novel to me- for the analysis of normal walking in people with PD.

Interestingly, one of the observations made in the PD gait research in Belgium was that ankle push-off power was reduced in PD gait in spite of the patients being on levodopa medication (Sofuwa et al 2005). There was also an indication that hip pull-off event might be reduced

but this was not clear from the study. This initial experience prompted my curiosity to further investigate these phenomena in PD gait in a future study and propose interventions specifically targeting these gait deficiencies. Actually, I considered the concept of a motion analysis system that provides real-time biofeedback on magnitudes of pull/push-off powers for PD patients hence being a beneficial adjunct in the rehabilitation of PD gait. Such intervention could be an area for future exploration by researchers in movement analysis. However, the issue of pull and push-off in PD gait needed to be first investigated and clarified before any intervention was explored.

Therefore, the overall aim of this present work was to characterise pull and push-off events in the gait of people with PD when they are walking at their usual speed. Whilst the present study was in progress, I hypothesised that if a reduction in pull or push-off power was observed it could have resulted from the slower walking speed usually associated with PD because power is a function of speed (Lelas et al 2003). It may be that power may be improved by fast walking. Thus, the changes that occur in pull and push-off powers generated when people with PD were asked to walk fast was also examined.

I think this research work is important because: 1) Deficiencies in pull/push-off powers in PD will definitely affect propulsion and progression when walking (Lehmann 1993, Kirtley 2005) therefore resulting in multiple difficulties such as loss of independence, daily functioning and possibly social isolation. Therefore, an understanding of how pull/push-off is affected in PD gait would inform management of the gait disorder in people with PD. 2) It is not known if all people with PD manifest deficiency with pull/push-off powers as there is little evidence around this topic 3). There is little information on other gait parameters associated with pull and push-off events in PD. Therefore, the findings from this present work will contribute to the database of gait analysis and management of people with PD.

Before examining pull and push-off in people with PD, it was imperative to carry out a first study to characterise and describe pull and push-off as they occur in healthy people. A gait analysis laboratory protocol was designed for this purpose that was reviewed for the second study to characterise and describe the pull/push-off in the gait of people with PD. The following sections presents the background and literature on previous investigations informing this study followed by the first and second studies.

1.2 Descriptors of Healthy Gait

Walking is accomplished through a complex coordinated pattern of nerve signals sent to the muscles that in turn move the joints, the limbs and the remainder of the body. Gait refers to the specifics of the general walking process that differentiates one individual from another. Irrespective of age, the healthy adult gait is a cyclic event. This means that there is a starting and an end point that is repeated when walking. The gait cycle has been systematically studied and certain prominent features have been highlighted that describe the consistent features of the gait cycle (Saunders et al 1953, Gage 1991, Perry 1992,).

There are several descriptors used for the events of the adult gait cycle in the gait literature such as the six determinants of gait i.e. pelvic rotation, pelvic tilt, knee flexion, hip flexion, knee-ankle interaction, and lateral displacement of the pelvis (Saunders, Inman, & Eberhart 1953). Whilst five prerequisites for normal gait have been proposed such as stability in stance, adequate foot clearance in swing, preposition of the foot in terminal swing, adequate step length and energy conservation have also been described (Gage 1991). However, it is generally accepted that a complete stride is the equivalent of a gait cycle. The description of the human gait cycle by Perry (1992) is perhaps the most common and widely accepted by the majority of people working in the field of gait analysis.

Perry (1992) explained that the gait cycle is divided into two phases: **Stance phase** (when the foot is on the ground) and **swing phase** (when the foot is moving through the air) (fig 1.1). During transition from stance to swing and later from swing to stance, both feet are simultaneously on the ground for a brief periods called initial and terminal double stance respectively. The period of a stride (or gait cycle) varies slightly for different people. However, during gait analysis, for the possibility of comparison, the gait cycle period is set to 100% irrespective of the persons being analysed. The stance phase constitutes about sixty

percent of the gait cycle period and the swing phase constitutes the remaining forty percent. Both phases are further divided into eight sub phases (Initial contact, Loading response, Mid stance, Terminal (late) stance, Preswing, Initial swing, Midswing, and Terminal swing).

1.3 Gait Parameters

With the development of sophisticated motion analysis systems, it is possible to use quantitative measures to describe changes that occur during the process of the cycle. These measures can be presented in three principal planes; sagittal, frontal and transverse planes at the pelvis, hip, knee and ankle joints (Whittle 2006). The outcome of the measures can reflect various walking disorders or abnormalities originating from various causes ranging from abnormal footwear, musculoskeletal conditions to neurodegenerative pathologies such as Parkinson's disease. The measurement categories commonly used in gait analysis are the spatiotemporal (gait velocity, stride length and cadence), kinematics (joint range of excursion, angular velocity) and the kinetics (Perry 1992, Kirtley 2005, Whittle 2006).

The kinetics include the joint moments of force and powers that move the joints to produce the spatiotemporal and kinematic features of gait (Perry 1992, Whittle 2006). Hence, measurement of the gait changes that occur on the kinetic level is important to the understanding of healthy or pathological gait. Most importantly, joint power (product of joint moment of force and angular velocity) is required during propulsive events such as pull and push-off at the hip and ankle joints.

The power across a joint is generated by muscular activity to move the limbs. The ability of muscles to generate power is affected by factors such as the cross-sectional area of the muscle, its length-tension ratio and degree of fatigue (Hof 2003, Mannion & Dolan 1996). With these factors being constant, there is a characteristic pattern where power is generated

or absorbed during the gait cycle. The measurement, interpretation and understanding of the power characteristics (especially with regards to pull and push-off event) is possible in motion analysis systems using a process called inverse dynamics (Whittle 2006) that involves the integration of the kinematics (e.g. angular velocity) and force (i.e. moment of force) generated across the joint.

1.4 Descriptors of Joint Power.

Most measurements of power generated or absorbed across a joint are usually graphically presented in 3-D motion analysis systems as peaks and troughs. The peaks represent power generated while the troughs are power absorbed. Winter (1991) gave a useful annotation that is increasingly being adopted by researchers in field of gait analysis (Fig 1.2). The power generated at each joints is profiled along the gait cycle (0-100%) with the peaks and troughs of the profile labelled according to the designation of the joint and the sequence of the peak/trough e.g. the first power peak (power generated) at the hip, knee, and ankle joints are H1, K2 and A2 respectively (fig 1.2). Only the hip joint has a second power generation in stance i.e. H3. The power absorbed (troughs) are represented as H2 (the hip), K1 and K3 (the knee) and A1 (the ankle). These joint profiles represent power generated in the sagittal plane i.e. plane of largest movement in the gait cycle. All the measurements of joints powers in humans are usually normalised to the weight of the body per unit kilogram (fig 1.2). Thus, joint powers as presented henceforth in the following chapters will be normalised powers i.e. in Watts/kg.

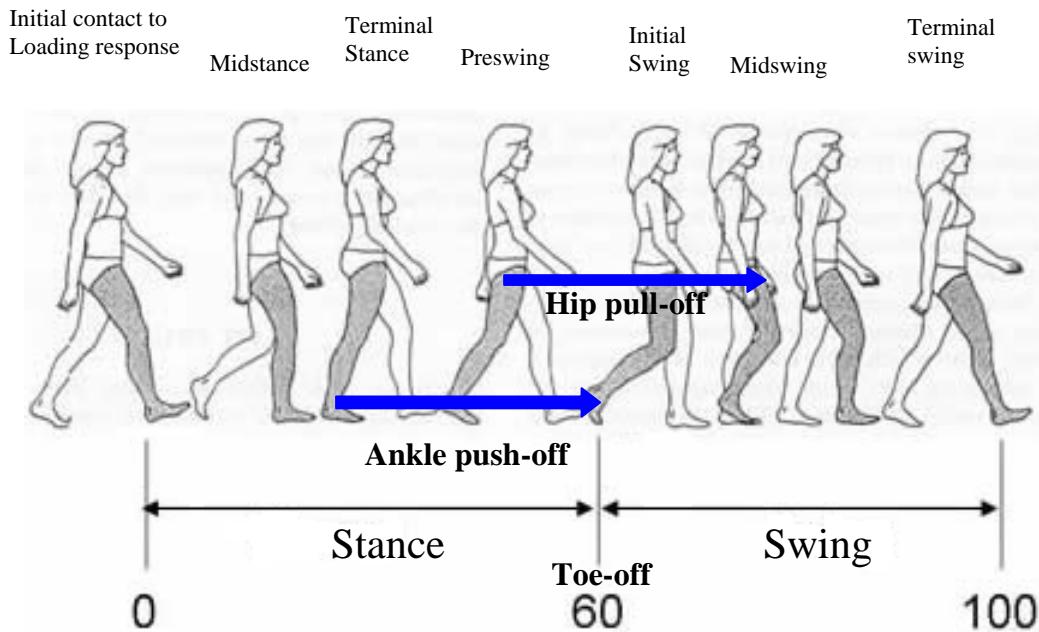


Fig.1.1 The Adult gait cycle (dark limb) demonstrating hip pull-off (preswing to start of midswing) and Ankle push-off (terminal stance to toe-off) events (adapted from Whittle 2006).

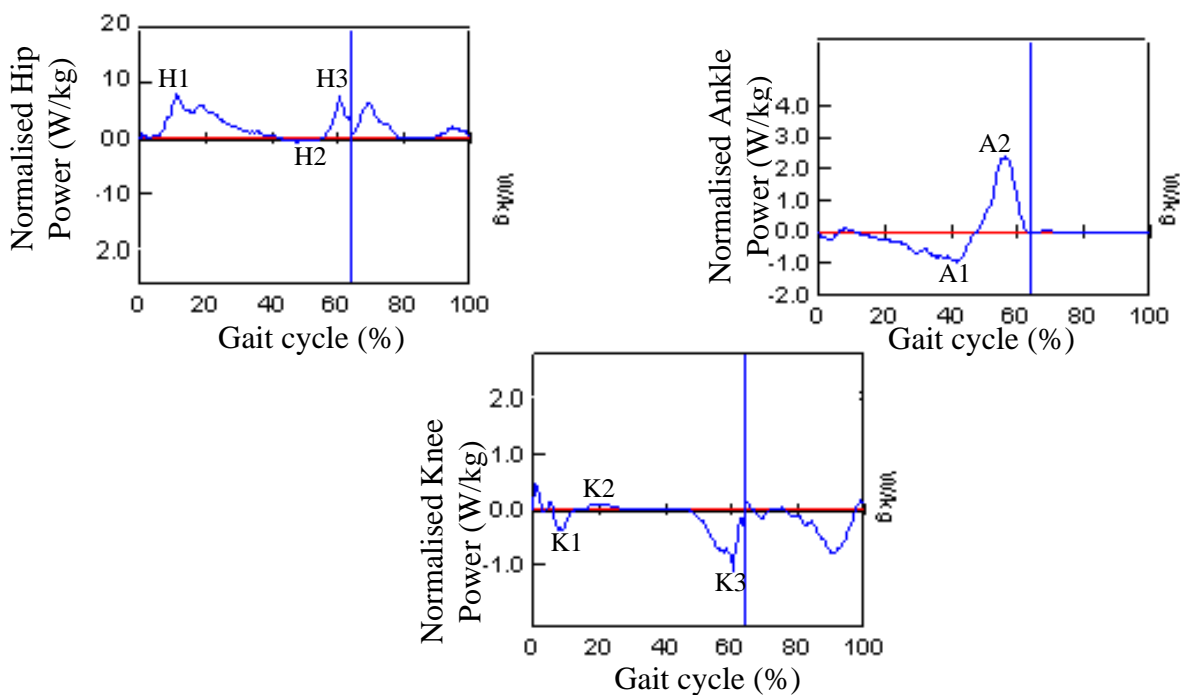


Fig 1.2. Joint Power profiles in the sagittal plane (Winter 1991). Y-axis is magnitude of power generated (+) or absorbed (-). X axis represent the complete gait cycle (0-100%). Middle vertical line about 60% represents the toe-off point. H3 = hip flexion (PULL-OFF) power, A2 = ankle power (PUSH-OFF) power generated in preswing. K3 = knee power absorbed in preswing.

1.4.1 Ankle push-off

The terms “push-off” and “pull-off” have been used in several studies that have investigated the kinetics of gait component either in the non healthy or healthy individuals (Perttunen et al. 2004; Shaw et al. 1998). Push-off has been used to describe the second peak of force i.e. F2 that is observed after the force of heel strike (F1) on a force platform measurement during gait analysis (Winter 1991). It describes when a person pivots on the ball of the foot as the heel is raised during walking. Others studies have used the term “reduced push-off” to describe reduced plantarflexion action either in the healthy gait (Hase & Stein 1998; Pijnappels et al 2005) or in pathologic gait (Hesse et al 1994; Pijnappels 2005). However, these studies assumed that the term "push off" was widely accepted within gait analysis domain therefore they were not compelled to give a description or definition specifically in relation to the periods of the gait cycle concerned. However, their methods indicate that what they were referring to is the event of plantarflexion action in the late stance period of the gait cycle.

The earliest description of push-off was by Winter (1991) who defined push-off as a component of gait late in the stance period where the plantarflexors generate a concentric ‘explosive’ burst of energy, causing the foot to rapidly plantarflex. Another description of push-off was given as “the action of the rapidly contracting plantarflexors in late stance period to provide the single largest burst of power generation in the gait cycle of adults without impairments”-(p147 McGinley et al 2003). Nadeau described push-off as occurring around the period of preswing i.e. about 40% and 60% of the gait cycle (Nadeau et al. 1999). Due to slight variations in gait between healthy individuals, it is not possible to name an exact common period in the gait cycle for push-off. Depending on the person concerned, it can be generalised to occur during the terminal stance period of the gait cycle.

Therefore, considering these previous descriptions, push-off in healthy people can be summarily described as *'the ankle plantarflexion that occur from terminal stance to toe-off of the gait cycle that contributes to lifting the foot off the ground'* (fig 1.1). The maximum power generated by the plantarflexors within this event is the **push-off power** i.e. A2 in ankle power graph (fig 1.2).

1.4.2 Hip pull-off

Hip pull-off is not a commonly used term as ankle push-off. It was also initially described by Winter (1991) to also begin in late stance just as is observed in push-off but a bit later i.e. preswing, around 50%. It ends in the early swing phase i.e. around 70% (Nadeau et al 1999) and it is believed to give added energy to the swing limb (Eng & Winter 1995). The power generated at the hip joint during pull-off i.e. **pull-off power** reaches its peak i.e. H3 (fig.1.2) in preswing just before toe-off point.

The end of ankle push-off phase and the beginning of hip pull off phases overlap to give an intra limb coordination such that as the action of the ankle plantarflexors is ending the hip flexor action begins. The combination of the ankle and hip powers towards the end of stance phase generates the required propulsion (hence progression) of the lower limb into the swing phase (Sadeghi et al. 2001).

For the purpose of the present study, hip pull-off in the gait of healthy people is described as *the hip flexion that occurs from preswing to midswing of the gait cycle that raises the thigh and contribute to lifting of the leg and foot off the ground* (fig 1.1). The maximum power generated by the hip flexors within this event is the pull-off power (H3) (fig 1.2).

1.4.3 The Knee - Its role during pull-off/push-off

The knee joint cannot be said to pull-off or push-off because unlike the hip and ankle joints during late stance/preswing phase, power is not generated but absorbed i.e. K3 (fig 1.2). Notwithstanding, the knee kinetics throughout periods of the gait cycle cannot be dismissed. When walking, the knee kinetics are observed to interplay with the other two joints i.e. ankle-knee coupling or hip-knee coupling. However, there appears to be more of hip-knee coupling which results in a higher variability of these two joints' kinetic output (Craig 1995). It has been observed that for an individual to be balanced dynamically when walking, there is more of trade-off between the knee and hip joint than with the ankle joint (Craig 1995; Winter et al. 1990). As the moment of force at hip joint changes to keep the support pattern and balance of the upper body, there must be an opposite change in the knee moment which is almost as variable (Craig 1995, Winter et al 1990). Thus, it can be inferred that changes in the power generated during hip pull-off i.e. H3 will occur with concurrent changes at the knee albeit as power absorbed i.e. K3 (fig 1.2). The power absorption observed at the knee (K3) during pull-off and push-off phases occurs as the knee rapidly flexes to unload the limb in preparation for swing phase. To control the speed of collapse at the knee, the quadriceps absorbs energy by contracting eccentrically hence absorbing energy (Perry 1992).

The joint forces and powers required to accomplish a normal gait are produced by muscles acting on the skeletal system-in this case the hip, knee and ankle joints. However, the control of magnitude and regulation of the joint force and powers is carried out by the central nervous system (CNS) (Jankowski et al 2009). The current evidence on neural control of movement provides some description of the role of the CNS in the control of gait. This has led to a key model that is widely accepted in the field of locomotor system research (Rothwell 2007). This model describe networks of systems and it is important to understand their complexities and relate their control to the features and changes in gait. The following

sections describe the neural network systems that significantly contribute to the control of the human gait, and the movement dysfunction that occur when such neural control is diminished as observed in Parkinson's disease.

1.5 Supraspinal Control of Gait

The present model of neurological control of gait show that primitive walking reflexes occur at the midbrain and subthalamic levels while selective motor control (a prerequisite of gait) is controlled at the level of the cerebrum (Lin & Gage 1989). This selective motor control by the cerebrum modifies the normal reflex or habitual pattern. However, the cerebrum does not work alone but by interaction with two other major regions: the cerebellum and the basal ganglia. Together these three brain regions influence lower motor neurons directly through the pyramidal system or indirectly via the extrapyramidal system (Rothwell 2007).

At the cerebral cortex, the primary motor cortex (including the premotor cortex and supplementary motor area-SMA) is utilised in generating and learning executive motor tasks such as gait in early life. Once gait is learned and reinforced (by repetitions) it is then transferred as a “programmed activity” or “engram” to the extrapyramidal system that include an essential group of subcortical nuclei called the basal ganglia (BG) (Kottke et al. 1978; Roland et al. 1980).

Inputs from peripheral receptors (e.g. muscle spindle and golgi tendon organs) to the cerebellum that then go to and from the cerebral cortex result in the programmed activity i.e. gait becoming fine tuned and smooth. Thus, the interconnections within the basal ganglia (BG) circuitry and its projection to the brain stem and spinal cord have been observed to be responsible for automatic control of gait (Rothwell 2007, Haber & Calzavara 2009).

1.5.1 The Basal Ganglia

Previous work by Morris (2000) on normal and pathological basal ganglia functioning led to the proposal that the basal ganglia function in two main roles in controlling the automatic movements mainly via their interaction with the supplementary motor area (SMA) located anteriomedial to the primary motor cortex. The first role is internal regulation and cueing to enable movement sequences to be carried out without attention. The second role is its contribution to cortical 'motor set'. This is achieved by aiding in the preparation and maintenance of motor plans in a state of readiness for action, enabling motor functions to be carried out functionally and appropriately.

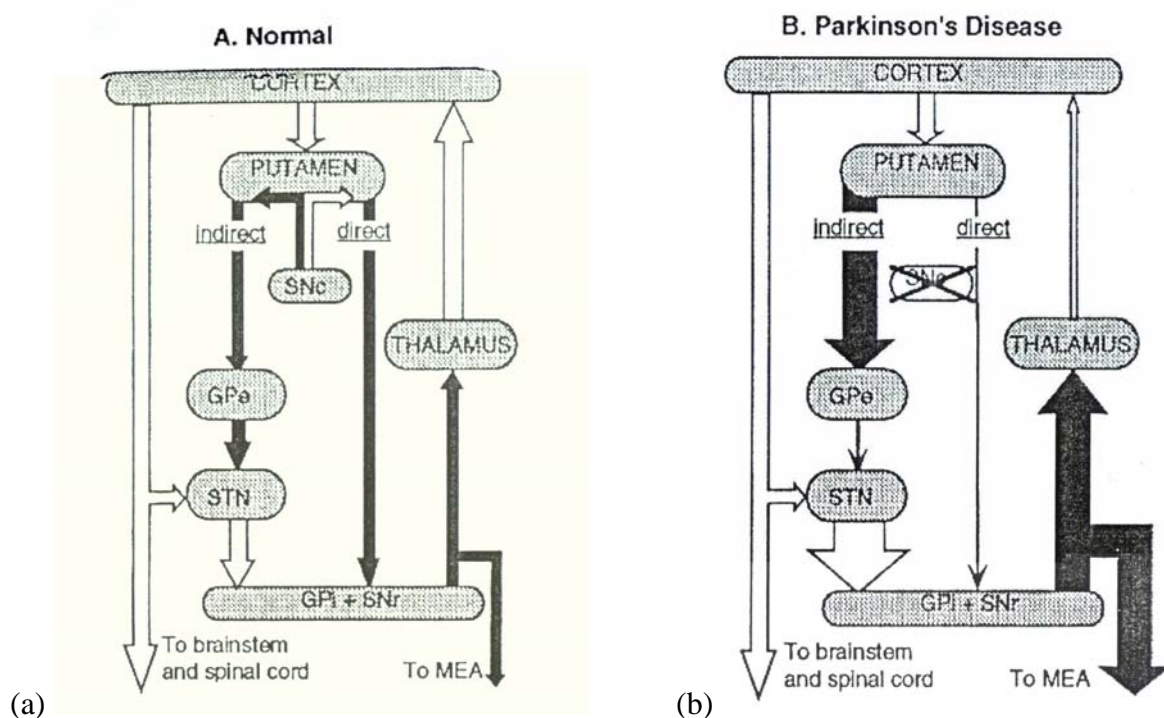


Fig 1.3. Basal ganglia in (a) healthy and (b) idiopathic Parkinson's disease (Pereira & Aziz 2006)

Direct and indirect pathways of the basal ganglia. White arrow = excitation, Black arrow = inhibition, SNc = substantia nigra pars compacta, SNr = substantia nigra pars reticulata, GPe = globus pallidus externus, GPi = Globus pallidus internus, STN = subthalamic nucleus, MEA = mid extra pyramidal area.

The BG nuclei classically consist of the substantia nigra pars compacta and reticulata, the caudate and putamen (corpus striatum), the subthalamic nucleus, the internal and external segments of the globus pallidus (fig 1.3a). The BG cells are either excitatory (white arrow-fig. 2.3) or inhibitory (black arrow) in their function. The excitatory and inhibitory function of the BG cells are operational via two classic pathways i.e. the direct and indirect pathways (fig 1.3a,b) (Pereira & Aziz 2006, Rothwell 2007). A range of degenerative diseases affect the BG such that clinical signs and symptoms are indicative of the dysfunction of a particular nucleus or its interconnection within the circuitry e.g. tremor indicates dysfunction of the Subthalamic nucleus (Sturman et al 2004). Most of the BG diseases present with features classified under movement disorders with associated cognitive and psychiatric manifestations (Weintraub 2004, Simuni 2008). A classic example is Parkinson's disease (PD). This will be discussed in the following section.

1.6 Parkinson's Disease

Parkinson's disease is a progressive neurodegenerative disorder that occurs due to depletion or interruption of supply of the neurotransmitter dopamine to the BG circuitry (Jones & Playfer 2007). This is mainly due to the death of the dopamine producing neuronal cells i.e. the substantia nigra. The diminished dopamine level results in increase or decrease in the inhibitory or excitatory output between the BG cells (fig 1.3b). The final consequence is an increased inhibition of the thalamic function thereby signals relayed through the pyramidal tracts are suppressed and diminished motor performance is observed (Pereira & Aziz 2006, Rothwell 2007, Jones & Playfer 2007).

The cause of the loss/death of dopaminergic nigral neuron has been unknown for a long time since the first description of the disease in 1817 by James Parkinson. However, some factors have been linked to the development of the disease; these include exposure to environmental

toxins such as MPTP in pesticides, carbon monoxide and manganese (Di Monte et al 2002; Cersosimo & Koller 2006). Recent advances in genetic research have increasingly identified hereditary factors as significantly contributory with environmental factors in the development of the disease (Dodson & Guo 2007; Solano et al. 2000).

The number of people affected with PD is difficult to ascertain due to different methods of data collection used by different epidemiology studies. However a systematic review by Twelves et al (2003) estimated PD incidence from three European countries (including the UK) to be approximately 16-19 per 100,000 per year but they warned that it “may be an underestimate because many patients remained undiagnosed without population screening”- pp28. Comparatively, Meara and Hobson (2000) estimated the incidence of PD in the UK to be 18 per 100,000 with a prevalence rate of 164 per 100,000 of the population.

1.6.1 Clinical Features

There are motor and non-motor symptoms that are used to identify a person affected with PD (Jones & Playfer 2007). The non-motor symptoms can include autonomic dysfunction, mood disturbance, loss of facial expression, breathing difficulties, sexual dysfunction and cognitive decline. However, many of the non-motor features only start to manifest after about 10 years of the disease duration. Conversely, the motor symptoms are the ones that feature earlier in the course of the disease. Four classic motor signs are used in identifying persons affected with PD. These are tremor at rest, rigidity, slow movement (bradykinesia) and loss of postural reflexes (Poewe 2006; Jones & Playfer 2007).

1.6.2 Motor Symptoms

Resting tremor

The first motor symptom often reported in about 75% of people with PD is the resting tremor (Clarke 2001; Selby G 1975). This tremor is seen to disappear when the person is active and moving. Hence, the ability to perform motor task such as writing, walking and grasping object are not influenced by tremor. The resting tremor is usually between 4-6Hz. However, less commonly some people present with action tremor that is between 6-8Hz observed when executing a task or moving. Tremor is asymmetrical at the initial stage of the disease but may spread to the other limb as the disease (Jones & Playfer 2007).

Rigidity

Rigidity is another prominent feature of PD often detected by slow passive movement of the affected body part (Jones & Playfer 2007).. However, active movement does not appear to be affected by rigidity as people with PD rarely complain about its presence even when it is rated as being severe on clinical examination. Rigidity is often experienced during clinical examination as a resistance that can be described either as “lead pipe” i.e. slow sustained resistance, or “cogwheel” rigidity i.e. ratchet like rigidity-caused by inclusion of tremor (Macphee 2001). It is believed that the rigidity is due to abnormal activation of long latency stretch reflex coupled with an increase in central reflex gain (Burke, Hagbarth, & Wallin 1977).

Bradykinesia

In contrast to the involuntary movements mentioned, voluntary motor symptoms occur in terms of bradykinesia, akinesia and freezing of movements (Jones & Playfer 2007). People with PD experience bradykinesia when movement amplitude and speed progressively decreases during execution of repetitive sequential movements of the limbs such as finger or

feet tapping. Growing evidence suggests that disruption of interaction between the globus pallidus of the BG and the supplementary motor area of the primary cortex (or between the globus pallidus and subthalamic nucleus) can cause bradykinesia (Berardelli et al 2001)

There are periods in which people with PD cannot start or initiate a movement (akinesia) or after movement have started, there is sudden cessation (motor blocks) partway through execution of the movement. Motor blocks are also referred to as “freezing” (Jones & Playfer 2007). Freezing has been observed to be triggered by certain situation with which the person with PD is exposed to or challenged with (Giladi et al 1997). For example, freezing has been observed to occur when attempting to walk through a narrow doorway or when walking onto a floor with different surface or texture e.g. wooden floorboard to carpet. As well having difficulty initiating movement is the difficulty of terminating movements. Morris (2000) attributed the difficulty ceasing actions such as walking, turning or speaking to a sustained discharge of the SMA rather than a rapid drop in neural activity.

Postural Instability

Postural instability usually appear during the later stage in the course of PD (Chastan et al 2008, Jones & Playfer 2007). It is known to result from the loss of postural or righting reflexes i.e. the reflex that allows people to readjust when balance is perturbed. At the stage that postural reflex is lost, the affected patients are also observed to present with a simian i.e. stooped posture that resulted from dominance of body’s flexor muscle tone over extensor tone (Jones & Playfer 2004). Characteristically, PD patients with impaired postural reflexes are also unable to stop falling backwards (retropulsion) or forwards (propulsion) thereby often increasing the risk of falls or injuries (Gray & Hildebrand 2000).

1.6.3 Diagnosis

Parkinson's disease usually starts insidiously therefore making early diagnosis difficult. Due to the progressive nature of the disease, the patients complain usually of the motor symptoms as they manifest often one at a time starting and usually unilaterally. The combination of two of the motor symptoms in a patient gives a clinical lead to suspect PD. The UK PD society Brain Bank criteria (Albanese 2003) is perhaps the most commonly used means for the diagnosis of PD. These criteria involve three steps in the diagnostic procedure as follows:

- 1) Presence or absence of Parkinsonian syndrome (e.g. bradykinesia and one of resting tremor, rigidity or bradykinesia)
- 2) Presence or absence of exclusion criteria (e.g. negative response to levodopa, head injury or Parkinsonian syndrome other than PD).
- 3) Supportive criteria for PD (e.g. unilateral onset of the disease, disease progression and positive response to Levodopa therapy)

1.6.4 Response to Levodopa

A positive response to the drug levodopa (a naturally occurring form of dopamine) is an important supportive criteria in the process of diagnosing PD, the drug remains the mainstay medication in the management of PD (Rezak 2007). The use of levodopa leads to marked improvement in Parkinsonian symptoms therefore patients can pursue their activity of daily living and improve on their quality of life. However, its use is associated with two major long term complications: fluctuations in motor performance and disabling involuntary movements.

The effect of levodopa can wear off within a short period of time (i.e. short half life) ranging from one to two hours hence the dosage has to be repeated over the course of the day (Nutt 2008, Pellicano et al 2009). With prolonged use of the drug over several years, the effect of

the daily dosage diminishes. Once this occurs, increasing dosage of the drug can lead to activation of involuntary movements affecting the face, tongue with choreo athetoid movement of the limbs (Obeso et al 2000). Response to levodopa can become unpredictable and patients can exhibit rapid switches from being 'on'-when the patients feels the effect of the medication and is able to move about- to the state of being 'off'-when the effect of the medication is not felt and carrying out activities of daily living become impossible. In some patients with PD, on-off syndrome can become unpredictable such that they are disabled and drug strategies are needed to cope with the problem (Obeso et al 2000).

The effect of medication on movement and functional capacity cannot be over emphasised. In the early stage of the disease, the response to drugs is very good and there is little residual deficit. However, as the disease progresses, inspite of medication, problems with functional ability in activities of daily living and difficulties with movement resurfaces. The movement problems that have been clinically identified in people with PD inspite of being medicated can be clinically categorised into problems with turning, transfer (e.g. sit to stand) balance and gait (Shulman et al 2008; Jones & Playfer 2007). With regards to gait problems in PD, investigative studies have reported changes in gait performance when testing the effect of intervention and strategies such as neurosurgery (Krack et al 2000), external stimuli (Rochester et al 2007) and levels of levodopa medication (Lubik et al 2006).

1.6.5 PD Gait and the Effect of Levodopa

People with PD experience considerable difficulty when walking. The difficulty in walking increases as the disease progresses such that a patient may be eventually immobilize and general well being affected. Slowness of gait (bradykinesia) in PD is often observed as decrease in speed and size (stride length) of gait. Some studies have been previously carried out to describe PD gait features in terms of spatiotemporal, kinematic and kinetics especially to test the effect of an intervention (Ferrarin 2005 et al, Frenkel-Toledo 2005, Van Wegen et al 2006). These studies were either conducted in the laboratory or in the hospital ward. They can be grouped into those that evaluated PD gait in the presence of (1) levodopa such as (a) in “de novo” condition or in the off phase (or end-of-dose) of their medication cycle (Koozekanani et al 1987) or (b) in the on phase (or peak dose) of medication cycle (Lewis et al 2000) and (2) intervention such as neurosurgery (Lozano et al 1995), exercise therapy (Goodwin et al 2008) and internal (Nieuwboer et al 1997) and external stimuli (cueing strategies) (Morris et al 1996, Lewis et al 2000). Many of the studies that investigate PD gait in the above conditions have mostly evaluated the spatiotemporal features than the kinematic and kinetic features.

In subjects who have never been medicated (de novo), it is common to observe subjects walk with marked reduced axial trunk rotation i.e. rigidity and a forward stooped posture (Van Emmerik et al. 1999). In addition, there is shuffling gait, reduced arm swing, stride length, speed, joint angular excursions at the hip, knee and ankle and ground reactions force with increased step to step variability (Ebersbach et al 1999, Koozekanani et al 1987, Murray et al 1978).

When subjects who have been placed on medication were tested, common changes were observed. In the testing conditions where medication was withdrawn or when there was an

end-of dose or 'off' effect of the medication cycle, the gait features were observed to be lower than that observed for healthy subjects (Kempster et al 1989). This was also demonstrated by Morris et al (1996) with subjects who were in their off phase (after 12 hour medication withdrawal) (Morris et al. 1994; Morris et al. 1996). They observed that the subjects walked more slowly (40-60m/min) than healthy age matched subjects (75-90m/min). This was inspite of the fact that it can take up to 3-4 weeks for the effect of levodopa to completely subside after withdrawal (Kempster et al. 1989). In addition, reduced stride length (0.4-0.9m) was also observed compared to that of healthy older people (1.2-1.5m) (Kerrigan et al. 1998; Ostrosky et al. 1994). Contrary to these diminished gait features when medication is withdrawn, cadence values remain within normal range of 100-110 step/min. Morris et al (1999) measured kinematics after withdrawal of medication (in a single case study) and observed that there were marked reduction in movement excursion across the hip, knee and ankle joints. These kinematic profiles were observed to be shifted towards flexion position with limited hip extension and plantarflexion ranges.

Inspite of walking in the L-dopa condition, PD subjects still have altered gait features when compared with healthy controls subjects (Lewis et al 2000, Sofuwa et al 2005). The PD gait characteristics during events of hip pull-off and push-off in the on phase of L-dopa medication is the focus of this present study. To describe and characterise PD gait in the medicated state, it is essential also to understand the features of the healthy adult gait as characterised from previous studies. The following chapter will examine the previous laboratory investigations on healthy adult gait and PD gait highlighting the events of push-off and pull-off.

Chapter Two

Laboratory Studies on Healthy and Parkinson's Disease (On medication) Gait

2.1 Introduction

In the measurement of usual or ‘normal’ gait in the healthy and people with pathological gait, there are factors to consider that make it difficult to have a consistent and specific description of gait. Factors that affect the our gait range from age to body dimension, physical fitness, psychological, regional or environmental, lifestyle or professional factors and the type of foot-wear (Arnadottir & Mercer 2000, Nurse et al 2005). These cause us to adapt to walk in a certain way. Even in instances where two people have been exposed to similar extraneous factors, intra and inter subject variability still exist. Therefore, attempts are made in gait laboratory test conditions to control or limit their influences. However, it was observed in the review of the body of work on gait analysis especially on the healthy and PD subjects that some differences still exist especially in the methods of acquiring gait data (e.g. laboratory set up and protocols) that could affect the measurements and objective interpretation of what is considered normal gait (Monaghan et al 2007, Gorton et al 2009). An examination of the methodological limitations in the previous studies on normal gait when a person is walking at usual (or fast speeds), will give an insight into the variability and comparability of the gait values. Thereafter, observations and findings between the studies on normal gait at usual speed and at fast speeds are converged and reviewed.

2.2 Gait Analysis Methods

Many of the differences between the studies are often associated with the differences between the subjects’ selected to participate in the studies, the laboratory protocol/set up, the instrumentation, and process and type of gait trial (test) analysis. These are discussed below.

The subjects groups recruited between the gait studies have differed with respect to different age limits of subjects. Some authors measured gait changes with increasing age (Auvinet et

al 2002, Blanc et al 1999, Bohannon 1997, Oberg et al 1993). Others investigators compared the young and the elderly (McGibbon et al 1999, Mills and Barrett 2001) while others compared intermediary ages (Cunningham et al 1982, Chao et al 1983). These different age groups across studies limit the pooling of findings to corroborate the effects of age on gait. Thus, to establish objective description of normal gait in healthy adults, there should be standard age categories for the elderly and young adults that are comparable across investigative gait studies. In PD gait studies, the effect of age on gait is usually controlled by matching the affected with similarly aged healthy elderly controls. However, within a PD age category, the disease severity may differ (e.g. Hoehn and Yahr 2 and 3), and often the average of different levels of severity is used. This could be resolved by having a large subject sample size that would allow further subgroup analysis on level of severity.

Different levels of physical activity or performance of subjects could cause differences between the studies. Some of the previous studies either screened and reported their subjects' level of physical activity or performance (Graf et al 2005, Watelain et al 2000, Allard et al 1996, McGibbon et al 1999, Bohannon et al 1997) whilst others did not screen nor gave any report (Shkuratova et al 2004, Vardaxis et al 1998, Oberg et al 1994, Jansen et al 1982, Zijlstra et al 2003, Murray et al 1969). In PD gait studies, it is not clear or quantified how the impact of other therapy the PD subjects may be receiving outside the studies could have affected movement and gait performance.

Another factor that have could caused the differences in findings on the gait parameter is the type of verbal commands (or instructions) issued during testing that could prompt modification or changes of gait by the subjects. Some of the instructions have varied between studies such as:

- “Walk at comfortable speed” (Bohannon 1997, Shkuratova et al 2004, Zijlstra et al 2003)
- “Walk at natural speed” (Watelain et al 2000, Vardaxis et al 1998, Allard 1996)
- “Walk at preferred rate” (Maki 1997)
- Unknown (Oberg et al 1994, Murray et al 1969)
- The exact instruction given not reported but authors analysed “Usual pace walking” (Judge et al 1996) or “self selected pace” (Eng et al 1994).

These different types of instruction could prompt subjects to walk at different gait speeds and may contribute to differences in gait features observed between studies.

Instrumentation between these studies also varied from the use of Interrupted light photography (Murray et al 1969) to stopwatches (Bohannon et al 1997, Oberg et al 1993), electrogoniometer (Oberg et al 1994), footswitches (Maki et al 1997, Shkuratova et al 2004), accelerometers (Auvinet et al 2002, Zijlstra 2004) and camera/video based 3-D motion analysis systems (Judge et al 1996, Graf 2005, McGibbon 1999).

The test environment also appears to be influential in outcomes of studies. There is a tendency for a person’s natural gait to depend on the length of the walkway. People tend to walk faster on a long walkways (Hausdorff et al 1999; Waters et al 1988; Murray et al 1970) and slower on short ones (Canning et al 2006; Oberg et al 1993). In addition to the length of walkway, the acceleration and deceleration walking distances (of varying lengths)-that should be excluded from final analysis- were either provided in some studies (Shkuratova et al 2004, Bohannon et al 1997, Oberg et al 1994 and Zijlstra 2004) or were either not reported or provided (Murray et al 1969, Graf et al 2005, McGibbon et al 1999, Allard et al 1996).

The number of gait trials (tests) analysed per subject also varied with most investigators not reporting if these were consecutive trials or gait cycles e.g. 1 trial (Zijlstra et al 2003), 2 trials (Murray et al 1969, Bohannon et al 1997, Watelain et al 2000), 3 trials (Vardaxis et al 1998, Oberg et al 1994, Shkuratova et al 2004, Graf et al 2005). Some studies properly distinguished the number of trials and limb analysed e.g. 2 right limb trials (Watelain et al 2000) and 3 trials per limb (Allard et al 1996, Judge et al 1996). It has been argued that the number of trials analysed depend on the parameter being measured i.e. spatiotemporal, kinematic or kinetics and the greater the number (average) of trials analysed the lesser the variability in the kinetics observed (Monaghan et al 2007). However, it is also believed that individual trials are more representative and should be interpreted separately for each subjects gait in association to other associated biomechanical events (Davis et al 1997). It is evident from these studies that the number of trials analysed is very variable and the reason (either clinical or practical) for number of trials selected for final analysis is often unknown.

It could be argued that the gait analyses studies were limited by different logistics available in addition to the different study design for the purpose of their investigations. However, the inconsistencies and differences make comparison within populations and between studies a difficult exercise. Further attempts should be made by clinical gait analysts to resolve many of these issues by having a consensus to standardized methods and protocols, analyses and units of measurement especially by those using the similar instruments and subject samples.

This review of the methods employed in the gait studies aim to highlight the limitations regularly involved and ignored in clinical gait analysis that could affect interpretation of the measured usual or normal gait in individuals. These methodological issues are areas to test and validate systematically in future studies. In the present study, a laboratory protocol/set up will be designed that tests and give detailed report on some of the aspects of the methods and

protocol omitted from the other studies: e.g. number of trials analysed per limb and provision of acceleration and deceleration distances. This will further inform clinical judgement on the outcome parameters of normal gait.

2.3 Normal Gait Parameters

Despite the limitations within and between studies, there are general gait patterns or gait descriptions that are observable from gait measurements using particular gait parameters. It is common practice by investigators in clinical gait analysis to describe people's gait often using spatiotemporal parameters such as velocity, stride length cadence and support period because these are readily observable and require minimal instrumentation to measure (Oberg et al 1993, Bohannon et al 1996). These parameters have been observed to be inextricably linked hence changes in one could affect the others in healthy individuals and pathologic cases. The spatiotemporal parameters are key descriptors of the physically demonstrable (or final) features of gait as opposed to the initial causative features of gait such as moment of force across a joint and the power generated such as pull-off and push-off powers. When these parameters are measured within a study they make it possible to differentiate between a normal healthy gait and a pathological deviation from the normal such as in PD. However, between studies variation exist in the reported normal values. Therefore, comparison of normal values may not provide an accurate description of an individual's gait. In this section, the findings from studies on the normative gait values during usual and fast walking speeds will be reviewed for both healthy and people with PD.

To observe the normal values of the gait parameters, most investigators usually test subjects at their preferred walking velocity as this will be naturally self selected (Cunningham et al 1982, Judge 1996). The role of velocity is relevant such that the other spatiotemporal (and even kinetics) are usually considered relative to the gait velocity.

2.3.1 Gait Velocity

It is a common view amongst investigators of gait in the elderly that the elderly walk at a lower speed or velocity in comparison to young adults (Blanke & Hageman 1989, Zijlstra 2004)(Blanke & Hageman 1989;Zijlstra 2004). This may have been reinforced by the observable depreciative effects of aging. Also, possibly by the fact that studies on the effect of age on movement (especially gait), usually compare two groups at the ends of the age profile of the populace without considering the intermediary ages e.g. the advanced aged adults in 60s and 70s versus the young adults in 20s. For example, Zijlstra et al (2004) used accelerometry (vertical and horizontal acceleration signals) to analyse the gait of a mixed gender sample (n=41) consisting of the young (mean age 23 years) and elderly adult (mean age 73.5 years) and recorded a lesser gait velocity in the elderly adults ($1.55\pm0.14\text{m/s}$) than the young adults ($1.80\pm0.15\text{m/s}$). In a similar study that used accelerometry (Auvinet et al 2002), findings on a larger mixed gender sample (n=282) showed that within both the male and female groups the young adults (20-29 years) walked faster than the elderly adults (60-69 years). This research has led to the conclusion that generally, the young subjects walked faster than the elderly.

Other investigators have observed a pattern of change in velocity with advancing age. Oberg et al (1993) attempted to determine the normative data for walking speed of adults of different age groups from 10-79 years old (n= 233). The investigators observed a pattern of decreasing walking velocity that started from the age of 50 years. Bohannon et al (1996) also reported a similar pattern of change in velocity with advancing age. These authors, using similar instrumentation as Oberg et al, also attempted to provide reference values for walking speed for healthy adults aged 20-79 years (n=230). They also provided data that showed decreasing velocity but from the age of 40-60 years (i.e. $1.35\pm0.5\text{m/s}$) compared to young

subjects from age 20 (i.e. $1.39 \pm 15.3 \text{ m/s}$). It is expected that a progressive decline in walking velocity would start to manifest due to physiological changes occurring with increasing age. However, Oberg's observation was even more counter intuitive as the first sign of velocity decline was observed at the fifth decade but males in sixth decade presented with a slightly higher gait velocity ($1.27 \pm 1.2 \text{ m/s}$) than those in the fifth decade ($1.25 \pm 1.8 \text{ m/s}$). However brief this increase in gait velocity was in those that were in their 60s, it may represent a compensative strategy or an attempt to "fight" against slowing effect of age.

In people with PD, reduced gait velocity corresponding to clinical observations have been reported in quantitative studies of PD gait (Ebersbach et al 1999, Frenkel-Toledo et al 2005, Morris et al 2005, Morris et al 1994b). In a study to test the capacity of people with PD to vary their gait velocity, Morris et al (2005) demonstrated that when PD subjects ($n=22$, mean Hoehn&Yahr grade 3) were instructed along with age matched healthy subjects to walk at their preferred natural speed, the gait velocity of the PD subjects was significantly lower (45 m/min) than the velocity of healthy controls (80 m/min).

It is generally accepted that people with PD walk with a slower velocity and it would be expected that the slow gait velocity recorded is invariably their comfortable and preferred velocity in all conditions (Morris et al 1994). However, it appears to change within the same study with different test conditions. Most of the studies that report reduced walking velocity usually test PD gait on walkways ranging from 8-10m length. However, it is seems that the preferred or comfortable walking velocity in people with PD increases on a longer walkway. Canning et al (2006) tested both PD ($n=16$, Hoehn & Yahr grade 2.4 ± 0.5) and age matched healthy subjects on both 14 and 30 metre walkways and found that PD comfortable walking velocity on the longer walkway ($1.52 \pm 0.28 \text{ m}$) exceeded that on the shorter walkway ($1.31 \pm 0.18 \text{ m}$). Although this pattern was also observed for the controls who walked faster

than PD subjects on both walkways, the observation suggest that the capacity of generating a greater magnitude of preferred or comfortable walking speed is preserved without having to walk within a velocity constraint imposed by test methods. It is therefore important for clinical gait analyst and therapist to always consider the capacity of people with PD to improve preferred walking velocity in the absence of any intervention.

2.3.2 Stride Length

Stride length is known to make significant contribution to gait velocity (i.e. $\text{velocity} = \text{stride length} \times \text{cadence}$) (Perry 1992). Therefore, to change a person's gait velocity, stride length is either increased or decreased. In the elderly, stride length is often reported to be significantly lesser than that of young adults (Blanc et al 1999; Shkuratova 2004; Winter et al 1990). Conversely, findings from other investigators suggest this may not always be the case. Cunningham et al (1982) did not observe any significant difference between the stride length of older adults (39-40 years) and younger adults (19-29 years). Likewise Jansen et al (1982) also did not report any difference in stride length between older (60-69 years) and younger adults (20-29 years). It is possible that the lack of significant difference was because both studies "normalised" the stride lengths of their subjects by dividing by their individual heights thus indicating the influence of subjects' height in differences in stride length. Moreover, Cunningham et al (1982) identified that some of their elderly subjects who were involved in regular physical activity may have affected the findings on (i.e. increase) the stride lengths.

In addition to the velocity-stride length relationship, the pattern of change in stride length with advancing age appears to mirror changes in gait velocity. In the study by Oberg et al (1993) on the different age categories, the stride length increased from age 20 years and started to decline by the age of 40 years (50s in women). The study by Bohannon et al (1997)

that reported similar findings on velocity pattern with age could not confirm Oberg's findings on stride length as they did not measure it.

In people with PD, small shuffling steps are often clinically observed that are readily corroborated with objective clinical gait analysis. The studies by Morris et al (1996,1998 & 2005) most prominently highlighted the stride length problem in PD. Other investigators aiming to improve PD stride length have also repeatedly reported stride length at baseline (on-phase of medication at preferred walking speed) to be significantly lesser in PD than healthy elderly control subjects i.e. $0.82\pm0.2\text{m}$ versus $1.27\pm1.2\text{m}$ (Peppe et al 2007) and $1.10\pm0.25\text{m}$ versus $1.42\pm0.18\text{m}$ (Lewis et al 2007). In a study to investigate movement amplitude disorder in PD, Morris and colleagues (2005) tested PD subjects (n=12, score 234 on UPDRS parts 2&3) in their preferred walking speed. They reported a smaller walking stride length in PD ($1.26\pm0.10\text{m}$) compared to that of age matched healthy subjects ($1.46\pm0.08\text{m}$). When PD subjects were asked to match their stride with markers placed on the ground, they were able to do so as well as healthy controls. Morris argued that the inability to generate sufficient stride length in absence of any external indicator was as a result of a central programming mismatch between the selected and actual amplitude of well learned repetitive movement sequence.

2.3.3 Cadence

The rate of stepping i.e. cadence is another function of gait velocity. In order to alter walking velocity, either stride length or cadence or both are altered. The common observation in gait studies of elderly people's cadence is that it remains relatively stable for most part of life (Auvinet et al 2002; Blanc 1999). The reports by Oberg and colleagues (1993) that demonstrated changes in gait velocity and stride length with advancing age using different age categories (20-79 years) did not show any significant change in cadence. This

observation was also corroborated by Stolze et al (2000) in subjects with wider age limits within the elderly (62-72 years) and the young (21-37 years).

Contrary to the findings that cadence remain unchanged in the elderly, evidence of reduced stride length in the elderly suggest that within a known walking distance, more steps could be taken. Menz et al (2003) reported on the gait of elderly subjects that were older (75-85 years) than those studied by the other investigators. It was observed that the elderly walked with a significantly greater cadence of 107 step/min than 103 step/min of the young adults (23-29 years). Although, many of the gait studies report that cadence is often unchanged in the elderly, it can be argued that this observation is maintained only to a certain age, e.g. up to 70 years, after which the declination in gait stride and velocity is compensated with increased step rate.

In people with PD, measures of cadence reported in gait studies have been conflicting. In a series of studies by Morris et al (1994) on the capacity of people with PD to regulate stride length and cadence. The investigators found that whilst PD subjects' stride length was significantly lesser than that of controls, their cadence was increased. Contrarily, in a later study by Morris et al (2005), there was no significant difference in cadence between PD subjects (118 ± 6 steps/min) and healthy elderly controls (124 ± 12 steps/min). The findings by Bowes et al (1990) supported Morris's second observation. Bowes et al tested the effect of the presence or withdrawal of levodopa therapy on PD gait and observed that whilst stride length changed with levodopa levels, cadence remained unaffected. The unchanging characteristic of cadence with different levodopa levels and in the on phase of medication give some credence to previous claims that the temporal feature of gait such as cadence is dopa resistant whilst stride length is dopa sensitive (Blin et al 1991; Blin, Ferrandez & Serratice 1990). However, it is very probable that the different observations made on PD gait

cadence in the on phase of medication is very dependent on whether the subjects did or did not exploit compensatory increase in cadence for reduced gait velocity or stride length.

2.3.4 Single and Double Support Periods

Within the gait cycle, double support period is the duration at which an individual has both feet on the ground (Perry 1992). It occurs either at the beginning or the end of the gait cycle (e.g. ipsilateral heel strike and contralateral preswing). The event after the double support period of the gait cycle is the single support period when only one foot is on the ground (Perry 1992).

As people get older, balance gradually deteriorates and the elderly tend to increase the double support period to increase stability (Kirtley 2005). In the early studies of healthy gait, one of the distinctive features of elderly gait was the greater time spent with both feet on the ground (double support). Murray et al (1969) noted that when the ratio of percentage swing period to percentage stance period during “free speed walking” was examined with increasing age of subjects, there was a decreasing amount of time spent with the foot in swing than in stance-on the ground. Conversely, Winter et al (1990) gave evidence supporting significantly greater single support period (65.5%) in the elderly subjects (mean age 68 years, n=15) than the young subjects (mean age 24, n=12) that had 62.3% stance period. The findings by Judge et al (1996) also revealed that the elderly (76 ± 6 years, n=26) spent lesser percentage period in swing phase ($37 \pm 3\%$) than the young adults (26 ± 6 , n=32) with $40 \pm 2\%$ swing period. Laufer et al (2003) also supported a greater percentage double support period in the elderly than the young in the study. Despite the conflicting findings by Mills et al (2001) and McGibbon et al (1999) who did not record any significant difference in the double support period of the elderly and young, there is more supportive evidence of a tendency to spend more time with the foot on the ground than in air possibly to increase stance stability in the elderly.

People with PD often clinically present with short or shuffling steps such that the feet barely clear the ground (Jones & Playfer 2007). This may have resulted from stride length deterioration as the disease progresses. In addition, postural instability could also have resulted in loss of postural reflexes. Therefore, there will be a tendency to gain balance by increasing double support period compared to healthy age-matched elderly. Greater double support period in PD than healthy controls have been reported by different investigators. Almeida et al (2007) in a study of the effect of dopamine on the gait of PD subjects (n=14) recorded a greater mean double support time (0.31s) than controls (0.28s). Likewise Peppe et al (2007) demonstrated a greater percentage double support period in PD before ($17.9 \pm 3.7\%$) and after rehabilitation ($16.9 \pm 8.2\%$) compared to healthy values ($14.1 \pm 1.8\%$). It can be interpreted from these studies that the PD pathology possibly increases instability coupled with the instability due to advanced age. The resulting effect will be an increased (compensatory) time spent with the feet on the ground for support.

Changes in the spatial (e.g. stride length) and temporal (velocity, support periods and cadence) are often accompanied by changes in the kinematic (joint angular excursion). This was also measured in some studies on normal gait as discussed in the next section.

2.4 Joint angular excursion

The information derived from change in joint position, angular excursion or functional range of motion (ROM) in gait is better understood in relation to the phase of the gait cycle in which it is measured. Some common findings and differences in joint range of motion and excursion measured during self selected, usual or comfortable gait due to factors such as age, gender and velocity of walking have been documented. The difference in joint angle excursion due to age was demonstrated by Judge et al (1996) who found that during natural gait, the older adults in comparison to young adults were observed to have a greater hip flexion at initial contact ($33\pm7^\circ$ versus $30\pm6^\circ$) and a lesser peak hip extension in stance ($-8\pm7^\circ$ versus $-11\pm7^\circ$). The elderly also had a lesser knee ROM, a slightly higher ankle dorsiflexion ($13\pm3^\circ$ versus $12\pm3^\circ$) but a significantly lesser plantarflexion i.e. $-13\pm5^\circ$ versus $-17\pm5^\circ$. This depicted a generally reduced functional range of motion in the elderly with indication of flexed posture at the hip joint. Kerrigan (2003) described this state of joint flexion in the elderly to have resulted from hip flexor contracture that translated to changes at the knee and ankle joints. Mills et al (2001) corroborated limited joint angular excursion owing to an initial joint flexor position or contracture in the elderly. They observed that at initial contact, a greater hip flexion in the elderly than the young adults ($25.4 \pm 0.7^\circ$ versus $22 \pm 1.8^\circ$) and at toe-off (i.e. the end of push-off), a slightly greater ankle angle than that of the young ($-11.6 \pm 1.0^\circ$ versus $-10.0 \pm 2.8^\circ$) although not statistically significant and a greater knee angle ($38.9\pm1.2^\circ$ versus $37.7\pm1.6^\circ$). Thus, indicating a state of greater initial joint flexion position in the elderly.

Changes in joint angular excursion have been observed to be influenced by changes in spatiotemporal gait measures such as the gait velocity. In the two studies cited above (Judge 1996 and Mills 2001), the higher joint angles in the elderly observed by Judge et al (1996) can only be attributed to the singular effect of aging and not the significantly lower gait

velocity and stride length with which the elderly walked. On the other hand, Mills et al's elderly walked with a higher velocity that could explain the higher joint angles. However, increases in joint angles during gait often translate to increase in stride length that in their case, was observed to be significantly reduced. Therefore, the higher gait velocity may have been caused by increased cadence and not as result of increased joint angular excursion that influences the stride length.

A clearer demonstration of the effect of walking velocity on joint angles was given by Kerrigan et al (1998b). These investigators measured the joint angles of young adults (28.5 ± 4.9 yrs) only at comfortable walking speed (1.37 ± 0.17 m/s) and compared it with the joint angles of the elderly (72.7 ± 5.5 yrs) walking at comfortable speed (1.19 ± 0.13 m/s) and then at fast speed (1.55 ± 0.20 m/s). At comfortable speed, the elderly had significantly lower joint range of movement (ROM) at the hip, knee and ankle than the young adults where it was persistently low when they were asked to increase their walking velocity. Persistently low hip extension and plantarflexion angle was also observed by Graf et al (2005) when they compared joint angles during comfortable gait of 25 healthy fairly active elderly (79 ± 6 years) with the joint angle in 27 low performance elderly (76 ± 4 years) during comfortable and fast walking speed.

Regarding PD gait, the studies that have measured changes in joint angles and position in PD gait have observed that the changes, as in healthy subjects, also accompany changes in the spatiotemporal parameters e.g. reduced joint position translates to reduction in stride length. Ferrarin et al (2005) measured the effect of medication and deep brain stimulation on PD gait. They observed that within the on phase of medication, the mean change in joint position was less than that of healthy control subjects at the hip ($39.8 \pm 9.8^\circ$ versus $46.2 \pm 5.5^\circ$), the knee ($47.4 \pm 11.8^\circ$ versus $58.2 \pm 1.3^\circ$) and the ankle ($19.3 \pm 2.8^\circ$ versus $24.5 \pm 4.5^\circ$). This

observation demonstrated that associated with slow walking velocity, there is a reduced joint angular excursion in the gait of people with PD. The reduced joint range of motion have been found to improve with external cueing and deep brain stimulation strategies but ankle plantarflexion range have been observed to be persistently lower than baseline values (Ferrarin 2005, Lewis et al 2000).

2.5 Hip pull-off Power

Joint power is known to vary with gait velocity. Hence, in association with a reduced walking velocity, a reduced joint power will be observed. McGibbon & Krebs's (1999) gave supporting evidence of a reduced hip pull-off power ($2.82 \pm 1.54 \text{ W/m}^2 \text{ kg}$ versus $3.14 \pm 1.45 \text{ W/m}^2 \text{ kg}$) in association with a slower gait velocity ($1.16 \pm 0.21 \text{ m/s}$ versus 1.36 ± 0.12) in elderly women ($n=16$, age 72.4 ± 5 years) when compared to young women ($n=16$, age 27.4 ± 5 years). Vardaxis et al (1998) highlighted the relationship between velocity and joint power further in the study. The authors using cluster analysis to classify the gait parameters of 19 young healthy males (25.3 ± 4.1 years), showed five distinct categories of gait that were mainly differentiated significantly by hip power generations either at heel strike or during pull-off. The fastest walking category of subjects (i.e. at $1.36 \pm 0.06 \text{ m/s}$) generated the greatest mean pull-off power (5.55 W/kg) while the slowest category ($1.15 \pm 0.05 \text{ m/s}$) generated the least power (1.74 W/kg). Although, this supports the association of velocity with joint power especially in the young, this pattern was also observed in the elderly. However, Watelain et al (2000), after gait measurements of a sample of adults consisting of both elderly and the young also used cluster analysis method to identify gait similarities and patterns in their subject samples. In contrast to Vardaxis' findings the authors observed only one category of young subjects but three categories in the elderly group that were identifiable by the hip powers strongly associated with the gait

velocities. Also, the fastest category of elderly subjects walked with comparable velocities and hip pull-off power as the young adults group.

Other investigators of the elderly gait could not find a reduced hip pull-off power with reduced gait velocity. Winter et al (1990) reported no statistical significant difference in the pull-off energy of the slower walking elderly compared to young adults ($0.098 \pm 0.032 \text{ J/kg}$ versus $0.090 \pm 0.027 \text{ J/kg}$). Moreover, Judge and colleagues (1996) in a study of the gait of elderly ($n=26$) and young adult ($n=32$) reported a greater hip flexor (pull-off) power in the elderly than the young adults ($0.92 \pm 0.27 \text{ W/kg}$ versus $0.87 \pm 0.29 \text{ W/kg}$) although the elderly walked at a slower velocity (1.03 ± 0.13 versus $1.16 \pm 0.13 \text{ m/s}$). After adjustment for difference in step length, the elderly had 16% more hip flexor power. The authors believed the high hip flexor power in the elderly compensated for a reduced ankle push-off power observed in the same group of subjects. With these conflicting findings, the questions remains if increased hip pull-off power as a compensatory mechanism is a feature in some or all of elderly gait and why was it not observed (nor exploited) in the elderly subjects of the other studies.

The findings and description of pull-off power in people with PD is very sparse. The only description on pull-off power in PD was by Morris et al (1999) on a 79 year old female subject. These authors demonstrated that when medication level was low, the hip pull-off power was significantly greater than that of healthy values. When tested in the peak (on-phase) medication state, the subject's hip pull-off power was not different from the healthy values. The authors explained that the greater hip pull-off power in the off phase was possibly a compensation for reduced joint power such as at the ankle. The study by Morris et al (1999) was limited to one subject sample with a 10 year history of right hip joint

replacement surgery that the authors claim did not adversely affect her gait whilst the healthy values (n=26) were from another study i.e. Judge et al 1996.

Other studies that measured joint power in PD gait either did not measure or record changes in the hip pull-off power (Lewis et al 2000) or were not clear as to which peak hip power within the gait cycle was being referred to (Ferrarin 2005; Sofuwa et al 2005).

2.6 Ankle push-off power

The common findings related to push-off power show that elderly adults consistently have a lower plantar flexion (push-off) power than the young adults. In the study by Judge et al (1996), elderly people walked with a push-off power that was significantly less than the young adults ($2.9 \pm 0.9 \text{ W/kg}$ versus $3.5 \pm 0.9 \text{ W/kg}$). Findings from other studies have corroborated the features of reduced push-off power in the elderly especially in association with reduced walking velocity: $0.19 \pm 0.05 \text{ J/kg}$ versus $0.29 \pm 0.05 \text{ J/kg}$ (Winter et al 1990), $2.82 \pm 1.54 \text{ W/m}^2 \text{kg}^{-1}$ versus $3.14 \pm 1.45 \text{ W/m}^2 \text{kg}^{-1}$ (McGibbon & Krebs 1999). There are indications that push-off power in the elderly remain less than that of young adults even at comparable walking velocity. In the study by DeVita & Hortobagyi (2000), elderly people were asked to walk at identical speeds ($1.484 \pm 0.11 \text{ m/s}$) as young subjects ($1.481 \pm 0.089 \text{ m/s}$). The authors reported a minimal contribution by the plantarflexors strength and more from hip extensors in midstance and not hip flexors during pull-off.

In people with PD, studies on the kinetics of PD gait are beginning to emerge that described the force generated during (or related to) the push-off event (Ferrarin 2005; Koozekanani 1987; Morris et al 1999; Lewis et al 2000). The common theme from these studies is that there is abnormal force generation during the push-off event. Nieuwboer et al (1997)

analysed footstep pattern using a pedodynograph to measure foot loading in PD patients and healthy elderly people. They observed that PD subjects, compared to healthy controls increased load at the mid foot than the heel (1st peak force) and fore foot (2nd peak force). This meant that they walked more with flat feet with reduced rocker action at the heel during loading response and reduced fore foot roll-off during the push-off event of gait. Such gait pattern is predicted to increase the risk of trips and falls, and may require greater energy expenditure to sustain than healthy people.

The earliest description of the joint power component of PD gait that gives some credence to the observation of force reduction during push-off event was the study by Morris et al (1999). Morris et al described changes in the joint power in the gait of a 71 year old woman at the end of dose phase (total UPDRS score 51) and at peak dose phase of L-dopa medication cycle (total UPDRS score 44) i.e. moderately affected by PD. The authors reported a reduced ankle push-off power in the subject. Unlike hip pull-off power, reduced push-off power was found to be persistent in both end of dose and at peak dose medication walking conditions. The authors did not measure healthy control values but compared their findings with healthy elderly from another study (Judge et al 1996) on healthy elderly (n=26, mean age 79 years). No statistical analysis was possible and the graphical comparison was within one standard deviation of the healthy values. Within two standard deviation, the differences in push-off power may not as such be highlighted. Although the other studies often refer to Morris's study, the limitations within the study require that the findings be reviewed.

Lewis et al (2000) supported the observation of reduced push-off power in the on phase PD gait. The investigators selected four of the 14 PD subjects (Hoehn & Yahr 2,2.5, 3 and 3 respectively) that demonstrated reduced stride length and observed a lesser push-off power generation compared to 14 healthy controls subjects. The basis of selection, however,

appears to be biased as stride length naturally correlates with joint power and reduction in both gait features should have been expected. They did however argue to support their method that consistent trend in gait power was difficult to observe. This raises the question on reduced push-off power as a common gait feature in PD or if PD subjects are similarly affected.

Ferrarin et al (2005) studied the effect of electrical stimulation on the basal ganglia of PD subjects (n=10, off-medication Hoehn & Yahr 3.7 ± 0.7) and observed that inspite of 'supra threshold' dose L-dopa medication and electrical stimulation, the ankle push-off power was persistently less than that recorded for the healthy controls. However, a persistently reduced ankle power generated could have resulted from factors that the investigators did not account for such as the deleterious effect of surgical procedure or electrode insertion into the brain that may affect motor control, also the effect of the initial practice test for the best stimulative voltage was not considered. There also could have been a depreciative effect of immobilisation due to hospital stay. Any of these factors or their combination could have been contributory to the reduced push-off power observed in the PD subjects compared to the healthy controls.

The common theme from these studies on usual or comfortable walking speed in the healthy and people with PD is that often the elderly presents with depreciated gait that is exacerbated with PD pathology. Overall, a distinguishing feature of a slow walking gait is presented. However, significant changes in gait features are observed when subjects aim to increase their walking speed (Kerrigan 1998b). Fast walking in PD has not been investigated as thoroughly as usual/comfortable walking especially in PD. The changes observed in fast walking require consideration in the approach to analysis and rehabilitation of elderly and PD gait and is discussed below.

2.7 Fast Walking in The Healthy and People with PD

The ability to vary walking velocity is a major feature of healthy adult gait. This is usually accomplished by changes in gait components such as cadence or stride length. Changes in the joint angular excursion and underlying kinetics have also been observed with the change in velocity. Kerrigan et al (1998b) studied the effect of aging and walking speed in the elderly (n=31, 72.7 ± 5.5 years). They found that when asked to walk fast, the usual velocity (1.19 ± 0.13 m/s) was significantly increased (1.33 ± 0.14 m/s) although not significantly different from the usual walking velocity of the young adults (1.38 ± 0.11 m/s) thus highlighting that although the ability to walk fast is preserved, the velocity is still influenced by aging. The investigators noted that older people were able to increase velocity mainly by increasing their stride length (1.20 ± 0.12 m to 1.33 ± 0.14 m). Although, they did not measure angular excursion, they recorded increments in peak joint position i.e. peak hip flexion ($26.1 \pm 4.6^\circ$ versus $29.6 \pm 5.1^\circ$), knee flexion ($60.1 \pm 4.7^\circ$ versus $57.9 \pm 4.6^\circ$), ankle plantarflexion (-15.6 ± 6.3 versus -16.3 ± 5.5) °. The joint powers were also observed to have increased: Hip pull-off power (-0.90 ± 0.22 to 1.51 ± 0.52 W/kg), knee power absorbed at preswing (-1.25 ± 0.33 to -2 ± 0.57 W/kg) and ankle push-off power (1.70 ± 0.23 to 1.88 ± 0.33 W/kg). Graf et al (2005) gave supportive evidence for the increment in the gait parameters with fast walking in the elderly. The authors demonstrated that when elderly subjects (with low physical performance) were asked to walk as fast as possible, there was a 23% significant increase in plantarflexion power (2.13 ± 0.58 to 2.59 ± 0.7 W/kg) and 67% significant increase hip flexion power in preswing i.e. 0.83 ± 0.39 to 1.39 ± 0.58 W/kg. These were associated with significant increments in joint angular excursion at the hip during pull-off and at the ankle during push-off events in addition to significant increments in step length (0.58 ± 8.2 to 0.64 ± 0.11 m) and cadence (105 ± 8 to 127 ± 15 steps/min).

Contrary to Kerrigan's and Graf's findings, Judge et al (1996) did not observe any significant change in plantarflexion power (i.e. 3.1 ± 1.2 to 3.2 ± 1.5 W/kg) in elderly subjects ($n=5$) who were asked to walk at maximal pace. However, increments in joint powers were found in the hip flexor power at preswing i.e. by 72% (1.1 ± 0.30 to 1.9 ± 1.0 W/kg) and knee absorption by 36% (-0.70 ± 0.03 W/kg). The associated velocity increased by 26% (1.12 ± 0.17 to 1.42 ± 0.36 m/s), cadence by 15% (117 ± 7 to 134 ± 9 step/min) and step length by 10% (0.67 ± 0.10 to 0.74 ± 0.09 m). Reduced plantarflexion power observed by Judge et al probably indicated that the healthy elderly mainly increased walking velocity by increased hip flexor power.

In people with PD, the ability to walk fast appears to be preserved (Canning et al 2006). This has usually been demonstrated in intervention studies that facilitated PD gait bradykinesia by the use of external speed control measures (e.g. treadmill) or cueing strategies (auditory, visual or verbal) (Lewis et al 2000, Nieuwboer et al). In a study on incremental speed-dependent training on PD subjects ($n=21$, age 71.8 ± 6.4 years, mean score 18 on motor UPDRS and 3 on Hoehn and Yahr scale), Cakit et al (2007) demonstrated that in the on phase of medication the PD subjects were able to tolerate a maximum speed of 1.9 ± 0.75 km/h which improved after exercise training to 2.61 ± 0.77 km/h.

In response to verbal instruction for people with PD to walk as quickly as possible, Camicioli and colleagues (2006) showed that the subjects ($n=29$, 83.6 ± 8.5 years, mean score 8 on motor UPDRS) were able to increase gait velocity (0.8 ± 0.2 to 1.06 ± 0.3 m/s) with increment in cadence (103 ± 16 to 127 ± 24 step/min) but no significant increase in stride length (0.93 ± 0.16 to 0.99 ± 0.17 m). This suggests that for PD subjects to increase velocity there was greater (or preferable) increase in step rate than stride length. Conversely, Behrman et al (1998) demonstrated that PD subjects could increase step length more than cadence. The PD

subjects ($n=8$, 72.9 ± 4.7 years old, Hoehn and Yahr stage 2-4) were given verbal instruction to walk fast, their gait velocity was increased by 65% i.e. 0.77 ± 0.23 to 1.27 ± 0.38 m, cadence by 30% i.e. 117 ± 29 to 153 ± 46 step/min and step length by 48% i.e. 0.85 ± 0.27 to 1.08 ± 0.41 m). With these conflicting findings, there is no clear evidence to suggest that PD subjects will either increase velocity more by preferred increase in stride length than cadence or vice versa .

Most of the studies on fast walking in the on phase of PD medication report on the spatiotemporal variables. Conversely, there is sparse evidence on the changes in joint power at preferred fast walking speeds. At usual walking speed, healthy adult joint power generated when walking is known to be associated with gait velocity (Lelas et al. 2003) whilst in PD gait reduced ankle push-off power (and probably hip pull-off power) was associated with reduced walking velocity. It is possible that the reduced joint power in PD gait is a result of their preferred walking speed (which lesser than that of healthy people) and that at preferred fast walking speed, the ankle push-off power will be normalised. The possibility of this will obviate the use of external cues that demand attention in gait training, and also inform rehabilitation of PD gait with respect to power and propulsion. Therefore changes in the joint power during preferred increase in walking speed will be investigated in this present study.

2.8 Summary of findings

Healthy gait studies have demonstrated that the greater the difference between the young and the old (e.g. 20 yrs versus 80yrs) the lesser the walking velocity of the elderly (Zijlstra et al 2004). With increasing age (approximately between 20 and 50 years), features of gait such as velocity and stride length show tendency to increase then starts to decline after this age range whilst cadence remains stable for most part of life (Oberg et al 1993). Conversely, double support period increases with advancing age mainly due to increasing instability (Kirtley 2005, Laufer 2003). Change in lower limb joint position during gait decreases with advancing age. In the elderly, as gait velocity increases, the joint angular excursion also increases but changes in ankle joint position remains persistently low (Mills et al 2001, Kerrigan et al 2003). In PD gait, there is an exacerbation of the gait features observed in age matched elderly people. The gait velocity and stride length are usually observed to be lesser in association to increase in single and double support periods whilst changes in cadence is unclear if it either increases or decreases.

In the measure of joint power during gait of healthy adults, the findings (some conflicting) indicate that the magnitude of hip pull-off and ankle push-off powers often vary with gait velocity and between studies (McGibbon & Krebs 1999). Reduced ankle push-off power is often compensated by increase in hip pull-off power (Judge et al 1996). Conversely, this compensation may not occur because it was also observed that hip pull-off power remain unaltered when ankle push-off power is low (Graf 2005). In PD gait studies, findings indicate that ankle push-off power is reduced but there is insufficient supportive evidence for this claim whilst evidence on changes in hip pull-off power is lacking.

Previous studies on healthy gait have shown an association between gait speed and joint power (Lelas et al 2003). The question is asked if a reduced pull or push-off power (if

observed) can be improved to levels comparable to healthy values when people with PD are asked to walk fast. Evidence is lacking on changes on joint power especially pull and push-off powers when people with PD walk at their preferred fast speed.

2.9 Gaps in Studies

Some of the gaps identified in the findings of the studies on normal gait in the healthy elderly and PD gait are:

- The measures of kinetic and associated kinematic (joint angular excursion) gait events at the three major lower limb joints during push-off and pull-off phases are missing from previous healthy gait and in PD gait studies. Furthermore, the information on hip pull-off event is scarce.
- The relationship between ankle push-off power, hip pull-off power and spatiotemporal factors such as velocity is not clear in studies on healthy elderly and PD gait
- Unlike studies that attempt to provide a database reference on spatiotemporal and kinematics, there is no power database for different age groups in the healthy individuals. Likewise there is a lack of longitudinal data on PD gait with disease progression.
- Some studies have indicated that gait in healthy people can be categorised according to speed and joint power (Watelain 2000, Vardaxis 1998). It may be possible to describe the healthy elderly and individuals with PD according to groups with similar kinetic (joint power) component type/pattern. There is insufficient

evidence to corroborate the existence of different joint power category in the healthy and PD population.

- In studies on fast walking in PD, there is insufficient evidence on how gait velocity is increased (predominantly by cadence or stride length?). It is also not known what the pull and push-off power changes will be when subjects walk at their preferred fast velocity. Will the hip pull-off and ankle push-off increase to normal values?

To address some of these gaps and answer the questions, the aims of the present study was

(1) To first design a laboratory protocol for analysis of healthy elderly gait that can be used later for the second study on PD gait and

(2) To characterise and describe hip pull-off and ankle push-off in the gait of the healthy elderly and people with PD. These will be by:

- Providing gait data on healthy elderly and people with PD within the age category of 50-80years.
- Measuring the associated spatiotemporal and kinematic variables especially changes in joint angles during hip pull-off and ankle push-off.
- Describing changes in voluntary fast walking with associated pull-off, push-off powers angular excursion and stride length and cadence.

As the first aim of the present study was to design a laboratory protocol/set up that would be used for the second aim (to measure, collect and analyse gait data in both groups of subjects), the following chapters will be presented in the following order:

- Chapter 3: Healthy Pilot study
- Chapter 4: Healthy gait study: Methods, results and discussion

- Chapter 5: PD Pilot study
- Chapter 6: PD gait study: Methods, results and discussion
- Chapter 7: Final discussion

The two pilot studies were necessary because the first pilot study was used to test the designed laboratory protocol/ set up before it was used in the healthy elderly study. The limitations and issues that arose with the use of the protocol in the healthy study were reviewed and tested in the second pilot study before being implemented in the PD study.

Chapter Three

Pilot Study

Gait analysis of Healthy Adult Gait

3.1 Introduction

The present investigative study was designed to be carried out at the Rehabilitation research gait laboratory located at the Southampton General Hospital. Before embarking on the main investigative study, the investigator familiarised himself with the use of the gait laboratory techniques and set up (equipment and materials), and the laboratory protocol for subject set up and gait measurement and analysis. The protocol for measurement involved subjects walking and stepping on a force platform (Kistler Instrument Ltd, Switzerland) with either foot without having to aim for it. Subjects would not be informed about the platform but to walk along a walkway with motion analysis cameras on either side. The feasibility of carrying out this procedure, the number of trials that could be acquired with the force plate data, within an arbitrary number of (i.e. 10-12) trials, and the duration of the gait analysis session were to be tested and determined. This test was also to identify any procedural problems that could be considered and addressed before the main study was conducted. The specific issues that needed to be addressed are discussed below.

In the laboratory protocol, the procedure of setting up of participants involved the placement of light emitting diodes (LED) markers on anatomical landmarks of the lower limb (fig 3.2.1). These markers are tracked by Coda mpx30 cameras (Charnwood Dynamics, Leicestershire, England) and their trajectories are used for lower limb 3-D segmental analysis. However, previous studies using motion analysis systems such Coda mpx30 have attributed poor reproducibility of gait data to inaccurate placement of markers on surface anatomical landmarks (Kadaba et al. 1989;Maynard et al. 2003). Therefore it is important in the present study to establish the reliability of placement of the markers correctly by the same examiner especially on different day intervals.

Furthermore, the raw data and signals collected from a laboratory session are meaningful when it is normalised to gait cycle events observed when walking. Normalization also enables the Coda gait report generator software to calculate the spatial and temporal variables (stride time, speed, cadence etc). The essential gait cycle events required for normalization are the initial contact (heel strike) and toe-off and subsequent heel strike periods of the selected data profile of each limb. Heel strike and toe-off is automatically marked by Coda on the limb that strikes the force plate. However, the subsequent heel strike (that completed the gait cycle) and the cycle of the other limb (that did not strike the force plate) have to be manually marked. Manual marking is done (post real-time) by visual inspection of the walking 3-D stick image of a subject. Cursors are placed and marked on the graph profiles that coincide with when the heel touches the ground (heel strike) and when the foot is off the ground (toe-off) and the next heel strike. This is required for all gait cycles selected for analysis. Therefore, the ability to carry out this procedure repeatedly with consistency needs to be established in a repeatability test. Hence, a pilot study was designed to address the above issues with the following aims:

1. To test out and familiarise the researcher with laboratory protocol and set up and to solve procedural problems that may arise before the main study.
2. To establish the reliability of placement of the markers correctly by the same examiner .i.e. the test retest agreement.
3. To test for reliability of manually defining the gait cycle events (heel strike and toe off).

3.2 Method

3.2.1 Subjects

Four healthy adults (subjects 1-4) were recruited for the pilot study. Two of the subjects (subjects 1-2) were used to test the research laboratory protocol/set up and the reliability manually marking the gait cycle events. The other two (subjects 3 and 4) were tested on the reliability of marker placement. The subjects were recruited from the population of the community of the University of Southampton. They were approached and informed of the purpose of the pilot study and their written and signed informed consent was received. The subjects were eligible to participate if they could walk unaided and were able to come to gait analysis laboratory at the Southampton General Hospital. They could not be included if they had any postural or gait deficiency or presence of any condition that affected the ability to walk such as musculoskeletal disorder, spinal or lower limb pain or fracture.

3.2.2 Equipment

The equipment used was located in the gait analysis laboratory of Southampton General Hospital (SGH). They consisted of two 3-D motion analysis i.e. CODA mpx30 (Charnwood Dynamics, Leicestershire, England) scanner units, 22 battery powered light emitting diode markers and a Kistler force platform situated midway along an eight metre walkway. These are further described below.

3.2.2a Coda mpx 30 and LED Markers

Cartesian Optoelectronic Dynamic Anthropometer i.e. Coda is a camera based motion analysis instrument that able to record different variables of movement e.g. positions in space, distances and velocities in linear and angular directions). Coda equipment consists of two scanner units each comprising of three cameras (fig 3.1a). The scanner units were

mounted on the walls opposite each other on either side of a walkway and are separated by a distance of 7.5m. The scanner units are able to track and record pulsed signals sent from 22 light emitting markers (fig 3.1b) powered by batteries (fig 3.1c) placed at specific location on a person's lower limb. The tracking rate (or frequency) of the scanner as a person walks increases with fewer number of markers on the subject. For 22 markers, the manufacturer recommended (sampling) rate is 200Hz that sufficiently tracks and capture all marker movements within the camera view. For the sampling rate, the recommended filtering rates (to smooth out data and limit artefacts) is 50, 20 and 50 Hz in the X, Y and Z coordinates of movements respectively.

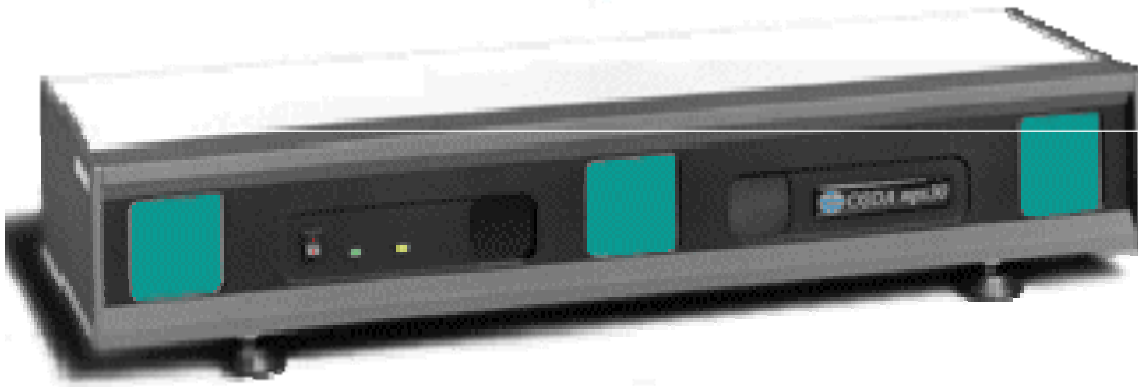
3.2.2b The Walkway and Force Platform

On the eight metre walkway the data capture area by the CODA cameras was approximately 3m (1.25m before and after a force platform). The force platform is a 60cm by 40cm aluminium Kistler force platform (Kistler Instruments Ltd, Switzerland). It was situated midway along and level with the walkway and is integrated and synchronised with CODA mpx30. Within the force platform are four piezoelectric force transducers corresponding with the four corners of the platform. When a person steps on the force platform, the ground reaction force vector are converted to electrical signals relating to their magnitude, direction and line of action across the lower limb to floor. These signals are collected as force data generated by the subject as the foot falls on the force platform.

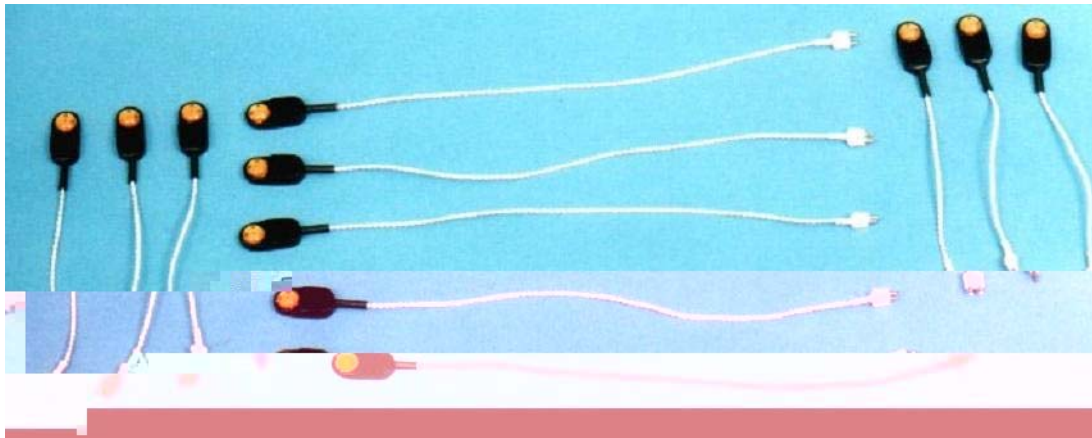
3.2.2c Anthropometric measurements

A height measure and weighing scales were used to measure barefooted height and weight of the subject. A tape measure was used to mark off specific location for LED markers. An alignment frame and T- bar were tools used after placing markers on specific anatomical

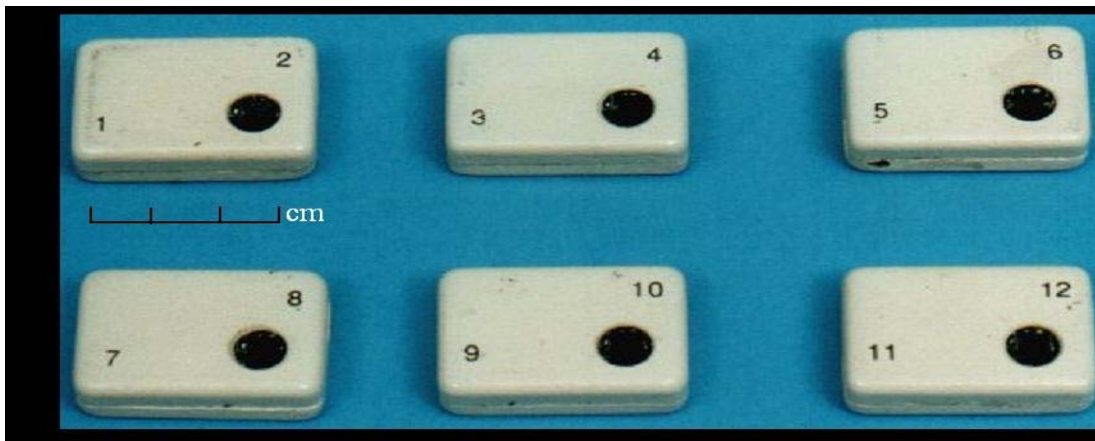
location. They were used to align the thigh and shin marker frames to lie perpendicular to the knee and ankle joint axis respectively. Velcro straps and sticky tapes were used as fasteners for markers, marker frames and marker battery boxes.



a)



b)



c)

Fig. 3.1. a) CODA scanner camera, b) LED Markers, c) Rechargeable battery boxes,

3.3 Aim 1: Testing the Laboratory Protocol/Set up

Data collection for each subject took place in one session in the gait analysis laboratory at the Southampton General Hospital. A record was taken of the age (years) and sex, height (m), weight (kg), width of ankle and knee, and pelvic depth and width. Subjects were required to wear shorts and be barefooted.

Twenty-two makers connected to designated battery boxes were placed bilaterally on the limbs directly on the skin surface and in alignment with frames that were strapped to the pelvis, thigh and leg segments. These markers were placed in alignment with specific anatomical locations on the lower limb (fig. 3.2.1) for tracking and segmental analysis of the lower limb joints and segments by Coda. Subjects were informed only about the necessity of the camera view of their walking and to ignore any floor markings (i.e. corresponding with the force platform). A practice trial of walking along the walkway was carried out. The preferred foot to initiate walking was noted and confirmed with the subject.

The specific instruction given to them was “starting with your left/right foot, walk as you usually do, whenever you are ready” (instead of being commanded or cued to do so). The subjects then walked barefooted in a straight line from one end of the walkway to the other (Fig. 3.2.2). At both ends of the walkway a laboratory assistant stood behind the subject and adjusted the start point if the foot did not wholly strike the force platform.

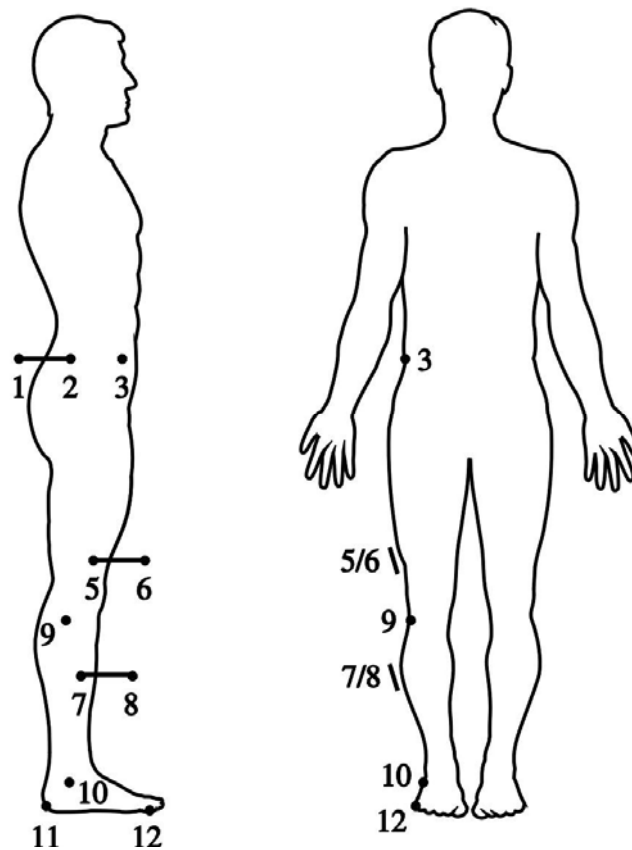


Fig. 3.2.1. Anatomical landmarks for marker placement on the lower limb.

1, 2, 3 = pelvic frame (sacral wand, posterior superior iliac spines and anterior superior iliac spines), 5, 6 = thigh wand (anterior femur, posterior femur), 9 = knee joint line, 7, 8 = shank wands (anterior tibia and posterior tibia), and 10, 11, 12 = lateral sides of the ankle, heel and fifth metatarsal.



Fig 3.2.2 A gait analysis trial with subject walking with markers set up.

All markers on a subject come into full view of the cameras at approximately 1m before and after the force platform. To allow for acceleration and deceleration distances when walking, the start point for walking was set at 2.5m away from the force platform on both sides. Hence, the gait cycle across the force platform would be approximately steady walking velocity. For each subject, an arbitrary number of ten trials were aimed to be conducted in a laboratory session. A gait trial was completed when the subject walked from one end of the walkway to the other.

3.3.1 Results

The gait sessions were completed within 75 minutes for each subject. The CODA and force platform equipment were fully functional and effective throughout both sessions of gait measurement.

In the first session for subject 1), 12 trials were recorded with three force plate data captured in trials 3, 5 and 8 (two trials recorded for the left leg, one for the right).

The second session (subject 2) also had 10 trials carried out of which six force plate data were captured i.e. trials 5-10 (alternate left and right legs force data) (Table 3.2.1).

Subject	Number of trials	Number + laterality of foot strike	Total Time spent in gait analysis session (minutes)
1	12	3L, 5L, 8R	75
2	10	5L, 6R, 7L, 8R, 9L, 10R	75

(3L =left foot struck force platform in trial 3; 8R = right foot struck force platform in trial 8)

Table 3.2.1. Number of trials for pilot subjects

Subjects did not report any discomfort or distress before during and after the gait sessions. However, at the end of the session, the second subject had stated that he had a different feel to walking barefooted in the laboratory although he believed this did not affect his style of gait.

After the gait sessions, the data acquired were stored in CODA filing system for extraction and analysis. All gait data files were screened after the laboratory session for any error or artifact. Only one was found and this was that the right and left anterior superior iliac spine (ASIS) markers had been misplaced for each other on the pelvic frame of the second

subject. Thus, the ASIS disconnection with the posterior superior iliac spine (PSIS) markers was easily detected on the pelvic model. The Coda function to reassign, reconnect and reanalyse the marker profile was used.

3.3.2 Summary

The instruction given to the subjects to walk was readily understood and complied with by the subjects. Without having to aim for the force platform the gait of the subjects can be considered normal. Although, some gait trials were without force data, it was demonstrated that within 10 trials, force platform data could be acquired for both left and right limbs in 3-6 trials. Therefore, in the main study, the target minimum number of gait trials for each subject will be 10 whilst a target maximum number of six trials with force platform data (three per limb) will be selected for analysis. This controls for the number of trials the subjects perform and the duration of the laboratory session.

A pelvic marker that distorted the data generated by the second subject was consequently corrected by re-designation of the markers in the CODA software program. The program repositions the marker and re analyses the gait profiles. This is possible after data collection for any specific marker to represent specific body anatomical landmarks.

Barefoot gait analysis is the option of preference for this study design despite the second subject's different feel to barefoot walking. This is because marker placement for gait analysis using CODA mpx 30 is designed (by default) to analyse barefoot gait. Barefoot gait protocol is a good controller across all subjects participating in the gait analysis. Moreover, other studies have observed that subjects walk slower when barefooted than with footwear (Arnadottir & Mercer 2000). Others have reported no clinically significant difference with or without footwear (Lord & Bashford 1996;Oeffinger et al. 1999). Hence, the wearing of

footwear in gait analysis is left to the discretion of the researcher (Whittle 2006). Therefore, this present study will use barefoot walking in its procedure.

3.4 Aim 2: Marking (Test-retest) Heel strike/ Toe-off Events

Marking the time of heel strike and toe-off points of the gait cycle was carried out on eleven gait trial data recorded for subject 1 and the 10 gait trials for subject 2. From the gait data output, the gait cycle of the subject's 3-D image (i.e. heel strike, to toe-off to next heel strike) occurring in the middle of the walkway was selected and the period of occurrence of the heel strikes and toe-off event were noted with a cursor and recorded. This procedure was repeated after a 10-day interval. The degree of repeatability of manually marking heel strike and toe-off was estimated by analysing the difference between the heel strikes (or toe-offs) on day 1 and day 10. This process of analysis was described by Bland and Altman (1986). The difference between repeated tests were plotted against the mean of the two tests and graphically displayed. The smaller the mean of difference between tests, the better the repeatability of the test.

In the graphs, the differences between tests (data points on graph) are expected to be distributed around the zero limit and mostly within the 95% limits of agreement. The 95% limits of agreement (upper and lower) were calculated using the mean \pm (2 times standard deviations).

3.4.1 Results

Test-Retest of Heel Strike Time

The table below shows the result of marking heel strike time in the gait cycles of subjects 1 (11 trials) and subject 2 (10 trials) on two occasions with a 10 day interval. The mean difference in the marked time for both subjects (-0.0039 and 0.0016 respectively) was not greater than the upper or lower limits of agreement. i.e. 95% of the data points (differences in heel strike times) were within the limits of agreement. The marked heel strike times were replicated (zero difference) in trials 3, 8 and 11 for subject 1 and in trials 1,2,4,8 in subject 2.

Subjects 1						Subject 2			
Heel strike times (sec)						Heel strike times (sec)			
Trial	Day 1	Day 10	Mean	Differences		Day 1	Day 10	Mean	Difference
1	1.508	1.517	1.5125	-0.009		0.923	0.923	0.923	0
2	2.995	3	2.9975	-0.005		0.626	0.626	0.626	0
3	2.184	2.184	2.184	0		1.378	1.374	1.376	0.004
4	1.39	1.4	1.395	-0.01		0.852	0.852	0.852	0
5	1.69	1.695	1.693	-0.005		1.449	1.445	1.450	0.004
6	1.926	1.93	1.928	-0.004		0.977	0.973	0.975	0.004
7	1.461	1.457	1.459	0.004		1.228	1.232	1.23	-0.004
8	1.89	1.89	1.89	0		1.044	1.044	1.044	0
9	1.466	1.47	1.468	-0.004		1.457	1.453	1.455	0.004
10	0.86	0.87	0.865	-0.01		0.994	0.99	0.992	0.004
11	1.34	1.34	1.34	0					
Mean of differences									0.0016
95% Upper limit of agreement									0.0072
95% Lower limit of agreement									-0.004

Table 3.3.1. Heel strike time marked on days 1 and 10 for subjects 1 and 2.

The differences between the marked time are graphically presented in fig 3.3.1 and fig 3.3.2. For both subjects all the data point (differences in marked time) were within the 95% limits of agreement i.e. 0.0053 and -0.0131 (fig 3.3.1), and 0.0072 and -0.004 (fig 3.3.2).

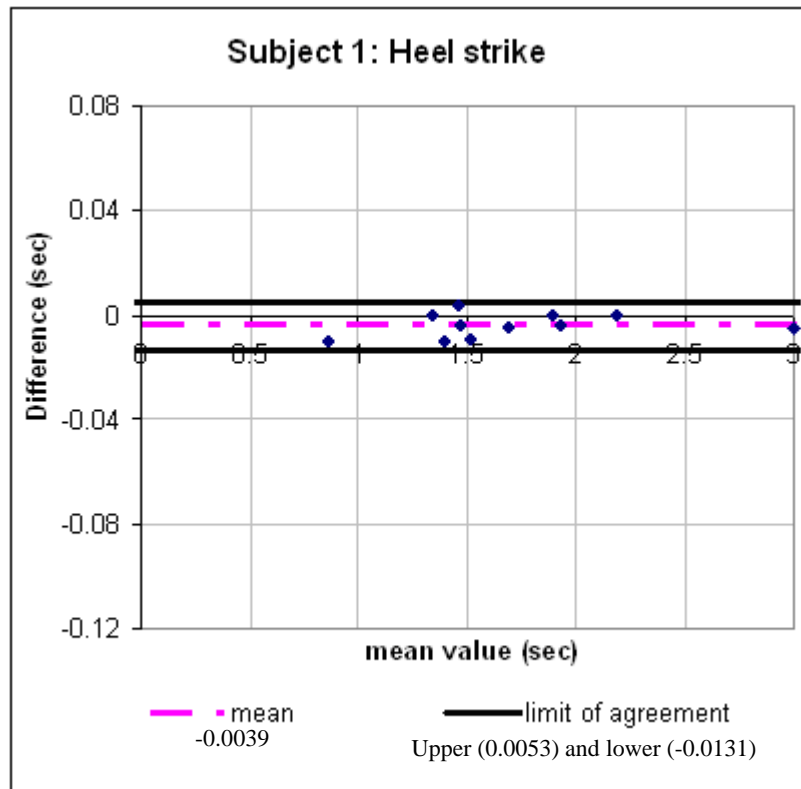


Fig 3.3.1 Graph plot of marked heel strike time for Subject 1. Mean of difference between test 1 and 2 = -0.0039, st dev = 0.0046, upper limit = 0.0053, lower limit = -0.0131. All data points were within limits of agreement (thick lines).

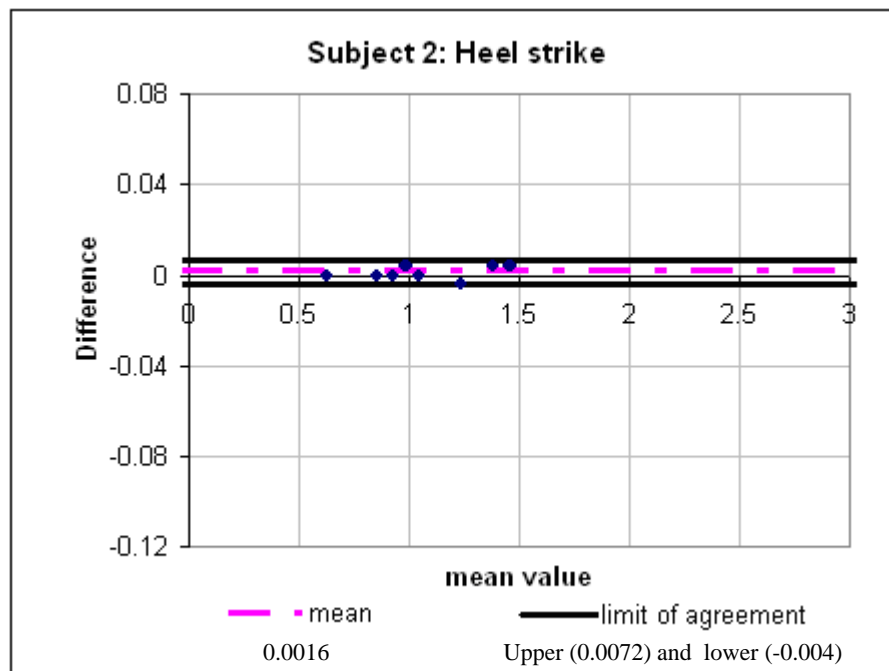


Fig 3.3.2 Graph plot of marked heel strike time for Subject 2. Mean of difference between test 1 and 2 = 0.0016, st dev = 0.0028, upper limit = 0.0072, lower limit = -0.004. All data points were within limits of agreement (thick lines).

Test-Retest of Toe-off Time

Table 3.3.2 shows the toe-off times marked for subjects 1 and 2 and on days 1 and 10. The mean of the differences in marked time indicate that at least 95% of the data points were within the upper/lower limits of agreement in both subjects. Subject 1 had one data point outlying the limits of agreement i.e. -0.1sec in trial 11. In subject 2, the marked toe-off times were replicated in trials 1,3,6,7,9 and 10.

Subjects 1						Subject 2			
Toe-off time (secs)						Toe-off time (secs)			
Trial	Day 1	Day 10	Mean	Difference		Day 1	Day 10	Mean	Difference
1	2.258	2.267	2.262	-0.009		1.62	1.62	1.62	0
2	3.72	3.71	3.715	0.01		1.29	1.282	1.286	0.008
3	2.923	2.906	2.914	0.017		2.067	2.067	2.067	0
4	2.13	2.14	2.135	-0.01		1.503	1.499	1.501	0.004
5	2.455	2.434	2.444	0.021		2.121	2.117	2.119	0.004
6	2.641	2.65	2.645	-0.009		1.653	1.653	1.653	0
7	2.154	2.15	2.152	0.004		1.942	1.942	1.942	0
8	2.583	2.57	2.576	0.013		1.716	1.72	1.718	-0.004
9	2.173	2.16	2.166	0.013		2.167	2.167	2.167	0
10	1.53	1.55	1.54	-0.02		1.658	1.658	1.658	0
11	2.0	2.1	2.05	-0.1					
Mean of differences									0.0012
95% Upper limit of agreement									0.0078
95% Lower limit of agreement									-0.0054

Table 3.3.2. Toe-off time marked on days 1 and 10 for subjects 1 and 2

The differences in the marked toe-off times for each gait cycle were plotted against the mean of the toe-off times. The graph plots (fig 3.3.3 and 3.3.4) for subjects 1 and 2 show that all (95%) of the plotted differences/mean were within limits of agreement for both subject with one outlier for subject 1.

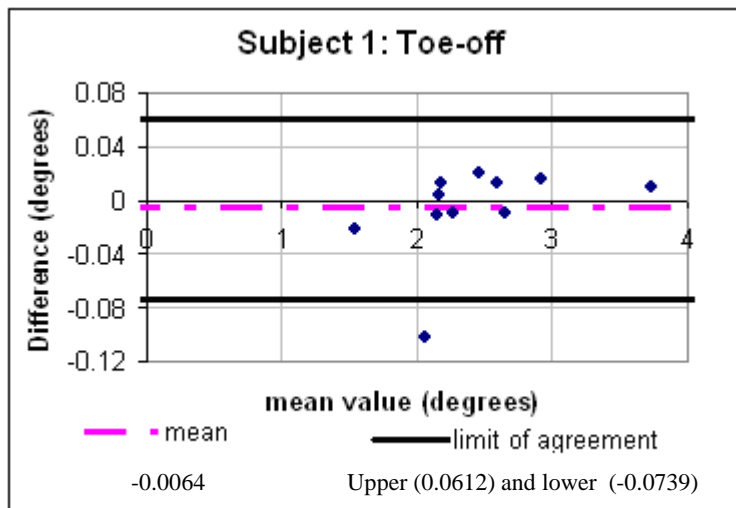


Fig 3.3.3. Toe-off Test-retest for Subject 1. Mean of difference between test 1 and 2 = -0.0064, st dev = 0.0338, upper limit = 0.0612, lower limit = -0.0739. All the data points (except one) were within the limits of agreement with one outlier i.e. -0.1 in trial 11

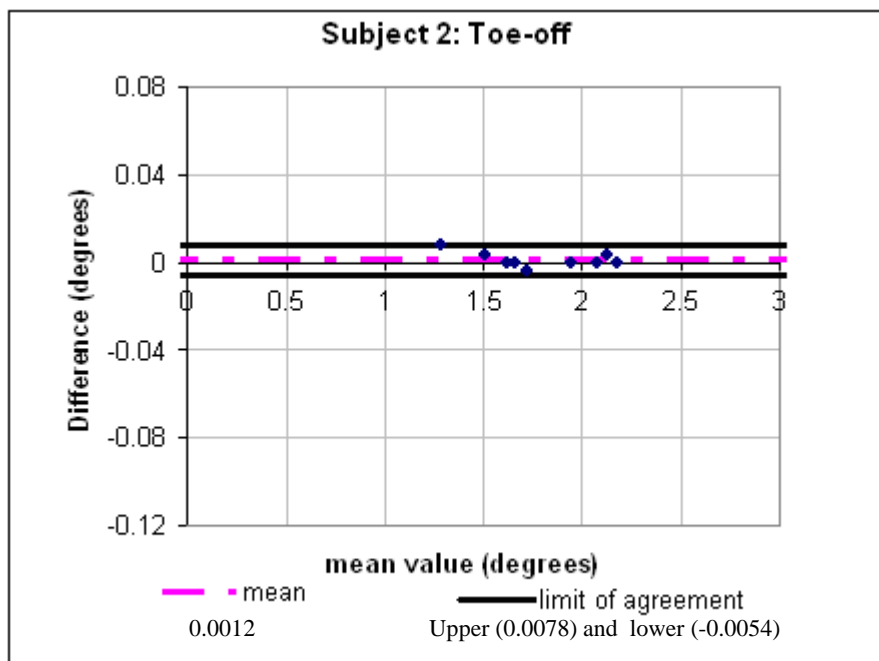


Fig 3.3.4. Toe-off Test-retest for Subject 2. Mean = 0.0012, st dev= 0.0033, upper limit = 0.0078, lower limit = -0.0054.

3.4.2 Summary

The marked heel strike times in both subjects were all within the limits of agreement and can therefore be considered as reliable measures. The lower mean difference in marked heel strike time for subject 2 (0.0016) than subject 1 (-0.0039) and the lower range of limit of agreement in subject 2 i.e. 0.0072 to -0.004 than that of subjects 1 i.e. 0.0052 to -0.013 indicated better repeatability in the values and better reliability of the tester in using the procedure again on subject 2.

In marking off toe-off times, the mean difference for subject 2 was, as observed for heel strike times, lower (0.0012) than that of subject 1 (0.0064) this was accompanied by a lower limit of agreement in subject 2 (0.0078 to -0.0054) than subject 1 (0.06 to -0.07). These values support an improvement in identifying and marking the time of the toe-off event with the second subject.

All data points for the mean differences were within limits of agreement apart from a data point for subject 1 that was outside the lower limit of agreement. In addition, the range of limit of agreement was relatively greater in toe-off than for heel strike in subject 1 and slightly lower in subject 2. These indicated that the marking of toe-off times was not as precise as marking heel strike time. However, with 95% of the differences data points within acceptable limits of agreement and with the improvement in marking the toe-off time on the second subjects, the effect on variability of gait data (step time, single/double support and cadence) can be considered negligible. Thus, the result of this test- retest of marking gait cycle event of heel strike and toe-off demonstrated that the procedure can be repeated in the present study with good reliability.

3.5 Aim 3: Test-retest of Marker Placement

In the gait analysis laboratory, LED markers were placed in alignment with anatomical landmarks according to marker placement protocol (fig 3.2.1). Subjects were then asked to stand still and erect at a specific marked location on the walkway midway between the two Coda scanner units facing the longitudinal direction of the eight metre walkway. A static 3-D data acquisition was then performed for a period of five seconds. This procedure was repeated 10 days afterwards for subject 3, and at 15 weeks for subject 4. Ten data points representing specific markers selected for the test were the ones placed on the left and right lower limbs in alignment with the heel, toe, ankle, knee and the PSIS.

The degree of repeatability of placement of markers was estimated by analysing the difference or change in the position of the limb markers (i.e. their vertical distance from the ground) on two different days (seven or more days apart). To corroborate this, the change in the relative distance of the markers to each other was also examined. This process of analyses of differences in a particular test was as described by Bland and Altman (1986). The difference between repeated tests were plotted against the mean of the two tests and graphically displayed. The more the mean of difference between tests approximates zero, the better the repeatability of the test. In the graphs, the differences between tests (data points on graph) are expected to be distributed around the zero limit and mostly within 95% limits of agreement. The 95% limits of agreement (upper and lower) were calculated using the mean \pm (2 times standard deviations).

3.5.1 Results

Marker Positions

Marker placement was tested on subject 3 on days 1 and 10 and on subject 4 on days 1 and 100 (15th week). Table 3.41 shows the vertical axis positions of the 10 markers on the two different days. The ten markers correspond to the (1) posterior superior iliac spine, (2) the knee joint, (3) the ankle joint, (4) the heel and (5) the toe of the left side (1-6) and right side (6-10). Subject 3 had a lesser mean difference in marker position i.e. 0.05cm than subject 4 i.e. 0.12cm. However, the mean differences in marker position for both subjects on the two different days were within the limits of agreement i.e. 1.07 to -0.97 and 0.99 to -0.75.

Marker positions (cm)									
Marker Type	Subject 3					Subject 4			
	Day 1	Day 10	Mean	Difference		Day 1	Day 100	Mean	Difference
1	106.12	105.68	105.90	0.44		103.46	103.67	103.56	-0.20
2	51.31	51.32	51.32	-0.01		49.20	49.42	49.31	-0.21
3	6.61	6.77	6.69	-0.16		4.49	5.15	4.82	-0.66
4	1.02	0.98	1.00	0.03		1.77	0.87	1.32	0.89
5	0.84	1.40	1.12	-0.56		1.28	1.10	1.19	0.18
6	106.02	104.80	105.41	1.23		104.84	104.83	104.84	0.007
7	50.27	50.66	50.46	-0.38		49.56	49.26	49.41	0.29
8	5.56	5.83	5.69	-0.27		5.43	5.19	5.31	0.23
9	1.89	1.61	1.75	0.28		0.62	0.53	0.57	0.08
10	0.99	1.09	1.044	-0.10		1.11	0.50	0.81	0.62
		Mean difference		0.05					0.12
		Upper limit		1.07					0.99
		Lower limit		-0.97					-0.75

Table 3.4.1 Marker positions on two days for subjects 3 and 4.

The graph plot of the mean marker positions against the differences in position on the two days showed all the data point were within the limits of agreement for both subjects except for one outlier in subject 3 i.e. placement of the right posterior superior iliac spine marker (figs 3.4.1 and 3.4.2).

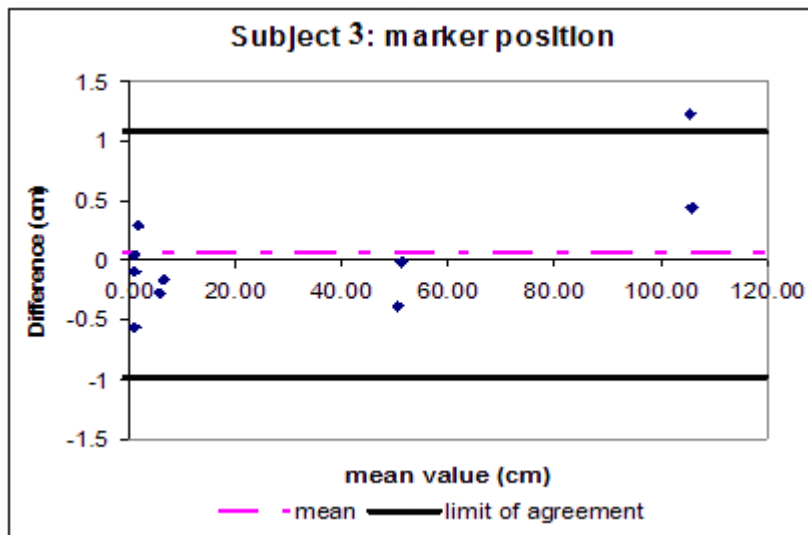


Fig 3.4.1. Graph plot for marker position (subject 3). Mean = 0.05, upper limit = 1.07, lower limit = -0.97. One point was outlying the upper limit (i.e. the right posterior superior iliac marker).

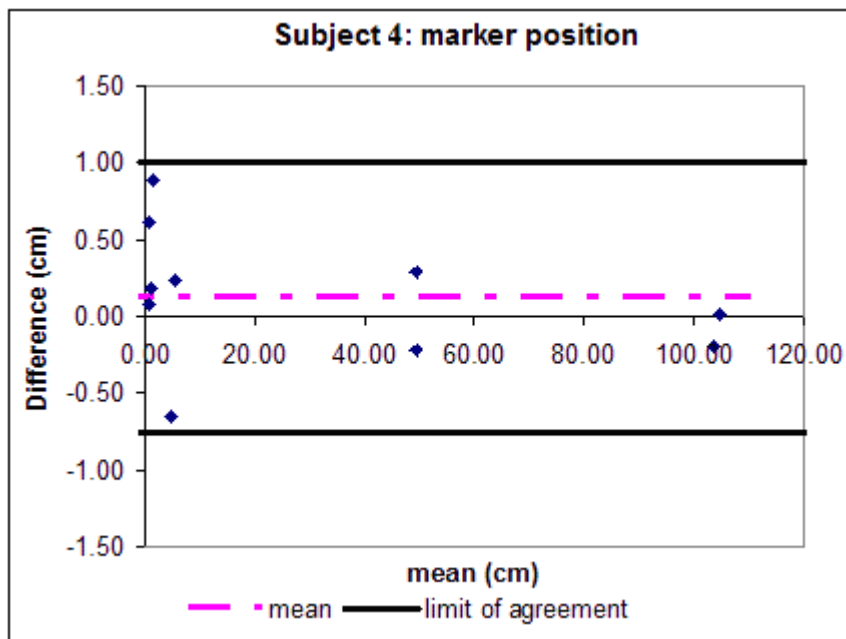


Fig 3.4.2. Graph plot for marker position (subject 4). Mean = 0.1231, upper limit = 0.998, lower limit = -0.751. All data points were within the limits of agreement.

Marker Separation

The results of separation (relative distance) between pairs of markers are presented (table 3.4.2) for subject 3 (days 1 and 10) and subject 4 (days 1 and 100). The marker pairs were: (1) sacral wand and left posterior superior iliac spine, (2) sacral wand and right posterior superior iliac spine, (3) left knee and left ankle, (4) left ankle and left heel, (5) left heel and left toe, (6) left toe and left ankle, (7) right knee and right ankle, (8) right ankle and right

heel, (9) right heel and right toe, (10) right toe and right ankle. The mean differences in marker pair separation for both subjects (0.07 and -0.03) were within limits of agreements. The difference in marker (pair 4) separation between in subject 4 was outside the limit of agreement (i.e.1.05cm)

Marker separation (cm)									
	Subject 3					Subject 4			
Marker Pair	Day 1	Day 10	Mean	Difference		Day 1	Day 100	Mean	Difference
1	30.33	30.60	30.46	-0.27		28.77	29.36	29.07	-0.59
2	30.69	30.46	30.57	0.22		31.37	30.87	31.12	0.50
3	45.89	45.79	45.84	0.09		44.89	44.53	44.71	0.37
4	7.07	6.98	7.028	0.09		6.13	7.18	6.65	-1.05
5	17.92	17.52	17.72	0.39		18.08	18.07	18.08	0.006
6	14.83	14.65	14.74	0.17		13.02	13.03	13.02	-0.003
7	45.90	45.78	45.84	0.11		44.88	44.62	44.75	0.26
8	6.45	6.79	6.62	-0.34		7.76	7.48	7.62	0.27
9	19.12	18.96	19.04	0.166		18.80	18.68	18.74	0.12
10	14.53	14.48	14.51	0.052		13.47	13.70	13.59	-0.23
		Mean difference		0.07					-0.03
		Upper limit		0.51					0.92
		Lower limit		-0.37					-0.99

Table 3.4.2. Marker separation on two days for subjects 3 and 4

The graph plot for the marker separations (mean separation against mean difference in separation) showed that all the data point were within limits of agreement for subject 3 (fig 3.4.3). This was similarly observed in subject 4 (fig 3.4.4) except for one data point representing distance between left ankle and left heel markers.

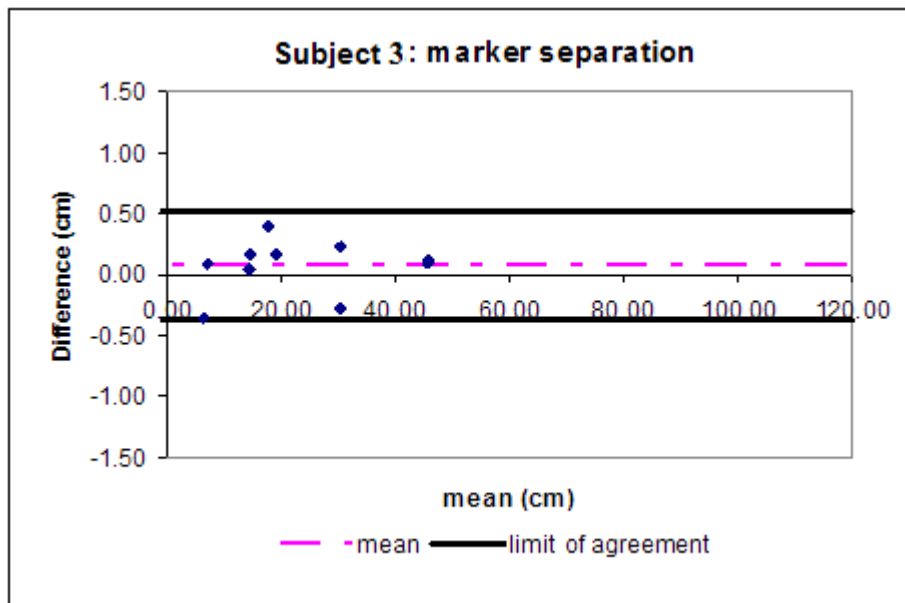


Fig 3.4.3 Graph plot for marker separation (subject 3). Mean = 0.0702, upper limit = 0.514, lower limit = -0.373. All data points are within limits of agreement.

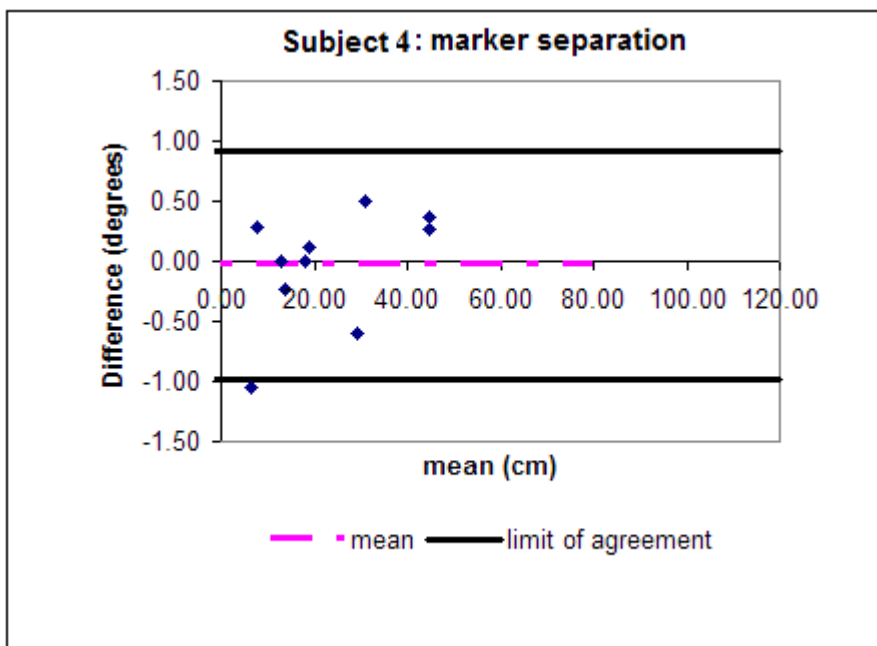


Fig 3.4.4. Graph plot for marker separation (subject 4). Mean = -0.033, upper limit = 0.921, lower limit = -0.9881. One data point (separation between left ankle and heel markers) was just below the lower limit.

3.5.2 Summary

The procedure of placement of markers on anatomical landmarks was tested by measuring marker position and separation on the subject's standing in similar position on a 10 and 100 day interval for subject 3 and 4 respectively.

The result showed that the changes (i.e. the mean differences) in marker position and separation between two test days were less than (or approximately) a millimetre in both subjects. This reflected a relatively greater reliability in positioning compared to others that observed approximately 5mm difference using the same equipment (Campbell, Ashburn et al 2001). Marker placement in this pilot study improved with the second subject i.e. all data points were within the limits of agreement. The difference in position of the right posterior superior iliac spine (RPSIS) marker in subject 3 can be attributed to a slight slant in the right arm of the pelvic frame whilst the change in heel to ankle marker distance on day 100 in subject 4 may be due to changes in skin or soft tissue position over the malleolus or heel. As in the previous section with test-retest of marking gait cycle events where the procedure improved with the second subject, the solution to limit the variability of marker placement will be subjectively practical. Thus, conscientious positioning of all the markers especially the RPSIS, ankle and heel markers will be carried out in the main studies. Although, changes in ankle-heel marker highlighted the fact that variability increases (or practice effect reduces) with a longer (100 days) duration for retesting on the same subject, it is important to note that the effect such a change with time has on the gait data/outcomes is controlled because marker placement will be carried out only once per subject. However, this pilot study established the reliability of marker placement procedure and highlighted the markers that required improvement in their placement.

3.6 Overall Pilot Summary

This pilot study was used to familiarize with the laboratory protocol and set up, and also test for procedural problems that could arise by utilising the protocol. The procedural problems were minimal and were corrected during and after the laboratory sessions as described earlier. The reliability of the procedures of marker placement and marking gait cycle events were tested and found to be mainly good, repeatable and acceptable to be utilised in the main study. Possible sources of increased variability were highlighted to be minimised in the main study. The laboratory protocol and set up that was tested in this pilot study were used in the methods in carrying out the main study on the hip pull-off and ankle push-off in the gait of the healthy adults. The methods and results of the main study are in the following chapter.

Chapter Four

Hip Pull-off and Ankle Push-off

In The Healthy Adult Gait

4.1 Introduction

The first aim of the overall study was to design a laboratory set up and protocol for the analysis of healthy subjects' gait that can be used later in the second study on PD gait. This protocol/set up was tested in a pilot study (in the previous section). The procedural adjustments that were made following testing the laboratory set up will be mentioned in this chapter. The second aim was to characterise ankle push-off and hip pull-off events in healthy gait. Thus, using the designed laboratory protocol the objectives were:

- a) To measure and describe hip pull-off and ankle push-off power.
- b) To measure and describe the other associated gait measures and their relationships with each other i.e. spatiotemporal and kinematic measures within the same gait cycle.

4.2 Methods

This was a prospective cross-sectional observational study designed to characterise hip and ankle function in the gait of healthy adult.

4.2.1 Recruitment

The approval to recruit and subjects and conduct the study was granted by the Research Ethics Committee of The School of Health Professions and Rehabilitation Sciences with sponsorship support by the University of Southampton (appendix 1,2,3). The healthy subjects that participated in this study were contacted and recruited via the Parkinson's disease society (PDS) of Winchester. They were the spouses and partners of members of the PDS. With the permission of the chairperson of the society, information packages (containing an invitation to participate, information sheet, consent letter reply slip and postage paid return envelope (appendix 4) were sent to healthy members' spouses and partners of PD society members.

To be eligible to participate, the subjects had to be (1) between the age of 50 and 80 years –to match the age of incidence of PD that usually rises from 50 years onward (2) able to walk independently unaided (because dependent gait and the use of walking aid alters the natural gait of individuals and confound the findings) and (3) able to travel to the Southampton General Hospital. Subjects were excluded if they had (1) a known diagnosis of PD or other neurological disorder and (2) any condition that affected the ability to walk such as acute illness, neurological disorder, musculoskeletal deformity and spinal/lower limb pain or fracture. The subjects gave written informed consent before participating in the trials. Sixteen subjects responded to the invitation and participated in this study.

4.2.2 Equipment

The equipment and materials used have been described in detail in the pilot study (chapter 3.1). They consisted mainly of the following:

- Two CODA mpx30 Scanner units
- Twenty-two body segment infrared markers (i.e. Light Emitting Diodes) and battery
- An 8m walkway and a force platform situated on the walkway.
- Anthropometers: external calipers, tape measure, height scale, electronic weighing scale, alignment frame and T-bar. Other materials are the cotton wool, ethyl alcohol, velcro straps, double sided sticky tapes.

4.2.3 Procedure

The procedure used in acquisition of gait data was as described in the pilot study section (chapter 3.2). It involved one gait data collection session per subject where subjects' demographic data were recorded and 22 markers were placed on anatomical landmarks of the lower limb (fig 3.2.1.). Subjects were then required to walk barefooted along the walkway.

The specific instruction given to them was that “starting with your left/right leg, walk as you usually do, whenever you are ready” (instead of being commanded or cued to do so). A gait trial was completed when the subject walked from one end of the walkway to the other.

The demographic data such as subject’s age sex height, weight, ankle, knee width and pelvic depth and width were loaded into data files of the motion analysis program used by Coda in processing of inverse dynamic modelling and clinical standard gait reports.

4.2.4 Data Analysis

Trials for analysis were checked and selected if they had the following:

1. Presence of force data
2. Absence of measurement artifact in gait data
3. Presence of at least one complete gait cycle for each limb

The number of gait trials with complete force platform data (i.e. the whole foot struck the force plate) was unequal across the 16 subjects. Some individuals achieved up to six successful trials while others only produced one. For data analysis, the first two trials for left and right legs were used for each subject. Two subjects were excluded due to insufficient data.

The gait cycle over the force plate made it possible for heel strike and toe-off points of the gait to be automatically detected and marked by Coda analysis software. All the gait components (kinetics: force, moment of force and powers, and Kinematics: joint/segment position and angular acceleration and velocity) were automatically calculated by the Coda system using the lower limb segmental analysis and inverse dynamics modelling of the

walking participants. The resulting output is usually in the form of a walking stick model of the participant and graph profiles of the gait components (on y-axis) plotted against time in seconds required to complete the gait cycle.

4.2.5 Outcome parameters

The outcome parameters were derived from graph profiles of gait components produced by Coda mpx30. The gait cycles (especially with a step on the force platform) of each participant were inspected for complete force platform data, complete marker trajectory, marker error and the presence of artifacts before being selected for analysis. The time component (on the x-axis) of the selected gait cycle was then “normalised” by automatically transposing it into a 100 percent (complete) gait cycle i.e. 0 (initial contact) to 100 (end of ipsilateral terminal swing/next initial contact). The following are graph profiles of the joint powers and angular excursions depicting the derivation of the outcome parameters i.e. A to J in Fig 4.1 to 4.6 during a gait cycle at the ankle, knee and hip joints.

- (1) **The peak ankle push-off power (APOP):** Derived as point “A” from the ankle joint power graph profile in late stance phase (fig 4.1). Y axis = normalised power in W/kg, X axis = % gait cycle (0-100%), Vertical line=point of toe-off.

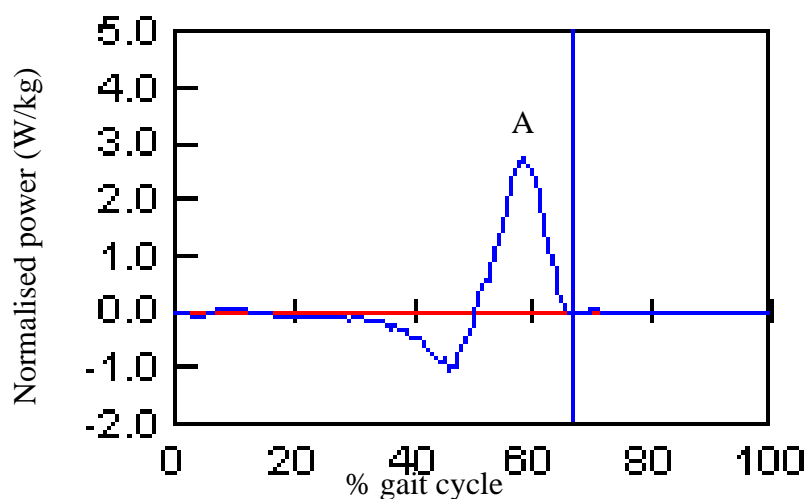


Fig 4.1 Peak plantarflexor/ ankle push-off power (APOP).

- (2) **The ankle angular excursion during push-off (ROMa):** Derived as the difference between point B and C in late stance phase of ankle angle profile (fig 4.2). B = peak dorsiflexion angle, C = peak plantarflexion at toe-off. The Y axis = dorsi-plantar flexion (angular position), X axis = % gait cycle (0-100%), Vertical line = toe-off point.

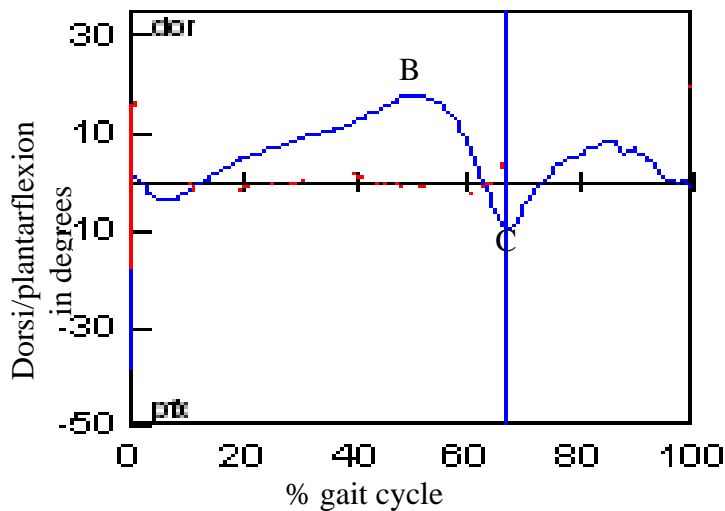


Fig 4.2. Joint angular excursion during push off at the ankle joint (ROMa)

- (3) **The peak hip extension power (HPEXP); and peak hip pull-off power (HPOP):**

Derived as point D and E respectively on the hip joint power profile (fig 4.3) during the midstance and preswing phase respectively. Y axis=normalised power in W/kg, X axis = % gait cycle (0-100%), Vertical line=toe-off point.

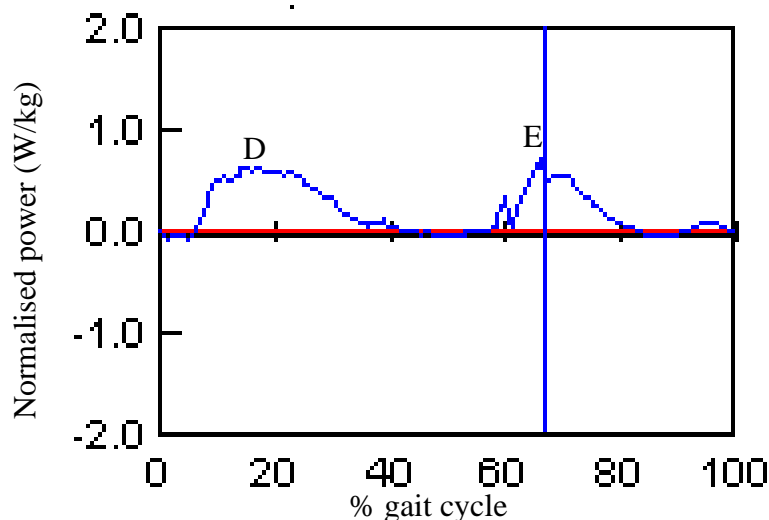


Fig 4.3. Hip extension power in midstance (HpExP) and Hip pull-off power (HPOP)- in pre-swing.

- (4) **The hip angular excursion during pull-off (ROMh):** Derived as the difference between point G and F on the hip joint angular graph (fig 4.4). F= maximum hip extension in terminal stance, G= maximum hip flexion in swing. Y axis=hip flexion/extension (angular position), X axis= % gait cycle. Vertical line=toe-off point.

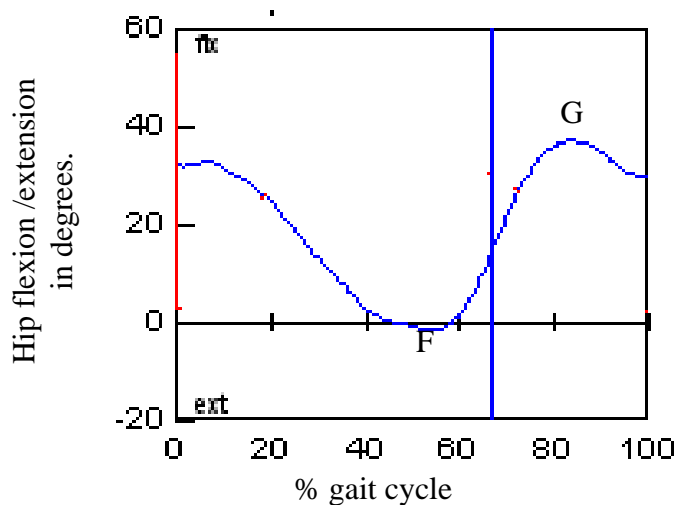


Fig 4.4 Hip joint angular excursion during pull-off (ROMh)

- (5) **The peak knee extension power (KnExP):** Derived as point H from the knee joint power profile (fig 4.5) during the preswing phase of gait

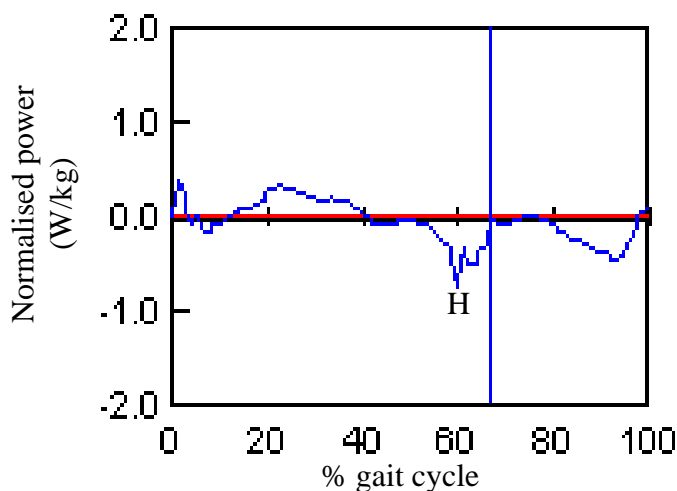


Fig 4.5. Knee absorption power in late stance (KnAbP)

(6) The knee angular excursion in late stance (ROMk): Derived as the difference between point J and I on the knee flexion/extension graph profile (fig 4.6). J= maximum knee flexion in swing, I= maximum knee extension in stance. Y axis= knee flexion/extension (angular position), X axis= % gait cycle (0-100%). Vertical line=toe-off.

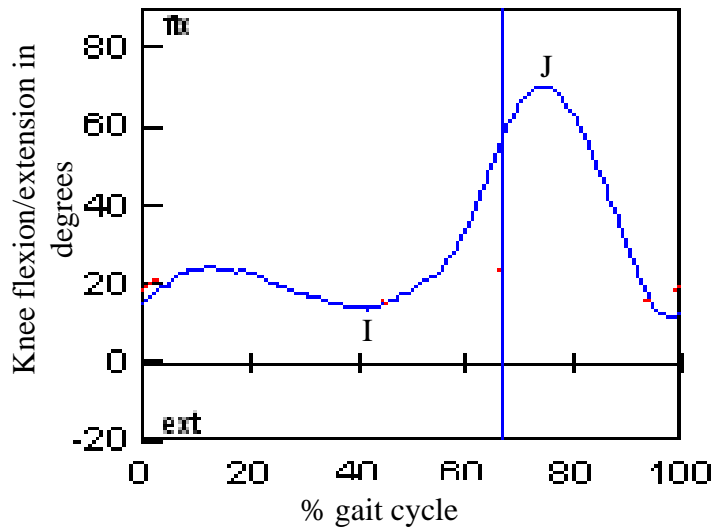


Fig 4.6. Knee joint angular excursion during push/pull-off events (ROMk)

The Spatiotemporal parameters

The parameters of stride length and time, cadence, gait velocity, single and double support were derived by converting the acquired gait files saved in “mdf” format to “mdr” format compatible with a Coda mpx30 report generator (i.e. MotionDB V2.25-UK) that is capable of calculating the spatial and temporal gait components. The software was able to utilise the data files with marked gait cycles events of heel strike and toe-off -of left and right limbs- carried out in the first part of analysis to estimate the following:

- (1) Stride time: The time duration of a gait cycle (or Stride) of both lower limbs
- (2) Stride length: The distance between two initial contacts(or heel strikes),
- (3) Gait Velocity: The velocity of a gait cycle (stride length against stride time),

- (4) Cadence: Estimated from number steps in seconds to number of steps in a minute
- (5) Support period: Time (secs) spent with both feet on the ground (i.e. double support) and time spent with only one foot on the ground (i.e. single support period)

The statistical analyses of the variables were carried out using SPSS 14.0 for Windows.

Descriptive statistics i.e. mean, standard deviation were used to estimate the push-off and pull-off events i.e. the peak pull and push-off powers, their corresponding joint angular excursion at the hip, knee and ankle joint and the spatiotemporal components (e.g. the gait velocity, stride length, cadence). The following section presents the results of the gait parameters on the healthy subjects.

4.3. Results

4.3.1. The Sample

The complete gait data of fourteen out of sixteen healthy subjects (age range 52-74 yrs) with complete gait data and profiles were finally analysed in this study (appendix 11). The mean age, height and weight of the subjects (5 males, 9 females) were 63.6 ± 7 years, 1.66 ± 0.1 m, 77.3 ± 11 kg respectively (Table 4.2.1)

Sex		Sample Size		Age (years)		Height (m)		Weight (kg)
Males		5		58.6 ± 8		1.77 ± 0.1		88.2 ± 9.5
Females		9		66.5 ± 6		1.61 ± 0.04		71.4 ± 5.8
Mean				63.6 ± 7		1.66 ± 0.1		77.3 ± 11

Table 4.2.1. Healthy Subject Sample characteristics (n=14 healthy adults)

From the table it can be that the females were older but lesser mean height and weight than the male subjects

4.3.2 Gait Parameters during Usual Walking

The primary outcome of hip pull-off and ankle push-off powers during steady state gait are presented in the kinetic results. The other gait measures are presented in the kinematic and spatiotemporal results.

There were no gender differences (t test) between the males (n=5) and the females (n=9) in the gait parameters e.g. ankle push-off power-APOP ($p=0.13$), hip pull-off power-HPOP ($p=0.76$), Knee absorption power-KnAP ($p=0.11$), Hip extension power-HpExp ($p=0.31$), gait velocity ($p=0.51$), stride length ($p=0.81$) and cadence ($p=0.21$). The mean values of the selected gait parameters for the group (both males and females) were analysed.

4.3.3 Joint Power

The greatest power generated in the lower limb (2.47W/kg) was at the ankle joint (peak push-off power). Mean hip pull-off power was 0.79W/kg whilst hip extension power was 0.60W/kg. The subjects' range of mean push-off power was also higher than that of mean hip pull-off power with the lowest push-off power recorded (1.43W/kg) being greater than the highest hip pull-off power (1.29W/kg) (Table 4.3.2).

Power (W/kg)	Mean	Std Dev	Median	Minimum	Maximum
HPOP	0.79	0.30	0.87	0.35	1.29
HpExP	0.60	0.26	0.59	0.26	1.23
KnAbP	-0.72	0.42	-0.65	0.4	-1.96
APOP	2.47	0.49	2.47	1.43	3.08

Table 4.2.2. Power generated (at the hip and ankle) and absorbed (the knee) during push-off and pull-off phases of gait.

In addition, there was more variability between subjects in ankle push-off power (± 0.49) than the hip pull-off power (± 0.30). There was a normal distribution of the HPOP, APOP and HpExP.

4.3.4 Joint Angular Excursion

The knee joint angular excursion during late stance was the highest followed by plantarflexion excursion during push-off and hip flexion during pull-off (Table 4.2.3).

Kinematics	Mean	Std Dev	Median	Minimum	Maximum
ROMh	36.1	4.9	36.4	29.3	45.0
ROMk	51.8	6.1	51.8	40.2	61.2
ROMa	-25.6	6.0	-25.8	6.52	36.7

Table 4.2.3. Change in joint position during Pull and Push-off

4.3.5 Spatiotemporal parameters

Table 4.2.4 shows the mean time-distance variables measured during the gait cycle of the healthy subjects. The mean gait cycle velocity, stride length and percentage period in stance (i.e. point of toe-off) were estimated for only fourteen subjects. This is because bilateral measures were required to produce these three gait measures. Variability appears to be greatest with the cadence than the other gait components.

Spatiotemporal	Mean	Std Dev	Median	Minimum	Maximum
Gait cycle velocity (m/s)	1.05	0.13	1.07	0.73	1.24
Stride Length (m)	1.16	0.11	1.20	0.90	1.29
Cadence (steps/min)	107	9	108	89	120
Single support (sec)	0.40	0.03	0.40	0.35	0.51
Double Support (sec)	0.14	0.02	0.13	0.11	0.20

Table 4.2.4. Temporal and spatial measures during gait of healthy adults

The spatiotemporal measures of stride length, cadence single and double support were observed to be normally distributed (skewness -1.01, -0.51, 1.20, 1.07, 2stES = 1.23). The distribution for gait cycle velocity was skewed (-1.23)

4.3.6 Relationship between the Gait Parameters.

A correlation analysis (Pearson) was performed on the healthy gait parameters of pull-off and push-off events and their corresponding kinematic, spatial and temporal parameters (table 4.2.5). All the parameters were normally distributed except for gait velocity with a negative skew hence non parametric analysis (Spearman correlation analysis) was performed.

	HPOP	HpExP	KnAbP	APOP	Gait Vel	Stride length	Cadence	ROMh	ROMk
HpExP	0.40								
KnAbP	-0.27	-0.21							
APOP	0.43	0.25	0.48						
Gait Velocity	0.31	0.33	0.23	0.78*					
Stride length	0.01	0.05	0.11	0.49	0.57*				
Cadence	0.64*	0.42	-0.23	0.68**	0.51	-0.06			
ROMh	0.71*	0.07	0.08	0.46	0.42	0.30	0.37		
ROMk	-0.02	-0.27	0.28	0.17	0.37	0.43	0.07	0.38	
ROMa	0.12	-0.04	0.20	0.54*	0.38	0.18	0.34	0.32	-0.32

Table 4.2.5. Correlation analysis of power and corresponding angular excursion, spatial and temporal gait parameters in healthy sample (n=14). *correlation is significant at the 0.05 level (2 tailed). **correlation is significant at the 0.01 level (2 tailed).

It was observed that HPOP correlated with hip joint angular excursion (ROMh) and cadence whilst APOP correlated with gait velocity, plantarflexion at push-off (ROMa) and cadence.

More importantly, gait cycle velocity was correlated with stride length. However, the lack of correlation between the other parameter may be due to the small effect size (n=14) used for the analysis.

4.3.7 Summary of Result

The aim of this study was to design a laboratory protocol that can be used analyse the gait of healthy adult subjects with the objectives of measuring and describing hip pull-off and ankle push-off power and their associated kinematics and spatiotemporal measures. Using the laboratory protocol on a sample of 14 subjects, the following were the results:

- The number of gait trials/cycles acquired with complete data was unequal across the study group. A consistent number of two gait trials per subject (total of Twenty eight) were finally analysed after exclusion of trials that did not meet with the selection criteria.
- The mean power generated at the lower limb joint during the gait cycle was greatest during ankle push-off followed by hip pull-off power. Between the subjects, the APOP was the more variable than HPOP measured. Both HPOP and APOP were normally distributed in the healthy adult sample studied.
- The change in joint rotation showed that the greatest angular excursion occurred at the knee joint in the late stance phase. The change in knee flexion in late stance was also more normally distributed than the change in flexion during hip pull-off and ankle push-off. As observed in power generated, change in plantarflexion during push-off was more varied between subjects than for change in hip flexion at pull-off.
- Of the spatiotemporal measures, cadence was more variable but normally distributed whilst distribution of gait cycle velocity stride length and other measures were skewed.

- Correlation analysis of the gait parameters showed that HPOP and APOP are related to their respective joint angular excursion during the pull-off and push-off events. Both powers were also related to cadence. However, only APOP was related to gait velocity. Gait velocity was also observed to be related to the stride length.

4.4.0 Discussion

4.4.1 Introduction

This study was aimed at characterising the ankle and hip function during healthy adult gait by describing the gait parameters (spatiotemporal, kinematic and kinetics) measured with a motion analysis system (CODA mpx 30). The primary objectives were to characterise the gait events of pull-off push-off in healthy gait by describing the joint powers, ranges of motion at the hip and ankle joint that are the prime actors during these gait events with the corresponding spatiotemporal measures. For this purpose, a laboratory protocol was designed that was first used on healthy subjects and later used in the study on PD gait.

The magnitude of gait components measured during pull-off and push-off events of the elderly gait are discussed and compared with findings from other studies. Thereafter, the appraisal of the use of the laboratory protocol and the issues that affected measurements on the subjects sample, gait trials and analysis in the course of the study are discussed. The findings of this study are important as future comparative values for the second phase of the study on people with Parkinson's disease. Therefore, the healthy individuals recruited for the present study were people that were within the age range that usually have a high incidence of PD

4.4.2 The Participants

The subjects that participated in this study can be classified as those advancing in age or the elderly as they were in their fifties (36%), sixties (43%) and seventies (21%) with the mean and the median age as 65.5 ± 6 and 67 years respectively. There was a predominance of healthy female subjects (64%) in the sample analysed. Since they were partners/spouses of people with Parkinson's disease, it may be indicative of the arguably higher incidence of PD

among men (Van Den Eeden et al. 2003;Wooten et al. 2004). None of the participants presented with any musculoskeletal dysfunction, pain or impairment that could affect their gait and were all able to walk and comply with the laboratory protocol and instructions.

4.4.3 Spatiotemporal at Usual Walking Speed

To characterise the elderly subjects' pull and push-off events with regards to the corresponding joint power, angular excursion and spatiotemporal components, it is essential to first consider the self selected (i.e. usual) gait velocity of the subjects because most of the other gait components are affected by the selected gait velocity .

The differences in usual gait velocity between the previous studies and ours may not only be due to the subjectivity of subjects' self selected gait velocity but also likely due to other factors that affect the velocity a subjects selects to walk with. These factors can be related to the methods utilised e.g. length of walkway i.e. the longer the walkway the faster a subjects walks. On an 8m walkway, the mean usual gait velocity the subjects in the present study walked with was 1.05 ± 0.13 m/s. Other researchers reported usual gait velocity that varied from 1.00 ± 0.25 m/s on 6m walkway (Brach et al 2001) to 1.17 ± 0.19 m/s on a 9m walkway (Stolze et al 2000), 1.19 ± 0.13 and 1.21 ± 0.21 m/s on 9m walkway (Kerrigan et al 1998 & 2000), 1.32 ± 0.23 on a 14m walkway (Hageman et al 1986) and 1.22 ± 0.18 on 10m walkway (Watelain et al 2001). A review study by Prince et al (1997) on elderly gait presented gait velocity values that ranged from 0.94 to 1.33m/s. Thus, the subjects in the present study appear to walk slower than that observed in the other studies. However, within this study's limits of acceptability, the characteristic elderly gait velocity approximated 1m/s especially as a reference for investigation with similar test conditions.

Stride length and cadence are principal gait components which influence gait velocity. Although stride length and cadence are independent of each other, they can unilaterally or conjointly change with gait velocity. It is therefore not surprising to have observed (as with gait velocity) that the subjects' mean stride length (1.16 ± 0.11 m/s) tended to be on the lower range relative to that previously observed i.e. 1.18 ± 0.14 m (Stolze et al 2000), 1.20 ± 0.12 m (Kerrigan et al 1998b), 1.35 ± 0.17 m (Shkuratova et al 2004) and 1.39 ± 0.14 m (Winter et al 1990). Similarly, the mean cadence recorded in the present study (107 ± 9 steps/min) was on the lower range relative previous findings i.e. 110.5 ± 7.3 (Winter et al 1990), 115 ± 6 (Sofuwa et al 2005), 115 ± 7 (Graf et al 2005), 119 ± 9 (Kerrigan et al 1998) and 129 ± 10 (Stolze 2000). There is an inverse relationship between cadence and stride length such that an individual walking with a short stride length often have a high step rate i.e. cadence. However, a lower stride length and/or cadence resulted in the lower gait velocity we observed in the current study. It could be argued that because cadence is often reported to be stable with advancing age, the clinical relevance of the healthy values reported from the previous studies could not be as emphasised as much as the stride length. It appears that the subjects in the present study walked with a shorter stride length albeit the mean height of the subject in this study (1.66 ± 0.1 m) is comparable to that of the other studies e.g. 1.65 ± 7 m (Shkuratova et al 2004) and 1.59 ± 0.1 m (Graf et al 2005). However, other contributory factors such as inter-rater differences between studies could affect measured stride length. The degree of human error in this procedure is often not reported and is unknown in most studies. In our study, the accuracy test of marking of the gait cycle event of heel strikes and toe-off (that determines the stride length) showed that the mean difference in marked times was less than a hundredth of a second when done repeatedly. Therefore, the stride length values measured in the present study can be considered to be within acceptable limits.

4.4.4 Joint Angular Excursion

Associated with ageing is the decrease in joint range of motion that may have resulted from muscle weakness and tightness hence the elderly joint angular excursion will not be similar to the magnitude observed in younger adults. The angular excursion we observed at push-off ($-25.6 \pm 6^\circ$) was within the range observed in other previous reports on elderly gait. Judge et al (1996) observed ($-26 \pm 5^\circ$), Sofuwa et al (2005), Kerrigan et al (1998) and Graf et al (2005) observed $-24.05 \pm 3^\circ$ and $-23 \pm 6^\circ$, $-26 \pm 6^\circ$ respectively. Judging from the minimum joint angular excursion observed from our study (19.6°) and from the reported values from other studies, the plantarflexion (push-off) excursion for the elderly can be considered to be approximately 20° .

Conversely, the hip joint angular range of excursion during pull-off has rarely been measured or reported, hence healthy elderly values for comparative purpose is lacking. Often, as with some of the other previous measures of ankle kinematics, the reports are usually based on range of motion throughout the entire gait cycle or peak joint positions but not on the propulsive phases of pull and push off events. The values for hip pull-off angular excursion presented in this present study ($36.1 \pm 4.9^\circ$) are therefore the representative values in the database of healthy elderly gait.

4.4.5 Pull and Push-off Power

The mean hip pull-off power observed in our elderly sample i.e. $0.79 \pm 0.3 \text{ W/kg}$ was less than (at the mean gait velocity of $1.05 \pm 0.13 \text{ m/s}$) that reported in previous studies. However, there was an indication that this may have been associated with self selected usual gait velocity of the study sample and not weakness at the joints. It is known that joint power is affected by a person's gait velocity. Therefore at a relatively greater mean gait velocity, $1.37 \pm 0.17 \text{ m/s}$,

Kerrigan et al (1998) reported a greater hip pull-off power ($0.90 \pm 0.22 \text{ W/kg}$) than that observed in our study. A relatively greater power with velocity was also observed in another study (Kerrigan et al 2000) when a mean power of $0.92 \pm 0.22 \text{ W/kg}$ was reported for a different group of subjects walking at greater velocity of $1.21 \pm 0.12 \text{ m/s}$. Conversely, at a lower gait velocity $1.03 \pm 0.13 \text{ m/s}$, Judge et al (1996) reported a greater hip pull-off $0.92 \pm 0.21 \text{ W/kg}$. Similarly, Graf et al (2005) reported a greater hip pull-off power of $0.91 \pm 0.21 \text{ W/kg}$ for their elderly walking also with a comparable mean gait velocity of $1.04 \pm 0.16 \text{ m/s}$. Therefore, there is an indication that the differences in hip pull-off were affected not only by the gait velocity but that other factors may have been influential. An examination of the ages of the subjects in the other studies did indicate that the depreciative effect of advancing age may have been a factor. The mean subject age in the previous studies were greater i.e. 79 ± 6 years (Judge et al 1996, Graf et al 2005), 73.2 ± 5.6 years, 73.2 ± 5.6 & 72.7 ± 5.5 years (Kerrigan 1998 and 2000) than the subjects in our study i.e. 63.6 ± 7 years. The greater hip pull-off power could have served as a compensatory strategy when the ankle push-off power is significantly diminished with age.

Indeed, with a lower ankle push-off power ($1.70 \pm 0.23 \text{ W/kg}$ -Kerrigan et al 1998) than that observed in the current study ($2.47 \pm 0.49 \text{ W/kg}$), a greater push-off power previously observed may have been a compensatory strategy. However, the other investigators reported greater ankle push-off power of $3.5 \pm 0.9 \text{ W/kg}$ i.e. Judge et al (1996) and $3.0 \pm 0.88 \text{ W/kg}$ i.e. Graf et al (2005) in addition to the greater hip pull-off power but comparable gait velocity. It therefore suggested from comparison with the previous findings that the effect of age is significant as our subjects can be considered as being 'younger' thus the greater pull-off and push-off powers others reported may have been to compensate the slowing down and maintain the gait velocity with advancement in age.

The relationships between the gait parameters indicated that the ankle push-off power changed with the gait velocity thus highlighting the role of ankle push-off power in influencing gait velocity. Interestingly, it was also observed that the ankle push-off power (and hip pull-off power) correlated with the step rate (cadence) rather than with the stride length. Previously, Judge et al (1996) had indicated that the ankle push-off power was the greatest predictor of stride length. Conversely, the findings of DeVita and Hortobagyi (2000) highlighted the tendency of the elderly to walk by emphasising the cadence than the stride length thus giving support to our findings in this study. The reason the elderly walk with emphasis on shorter steps (with high step rate) is often due physiological and biomechanical changes at the musculoskeletal level that occur with age that could lead to decreased joint angular excursion in addition to gradual deterioration of balance. However, joint angular excursion during pull-off and push-off were related to the respective powers generated at the joints. This suggests that the hip pull-off and ankle push-off powers effectively translated to spatial (angular) changes at the joint. These indicated that restricting joint factor that can result from contracture and ageing thereby diminishing joint active range of motion were minimal in our study sample. Therefore, the joint angular excursion values recorded in this study can be considered as healthy values for these subject samples.

It was interesting to note that ankle push-off power and stride length each correlated separately with gait velocity but not with each other. This can actually mean that the subjects with high power generation had accompanying high step rate but that any change in gait velocity was significantly dependent on the stride length. This suggests that in elderly gait rehabilitation, stride length remains the predominant factor to gait velocity and clinicians could consider implementing goals to redirect the ankle and hip propulsive powers at improving the stride length instead of the self direct powers towards cadence.

4.4.6 Using the Laboratory Protocol

Some limitations to the laboratory measurements were encountered which affected the quantity and probably the quality of the trials conducted during the laboratory sessions. Most of these limitations had to do with markers that were faulty or obstructed from the view of the cameras. Faulty markers were readily detected and replaced but markers obstructed by a stationary arm (not swinging) gave a data output that was seen as a discontinuity in the trajectory of the affected marker. Trials with such data would normally not be valid for final analysis even when the primary data from the force platform is available. However, brief discontinuities caused by a swinging arm were automatically reconstructed or “filled in” by CODA software by producing a continuation of the regular pattern produced earlier in the trajectory before the obstruction.

This raises the question of the validity of the reconstructed profile. It could be reasoned that healthy people would reliably produce a regular pattern that can be easily reproduced by CODA but what happens when measuring subjects with disorders of gait that may not follow a regular pattern?. Furthermore, in instances when subjects do not swing the arms characteristically (e.g. Parkinson disease) there may be a prolonged marker obstruction that will not be resolved automatically by CODA.

A solution is to avoid repeating trials as much as possible by strictly checking for markers and battery boxes that are faulty or have exceeded their lifespan. Also when testing people with gait disorders, a pilot study will be required to examine the occurrence of procedural problems with equipments such as limb markers that can affect analysis. Alternatively, only trials that do not have trajectory discontinuities or distorted profiles could be used. However, this would limit analysis to fewer trials.

Force platform data was acquired without instructing or informing the subject to step on the force plate. This was to ensure that there was no aiming for the force platform that would require the subject to alter his gait thereby affecting the data. The pilot study had indicated that it was possible to record six trials with force platform data without having to ask subjects to aim for it. However, in the main study, this strategy contributed to an increase in the number of trials more than the six times as subjects often missed the force plate. Future studies could limit the number of trials by informing subjects about the force platform and that they should walk as normally as possible without aiming for it. However, some studies that instructed their subjects to aim for the platform observed that it does not adversely affect measurement of the ground reaction force vector (GRF) particularly when the starting point on the walkway is fine tuned (Sanderson et al 1993, Grabiner et al 1995, Paul 1996, Wearing et al 2000). Nevertheless, they still recorded trials where the subjects were observed to have altered their stride. This questions the validity of the measurements (as changes in joint angle thus stride, can affect power generated). Thus the issue of the constraints placed on natural gait by force plate targeting requires careful consideration. The other contention is if this strategy will be applicable in people with PD that have diminished automatic/initiative motor processes and therefore may utilise attentional strategy on a visual cue (the force platform) to augment their gait. The only way to know if the PD gait was affected by informing subjects about the force plate would be to keep an account of the events of each trial and consider them during analysis e.g. by asking if the PD subject felt he/she reached for the platform or the researcher observed a reaching of the leg for the platform.

In this present study, missing the force platform or stepping on its edge after defining the start point did not occur very often but more frequently than desired in about two subjects. This could be resolved by asking the subject to target the force platform as mentioned in the paragraph above. Moreover, the start point could further be fine tuned if at first the subject

stands on the force platform and walks away to the end of the walkway where the would-be start point is then marked.

The eight metre walkway in the gait laboratory used in this study can be considered relatively moderate in length despite the limited data capture area by the CODA cameras. Comparatively, other studies have used different lengths of walkways varying from 7.25m (Bohannon 1997), 9m (Kerrigan et al 1998), 10m (Oberg et al 1993), 14m (Hageman et al 1986) and 20m (Menz et al 2003). Many of these studies included acceleration and deceleration distances as gait is often unsteady during the period and therefore is excluded from analysis. In this present study, analysis of strides over the force platform and the 2.5m camera capture area allowed for acceleration and deceleration distances. Generally, in gait analysis and laboratory studies, there has been no consensus as to what the length of a walkway should be, how long an individual is required to walk to achieve steady state gait and the acceleration and deceleration distances to be included. It appears that different studies decide and design these measures not on a common standard but on logistical and practical reasons.

4.4.7 Conclusion and Summary

This study set out to characterise gait in healthy adult by the describing the events of push-off and pull-off. The laboratory protocol designed for this purpose was successfully used in achieving this aim. Data were provided on the kinetic and the associated kinematic and spatiotemporal measures of hip pull-off and push-off. These were comparatively similar to some of the healthy values separately measured in previous studies on healthy adult gait. Moreover, other measures such as change in joint position during pull-off and push-off at the

hip, ankle and in the knee at late stance that were otherwise not available in the previous studies, adds to the reference database of gait in the healthy adults.

In general, most subjects walked at a velocity greater than 1m/s with the HPOP and most especially the APOP being a significant factor in affecting the gait velocity via changes in joint angular excursion and the stride length. Due to intersubject variability in gait patterns, generalised description of healthy gait may be limiting.

Recommendations for future studies on healthy adult gait are that a larger subject sample size be analysed with characterisation for different age groups and gender. The effect of other factors such as level of physical activity and laterality on the gait findings should be investigated. Sagittal plane data were extracted and used for analyses because primarily, the main outcome measures of ankle push-off and hip pull-off powers are both generated in the sagittal plane. This is the plane of largest movement when walking and it was observed to account for more than 80% of the power developed in the lower limb (Eng & Winter 1995, Allard et al 1996). Although, it has been estimated that the influence of out-of-plane movement such as abduction/adduction on flexion and extension in healthy gait is negligible (Cheng & Pearcy 1999), pathological cases such as PD where gait compensations may occur should be carefully considered.

The laboratory protocol utilised should be optimised to increase number of trials with force platform data and decrease the total number of trials carried out. In addition, clinical motion analyst and experts in field of motion analysis should reach a consensus and provide recommendation on the laboratory protocol such as instruction to walk, length of walkway, number of trials required for analysis, acceleration/deceleration distances and method of force data collection (e.g. aiming versus not aiming).

Although, the present study was conducted in a controlled laboratory condition, it utilised the gait measures and tools that are otherwise not currently available in outdoor walking tests. Therefore, it was intended that the data provided will serve as reference for normal gait in healthy people in gait laboratory studies. In designing and using the laboratory protocol, the issues that arose and their solutions were considered and the necessary modifications were made for the utilisation of the protocol in the next study on the characterisation of gait in Parkinson's disease. The following chapter presents the testing of the issues and modification made in a pilot study followed by the implementation in the methods of the main study on people affected with PD.

Chapter Five

Pilot Study

Parkinson's Disease Gait Analysis

5.1 Introduction

One of the aims of the previous study on healthy gait was to design a laboratory set up and protocol that was to be used firstly on healthy subjects then later in the study of PD gait. In carrying out the aim of the first study some issues arose that were highlighted with modifications needed for the PD gait study. The areas that were addressed were:

1. **Pelvic (ASIS) marker obstruction:** The Coda camera view of the pelvic (especially the ASIS) marker was often obstructed by a stationary (unswinging) arm. Therefore, the pilot subjects will be tested in different conditions that improve Camera-Marker view.
2. **Missing the force platform:** The healthy subjects often missed stepping on the force platform even after fine tuning the start point. To improve the rate of foot-on-platform, the pilot subjects will be informed about the platform (unlike the healthy subjects in the previous study). To minimise subjects aiming for the platform, they will be asked not to aim for it but that the camera view was more important.
3. **Fast walking test:** Unlike the previous study on healthy gait, the present study on PD gait included testing the PD participants at fast walking speed. In fast walk test condition there is the risk of trips and falls that must be minimised. Therefore, a safety harness was introduced to the set up for both usual and fast walking speeds. It was important to test the feasibility of the modified set up.
4. **Manual marking of gait cycle events:** As was carried out in the healthy gait study, a test-retest of marking the gait cycle events of heel strike and toe-off was carried out. This is to examine that the marking process was maintained with good reliability but most especially because the point of toe-off may be a potential source of variation in spatiotemporal outcome measures in people with PD particularly in those presenting with shuffling gait.

5.1.1 Aim and Objectives

The aim of this pilot study was to examine the above issues with their modifications. Therefore, the objectives were: (1) For marker obstruction, to test subjects in different walking conditions e.g. walking with arms folded, arms swinging and with extension of the arms of the pelvic frame. (2) For stepping on the force plate, subjects were informed of the platform but not to aim for it whilst the start point is fine tuned when the plate is missed. (3) For fast walking, subjects are tested with the use of the safety harness to prevent trips/fall and (4) For manually marking the gait cycle events, a test-retest of the procedure will be carried out.

5.1.2 Recruitment

To test the modified laboratory protocol and the marking procedure, ethical approval for the pilot study was given by research ethics committee of the School of Health Professions of the University of Southampton. Seven subjects diagnosed with idiopathic PD were recruited out of which two were used for test-retest of the protocol of marking the gait cycle event. The PD subjects were contacted through the local Parkinson's disease society of Winchester and Southampton. Eligibility to participate was according to the inclusion and exclusion criteria for recruitment in the main study (please see chapter 6.1.2). They should be able to follow instruction walk unaided and able to make the trip to the laboratory. There was an initial home examination session to screen for eligibility to participate by taking the subjects' history and disease specific tests i.e. Unified Parkinson's disease rating scale and mini-mental state examination (as detailed in section 6.1.4). All subjects that participated in the pilot study were given the information package and they gave their written and signed informed consent before taking part.

5.1.3 Instruments

The instruments used were: The Mini-Mental State Examination test, the motor section of the Unified Parkinson's disease rating, Codampx30, a force platform, 8m walkway, and a safety harness for the gait assessment. These are described in detail in the method section (chapter 6.1.4)

5.2 Aim 1: Manual Marking of Gait cycle Events (A Test-retest).

The procedure was carried out as in the test retest of the healthy study (chapter 3.3). The first ten trials of two of the recruited PD subjects were tested for reliability of marking the gait cycle events. On the animated stick figure constructed by Coda from markers placed on specific anatomical landmarks, cursors were used to mark the time in the gait cycle corresponding to the periods of heel strike and toe-off. This procedure was repeated after a 7 day interval. Using the Bland-Altman method of analysis, the difference between the values on the two test days were plotted against the mean of the values of the toe-off times on both days. Good agreement of the values is attained when they are within the 95% limits of agreements.

5.2.1 Results

5.2.1a Heel Strike Time (Test-retest)

The table below shows the result of marking heel strike time in the gait cycles of subjects 1 and subject 2 on two occasions with a 7 day interval. The mean differences in the marked time for both subjects (0.00045secs and 0.0065secs respectively) were within the 95% limits of agreement. The marked heel strike times were replicated (zero difference) in trials 1,4,5,7 and 9 for subject 1 and in trials 1,3,5,7,9,11 in subject 2.

Heel strike times (sec)									
	PD Subject 1					PD Subject 2			
	Day 1	Day 7	Mean	Difference		Day 1	Day 7	Mean	Difference
1	1.74	1.74	1.74	0		0.54	0.54	0.54	0
2	1.64	1.63	1.635	0.01		1.2	1.22	1.21	-0.02
3	1.07	1.06	1.065	0.01		2.24	2.24	2.24	0
4	1.68	1.68	1.68	0		1.24	1.265	1.2525	-0.025
5	1.28	1.28	1.28	0		0.86	0.86	0.86	0
6	0.525	0.52	0.5225	0.005		1.26	1.28	1.27	-0.02
7	1.08	1.08	1.08	0		1.22	1.22	1.22	0
8	0.96	0.98	0.97	-0.02		1.08	1.06	1.07	0.02
9	0.4	0.4	0.4	0		2.6	2.6	2.6	0
10	1	1.02	1.01	-0.02		1.14	1.16	1.15	-0.02
11	0.32	0.3	0.31	0.02		0.54	0.54	0.54	0
Mean of differences				0.000455					-0.0065
95% Upper limit of agreement				0.02431					0.021804
95% Lower limit of agreement				-0.0234					-0.0348

Table 5.2.1 Heel strike times marked for subject 1 and 2

The graph plots of the differences between the marked times are presented in fig 5.2.1 and fig 5.2.2. For both subjects all the data point (differences in marked time against the mean of marked time) were within the 95% limits of agreement i.e. 0.0053 and -0.0131 (fig 5.2.1), and 0.0072 and -0.004 (fig 5.2.2).

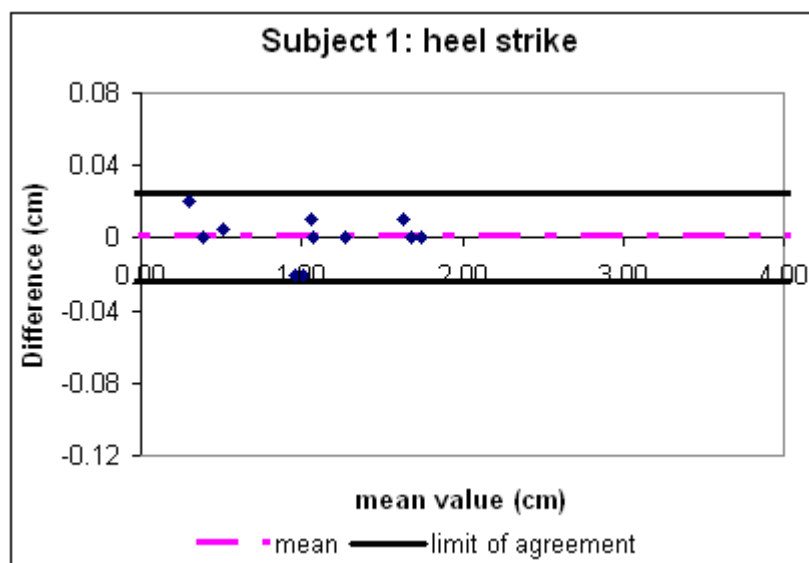


Fig 5.2.1. Graph plot of heel strike time difference for PD subject 1. Mean = 0.0004, st dev = 0.0119, upper limit = 0.024, lower limit = -0.0234. All data points were within the limit of agreement. Five of the data points were exactly repeated (zero difference).

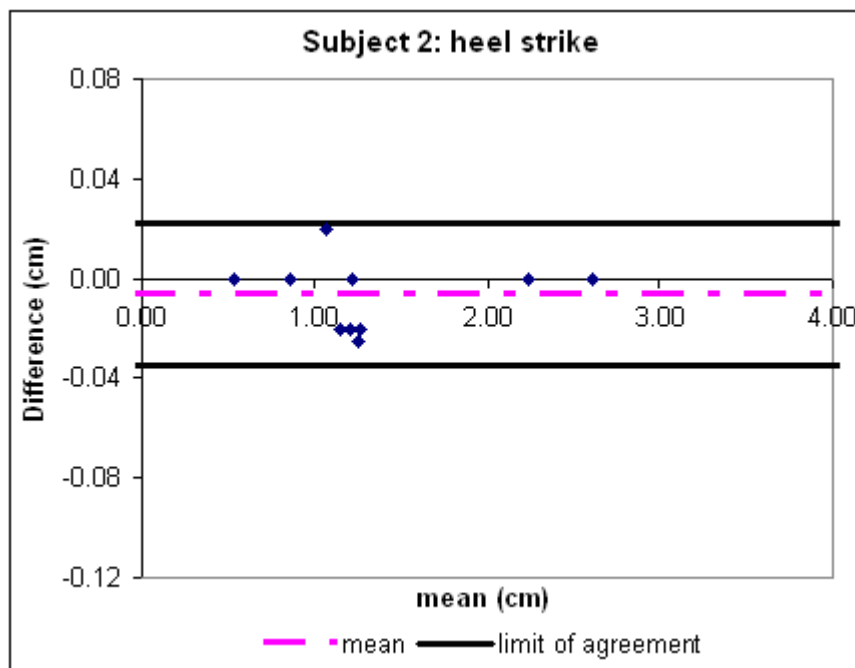


Fig 5.2.2 Graph plot of heel strike time difference for PD subject 1. Mean = -0.0065, st dev = 0.0141, upper limit = 0.0213, lower limit = -0.0348. All data points for were within limits of agreement. Half (5) were exactly repeated (zero difference)

5.2.1b Toe-off Times (Test-retest)

Table 5.2.2 shows the toe-off times marked for subjects 1 and 2 and on days 1 and 7. The mean of the differences in marked time indicated that the data points were within the upper/lower limits of agreement in both subjects. Marked toe-off times were exactly repeated i.e. zero difference in trial 7 in subject 1 and trials 1, 5, 8-11 in subject 2.

Toe-off Times (secs)									
	PD Subject 1					PD Subject 2			
	Day 1	Day 7	Mean	Difference		Day 1	Day 7	Mean	Difference
1	2.56	2.58	2.57	-0.02		1.32	1.32	1.32	0
2	2.48	2.49	2.485	-0.01		2.06	2.08	2.07	-0.02
3	1.88	1.94	1.91	-0.06		2.9	2.92	2.91	-0.02
4	2.36	2.38	2.37	-0.02		1.98	1.98	1.98	-0.005
5	1.96	2	1.98	-0.04		1.6	1.6	1.6	0
6	1.325	1.34	1.3325	-0.015		2.06	2.08	2.07	-0.02
7	1.88	1.88	1.88	0		2	2.02	2.01	-0.02
8	1.68	1.7	1.69	-0.02		1.78	1.78	1.78	0
9	1.2	1.22	1.21	-0.02		3.34	3.34	3.34	0
10	1.68	1.7	1.69	-0.02		1.84	1.84	1.84	0
11	1	1.06	1.03	-0.06		1.32	1.32	1.32	0
Mean of differences				-0.02591					-0.0085
95% Upper limit of agreement				0.012774					0.011
95% Lower limit of agreement				-0.06459					-0.028

Table 5.2.2 Toe-off times marked for PD subject 1 and 2.

The graphs below (figs 5.2.3 and 5.2.4) show the distribution of the plots of the mean of marked toe-off times against the difference in marked times on two different days. All the data points for subjects 1 and 2 were within 95% (upper and lower) limits of agreement for both subjects.

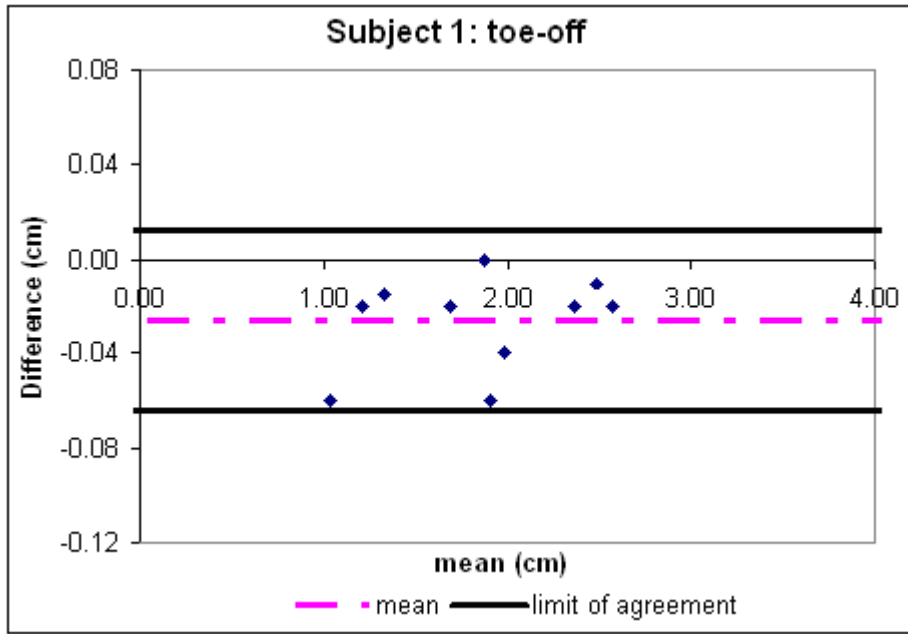


Fig 5.2.3 Graph plot of toe-off time difference for PD subject 1. Mean = -0.0259, st dev = 0.02, upper limit = 0.0127, lower limit = -0.06. All data points were within the limits of agreement. One toe-off point was exactly repeated

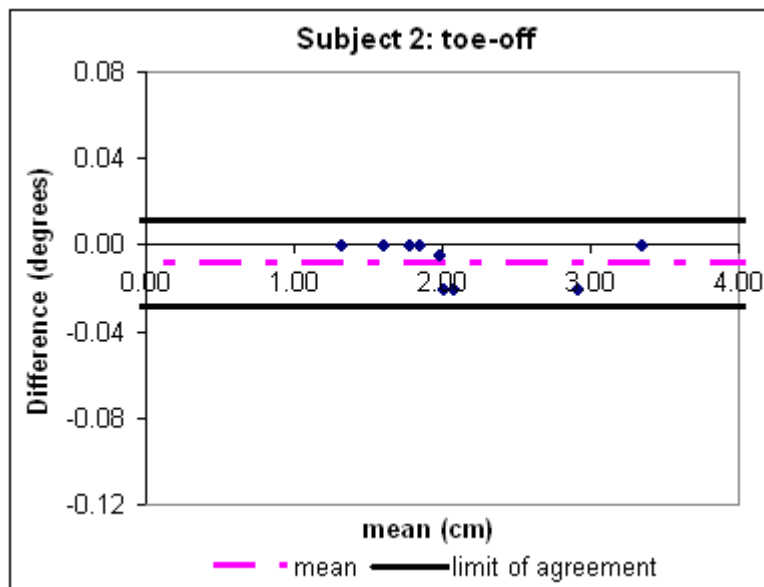


Fig 5.2.4. Graph plot of heel strike time difference for PD subject 2. Mean = -0.0085, st dev = 0.010, upper limit = 0.011, lower limit = -0.028. All data points were within limits of agreement. Half (5) were exactly repeated (zero difference)

5.2.1c. Summary

In the 7-day interval test-retest of manual marking of gait cycle events, all the repeated data points for heel strike and toe-off events were within the limits of agreement in both PD subjects tested thus proving good agreement of data and repeatability of the method. The mean differences in marking the timing of heel strike in both subjects on the two test occasions (an estimate of average bias) were less than six millisecond whilst the mean difference in marking of toe-off in both subjects were greater i.e. 25 and 8msecs. This is most likely because the position of the toe marker was on the head of the fifth metatarsal and not the hallux hence the variation occur in determining the exact time the hallux is lifted off the ground. However, 50% of the marked heel strike times were exactly replicated (zero difference in marked time) in both subjects whilst 50% of the toe-off times were replicated in the second subject thus showing an improvement by the investigator on toe-off times on the second subject especially in marking toe-off times. Therefore, this gait event marking procedure was within reliable limits and is repeatable. Hence, the influence of variation by the investigator in implementing the procedure can be seen as negligible especially on gait variables such as stride length and gait cycle velocity that are dependent on a determined gait cycle period. This procedure can be reliably applied in the data analysis of the main study.

5.3 Aim 2: Testing of the Modified Gait Laboratory Protocol

In contrast to the previous study on healthy gait, the present laboratory protocol was designed to analyse fast gait in addition to usual walking speed in people with PD. This was to test for the effect of increased walking speed on joint power in PD gait. Thus, the fast gait trials will also be tested in this pilot study. The use of the protocol in the previous study and the aim of the present study gave rise to issues that were considered and tested in the pilot study. Such issues are:

1. Obstruction of pelvic markers in subjects with non-swinging arm(s).
2. Missing the force platform by subject's foot.
3. Possibility of falls when PD subjects are asked to walk fast.

To address the above potential limitations the laboratory protocol was modified. The aim was to test these modifications on the other five PD subjects randomly (i.e. excluding the two from the previous test) during normal and fast walking. LED markers were placed on anatomical landmarks of the lower limb. The obstructed pelvic markers were often the anterior superior iliac spine marker and sometimes the posterior superior iliac spine marker. It was expected that people with PD will have reduced or no arm swing (Nieuwboer et al. 1998) hence it could constitute a greater obstruction to signals from the marker than in the gait analysis of healthy adults. Thus to improve marker-camera signal transmission the PD subjects were tested with three conditions: (1) arms free-to swing (2) arms folded (3) anterior extension of the arm of the pelvic frame using thin aluminium bars . Subjects were randomly assigned to these conditions and tested at both usual and fast walk speeds e.g. subject 1 & 3 (arms free gait only), Subject 2 & 5 (arms free & arms folded gait), Subject 4 (arms free + extended ASIS markers), and Subject 6 (extended ASIS markers).

To facilitate stepping on the force platform, the PD subjects were informed about the force platform but were asked to walk straight ahead and not to aim to walk on it because the start point will be adjusted. However, they performed 4-5 test walk to fine tune their start position to enhance stepping on the plate and to limit gait alterations.

To prevent falls and its consequences when the PD subjects were asked to increase their walking speed, they were required to wear a safety harness connected to an overhead carriage that runs parallel with the walkway. The harness was worn both in normal and fast gait trials as a study control measure across gait trials.

5.3.1 Results

The modified gait laboratory protocol was used in five sessions for five PD subjects respectively. The mean age, weight, height, UPDRS, Hoehn & Yahr and Mini mental scores of the subjects are as described in the table 5.3.1.

Subject	Sex	Age (years)	Weight (kg)	Height (m)	UPDRS (motor)	Hoehn & Yahr	MMSE
1	M	73	85	1.71	19	1.5	30
2	M	60	72	1.76	9	1	30
3	F	64	65	1.66	22	2	30
4	M	73	78	1.77	17	2.5	30
5	M	65	102	1.77	7	1.5	27
Mean		67.0	83.4	1.73	14.8	1.7	29.4
St dev		5.7	14.1	0.05	6.5	0.57	1.34

Table 5.3.1 PD Pilot subjects characteristics

The results of the total number of gait trials conducted per session are presented in tables 5.3.2-5.3.6. The trials include those with good step on the force platform, the trials with obstructed markers (hence not useful) and final useful trials after screening of the gait data. Some of the laboratory sessions were not completed due to development of technical fault

hence they had a lower number of total trials recorded especially in the fast gait trials i.e. in subjects 2, 3 & 4. The session and trials successfully conducted per PD subject are as presented below.

Arms Free Condition

Subject 1	Total trials	Trials with good force data	Number of trials with markers out of view	Useful trials
Usual gait	10 arms free	2	6	0
Fast gait	9 arms free	1	8	0

Table 5.3.2 PD subject 1 tested during usual and fast gait with the arms free to swing.

Subject 3	Total trials	Trials with good force data	Trials with markers out of view	Useful trials
Usual gait	11 arms free	4	5	3
Fast gait*	4 arms free	3	0	3

Table 5.3.3 Subject 3 tested with arms free to swing condition during usual and fast gait. *Stopped testing due to technical fault.

Arms Free and Folded Condition

Subject 2	Total trials	Trials with good force data	Trials with markers out of view	Useful trials
Usual gait	7 arms free	3	5	1
	4 arms folded	0	0	3
Fast gait*	6 arms free	3	6 during arms free testing	0

Table 5.3.4 Subject 2 tested in trials with arms free and folded. *Stopped testing due to technical fault. Arms free testing in all fast trials.

Subject 4	Total trials	Trials with good force data	Trials with markers out of view	Useful trials
Usual gait	9 arms free	9	8	1
	5 arms folded	4	0	4
Fast gait	6	5	0*	5

Table 5.3.5 Subject 4: tested in *arms free and arms folded trials.

Pelvic Frame Extension Condition

Subject 5	Total trials	Trials with good force data	Trials with markers out of view	Useful trials
Usual gait	10	10	4	6
Fast gait	10	8	0	8

Table 5.3.6 Subject 5 tested with extension of pelvic frame during usual and fast gait.

5.3.2 Summary

The gait analysis laboratory protocol tested in this pilot study was modified from the protocol used in the previous study of healthy subjects. The modification were made in order to achieve the aim of the main study which therefore required consideration of both the motor disability of the PD subjects and laboratory test condition i.e. a) possibility falls in PD when walking fast, b) influence of dual task and visual cues, c) reduced of arm swing that obstruct pelvic markers, and c) stride length variability. Thus, to minimise their influence on the acquisition of standard PD gait analysis, the use of overhead harness, informing on the presence of the force platform, extension of ASIS markers and were employed and tested.

The use of the overhead harness to prevent falls was used successfully as no fall was recorded in any subjects except a near trip in subject 3 during the third fast trial. The harness presented its own limitation because it was originally designed for safety and weight relief in gait rehabilitation and not for standard gait analysis. Although it runs in the midline parallel with the walkway, the subjects were aligned lateral to the midline to ensure only one foot struck the force platform. There was an initial tug of the harness's overhead carriage during gait initiation with the first step. However, the carriage automatically moves at the subject's walking speed hence no external influence or impedance to natural gait was added. The size of the harness strap around the subject trunk was a cause for concern as subject 2 reported difficulty in walking as naturally as desired. However, the safety of the participants was paramount throughout the study. Therefore, the harness was used for the main study with consideration of the limitations.

All the PD subjects were informed about the force plate measurements but not to aim for it as the walkway start point will be finely tuned if they missed it. Subject 3 (in the arms free condition) reported difficulty of walking naturally when trying to walk in line with the

platform and was observed (in trial 4 and fast trial 2) to the adjust stride length over the platform. Other subjects when asked reported no step adjustment. Other subjects reported not to focus on the plate but were more concerned with walking straight to the end of the walkway. It was concluded that although subjects will be made aware of the force platform, emphasis will be placed by the researcher on the single task of straight line walking and fine tuning of the start position. However, it is clear that the number of foot strikes on the force plate will vary between subjects within a group. Thus, final analysis of the gait would require consideration of the control number of trials that will be valid within the study's limitations

The obstruction of pelvic markers (especially the anterior superior iliac spine-ASIS) was variable across the sample studied. The subject 1 tested with the arms free to swing had more than half of the gait trials with obstructed ASIS marker (including the trials with force data). However, at fast walking almost all (8 out of 9) trials were obstructed indicating that this subject had little or no arm swing as would be expected in a person with PD presenting with rigidity and motor disability (motor UPDRS of 19). Similarly, subject 2 (motor UPDRS of 22) had five out of 11 trials obstructed. Interestingly, at fast walking speed, this subject had no trials with the view of camera on the pelvic marker being obstructed. This was also observed in fast walking of subject 4 thus depicting increase in upper limb mobility when speed of walking is increased.

The number of useful trials for analysis increased in the arms folded condition in for subjects 2 and 4, because the arms did not obstruction camera view. Similarly, in subject 5, with extension of the arm of the pelvic frame beyond the free swing arm, the number of useful trials also increased. However walking with arms folded has been documented to alter gait (Eke-Okoro, Gregoric, & Larsson 1997; Marks 1997) and it could contribute to differences

when compared to healthy adult gait measured in the previous study. This made the pelvic frame extension the most preferable option to be used in the main study of PD gait.

5.4 Pilot Study Summary

The procedure of marking gait cycle events by the researcher was reliably repeated hence measurement errors due to marker placement will be negligible and are not expected to affect gait data in the main study.

The PD subjects are less at risk of falling when test at fast walking condition with the use of a safety harness.

It is possible to inform PD subjects about the force platform and get them to walk without having to aim for the force plate when the start point is fine tuned. The number of gait trials with force platform data varied between subjects therefore a control number of trials for final analysis will be required.

Extension of the arm of the pelvic anteriorly brought the ASIS markers out of obstruction by the arm into camera view and thereby increased the number of useful trials. This will be the preferred method to be utilised in the main PD study.

The modifications on the laboratory protocol/ set up were used in the methods for gait analysis in the PD study. The methods and results of the PD are presented in the following chapter.

Chapter Six

Hip Pull-off and Ankle Push-off In Parkinson's Disease Gait

6.1 Methods

6.1.1 Introduction

Similar to the first study on healthy gait, the present study tested people with PD at their usual walking conditions. In addition, PD subjects were also tested at their fast walking speed to study the effect of increased walking speed on the suspected diminished joint power generation. There was an initial (home) examination session to screen potential participants with PD before they participated in the laboratory session. The laboratory protocol and set up for this purpose was used previously (in the first study) for the analysis of gait of healthy people. This gave rise to issues on the laboratory protocol/set up that required modification before testing people with PD. The suggested solutions to these issues were tested in a pilot study (chapter 5) and were incorporated into the methods of carrying out the present study on pull and push-off events during usual and fast PD gait. Therefore the following are the aims and objectives of this study:

6.1.2 Aim and Objectives

The aim of this study was to characterise hip pull-off and ankle push-off events in the gait of people with PD during usual and fast walking speeds. This was mainly by (1) describing the peak joint powers generated during the events off pull and push-off (2) describing the corresponding joint angular excursion and spatiotemporal component (3) describing the relationships between the gait parameters measured during the push-off and pull-off events. These objectives were carried out for both usual and fast walking speeds.

6.1.3 Recruitment

The approval to recruit and carry out this study was received from the research ethics committee of the School of Health Professions and Rehabilitation Sciences and sponsorship provided by the University of Southampton (appendix 5-7). A substantial number of 30 PD subjects was aimed to be recruited because previous studies that described joint powers in PD gait analysis had small sample sizes i.e. $n = 1, 4$ and 10 (Lewis, Byblow, & Walt 2000; Morris et al. 1999). The sample of people with PD was identified through the local Parkinson's disease societies of Southampton and Winchester. After obtaining permission from the chair persons of the societies to include the members in the study, a total of fifty postage paid information packages were sent to each society branch for distribution to members. The information package contained an invitation to participate in the study, an information sheet, a reply slip and a postage paid addresses return envelope (appendix 8).

Participants that expressed their interest to participate by filling and returning the reply slip were contacted via telephone and an initial appointment was made for the researcher to visit them at home. This was to carry out a clinical examination of the participants and to make a subsequent appointment for clinical gait analysis at the gait analysis laboratory of the general hospital.

To be eligible for inclusion into the study, participants needed to be between the ages of 50 and 80 years and had to have been diagnosed with PD (at least two years) with no other condition that affected their gait. They should be able to walk independently unaided and able to travel to Southampton General Hospital. They should also be able to understand and follow simple instructions and be medically stable. Subjects were ineligible to participate if they had any musculoskeletal or neurological dysfunction (other than PD) and other disability that can affect their gait. Potential participants were screened at home as having

Parkinson's disease by self-report and assessment/examination using the modified Unified Parkinson's disease rating scale (i.e. UPDRS, inclusive of the Hoehn and Yahr scale). Their cognitive ability (e.g. ability to follow instructions) was also assessed using the mini-mental state examination (MMSE) using a cut off score of 24 for eligibility to participate. These instruments are described further in the following section.

6.1.4 Instruments

The Unified Parkinson's Disease Rating Scale (UPDRS)

The UPDRS was used in this study to assess and screen into the study subjects that were affected with PD. The UPDRS (Martinez-Martin et al 1994) is a validated assessment tool for motor and non-motor signs and symptoms in PD (appendix 9). It consists of four sections pertaining to (1) mentation, behaviour and mood, (2) Activities of daily living (3) motor function and (4) complications of dopaminergic therapy. PD subjects are rated on items in each section on a score of 0-normal to 4-severe. The sections 1, 2 and 4 are interview based and PD subjects are required to provide answers representative of their condition over preceeding weeks. Conversely, section 3 is based on clinical examination of the motor function and represents the present clinical presentation of subjects' disabilities. The UPDRS also encompasses the Schwab and England activities of daily living scale and the Hoehn and Yahr (H&Y) staging scale. The H&Y scale (Goetz et al 2004) gives a general overview of the PD pathology by describing the level or stage of severity of the disease on scale of 1-5. H&Y 1 represents the mildest state of the disease with unilateral symptoms and 5 represents the state at which the PD subjects is bedridden or using a wheelchair fulltime. The intra and inter-rater test-retest reliability and the validity of the UPDRS has been previously examined and reported to be high for motor assessment in PD (ICC 0.9) (Martinez-Martin et al. 1994;Metman et al. 2004).

Mini Mental State Examination (MMSE)

The MMSE (Folstein et al 1975, Kahle-Wroblewski et al 2007) was used in this study to assess cognitive function especially the ability to follow instruction (as will be given in the gait laboratory) (appendix 10). It tests subjects in areas such as orientation, attention, memory, calculation, response to oral and written commands (totalling 30 points). Inter rater test-retest reliability of using MMSE has been reported to be high (ICC 0.98) (Bassuk & Murphy 2003). An MMSE score of less than 24 is considered as indicative of cognitive impairment. This was used as the cut-off point for exclusion from the study

Gait Analysis Laboratory

The gait analysis laboratory set up used in the first study with healthy subject was used in this study with people with PD (chapter 3). It is equipped with a two camera 3-D motion analysis system (Coda mpx 30) and force platform. These were able to acquire motion in terms spatiotemporal (gait cycle time and length, cadence and support period), kinematics (joint angular excursion) and kinetics (joint forces, moment and power). In addition to the previous laboratory set up for the healthy study, a safety harness system was used for the PD study. This consists of straps worn around the trunk and connected to an overhead carriage. The carriage, via an inbuilt motor, automatically followed the subject on a rail parallel to the walkway once movement is initiated instead of being pulled along.

6.1.5 Data collection Procedure

Session 1 (Home session)

Data collection was divided into two sessions i.e. at the subject's home and at the gait analysis laboratory to avoid overburdening the subjects in one long session. The first session at the subject's home was clinical examination and screening the subject for eligibility to

participate in the study. Disease specific information were taken such as the PD disease duration, disease severity and cognitive ability using the Unified Parkinson's disease rating scale (UPDRS), the modified Hoehn and Yahr scale and the mini mental-state examination (MMSE) respectively. Other preliminary information was documented such as informed consent, subject's age, sex, history of disease and medication.

Session 2 (laboratory session)

The second session of data collection was at the gait analysis laboratory of Southampton General Hospital and was scheduled to coincide with the mid-dose phase of PD medication cycle (i.e. approximately 1-2 hours after taking medication). The subjects' height, weight and width of the knee and ankle and pelvic depth were measured for use in gait data analysis. The subjects were required to be in shorts and bare footed for the gait analysis session. Twenty-two markers powered by designated battery boxes were placed bilaterally on the limbs either directly on the skin surface or on frames aligned with the pelvic thigh and leg segments (fig 3.2.1). In both normal and fast walk test conditions, subjects were required to wear a safety harness connected to an overhead carriage above the subject. This is to ensure against the risk of falling during fast walk trials and to serve as a control factor across both usual and fast walk conditions.

The markers placed on the PD subjects come into full view of the Coda cameras at approximately 1m before and after the force platform. Therefore to allow for acceleration and deceleration distances, the start point to walk was set approximately 2m away from the force platform. The start point was aligned such that it was lateral to the midline of the force platform so that only one foot struck the plate when the subject walk along the walkway and

the other foot the returning. Thus, the subject will not have to adjust the gait to step on the plate. A practice trial was carried out to fine tune the start point when the foot missed the plate and to determine the preferred leg to initiate walking.

It was planned that ten trials each would be collected for usual and fast walking speed tests. The usual walking speed trials were conducted first followed by fast walking speed trials to avoid carry-over effect of change in walking speed. The subjects were instructed to walk in a straight line along the walkway towards marks placed at either end of the walkway. At the start of a gait trial, the instruction given to subjects was ‘whenever you are ready, starting with your left (or right) limb, walk in your usual (or fast) way towards the other end of the walkway’. The PD subjects were given the opportunity to rest anytime in between trials whenever they felt they needed to especially between the usual and fast walk sets of trials.

6.1.6 Data Analysis

The kinetic and kinematic gait variables were extracted from the profiles generated within the selected normalised gait cycle of each limb of each subject. The data points were extracted from the late stance/preswing phase (i.e. pull-off and push-off events). All the individual gait trials were screened and selected for analysis if there was:

1. Complete force platform data (i.e. power is the product of moment of force and angular velocity)
2. Complete kinematic profile. (i.e. data is missing in discontinued profile hence leading to error in analysis)
3. Presence of at least one complete gait cycle for each limb (left and right limb data are used in final analysis)

4. No marker profile discontinuity (i.e. markers are linked for segmental analysis hence, a discontinuity distally affects proximal segments)
5. No contamination of data with measurement artifact (aberration in data can occur due to external influence such surrounding light or vibrations).

Similar to the healthy gait study, the number of complete gait trials with force platform data (where the whole foot struck the force plate) and absence of artifact and marker discontinuity was unequal across the subject group. Some individuals achieved up to six successful trials while others only produced one. For data analysis, the first two trials for left and right legs were used for each subject. After selection, the trials were processed and the parameters were extracted.

6.1.7 Outcomes Parameters

As was measured in the first study on healthy gait (chapter 4), the primary gait parameters that were measured during the usual and fast walking speeds trials as integrated by the motion analysis system were the peak powers and the associated joint angle changes during the pull and push-off gait events with the associated spatiotemporal variables i.e.:

1. The peak hip pull-off power (HPOP), ankle push-off power (APOP) and knee absorption power (KnAP)
2. The corresponding change in joint position (or angular excursion) i.e. at the hip joint (ROMh), the knee joint (ROMk) and the ankle (ROMa), during the events of pull and push-off.
3. The corresponding spatiotemporal variables associated with the powers and joint angular changes i.e. the gait cycle velocity, stride length, and cadence.
4. A secondary variable of Hip extension power (HExP) was also measured.

5. Pearson/Spearman correlation coefficient between the variables HPOP, APOP, ROMh, ROMa, gait velocity, stride length and cadence.

The outcome variables were analysed using SPSS 14.0 for windows. Descriptive statistics such as frequency statistics i.e. mean, standard deviation, histogram (with the curve of distribution) were used to describe the push-off and pull-off powers and the associated kinematics and spatiotemporal.

Cadence was derived from the time required to take left and right steps and then the equivalent numbers of steps per minute was estimated. The spatiotemporal variables were derived from the selected gait cycles (normalised to 100%) for the right and left sides of each subject and further processed by Coda report generator software. The following section presents the results of the main gait parameters of pull and push-off events and the corresponding spatiotemporal parameters.

6.2 Results

6.2.1. Sample characteristics

The data of eleven out of twelve people with PD, age ranging from age 50 to 70 years with a median age of 67 years were analysed for both usual and fast gait tests in this study (appendix 11). Almost all were males with disease duration less than 10 years i.e. from 3-9 years. However, the mean motor UPDRS score indicated moderate motor disability.

Sample (M/F)	10/1
Age (yrs)	66.4 ±5.07
Weight (kg)	75.09±16.4
Height (kg)	1.73±0.07
Disease duration (yrs)	6±3.1
UPDRS (motor)	24.3±9.98
Hoehn &Yahr	2.14±0.5
Mini mental score	28.9±1.5

Table 6.2.1 Demographics for PD subjects (n=11)

The mean Hoehn and Yahr score indicate that the group was mostly bilaterally affected with the disease with slight disturbance of balance. The subjects also had a high mini-mental score of (28.9±1.5) that suggested that their cognitive function was sufficient for participation.

The gait parameters during push-off and pull-off events in the usual and fast walking test were analysed for all the trials with complete data. These analyses are presented and compared in the following three sections as: (1) usual walking speed (2) fast walking speed and (3) Comparison of usual and fast PD gait with healthy usual gait. In both usual and fast walking speeds, the first two complete trials i.e. left and right sides (as carried out in the healthy study) were selected per subject for final analysis.

6.2.2. Usual Walking Speed in PD

6.2.2a. Hip Pull-off and Ankle Push-off

At usual walking speed, the mean peak power generated during pull/push-off events was greatest in the ankle push-off power (APOP) i.e. 2.77 ± 0.69 W/kg (tables 6.2.2) followed by the hip pull-off power (HPOP) i.e. 0.84 ± 0.24 W/kg. APOP also had the greatest power range (3.99-1.25W/kg) and with the standard deviation, the greatest variability within the PD group.

Power (W/kg)	Mean	Std Dev	Median	Minimum	Maximum
HPOP	0.84	0.24	0.85	0.48	1.23
HEXP	0.77	0.38	0.79	0.19	1.48
KnAP	-0.79	-0.41	-0.67	-1.65	-0.31
APOP	2.77	0.69	2.77	1.25	3.99

Table 6.2.2. Peak joint power generated during usual gait in PD (n=11)

6.2.2b. Joint Angular Excursion

The joint angular excursion during pull and push-off and knee flexion in late stance are presented in table 6.2.3. Angular excursion during hip pull-off (i.e. ROMh) was greater than angular excursion during push-off (ROMa). However, the greatest joint angular change was at the knee joint. The greatest variability (st dev) within the group in joint angular excursion was during hip pull-off event. The magnitudes of joint angular excursion across the three joints were normally distributed within the group.

Change in Joint angle (°)	Mean	Std Dev	Median	Minimum	Maximum
ROMh	39.7	10.2	39.2	24.2	62.3
ROMk	55.6	7.9	56.3	43.9	64.3
ROMa	28.7	7.1	26.5	19.5	40.2

Table 6.2.3. Associated joint angular excursion at the hip, knee and ankle

6.2.2c Spatiotemporal

At usual walking speed, the PD group walked with a mean gait cycle velocity with corresponding mean stride length and cadence as shown in table 6.2.4

Parameters	Mean	Std Dev	Median	Minimum	Maximum
Gait velocity (m/s)	0.97	0.24	0.95	0.54	1.39
Stride length (m)	1.11	0.19	1.11	0.89	1.41
Cadence (steps/min)	105	13	103	72	127
Single support (sec)	0.42	0.06	0.41	0.34	0.55
Double support (sec)	0.17	0.04	0.17	0.11	0.29

Table 6.2.4 Associated spatial and temporal values.

The gait velocity, stride length and cadence within the group were all normally distributed.

6.2.3 Fast Walking In PD

The following are the results of eight subjects that had trials with complete data during fast walking tests. Two trials were selected and analysed per subject i.e. a total of 16 trials were analysed for fast walking test.

6.2.3a Joint Powers

With increase in walking speed, the hip pull-off power (HPOP), ankle push-off power (APOP), knee power absorbed and hip extension power were all increased (tables 6.2.5). However APOP was less variable ($\pm 0.55\text{W/kg}$) within the PD group than was observed at usual walking speed ($\pm 0.64\text{W/kg}$). Conversely, HPOP was more variable than at usual walking speed.

Power (W/kg)	Mean	Std Dev	Median	Minimum	Maximum
HPOP	1.45	0.55	1.57	0.56	2.03
HExP	1.62	1	1.32	0.57	3.7
KnAP	-1.31	0.38	-1.32	-0.76	-1.99
APOP	3.39	0.53	3.2	2.84	4.43

Table 6.2.5 Joint power generated during fast walking speeds

The distribution of power generated across the PD group for HPOP, APOP, knee absorption and hip extension power were all normal.

6.2.3b Joint Angular Excursion

The values for joint angular excursion during hip pull-off and ankle push-off at fast walking speed are presented in table 6.2.6. The angular excursion during hip pull-off ($41.1 \pm 10.5^\circ$) was greater than ankle push-off excursion ($32.1 \pm 5.7^\circ$). There was a normal distribution of the angular excursion during pull and push-off at the hip, ankle and knee.

Joint angular excursion ($^\circ$)	Mean	Std Dev	Median	Minimum	Maximum
ROMh	41.1	10.5	41.8	22	54.7
ROMk	54.4	17.6	58.6	16.1	71.6
ROMa	32.1	5.7	31.3	24.3	40.5

Table 6.2.6 Joint angular excursion during fast walking speed

6.2.3c Spatiotemporal

At a faster walking speed, there was increased gait velocity, stride length and step rate (cadence) to values greater than at usual walking. The minimum gait velocity and stride length were greater than 1m/s and 1m respectively.

Parameters	Mean	Std Dev	Median	Minimum	Maximum
Gait velocity (m/s)	1.43	0.27	1.37	1.15	2.06
Stride length (m)	1.31	0.11	1.26	1.18	1.52
Cadence (steps/min)	129	16.3	125	108	164
Single support (sec)	0.35	0.04	0.34	0.30	0.45
Double support (sec)	0.10	0.30	0.11	0.05	0.16

Table 6.2.7. Associated spatiotemporal values during fast walking speed

The distributions of the spatial and temporal values within the PD group during fast walking were all normal except the gait velocity that was positively skewed.

6.2.4. Relationship between the PD Gait Parameters

During usual walking speed, there was a correlation of APOP with ankle joint angular excursion (ROMa), stride length, gait cycle velocity and cadence (table 6.2.8a) whilst HPOP also had a relationship with ROMa and cadence. Stride length was related to gait velocity.

Usual speed (n=11)	HPOP	HpExP	KnAbP	APOP	Gait Velocity	Stride Length	Cadence	ROM hip	ROM Knee
HpExP	0.47								
KnAbP	0.22	0.26							
APOP	0.60*	0.53	0.21						
Gait Velocity	0.48	0.64*	0.16	0.78**					
Stride length	0.31	0.50	0.18	0.63*	0.93**				
Cadence	0.68*	0.56	0.12	0.83**	0.81**	0.56			
ROMh	0.30	0.08	0.13	0.56	0.57	0.62*	0.39		
ROMk	0.39	0.07	0.18	0.50	0.65*	0.66*	0.51	0.84*	
ROMa	0.70*	0.40*	0.18	0.64*	0.39	0.40	0.34	0.42	0.38

Table 6.2.8a Correlation coefficients of pull and push-off powers with associated spatiotemporal and joint angular excursion. *Correlation is significant at the 0.05 level (2 tailed). **Correlation is significant at the 0.01 level (2 tailed).

In the fast walking test, there were no relationships between HPOP/APOP and the other gait components (likely due to the small sample size-8). However a moderate effect size correlation was observed between HPOP and gait velocity; ROMa and HPOP/ APOP/ KnAbP; gait velocity and stride length/ cadence/ROMk, Hip extension power and stride length (table 6.2.8b).

Fast speed (n=8)	HPOP	HpExP	KnAbP	APOP	Gait Velocity	Stride Length	Cadence	ROMh	ROMk
HpExP	-0.1								
KnAbP	0.66	0.05							
APOP	0.23	-0.37	0.21						
Gait Velocity	-0.52	0.28	0.47	-0.16					
Stride length	-0.41	0.66	0.14	-0.32	0.61				
Cadence	-0.54	0.19	0.64	-0.26	0.69	0.14			
ROMh	0.23	-0.63	0.42	0.45	0.000	-0.09	-0.50		
ROMk	-0.26	0.02	0.16	0.23	0.54	0.69	-0.11	0.61	
ROMa	-0.58	0.1	0.64	-0.59	0.000	-0.18	0.42	-0.41	0.52

Table 6.2.8b Correlation coefficients of pull and push-off powers with associated spatiotemporal and joint angular excursion at fast walking speed. Correlations were not significant at either 0.01 or 0.05 level (2 tailed).

6.2.5 Comparison between Usual and Fast Gait

The result from the measurement of the main parameters of HPOP and APOP, gait velocity, stride length and cadence during usual PD gait were statistically compared (t test) with findings from usual healthy gait (presented in chapter 4) and fast PD gait.

6.2.5a Usual Gait: PD versus Healthy

At usual walking speed, there were no statistical significant differences between the PD and healthy group in the analysis of the first two trials per subject (table 6.2.9).

Parameter	PD (n=11) usual gait n trials = 22	Healthy usual gait (n=14) n trials = 28	P value
HPOP (W/kg)	0.84 (0.24)	0.80 (0.30)	0.75
HEXP (W/kg)	0.78 (0.37)	0.60 (0.27)	0.20
KnAbP (W/kg)	-0.78 (-0.41)	-0.72 (0.43)	0.72
APOP (W/kg)	2.77 (0.66)	2.48 (0.49)	0.23
Gait velocity	0.98 (0.24)	1.05 (0.13)	0.36
Stride Length (m)	1.11 (0.18)	1.17 (0.11)	0.36
Cadence (steps/min)	105 (14)	107 (9)	0.49
ROMh	39.7 (10.2)	36.1 (4.9)	0.61
ROMk	55.6 (7.9)	51.8 (6.1)	0.24
ROMa	28.7 (7.1)	25.6 (6)	0.24

Table 6.2.9. Healthy versus PD usual walking speed

6.2.5b. PD Usual and Fast Gait

Eight of the 12 PD subjects with usual gait data had corresponding fast gait trials. Their results showed that when people with PD walked fast, HPOP, APOP, gait velocity, stride length and cadence significantly increased except for the change in joint angular excursion (table 6.3).

Parameter	PD usual (n=8)	PD fast (n=8)	P value
	n trials = 16	n trials = 16	
HPOP (W/kg)	0.82 (0.30)	1.45 (0.56)	0.015
HE _x P (W/kg)	0.68 (0.36)	1.62 (1)	0.04
KnAbP (W/kg)	-0.79 (0.29)	-1.31 (0.38)	0.008
APOP (W/kg)	2.70 (0.78)	3.4 (0.53)	0.05
Gait Vel (m/s)	0.93 (0.25)	1.43 (0.27)	0.001
Stride Length (m)	1.08 (0.17)	1.31 (0.12)	0.005
Cadence (steps/min)	104 (16)	129 (16)	0.009
ROM _h °	35.8 (7.5)	41.09 (10.57)	0.72
ROM _k °	53.7 (8.1)	54.4 (17)	0.99
ROM _a °	28.7 (7.14)	32.12 (5.73)	0.31

Table 6.3. Usual versus fast gait walking speed in PD subjects.

6.2.6 Individual Data and Results

The individual demographic data, clinical presentation (table 6.4) and gait results (table 6.5a,b) of the gait of the eleven PD participants are presented in this section. There were ten males and one female PD participants in the group. Their ages ranged from 59-73 years with disease duration ranging from 2 to 11 years. Their measure of motor dysfunction (by UPDRS motor section-56 points) ranged from 7 to 36 - with most scoring above 20 (n=7). Most of the participants (n=9) can be considered bilaterally affected (2-2.5 on the Hoehn and Yahr scale). Their mean cognitive ability as measured with the 30 points Mini-Mental State Examination test (MMSE) ranged from 27-30 (greater than the cut off point of 22).

S/N Participants	Sex	Age Yrs	Weight Kg	Height M	UPDRS (56)	Hoehn & Yahr	MMSE	Duration Yrs
PD1	M	70	86	1.80	19	2	29	9
PD2	M	60	65	1.74	33	2.5	30	9
PD3	M	71	71	1.67	29	2.5	30	6
PD4	M	59	47	1.66	26	2	27	9
PD5	F	69	53	1.56	27	2	29	3
PD6	M	65	97	1.77	35	2.5	27	11
PD7	M	72	85	1.73	36	2.5	30	4
PD9	M	67	70	1.81	29	2.5	30	4
PD10	M	60	72	1.76	9	1	30	2
PD11	M	73	78	1.77	17	2.5	30	3
PD12	M	65	102	1.77	7	1.5	27	5
mean		66.4	75.1	1.73	24.3	2.14	28.91	6
Std		5	16.9	0.07	9.9	0.50	1.50	3
median		67	72	1.76	27	2.50	30	5

Table 6.4. PD participants' individual demographics and clinical characteristics

As was observed in the healthy group, during usual walking speed, the PD participants had associations between the parameters of ankle push-off power (APOP), hip pull-off power (HPOP) and the gait velocity thus permitting categorisation of the participants according to these three parameters. Thus, participants had the three gait parameters as either above the group mean/median i.e. High power category, or below the mean/median i.e. Low power category, or one of HPOP or APOP above or below the mean/median i.e. Mixed power category (table 6.5a,b)

		HPOP	
		Lower than mean-median	Above median
APOP	Lower than mean-median	n=3 Low Power Generators PD1, PD3, PD9	n=2 PD4, PD6
	Above median	n=2 PD2, PD7	n=4 High Power Generators PD5, PD10, PD11, PD12

Table 6.5a. PD participants in categories of High, Low and Mixed powers.

	Sex	APOP	KnAb Pow	HPOP	HpEx Pow	Vel (m/s)	Stride L (m)	Steps/ Min	ROMh	ROMk	ROMa
Low Power											
PD1	M	2.38	-0.31	0.84	0.79	0.86	0.91	110	38.45	58.00	19.50
PD3	M	2.64	-1.26	0.79	0.49	0.81	0.96	101	38.55	55.15	30.60
PD9	M	1.25	-0.48	0.48	0.40	0.54	0.89	72	28.30	43.90	20.85
Mixed Power											
PD2	M	2.60	-0.73	1.08	0.62	0.96	1.15	103	39.20	64.30	38.65
PD4	M	2.88	-0.68	0.49	0.19	1.02	1.22	102	46.90	62.10	23.25
PD6	M	2.77	-0.62	0.58	0.88	0.83	0.95	102	24.20	44.20	25.10
PD7	M	2.32	-1.17	0.93	0.56	0.81	0.96	102	31.15	45.55	25.00
High Power											
PD5	F	3.99	-0.86	1.23	0.96	1.08	1.12	121	41.80	56.30	40.15
PD10	M	3.18	-0.31	0.86	1.48	1.15	1.30	103	43.55	55.05	37.05
PD11	M	3.25	-1.65	0.89	0.87	1.30	1.41	111	42.80	63.95	30.05
PD12	M	3.17	-0.54	0.98	1.32	1.39	1.38	127	36.04	57.95	26.25
Median		2.77	-0.68	0.86	0.79	0.96	1.12	103	38.55	56.30	26.25
Mean		2.77	-0.78	0.83	0.78	0.98	1.11	105	37.36	55.13	28.77
st dev		0.69	0.42	0.24	0.39	0.25	0.19	13	6.93	7.52	7.17

Table 6.5b. Gait characteristics of individual PD participant in categories.

6.2.6a The Low Power category (PD 1,3,9)

The PD participants in this category generally walked with lower gait velocity, pull and push-off power when compared to PD participants in the high power category. They had a Hoehn and Yahr score of 2-2.5 i.e. bilaterally affected and higher UPDRS motor score of 19, 29 and 29 respectively. However, their disease duration appear to be in reverse magnitude i.e. 9, 6 and 4 years to the UPDRS score (table 6.5b). These participants appear to be more affected than the high power category of subjects. Within the category, PD9 was the youngest of the three participants (i.e. 67 years versus 70 and 71 years) and the tallest (1.81m). Although, the participant had one of the lowest disease duration in the entire PD group and the least in the low power category (i.e. 4 years compared to 9 and 6 years of PD 1 and PD 3), he presented with about the poorest gait feature (gait velocity, stride length HPOP, APOP) within the low power category and the overall PD group.

6.2.6b The Mixed Power category (PD2,4,6,7)

This category of participants had either only the APOP or HPOP associated with the gait velocity but not both powers simultaneously. They tended to have greater disease duration (9, 9 and 11 years) than those in the low power category except for the PD7 with a 4 years disease duration. The most common feature of the four participants in this category was that they generally presented with higher UPDRS motor scores i.e. (33, 26, 35, 36 respectively) compared to participants in the other categories.

Participant PD6 (65 years old) was the tallest within this group (i.e. height 1.77m/s). In spite of presenting with a high UPDRS score of 35 and 11 years disease duration, he walked with high APOP but had low HPOP, gait velocity and stride length and cadence. This was in contrast to the features observed in participant PD7, who was older (aged 72 years), shorter (1.73m) and with a slightly higher UPDRS motor score (UPDRS: 36) but 4 years disease duration. PD7 walked with lesser APOP but had a greater HPOP compared to PD6. Nevertheless, the above two participants (i.e. PD6 and PD7) walked with lower gait velocity, stride length and cadence in the category.

6.2.6c The High power category (PD5,10,11,12)

At their usual walking speed, participants in this category generated the highest power magnitude during push-off, pull-off and had the highest gait velocity. It is of note that although PD5 and PD11 had moderately high UPDRS motor dysfunction scores of 27 and 17 respectively, all four participants in this category were the ones with low disease duration of 3,2,3,5 years respectively. They also had lower Hoehn and Yahr score was between 1 and 2.5. However, the only female participant (PD5-aged 69years) presented with a high motor dysfunctional score of 27 that contrasted against her generating the greatest APOP (3.99W/kg) and HPOP (1.23W/kg) in the entire PD group. This also contrasted with fact that

she was the shortest (1.56m) in the entire group. Although her gait velocity and stride length were the lowest in the high power category, they were greater than the magnitudes presented by nearly half of the participants in the entire PD group.

6.2.6d Fast Walking In PD

Eight PD participants tested during fast walking had viable gait data that was appropriate for analysis. The usual and fast gait parameters were compared for the eight participants i.e. PD 3 and 9 (Low power), PD 2,4,6,7 (Mixed Power) and PD 5 and 12 (High Power). All the 8 participants significantly increased their gait velocity, stride length and cadence (table 6.6).

Fast walk	APOP (W/kg)	PkAbP (W/kg)	HPOP (W/kg)	HpExPow (W/kg)	Vel (m/s)	St L (m)	Cad (s/min)	ROMh°	ROMk°	ROMa°
PD2	2.6	-0.73	1.08	0.62	0.96	1.15	103.48	39.2	64.3	19.5
Fast	3.41	-1.52	1.9	2.14	1.44	1.43	120.19	54.75	71.6	40.5
Change	0.81	-0.79	0.82	1.52	0.48	0.28	16.71	15.55	7.3	21
PD3	2.64	-1.26	0.79	0.49	0.81	0.96	101.73	38.55	55.15	30.6
Fast	4.43	-1.99	1.38	1.06	1.27	1.22	124.36	45.5	58.9	35.15
Change	1.79	-0.73	0.59	0.57	0.46	0.26	22.63	6.95	3.75	4.55
PD4	2.88	-0.68	0.49	0.19	1.02	1.22	102.57	46.9	62.1	23.25
Fast	3.24	-0.76	0.56	0.57	1.46	1.39	127.72	52.1	67.8	26.55
Change	0.36	-0.08	0.07	0.38	0.44	0.17	25.15	5.2	5.7	3.3
PD5	3.99	-0.86	1.23	0.96	1.08	1.12	121.33	41.8	56.3	40.15
Fast	3.92	-1.52	2.03	0.84	1.37	1.24	125.06	44.7	58.45	38.95
Change	-0.07	-0.66	0.8	-0.12	0.29	0.12	3.73	2.9	2.15	-1.2
PD6	2.77	-0.62	0.58	0.88	0.83	0.95	102.63	24.2	44.2	25.1
Fast	3.02	-0.93	1.2	1.42	1.38	1.18	136.65	33.35	16.15	24.3
Change	0.25	-0.31	0.62	0.54	0.55	0.23	34.02	9.15	-28.05	-0.8
PD7	2.32	-1.17	0.93	0.56	0.81	0.96	102.63	31.15	45.55	25.2
Fast	3.3	-1.37	1.96	2.02	1.34	1.28	126.33	38.95	47	31.05
Change	0.98	-0.2	1.03	1.46	0.53	0.32	23.7	7.8	1.45	5.85
PD9	1.25	-0.48	0.48	0.4	0.54	0.89	72.97	28.3	43.9	20.85
Fast	2.84	-1.28	1.78	1.22	1.15	1.26	108.33	37.4	50.1	28.9
Change	1.59	-0.8	1.3	0.82	0.61	0.37	35.36	9.1	6.2	8.05
PD12	3.17	-0.54	0.98	1.32	1.39	1.38	127.89	36.04	57.95	26.25
Fast	2.99	-1.11	0.82	3.7	2.06	1.52	164.42	22.05	65.15	31.55
Change	-0.18	-0.57	-0.16	2.38	0.67	0.14	36.53	-13.99	7.2	5.3

Table 6.6. Gait parameters of PD participants (n=8) during usual and fast walking speeds

There were changes in the joint power generated at the ankle and hip, and in the power absorbed at the knee. These power changes were mostly increased for each participant. However, two participants presented with reduced power when walking fast i.e. PD 5 and 12. Whilst PD5 had reduced APOP (i.e. usual: 3.99W/kg to fast: 3.92W/kg) accompanied by reduced plantarflexion excursion (i.e. 40.15° to 38.95°), PD12 also presented with reduced HPOP (0.98W/kg to 0.82W/kg) accompanied by reduced hip flexion during pull-off (36.04° to 22.05°). Contrary to expectation, PD12 did not present with reduced plantarflexion excursion (ROMa) but walked with reduced APOP (3.17W/kg to 2.99W/kg). In addition to the observations on PD5 during fast walking, there was a reduction in hip extension power (0.96W/kg to 0.84W/kg).

Another notable feature during fast walking was the reduction in the joint angular excursion in PD6. The participant presented with a 11 year disease duration (the highest within the PD group) and had one of the highest motor dysfunction i.e. motor UPDRS score of 35 (after PD7 with 36 score). This participant presented with an apparently great reduction in knee flexion (ROMk: 44.2°-16.1°) and reduction in plantarflexion at push-off (ROMa: 25.1°-24.3°). However, in spite of these reductions, as was observed throughout the PD group, PD6 was able to increase gait velocity, stride length and presented with the greatest change (increase) in cadence.

6.2.7 Summary of Result

The gait analysis result of 11 PD participants with age range 59-73 years, disease duration of 6 ± 3.1 years with mean Hoehn and Yahr stage of 2.14 ± 0.5 were presented.

All the gait measures generated within the PD sample were normally distributed. The PD participants walked with a mean gait velocity below 1m/s. The mean power generated during push-off and pull-off event was greatest at the ankle i.e. push-off power. The standard deviation indicated that there was a greater variability in ankle push-off power within PD group than the hip pull-off power and knee absorption power. Conversely, the corresponding change in joint angular excursion was greatest during the event of hip pull-off than ankle push-off. There was also a greater variability (st deviation) in angular excursion at hip pull-off than at ankle push-off.

In the fast walking speed test, a total of 16 gait trials with complete data were analysed in a group of eight PD participants. There was a normal distribution of the gait parameters during the fast walking in the PD group with the exception of the gait cycle velocity. The PD subjects walked at an increased speed that was significantly greater than that observed at usual walking speed. There was a greater variability in cadence in fast walking as was observed at usual walking. Significant increase was also observed in the HPOP and APOP with increased variability in HPOP and decreased APOP variability in contrast to findings at usual walking speed. The mean change in joint position (angular excursion) corresponding to hip pull-off and ankle push-off were not significantly changed during the fast walking speed.

Correlation analysis showed that at usual walking speed, push off and pull-off powers had a relationship with ankle angular excursion at push-off. Ankle push-off power also correlated

with stride length and gait velocity but these relationships were not observed at fast walking speed with a smaller sample size for analysis except between Hip extension power and cadence.

The results of the eleven PD participants can be categorised according to three categories of High, Low and Mixed power generators based on the participant's gait values relative to the groups' mean of HPOP, APOP, and gait velocity i.e. above or below the group mean of these gait parameters.

The participants in the Low power category (PD1,3,9) walked slower and generated less APOP and HPOP than other PD participants in the entire group. They presented with greater UPDRS (motor dysfunction) score than those in the high power category.

The participants in the high power category generated greater APOP, HPOP and walked faster than other PD participants. They commonly had low disease duration and Hoehn and Yahr scores than other PD participants.

In the entire PD group, the participants in the Mixed power category were mostly the ones with the greatest motor dysfunction scores with mostly high disease duration than other members of the PD group. They either walked as well as the participants in the high power category or as poorly as those in the low power category.

During the fast walking test, all 8 participants significantly increased their gait velocity but PD5 and PD12 (high power category) had reduced APOP whilst PD12 alone had reduced APOP and HPOP. PD6 from the Mixed power category also had a reduced KnabP at fast walking but with accompanying reduction of joint angular excursion at the knee and ankle.

6.3 Discussion

6.3.1 Introduction

The purpose of this study was to characterise hip pull-off and ankle push-off events when people with PD are walking at their usual and fast walking speed. Whilst people with PD are known to characteristically walk slowly, it is also known that there is a relationship between walking speed and joint power (Lelas et al 2003). It can be argued that reduced ankle push-off power (and possibly hip pull-off power) in PD gait results from their slow (self selected) walking speed. Therefore, in this study, the effect of increased walking speed on the gait components especially pull and push-off powers was tested. To help understand PD usual and fast gait the following questions were asked (1) Are pull and push-off powers reduced in PD gait as indicated from the previous studies? (2) If joint powers are related to walking speed, will fast walking speed improve the diminished power to normal levels? (3) How will people with PD alter their gait to walk fast. Is it by an increase in cadence or stride length or by both in similar proportions? Therefore, the gait analysis laboratory protocol designed for this study was used to quantitatively measure the PD gait parameters at their usual and fast walking speed during the period at which they were in their individual on-phase of PD medication.

6.3.2 Participants

The age of the PD participants in this study ranged from sixty years (55%) to seventy years (34%) and one subject in the fifties (9%). The mean and median age was 66 years and 72 years- depicting the typical age prevalent in PD. The previous investigations on gait kinetics in PD recruited subjects that were comparable in age to the present study. Their subjects ranged from 52-68 years (Ferrarin et al 2004), 52 to 84 years (Lewis et al 2000) and mean

age 63 ± 8 years (Sofuwa et al 2005). Furthermore, the disease severity (on phase medication testing) presented by the participant recruited in the present study was 2.14 ± 0.5 i.e. predominantly 2.5 ($n=6$) and 2 ($n=3$) on the Hoehn and Yahr scale compared to 2.8 ± 0.8 (Lewis et al 2000), 2.6 ± 0.6 (Sofuwa et al 2005) and 3.7 ± 0.7 (off-medication, Ferrarin et al 2004). Thus, suggesting that although the subjects in the present study were bilaterally affected with some instability there was some similarity to the subjects in the other studies. However, the lesser mean disease duration of six years compared to others with greater length of disease duration i.e. 16.9 ± 5 years (Ferrarin et al 2004), 11.2 ± 3.8 (Sofuwa et al 2005) and 9.1 ± 5.7 years (Lewis et al 2000) may indicate that the subjects in the present study were either less affected or the rate of disease progression was greater. The measure of motor disability as reported only by Sofuwa et al i.e. motor UPDRS score of 16.1 ± 6.4 , gave support that the subjects in the present study did present with considerable disability (motor UPDRS 24.3 ± 9.98) at mean duration of 6 years compared to that of other studies at greater length of duration.

Motor blocks such as freezing of gait did not feature during laboratory measurements. However, two of the subjects suffered from dyskinesia which commonly featured at the end-of dose of the PD medication cycle. Dyskinesia can be disabling especially in its severe form and could further inhibit gait. However all the timings of laboratory measurements were planned to coincide with the mid-dose phase (1-2 hours after) of their medication cycle. Ultimately, in the present study, it was only those with stable condition that were able to walk unaided that participated in the gait laboratory trials.

6.3.3 Pull and Push-off in PD Usual Gait

Contrary to expectations, the gait features of the PD subjects were not worse than the healthy subjects measured in the first study (Chapter 4). This was an unusual finding. One possible

reason for this observation is the variability of gait between the PD subjects. Inspection of the PD group's standard deviation indicated that the gait of some PD subjects had values that were comparable to healthy values. However, only two of the PD subjects can be referred as being mildly affected as they had motor disability UPDRS score of 7 and 9 whilst others had greater disability that ranged from 17 to 19 and 40. Consideration of the mean gait parameters indicated that the PD subjects tended to walk relatively slower than the healthy subjects because they had lesser mean gait velocity (0.98m/s versus 1.05m/s), a lesser stride length (1.11m versus 1.17m) and a lesser cadence (105 steps/min versus 107steps/min). Interestingly, this lesser mean gait feature was not observed for hip pull-off and ankle push-off powers as expected. Instead, the mean pull and push off powers were greater than healthy values (i.e. pull-off power of 0.84W/kg versus 0.80W/kg and push-off power of 2.77W/kg versus 2.48W/kg) and also greater than what was previously observed in other studies.

Only two studies (Ferrarin et al 2004, Sofuwa et al 2005) have actually quantitatively reported ankle push-off value in PD during usual (self selected) walking speed within the on phase of medication. In the study by Ferrarin et al (2004) on the effect of deep brain stimulation (DBS) on PD gait, the push-off power recorded (1.24 ± 0.44 W/kg) when PD subjects (n=10) were in the on phase of medication state (without DBS) was less than that observed in our study (2.77 ± 0.69 W/kg). Their reduced ankle push-off power was recorded with reduced gait velocity and stride length (i.e. 0.94 ± 0.21 m/s and 1.02 ± 0.24 m) than that of the present study (0.97 ± 0.24 m/s and 1.11 ± 0.19 m) although their subjects had comparable motor disability (motor UPDRS 24.4 ± 14.4) as the subjects in this study (24.3 ± 9.9). It can be argued that the self selected (usual gait) was less than that observed in the present study which may have led to the reduced powers. However, there are other factors that could have effected the lesser gait features observed in their subjects. These could be related to the depletive effect of surgery, the carry over effect of electrical stimulation (which can be

inhibitory or excitatory along an electrode pathway) or the pre and post operative (prolonged) bed rest, none of which were accounted for by the investigators. The study by Sofuwa et al (2005) eliminated most of these limitations by measuring the gait of PD subjects ($n=15$) also in the on phase of medication at baseline (before any intervention). Although their subjects were younger (63.1 ± 14 years versus 66.4 ± 5 years), less affected (motor UPDRS 16.1 ± 6.4 versus 24.3 ± 9.9) and walked on a similar length (eight metres) of walkway as subjects in the present study, they were observed to walk with slower gait velocity ($0.94\pm0.27\text{m/s}$ versus $0.97\pm0.24\text{m/s}$), lesser stride length ($1.03\pm0.16\text{m}$ versus $1.11\pm0.19\text{m}$) and lesser push-off power ($2.33\pm0.15\text{W/kg}$ versus $2.77\pm0.69\text{W/kg}$). From these previous observations and the present findings it can be suggested that the PD subjects in the present study tended to walk faster and generated greater ankle push-off power.

Increased power generation is often associated with increased gait velocity but in the present study, the PD subjects' slower walking velocity in relation to the healthy subjects indicated that other factors might be responsible for the PD pull and push off powers not being significantly less than the healthy values. It is possible that PD pull/push-off powers were increased to compensate for impairment in gait. Indeed in the PD group, both pull and push-off powers had positive relationships with the ankle joint angular excursion during push-off but neither pull or push-off powers had any relationship with the hip joint angular excursion during pull-off. This was contrary to the observation in healthy group where there was a relationship between the hip pull-off power and its corresponding hip joint angular excursion during the pull-off event and also the relationship between ankle push-off powers and its corresponding ankle joint angular excursion during push-off. This suggests that the PD subjects with low ankle joint excursion (plantarflexion) during push-off generated both low push and pull-off powers and vice versa suggesting the magnitude of movement at the ankle is not only dependent on ankle push-off power but also on hip pull-off power. The role of hip

pull-off power in this regards appear to be compensatory by aiding forward lift of the foot into swing.

6.3.4 Fast Walking Speed

When asked to walk fast, the PD subjects were able to significantly increase all components of their gait compared to usual walking. The PD subjects altered the mean gait velocity from 0.93m/s to 1.43m/s i.e. about 54% increase. This was accompanied by increase in both mean stride length (by 21% i.e. 1.08m to 1.31m) and cadence (by 24% i.e. 104 to 129 step/min) suggesting an almost proportional increase but a tendency to use more of cadence than stride length to alter their gait. The tendency in people with PD to increase cadence to walk fast has been previously observed. In a series of tests by Morris et al (1994b) it was observed that “when given verbal encouragement to walk faster”, the PD subjects were able to increase their gait velocity to the preferred walking speed of healthy controls with significant increase in cadence but significant decrease in stride length. However, they also observed that in the absence of verbal feedback, fast walking in the PD subjects was significantly slower than fast walking in healthy controls in terms of cadence as well as gait velocity and stride length. Conversely, in another study (Morris et al 1994a) the authors reported that at preferred fast walking speed, there was no significant difference in cadence between PD and healthy controls. However, the increased PD stride length and gait velocity were less than healthy values albeit indicating as observed in the present study that people with PD can alter their gait features to walk fast.

The generation of increased pull and push-off powers during PD fast walking test gave some indication that joint power does change with gait velocity. Interestingly, the relationships between ankle push-off power with ankle angular excursion, and gait velocity observed during usual walking velocity were not observed at fast walking speed. Neither was there any

relationship between pull-off power and the other gait components. It is possible that the lack of any relationship between the powers and the fast gait velocity and the other gait components was because other factors were contributory to the fast walking in PD gait. Indeed, within the gait cycle of healthy people, there is a third power burst that is generated during midstance phase that corresponds to hip extension. In the PD group, the hip extension power during usual walking speed was lesser than pull and push-off powers (i.e. $0.68 \pm 0.36 \text{ W/kg}$ versus $0.82 \pm 0.3 \text{ W/kg}$ and $2.70 \pm 0.8 \text{ W/kg}$). However, at fast walking speed, this hip extension power ($1.62 \pm 1 \text{ W/kg}$)- although still less than ankle push-off power ($3.4 \pm 0.53 \text{ W/kg}$)-was greater than the hip pull-off power ($1.45 \pm 0.56 \text{ W/kg}$). It also had the greatest proportional increase (139 %) compared to 77% pull -off power (i.e. $0.82 \pm 0.3 \text{ W/kg}$ to $1.45 \pm 0.56 \text{ W/kg}$) and 25 % (i.e. $2.70 \pm 0.78 \text{ W/kg}$ to $3.4 \pm 0.53 \text{ W/kg}$) of push-off power. The increased role of hip extension power was further corroborated in the greater effect size association with stride length (correlation analysis; $r = 0.66$) that was not observed with either pull or push-off powers. This suggests that during fast walking speed, as more propulsion is required, more power is recruited not only as increase in pull/push-off powers but with significant contribution from the hip extension power. In a previous study on fast walking speed of healthy people (mean age 79 ± 6 years), Judge et al (1996) observed that the only significant contribution to power was by hip pull-off power that increased from $1.1 \pm 0.3 \text{ W/kg}$ to $1.9 \pm 1 \text{ W/kg}$ i.e. by 72%. However, unlike the present study, the authors did not observe any significant increase in ankle push-off power and hip extension power. Despite this observation, the ankle push-off power they recorded at fast walking speed remained the power with the greatest magnitude whilst the hip extension power was the least. This contrasted with the findings in the PD group in the present study where the hip extension power was greater than the hip pull-off power. The comparative differences between the two studies suggest that the strategy utilised to increase gait speed differs between healthy and PD subjects i.e. PD utilises increase in pull and push powers with

increased role of hip extension power whilst healthy subjects utilise only increase in hip pull-off power.

Currently within the PD gait rehabilitation literature, it has been reported that in PD patients presenting with dysfunctional gait, walking can be improved using frontal cortex attentional strategies resulting in movement being carried out not automatically as in healthy cases but “consciously”(Nieuwboer et al 1997, Morris 2000). This enables signals to by-pass the defective basal ganglia. However, in the present study, the ability to increase gait component without being instructed as to the specifics of the increment suggests the role of mental specification of gait that may have been neglected in current PD gait investigative studies and rehabilitation. In PD gait rehabilitation what may be required is to ask subjects to walk fast in addition to informing subjects (with reduced pull/push-off powers) of the relationships between gait speed and joint power and consequence of diminished power generation. A real time feedback mechanism to the PD subject may be required for this purpose that quantifies power generated with the subjects having a target value to attain optimum (healthy) levels at a particular walking speed.

Due to the present study limitations, self selected fast walking in healthy subjects was not examined. Hence, to be conclusive, it is recommended that the mechanism of fast walking in both PD and healthy subjects be further investigated in future studies using a greater subject sample size in similar study conditions to examine and/or confirm what the findings of the present study suggests.

6.3.5 Individual Gait Data

Gait characteristics of all the PD participants in the present study were measured during their usual walking speeds. Individual variations were observed in the gait characteristics among

participants and no two participants walked exactly the same way. However, there were trends in gait characteristics that could be related to the clinical or physical presentation in individual participants. The trend observed was consistent with the association between gait velocity and the power generated during ankle push-off and hip pull-off events. Therefore, by exploring these associations, some participants were at the lower end of the “gait spectrum” i.e. exhibiting low velocity, low pull and push-off power (low power category). At the other end were the participants who had high gait features of pull/push-off power and high gait velocity (high power category). Participants in the mixed power category had only one of pull or push-off power as a high gait characteristic.

The common feature of those in the low power category was that they had greater motor UPDRS (motor dysfunction) score than those in the high power category. Hence, their incapacity to walk as well and generate sufficient joint power was related to their clinical presentation. In spite of one of the participants (PD1) having a relatively high cadence (110 steps/min) compared to the group average (103 steps/min), their stride lengths were generally lower than the group average. A high knee joint angular excursion in PD1 (contrary to low ankle and hip joint) did not sufficiently affect the stride length. Neither did a high hip joint angular excursion affect stride length in PD3 (contrary to low knee and ankle joint). Also, an overall low joint angular excursion in PD9 was associated with a low stride length. Hence the participants had variable joint angular excursions that were differentially reflected in their low stride lengths.

In contrast to the participants in the low power category, participants in the high power category presented with clinical characteristics that indicated lesser motor dysfunction within the PD group with disease duration less than 5 years and Hoehn and Yahr score between 1-2.5. Their gait was affected to a lesser extent when compared to other PD participants. All

the participants in the high power category were able to walk with relatively greater gait velocity in association with the stride length and cadence. However, decreased motor dysfunction (resulting from lower disease severity) may not always explain the high power generation observed. Most notable was the female participant (PD5) with contrastingly high motor UPDRS score in comparison to the others in her category (i.e. UPDRS score 27 versus 9, 17 and 7) and the shortest (1.56m) in the overall PD group. In spite of her physical and clinical features that could contribute to diminished the kinetic and kinematic gait features, this participant presented with the overall greatest pull and push-off power (in the PD group), high velocity, stride length cadence and joint angular excursion. It is possible that the strategy used by this participant was to generate an excessively high power at both pull and push-off to counteract the other factors that could limit her gait such as joint stiffness resulting from rigidity. A more plausible explanation is the difference in gender i.e. gait between males and females may have been differentially affected by PD. It is known that in female PD patients, there is a neuroprotective effect of the hormone oestrogen (Behl & Manthey 2000, Shulman 2007) possibly translating to the degree of movement disorder presented in female patients. However, the process of how motor functions are wholly or selectively affected between the genders require further investigation in future studies.

The other participants presenting with different characteristics were those in the mixed power category. The prominent feature was that majority of participants within this group had a high motor disability score (UPDRS score 33, 26, 35 and 36) when compared to the overall PD group. Participants PD2 and PD4 who had a lesser motor disability within the category (score 26 and 33) walked with a higher gait velocity and stride length than participants (PD6 and PD7) with higher UPDRS scores (35 and 36) had low gait velocity and stride length and joint angular excursion. This highlighted the effect of the disease severity on their gait performance.

The significant feature of participants in this category was that although they mostly had greater motor dysfunction than those in the low power category they did not all walk with gait velocities as low as those in the low power category. The lower stride length observed in PD6 and PD7 (with the greatest motor dysfunction) is consistent with the dysfunction in stride length generation that has been reported in PD gait studies (Morris et al 1994, Morris et al 1996). The high joint power the two participants generated at either hip (or ankle) appeared not to be reflected in the spatial-temporal gait features (gait velocity and stride length). High power generation may be used to facilitate gait or enhance compensatory gait strategies. However, neither PD6 nor PD7 appeared to utilise the any strategy to affect stride length or gait velocity. Therefore, the efficient translation of neither joint power into gait with adequate stride length and velocity is questionable in the two participants. Thus, in biomechanical analysis of gait in PD, it is relevant to note that the patients walking poorly and clinically presenting with high clinical high motor dysfunction may be generating a high joint power that may be indicative of another dysfunction (e.g. poor foot-floor clearance). Therefore further assessment of the associated joints with their gait components will be relevant in differentially identifying the type of gait dysfunction clinically presented and the possible causes.

Overall, in consideration of the clinical and gait presentations of the participants in the present study, the trend seemed to be that those with mild disease severity (as reflected in degree of motor dysfunction) were in the early stage of the disease and therefore had minimal gait impairment i.e. they generated high power. As motor dysfunction increases, the gait features decreased. Therefore, the affected individuals will most likely be characterised into the low power category. However with further increase in disease severity (irrespective of rate of progression), participants may utilise strategies of high power generation. The high

power generated may not be as effective in some patients to walk as well as those with minimal disease severity.

The relevance of these gait power categories is that whilst the clinical presentation of most of the patients was associated with their gait's speed-power relationships, there were exceptions where the clinical or physical features did not match the presented gait features. Such cases would have been masked by those with predominant gait features when findings are generalised as done in larger studies. Thus, clinical examiners of PD gait should be aware of such possibilities of 'inconsistent' cases and examine the possible causative factors in such cases.

Fast walking

In the fast walking test, all the participants appeared to have preserved their capacity to increase their gait velocity with increase in joint power, spatial and temporal characteristics. However, there were some interesting observations. The most prominent was in the participants that generated high power initially during the usual walking speed test. At fast walking speeds, the two participants from the high power category (PD5 and PD12) had a reduction in their joint powers. Push-off power and hip extension power were reduced in PD5 whilst both pull and push-off powers were reduced in PD12. There was also a resultant effect on the joint angular excursion as PD5 had a reduction in plantar-flexion at push-off whilst hip flexion at pull-off was reduced in PD12. Both participants appear to have utilised a power reduction strategy to facilitate their walking speeds. This was not expected. Although, their joint powers were reduced, their stride lengths and cadence were not adversely affected indicating the power reduction may have been utilised for another function associated with fast walking. A possible theory is that in considering the principle of lower limb segmental energy transfer and net balance across the joints (Winter 1991), power may have been

absorbed for other critical functions such as coordination and balance (Sadeghi 2001). This may have been very critical in both participants especially as they were already generating the highest pull-off power and gait velocity within the entire group during usual walking test that may have predisposed them to some instability. However, a more objective segmental analysis to examine this possibility should be explored in future studies.

6.3.6 Conclusion and Summary

Testing of the PD gait at usual walking speed demonstrated no significant difference from the healthy subjects earlier studied. This could have been affected not only by the sample size of the PD group but also by the inter-subject differences in addition to the PD sample being moderately affected by the disease.

At usual walking speed, PD subjects tended to walk faster than subjects in previous studies and also generated greater power at ankle push-off. The power generated during hip pull-off was associated with ankle angular excursion at push-off thus suggesting a compensatory strategy to aid plantarflexion.

The PD subjects were able to voluntarily significantly increase their walking speed by altering both stride length and cadence. The hip extension power and hip pull-off power appear to play a increased role in fast walking and exhibited greater percentage change than the ankle push-off power. Albeit ankle kinetics appear to be most important in altering walking speed with push-off power remaining the major power burst at both usual and fast walking speed.

The ability to voluntarily walk fast is still preserved in PD in the presence of minimized external triggers and cues such as verbal commands. The change in walking speed was via cadence and stride length with a slight tendency for the PD group to employ more steps (cadence) than stride length.

The categorisation of the gait of individual PD participants was associated with their clinical presentations. Thus, the gait features presented by patients could facilitate the examination and identification of the state of motor dysfunction and disease severity of PD patients.

Future studies on power generated in PD gait especially on push-off and pull-off gait events should further explore these observations. This will help to further clinically elucidate the different gait features presented in the PD pathology.

Chapter Seven

Overall Discussion

7.1 Introduction

This investigation grew out of the lack of clarity and insufficient information on the pull-off and push-off events of people with PD during walking. The overall aim was to explore these two gait events with the view that an understanding of these events might contribute to the body of knowledge on PD gait.

Push and pull-off events are believed to be contributory to forward propulsion and progression of the lower limbs. Hence it was argued that the difficulty of the individuals affected by PD in adequately carrying out gait specifics such as stride length and gait velocity is related to deficiencies in the performance of the pull and push-off events. In addition, it was theorised, based on the relationship between walking speed and power, that when people with PD are instructed to walk fast they will improve their pull/push-off power. For these reasons, the investigation aimed to highlight pull/push-off events during usual and fast walking speeds as very little research had been carried out in those area. The result showed no significant difference in the pull/push-off of healthy and people with PD at usual walking speed. This did not corroborate the previous studies that investigated the PD gait measuring mainly the spatiotemporal parameters.

There are plausible reasons for the conflicting findings that could have affected the present study's results in addition to other factors that were considered as the study was developed and carried out. These factors could have been methodological, for example, sample characteristics (size, demographics and presentation e.g. disease severity), instrumentation, measurements and type of analysis. There were other possible factors affecting PD gait that may have interacted with the study's methodological factors that suggest that the gait observed in most studies should be cautiously considered. Such factors include the influence of subjects' cognitive capacity and ability to compensate for gait deficiencies.

7.2 Compensation in PD Gait

Gait compensation is an alteration in function of any of the determinants or factors of gait (either centrally (CNS) or peripherally) to adjust for or accommodate a dysfunction caused by another determinant of gait. This accommodation of the dysfunction is often towards a functional walking pattern that may appear abnormal but is useful in the presence of limiting pathological factors. The use (and effect) of compensatory strategies by PD patients with dysfunctional gait in most PD clinical gait studies cannot be overruled. There appear to be two types of compensation reported in PD studies i.e. (1) those observed to occur as result of the patient's response to the deficiency caused by the PD pathological process without therapeutic intervention and (2) those that occur following some sort of therapeutic intervention.

In the absence of intervention on PD gait, some types of gait compensation have been reported in PD gait literature (Morris et al 1994a,b., Nieuwboer et al 2004, Jobges et al 2004, Rocchi et al 2006). Morris et al (1994a, 1994b) reported that due to the difficulty in generating sufficient stride length by people with PD gait hypokinesia (i.e. slowness), there was a tendency to compensate by increasing their step rate/frequency i.e. cadence. Their findings also suggested that cadence was the gait feature least affected by PD pathology and is readily modulated in PD gait dysfunction to adjust for gait hypokinesia. Jobges et al (2004) also reported a similar gait feature where they observed that the ability to modulate the step rate even as a compensatory means i.e. taking a high number of steps was utilised by PD sufferers with postural imbalance to recover from triggered instability. Rocchi et al (2006) reported that in PD patients with difficulty in initiating gait, the patients maintained a narrow stance width to compensate for the difficulty and slowness in step initiation when starting with a wider stance.

An EMG study on gait of people with freezing of gait reported compensatory (increased) tibialis anterior muscle activation that aided the pulling of the foot into the swing phase of gait for a better foot-floor clearance (Nieuwboer et al 2004). Whilst the above mentioned compensatory strategies appear to contribute to attaining a gait pattern useful in ambulation, they may lead to consolidation of abnormal gait patterns if not corrected thus possibly contributing to complications such as falls (in case of narrow stance) and festination/freezing of gait (in case of increased cadence). Although, the use of a compensatory strategy is not reported in all PD gait studies, the possibility of it already being utilised by PD patients in response to the pathology should not be overruled.

The most demonstrable form of compensation in people with PD has been reported in studies that used intervention strategies to facilitate a patient's capacity to by-pass the utilisation of the defective basal ganglia neural system. This was achieved by attentional strategies e.g. instructing the patient to focus their attention on (or think about) the specific aspects of movement. e.g. "walk with large steps", "take better heel strike (or toe rise)" (Morris et al 1996, Behrman et al 1998). This strategy is known to have a dependency on the fronto-cortical regions (cognitive mechanism) of the brain (Morris 2000).

An alternative to the internally generated (attention) mechanism is the use of external stimuli such as cueing modalities. Researchers investigating the use of external cueing modalities have shown that such modalities (auditory, visual and somatosensory) can serve as beneficial compensatory strategies in improving gait in PD sufferers (Lewis et al 2000, Van Wegen et al 2006, Baker et al 2007, Ferrarin et al 2008).

PD gait is known to worsen with the performance of dual motor tasks (e.g. walking and talking/cognitive challenge) that compete for attentional resources (Morris et al 2000,

Rochester et al 2004). However, it has been observed that the use of an external e.g. auditory cue (also requiring attention like the attentional strategies) while performing another task places less demand on the attentional resources. More importantly, it is believed to facilitate the allocation of attention on critical aspects of gait thus improving gait in presence of the other tasks competing for attention (Rochester et al 2004, Rochester et al 2007). The external provision of such constant and continuous critical aspect of gait such as the spatial components (from visual cues) and temporal component (from auditory rhythmic cues) may reduce the demand on planning and preparation and monitoring required for movement. This could prove very useful especially in cases where the ability to use cognitive (executive) function have declined with disease severity.

PD is known to affect cognitive function (attentional and executive) with its deterioration with the disease severity and progression. It was therefore critical in the present study that participants were able to follow instructions and carry out the tasks of walking according to the protocol of investigation. Therefore, none of the PD participants recruited had cognitive impairment as they all scored above 24 on the Mini-mental state of examination tests. Hence, they would have been able to utilise their cortical attention mechanism if required. This may have been contributory to the lack of significant difference between the gait of PD and healthy participants because the PD participants may have had their gait improved much more than their “natural” gait due to heightened (cortical) awareness or vigilance to comply with the laboratory protocol thus drawing their attention to the single task of walking. Morris et al (1996) observed a similar pattern when their PD patients used (learned) attentional strategies when there was an external cueing demand to do so. However, the patients then reverted to their typical hypokinetic gait when they perceived they were not being monitored. It is therefore possible that the participants in the present study were involuntarily compensating with attentional mechanism albeit in the absence of specific gait modifying

instructions. The possibility of such occurrence in PD gait studies needs to be taken into account. Alternatively, in future studies, gait analysis could be carried out in the environment of activity of daily living of the participants with limited observer presence to achieve a gait that is near as possible to a participant's usual pattern. In conclusion, it is possible that what is being presented and measured as the usual gait of people with PD may be gait that has been compensated for or adapted (albeit temporarily) to the environment and/or pathology. Thus, future studies should endeavour to limit or account for these influence and extraneous factors.

In the present study, there were PD participants who generated high hip pull and ankle push-off powers with analysis showing an association between hip pull-off power and ankle plantarflexion at push-off. It is possible that the range of ankle plantarflexion could have been affected by hip joint power to lift the limb into swing as a compensation for any limitation in ankle joint angular excursion. It can therefore be stated that a key observation from the present study is that:

Ankle push-off and hip pull-off power are not always diminished in PD but may be altered (e.g. increased) especially when compensatory strategies are exploited.

All PD subjects were able to walk fast with increment in gait components. Therefore, it can be stated from the outcome of the present study that:

The capacity to significantly increase gait speed is preserved in PD with capacity to increase cadence and stride length.

Also preserved is the ability to change gait features such as power more than the usual and healthy gait values. There was a tendency to increase more of cadence than stride length although inspection of the data showed that some subjects had more percentage increase in

stride length than cadence. Thus, the internal specification of gait features (as opposed to external specification by stimulus) to and above normal levels is preserved and attainable in PD during self selected fast walking speed.

‘Asking’ a person to voluntarily walk fast by their self selection of speed, stride length and/or cadence could also have triggered the utilisation of an attentional strategy that by-passes the use of the defective basal ganglia with internal specification of gait that alters the external gait components such as stride length and/or cadence. The limitation to this strategy is that the attentional resources are naturally limited; therefore in the presence of an additional demand (i.e. environmental or task) that surpasses such resources can increase the difficulty of walking in PD. However, as earlier mentioned, external cueing strategies compared to the internal attentional mechanism provide a consistent and continuous flow of temporal and spatial information that place less demand on attentional resources (Rochester et al 2004, Rochester et al 2007). Such strategy would require constant monitoring (Morris et al 1996) and the constant availability of the modalities whenever in demand. Patients can benefit from both internal-attentional and external strategies because the combination of both strategies has been found to be equally effective (Baker et al 2007). This implies that in the rehabilitation of PD, gait training programme can be used to harness the preserved capacity in fast walking in combination with external cueing modalities to emphasize, improve and reinforce the component of PD gait that is deficient especially early in the disease stage before cognitive capacity and positive pharmacological effect begin to decline.

7.3 Factors and Limitations Affecting the Studies

The Sample

Recruitment

At the onset of the investigation, the difficulty of recruitment of PD subjects was not fully anticipated. Beside the strict selection criteria for participation, it was considered a very optimistic option to invite potential participants from the PD societies that had a high membership. It was expected that the proposed investigation into the problem with PD gait would generate sufficient interest and encourage participation. However, the response to invitation was lower than expected. People living with PD may show disinterest in participating in activities especially outside the home environment (Dujardin et al 2007, Levy & Czernecki 2006). This may have influenced the overall response rate.

Conversely, another factor not accounted for but is questionable is the issue of motivation of participating subjects during the test sessions. It could be that the participants were the enthusiastic ones that responded to invitation to participate. Thus, their gait deficiencies may have been masked by improved performance. Willingness to participate may be linked to subjects' expectations e.g. gratification (or cost-benefit) from their personal contribution to the advancement of research into PD.

The sample size

The study presented with a small sample size that diminished the power to detect any statistical difference between the gait of the healthy and people with PD. In addition, the lack of homogeneity in the clinical presentation and demographics of each group may be related to the variability in the gait outcomes. The absence of homogeneity in the clinical presentation (especially in those with the same disease duration but significantly different

disease severity) and the gait outcome may be indicative of the multivariate nature of the disease. Thus, it will be relevant in future studies with the same test conditions, to do a power analysis to derive a sample size with sufficient power to detect differences between PD and Healthy gait. This will permit the examination and sub-analysis of gait under other factors considered to affect gait (e.g. age and gender) in addition to studying the effect of possible co-variants such as disease duration and severity. By analyzing the gait outcome of a larger sample, those with common features can be readily identified and characterised.

In the testing of fast walking in PD participants, the absence of fast walking data for the healthy group in similar testing conditions makes it difficult to conclude on the findings on PD fast gait. For example, it is not known if the strategy used by the PD participants (that generated high power at usual gait speed) in reducing their joint powers during fast walking is similar to the strategy utilised by most of the healthy participants. It is therefore suggested that future investigative studies on healthy gait should explore, describe and establish the kinetic strategies utilised by healthy people tested in similar conditions of the present study. However, within the PD group ($n=8$), the effect size of the change from usual to fast walking was sufficient to detect differences in pull/push-off powers, gait velocity and stride length. To highlight the significant correlations between the gait variables, a bigger effect size (e.g. $r = 0.7$) will be necessary in future studies. This means that in future gait analyses studies larger sample sizes with sufficient power to detect not only gait changes but also associations between the gait parameters is used. A larger sample size will also permit the analysis of differences in gait due to gender, age and discrepancies between left or right lower limb that may have been masked due to pooling of the data.

Gender Differences

The predominant male gender of the final participants could have been influenced by the presence or absence of the carers such as partners/spouses living with them. According to the PD society (Parkinson's Disease Society UK, 2008), the strain involved in the care of partners with disabilities affects the quality, dynamics and most likely the length of their relationship. This could mean a longer duration of stay with partners by women than men. As males are often the car drivers in the home, females with PD without partners/spouses may not have the required assistance to travel and participate in the study. This is coupled with the fact that men are more affected with PD than women (Shulman 2007). Therefore, I often got responses to participate from male PD sufferers accompanied by female spouses. This is not unique to the present study as most of the other previous studies on PD often involved more male participants than females (Morris et al 2005, Ebersbach et al 1999 & Nieuwboer et al 2001). Thus, in the present study, the healthy group had more females than males (9:5) in contrast to the PD group with only one female. This may indicate that the results were more reflective of a comparison between healthy females against affected males. Evidence from other studies suggests that females walk differently to male. Females have been observed to walk with reduced stride length and higher cadence than males (Auvinet et al 2002, Blanc et al 1999, Sutherland et al 1998) and sometimes with greater ankle power associated with the cadence (Kerrigan et al 1998).

On the basis of these previous observations, it may be possible in the present study that the gait of the healthy group (predominantly female) will not be significantly different from the PD participants (mostly male) whose otherwise greater gait features such as gait velocity, stride length and joint power have been attenuated by the PD pathology. Some findings suggest that gender differences may play a role or modulate symptomatic presentation and motor performance/deterioration in PD (Scott et al 2000, Haaxma et al 2007, Rochester et al

2008). However, little is known on how this is translated into PD kinetics i.e. kinetics differences (joint moments and powers) between genders affected with PD. Future studies that provide such evidence will highlight and elucidate any contributory effect of gender on PD gait such as increase/ decrease in the cadence or joint power.

To limit a predominantly male gender type of subject participation as occurred in the present study, I suggest that a home based investigation be explored and developed in future studies. Not only would this improve participation levels of both genders but also of potential female participants who are not be able to participate because of the reasons mentioned. This method will provide a more representative sample with gait features measured in the environment of subjects' daily function. A limitation to a home based investigation is the sheer sophistication and size of the equipment involved. A single unit portable Coda (camera) system is available that can be used to record the kinematics however there remains the problem of force platform measurements and its synchronization with the more easily measured spatiotemporal parameters and the possibility of limited camera view.

Age differences

It is well known that the effect of age on gait is significant irrespective of the gender being studied. Generally, older people walk slower with shorter stride length than their younger counterparts (Blanke and Hageman 1989, Judge 1996). The age criterion for participation in the present study was 50-80 years. This was because PD predominantly affects people of this age range. The selected age range encompassed three categories (50-60 yrs, 60-70 yrs, 70-80 yrs) that could permit sub-analysis of these age categories and allow the effect of (or differences due to) age progression even among the elderly to be explored. However, the number of individuals that participated and were used in the final analyses was small thereby limiting the possibility of such analysis. Examination of the ages of the participants showed

that the number of participants in the healthy and PD groups between 60-70 years were similar (n=6:6). However, the healthy group had slightly less participants between ages 70-74 years (n=3:4) but had more participants than the PD group between ages 50-60 years (n=5:1). What this reflects is that not all the participants were exactly matched in age due to the smaller sample size of the PD group. The results from the healthy group with more participants in the 50-60 years category could have been influenced by the less diminished gait features of a “younger” and faster participants (in terms of speed and stride length) than in the PD group. It would be expected that a group comparison would show significant difference between the healthy and PD groups. However, the power to detect the difference due to the small sample size for both groups was lacking.

Pooling of left and right leg data

In the measurement of gait of healthy individuals, asymmetry of left and right sides can be seen in varying degrees. In gait analysis, it is not uncommon to find that the left and right side data are not exactly the same as observed in the present study. Some authors (Allard et al 1996, Sadeghi et al 2000) have suggested that possible differences in gait values between limbs (however negligible) may reflect individual functional differences of the limbs e.g. laterality. It is also possible that when an individual is walking the consecutive left and right gait data are representative of the gait features at a specific position and time of measurement. Thus, the use of one force platform meant that only one step on the force plate was measured in a trial (a single journey along the walkway) while the other limb's force data was measured in another trial- usually the return journey. Therefore, gait data of either limb may differ because they were of different trials or of different measurement time in a laboratory session-although time intervals (for rest and positioning) between trials were relatively small (approximately 15-20 seconds). However, it can be assumed that healthy individuals walk with a consistent gait. Although, gait data can be affected by any alteration

or adjustment in step by the participant, this would have been identified by visual monitoring during the testing sessions and by data inspection afterwards on the 3D segmental analysis output. In contrast, any kinetic asymmetry can only be detected by using more than one force platform to measure consecutive steps thus limiting (and accounting for) differences between limbs due to analysis of force data from different trials.

The inclusion of PD pathology to the testing conditions may accentuate any existing asymmetry of gait features in the individuals. PD is known to have a unilateral onset and may dominantly affect a side (Toth et al 2004; Katzen et al 2006). Thus, a participant who has right side laterality and a right side disease onset (or dominantly affected side) would present with co-confounding factors of the gait outcome depending on which of the factors is primarily examined. This may contribute to the difficulty in characterisation of PD with the gait features. In future studies, the solution will be to have PD case studies that chart the left and right gait data over a period. Thereby, the differences can be attributed to disease severity or progression rather than the laterality that is usually constant.

In the present study, the decision to pool left and right leg data was based on the consideration of the study's limitation of the number of trials with complete data during single session measurements, and the lack of consecutive contralateral limb's force data that would have presented comparable gait features between overlapping gait cycles in the same trial. However, pooling left and right gait data may be more representative of an individual's characteristic gait generally affected by PD. Future studies should consider individual cases to highlight individual difference in left and right gait data.

The equipment

Another factor that may have affected the performance of PD subjects was the use of the safety harness. Whilst the use of this equipment met the ethical requirements for subject-researcher safety in the laboratory, the overall effect in affecting the level of confidence and fear of falling could not be estimated. It is possible that in conjunction with the PD subjects' level of motivation, their confidence was enhanced by such safety precaution.

The inverse dynamics technique utilised by the motion analysis (Coda mpx30) system in this study (as with other studies on kinetics) provides a vast amount of information which helps to understand motion and its causative forces. The sophistication of the technique in gait analysis is often matched by the vast amount of data that is yet to be utilised in clinical analysis and rehabilitation. Even with such potential to provide objective insight into movement dysfunction, what are not always highlighted in gait analysis studies using inverse dynamics based systems like Coda are the assumptions and limitations inherent in such systems. The 3-D models created by these systems assume that the human joints are frictionless and that the lower limbs are rigid with uniform distribution of mass. In addition, the centres of rotation of deep joints like the hips are estimated since markers cannot define them. Most of the lower limb segment parameters are estimations and approximations with ratios derived from cadaveric measures that are then used in gait analyses of different people irrespective of body morphology. Therefore, like most advanced technology systems, it is yet to be perfected but nonetheless it provides useful estimations in practice of clinical motion analysis.

The analysis

Analysis of the gait variables after laboratory measurements presented with the issue of the associated number of trials that will provide the most valid outcome. Some have argued that

the fewer the trials the less the variability and the more the reliability (Davis 1997, Morris et al 1999). Others have argued for more numbers of trials to reduce variability and increase the reliability (Davis 1997, Monaghan et al 2007). It appears that there is no standard number of trials but decisions appear to be made based on the study design and conditions. In the present study, the highly unequal number of useful trials available between subjects gave rise to the question of which number of trials was valid for final analysis. Therefore, I had to carefully consider and decide on the control number of trials that was suitably valid for analysis within the limitations of this study. I suggest that instead of having a recognised standard number of trials used in the gait analysis, different standard number of trials for final analysis should be assigned and established for different pathologies with consideration of their gait variability and/or consistency.

The issue of variability cannot be over emphasised. In individuals, it can indicate the effect of intrinsic factors (e.g. pathology) or their response to extrinsic (environmental) factors. Whilst averaging values can give strong indication of the predominant features in the study sample, I have questioned the rationale of group averaging especially when there is the possibility of subjects being differentially affected by the disease. The dominant ones will mask individual features. Thus, one of the challenges to investigators who study PD is the problem of characterising the sample. However, studies in PD gait analysis should attempt as much as possible to characterise and identify the different features inherent within the study group.

7.4 Conclusions

All the measurements and observations made in this study was during the self selected walking speeds for the healthy subjects and PD subjects in the on phase of their medication cycle. Group average analysis did not show any difference in the pull and push-off events between both study groups thereby suggesting the possibility that the expected reduction in ankle push-off power may not be applicable to all cases of PD as some subjects have gait values greater or lesser than healthy values. The fast walking test highlighted the fact that the PD subjects still preserved the capacity to walk fast with the ability to significantly alter their gait features most especially the joint powers to increase gait velocity.

7.5 Clinical Implications

At the stage people with PD become bilaterally affected and are still independently ambulating, they may present with diminished or increased gait components. The increase in a gait component such as joint power as observed in some of the PD subjects can result in high energy expenditure (power is also energy produced per unit time) and be contributory to tiredness often experienced in some people with PD during community ambulation with longer walkways. This ultimately may have a negative impact on their quality life and daily activities. Therefore in PD clinical (gait) examination and management, clinicians/therapist could focus more attention on the ankle joint active range of motion and its limitations with the aim to reduce any limiting factor necessitating a subject to adopt compensatory strategies such as increase in hip pull-off power. In addition to clinical examination, there could be assessment of PD joint power generated and the energy expenditure when walking with the clinical goal to balance power (limit excess power generation) between the lower limb joints and minimise energy expenditure.

It is inevitable that PD gait deteriorates as the disease progresses. As part of the progressive paradigm of physiotherapy management of PD, the capacity to increase gait speed could be maximised and/or maintained from the onset of subclinical symptoms. Motor training and reinforcement could be directed at gait component such as stride length that can be translated into motor function in the later stages of the disease to minimize gait deterioration.

7.6 Recommendation for Future Studies

In the present investigation, there were limitations that could be improved on to corroborate the present findings:

1. The healthy and PD study sample were small. Future investigation could significantly increase the number of participants into the study thus also allowing for subgroup analysis on gait differences due to age, gender, disease severity and duration.
2. It is recommended that changes in pull and push-off events be studied in individual healthy and/or PD subjects instead of group analysis.
3. A longitudinal study of a cohort of subjects will make it possible to chart and characterise pull/push-off over the period of the disease progression and stages instead of a single session in time as performed in this present study.
4. It will be more informative and beneficial to study the relationship the power generated for propulsion and progression have with energy expenditure and endurance to identify the efficiency of the gait presented by people living PD.
5. Repeated exposure to gait training conditions and strategies that can utilize motivation and reduced fear of falling may prove valuable in reinforcing positive gait features that are self selected by patients. This should be in conjunction with constant assessment by therapist to minimise abnormal gait pattern.
6. Effort should be made by clinical gait analyst and rehabilitation specialist to examine gait in conditions similar to the daily (home) conditions to which participants are subjected to than in a controlled environment the gait analysis laboratory provides.

7. The gait variables used and often described in studies are not yet used to determine how disabled a subject with PD is. It would be very useful if a factor system is developed that integrates gait features of velocity, joint power, stride length and distance walked with other demographic features and/or information from other clinical tools. This will make it possible to grade and characterise gait disability thus facilitating diagnosis and treatment.

These recommendations are what I view as potential follow up to the present study and I suggest that clinical investigators and analyst in the research domain of PD and gait analysis explore and/or modify these recommendations because such could usefully inform rehabilitation in Parkinson's disease and related disorders and add to the body of knowledge and database of investigation into PD and gait analyses.

Appendix 1



University
of Southampton

School of Health Professions
and Rehabilitation Sciences

Tel+44 (0)23 8059 2142 Fax+44 (0)23 8059 5301
Web www.sohp.soton.ac.uk/sohp/

University of Southampton
Highfield
Southampton
SO17 IBJ
United Kingdom

7 July 2005

Olu Sofuwa
School of Health Professions
University of Southampton

Dear Olu

Submission No: PO5/04-01

Title: Hip pull-off and ankle push-off

I am pleased to confirm **full approval** for your study has now been given.

The approval has been granted by the School of Health
Professions and Rehabilitation Sciences Ethics Committee

You are required to complete a University Research Governance Form and to receive insurance clearance before you begin data collection. I enclose a form which must be completed and sent to Jennifer Roemer in the Research Support Office (RSO) at the University along with a copy of this letter. Your project will be registered at the RSO, and then automatically transferred to the Finance Department for insurance cover. **You can not commence data collection until you have received a letter stating that you have received insurance clearance.**

Please note that you have ethics approval only for the project described in your submission. If you want to change any aspect of your project (e.g., recruitment or data collection) you must discuss this with your supervisor and you may need to request permission from the Ethics Committee.

Yours sincerely

Dr Emma Stack (Chair, SHPRS Ethics Committee)

Appendix 2



**University
of Southampton**

Research Support Office

Dr Peter Hooper, Director

University of Southampton Highfield
Southampton SO 17 1BJ United Kingdom

4 August 2005

Mr Olumide Sofuwa Rehabilitation Research (SHPRS) Mailpoint 886 Level E,
Centre Block Southampton General Hospital Southampton SO166YD

Dear Mr Sofuwa

Project Title: Hip pull off and ankle push off

I am writing to confirm that the University of Southampton is prepared to act as sponsor for this study under the terms of the Department of Health Research Governance Framework for Health and Social Care (2001).

The University of Southampton fulfils the role of research sponsor in ensuring management, monitoring and reporting arrangements for research.

I understand that you will be acting as the Principal Investigator responsible for the daily management for this study, and that you will be providing regular reports on the progress of the study to the School on this basis.

I would like to take this opportunity to remind you of your responsibilities under the terms of the Research Governance Framework for researchers, principal investigators and research sponsors. These are included with this letter for your reference, in this regard if your project involves NHS patients or resources please send us a copy of your NHS REC and Trust approval letters when available.

Please do not hesitate to contact me should you require any additional information or support. May I also take this opportunity to wish you every success with your research.

Yours sincerely

Dr Martina Dorward

Research Governance Manager

Cc .File
Ruth McFadyen

Appendix 3



From; Ruth McFadyen Ext; 22417

E-mail: lirm@soton.ac.uk

To: Olumide Sofuwa

Dept: School of Health
Professions' and Rehabilitation
Sciences

Date: 4 August 2005

CLNTRL/HRM/GFT

Reference

Professional Indemnity

Insurance Project No:

P05/04-01

Hip Pull Off and Ankle Push Off

Thank you for forwarding the completed questionnaire and attached papers,

Having taken note of the information provided, I can confirm that this project will be covered under "the terms and conditions of the above policy, subject to written consent being obtained from the participating volunteers.

Ruth McFayden
Insurance Services Manager

Appendix 4



**University
of Southampton**

**School of Health Professions and Rehabilitation
Sciences**

**Rehabilitation Research Unit, Mailpoint 886
Southampton General Hospital
SO16 6YD United Kingdom
Tel +44 (0)23 8079 6469
Fax +44 (0)23 8079 4340
Web www.sohp.soton.ac.uk/sohD/**

Hip and Ankle Function in Walking

Ethics: P05/04-01

Dear Sir/Madam,

I am a chartered Physiotherapist working with Professor Ann Ashburn and Dr Emma Stack at the University of Southampton. I am conducting research on the way people with Parkinson's disease (PD) walk, in particular, the way in which the muscles around the hip and ankle function. Many people with PD walk with slow shuffling steps when compared with other people of their age category. Therefore, I would like to carry out two studies, the first on the way healthy people without PD walk, then I go on to investigate people with PD in detail.

As a spouse, partner or friend of a person with PD, I am writing to ask if you would like to participate in my initial study, which would involve you coming only once to our laboratory at Southampton General Hospital. I will ask you to change into shorts, and then I will stick some small markers on your legs and feet and ask you to walk up and down between our cameras. You would spend approximately two hours with us.

Please read the information sheet accompanying this letter. If you are interested in taking part, please complete and send back the return slip attached to this letter or contact me by telephone or e-mail. Thank you.

Olumide Sofuwa,
oas1@soton.ac.uk, phone: 023 8079 6466,

Return slip
Hip and Ankle Function in Walking
P05/04-01

Dear Researcher,

I am interested in taking part in the above study and am happy for you to contact me using the details below to discuss it further.

Name

Address

Postcode.

Telephone.

Mobile

Email..

Post to the following address:

Olumide Sofuwa Rehabilitation Research University of
Southampton, Mailpoint 886, Level E Centre Block
Southampton General Hospital, Tremona Road,
Southampton, SO166YD



**University
of Southampton**

**School of Health Professions and Rehabilitation
Sciences**

Rehabilitation Research Unit, Mailpoint 886
Southampton General Hospital
SO16 6YD United Kingdom
Tel +44 (0)23 8079 6469
Fax +44 (0)23 8079 4340
Web www.sohp.soton.ac.uk/sohD/

Research Participant Information sheet

Hip and Ankle Function In Walking

My name is Olumide Sofuwa and I am a chartered Physiotherapist working with Professor Ann Ashburn and Dr Emma Stack at the University of Southampton. I am writing to invite you to take part in a research study. Before you decide, it is important for you to understand why we are conducting the research and what it will involve. Please take time to read the following information carefully and discuss it with others if you wish. Ask us if there is anything that is not clear or if you would like more information. Take time to decide whether you wish to take part.

What is the purpose of the study?

The purpose of this study is to measure normal walking patterns in healthy adults aged 50-80 years. Once we have this information we will then use it in another larger research study to compare the information with walking patterns in a group of participants with Parkinson's disease (PD).

To collect the information about how people walk I will need to measure what happens at the ankle, knee and hip during walking. This information will be collected using special equipment in the gait analysis laboratory of the Southampton General Hospital. The equipment to be used is a motion analysis device that measures the angles at joints, a force platform that measures the contact of your foot with the floor and special electrodes that will tell me when your muscles are working.

Why have I been chosen?

I am contacting healthy people who are the spouse or partners of people with PD because most partners or spouses are within the age range of people with PD who will be involved in the next phase of the study.

Thirty healthy individuals are needed for this initial study. You should be a person between the age of 50 and 80 years, able to travel to Southampton General Hospital and be able to walk without assistance or using a walking aid. You will not be able to take part if you have PD yourself or any condition that

affects the ability to walk such as acute illness, neurological disorder, spinal/lower limb pain or fracture.

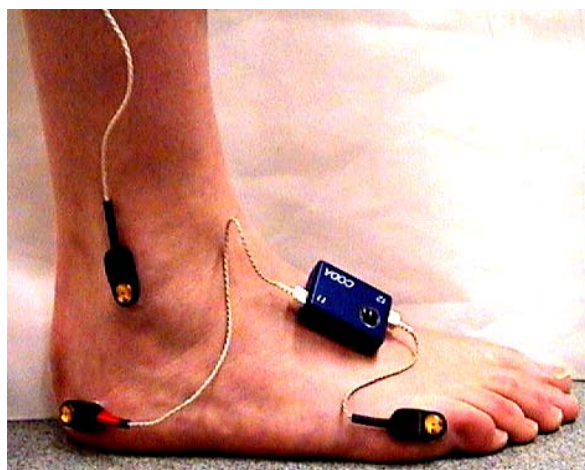
Do I have to take part?

It is up to you to decide whether to take part or not. If you decide to take part, you will be asked to sign a consent form. If you decide to take part, you are still free to withdraw at any time and without giving any reason. Your decision to take part in this study will not affect the care of your partner or spouse who has PD.

What will happen to me if I take part?

If you respond to this invitation to show that you would like to participate, Olumide Sofuwa (Olu) will either telephone or write to you to discuss the project further and answer any questions you may have. If you are happy to take part, a convenient time will then be arranged for you to visit the laboratory at Southampton General Hospital. You will only need to make one visit and this should take no more than 1 hour. 15 minutes.

When you arrive at the hospital, you will be met at reception. At the laboratory, a record will be made of your weight and height and you will be asked to change into shorts. Two researchers will be present (Olu Sofuwa & Malcolm Burnett) and they will place reflective markers over your ankles, knees and hips on both legs. Sticky button- like electrodes (two each) will also be placed on the skin in the front and at the back of e lower thigh and legs. Small areas of the skin may need to be shaved and cleansed with an alcohol wipe before the electrodes can be attached. The picture shows the type of markers we will need to attach. Once all the markers are in place, you will then be asked to walk barefoot back and forth on an 8-metre walkway (about 8-10 times). Special cameras will film the markers on your legs whilst you are walking.



Refreshment will be provided and you can rest at anytime during the session. If you like, you can bring along a friend or family member with you.

What are the possible side effects, disadvantages and risks of taking part?

There should be no side effects to this study, but please tell us if you are allergic to alcohol wipes or sticky plasters. You may become a little tired from the walking although you will be given sufficient time to rest.

What are the possible benefits of taking part?

There is no clinical benefit to participants from taking part in this study however, we hope the information we get will help in the future to explore interventions that help people with PD walk better.

What happens when the research study stops?

The results will contribute towards my PhD studies and will be presented in a scientific conference and published in a scientific journal. All data will be retained at the University of Southampton for fifteen years. If you would like to know the outcome of the study, you can let us know and we will provide you with a summary.

What if something goes wrong?

In the unlikely event that you are harmed by taking part in this research project, or if you have any concerns about any aspect of the way you have been approached or treated during the course of this study, the normal University complaint mechanisms are available to you. If you have a complaint please contact the research supervisor who is Professor Ann Ashburn, on 023 8079 6469.

Will my taking part in this study be kept confidential?

All information that is collected will be kept strictly confidential. Any information about you will have your name and address removed so that you cannot be recognised from it. All information will be kept in a secure locked drawer and data will be stored in accordance with the University of Southampton's guidelines.

Who has reviewed the study?

The project has been reviewed by the School of Health Professions and Rehabilitation Sciences Ethics Committee.

Contact for further information.

If you have any further queries, please contact either
Olumide Sofuwa (Main researcher) 023 8079 6466, oasl@soton.ac.uk

Or the research supervisors

Prof Ann Ashburn (023 8079 6469, ann@soton.ac.uk)

And Dr Emma Stack (023 8079 8669, elsl@soton.ac.uk)

Thank you for your interest in the study. If you are interested in taking part, please complete the attached reply slip and return it to us or contact us via the email address: oasl@soton.ac.uk

Ethics number: P05/04-1

Date: 07/07/05

Name of participant

SignatureDate:

Name of Researcher .

SignatureDate:

(1 copy to be kept by researcher and 1 copy for participant)

Ethics No: P05/04-01

Appendix 5



Rehabilitation Research Unit, Mailpoint 886
Southampton General Hospital
SO16 6YD United Kingdom
Tel +44 (0)23 8079 6469
Fax +44 (0)23 8079 4340
Web www.sohp.soton.ac.uk/sohD/

31 July 2006

Olu Sofuwa
School of Health Professions & Rehabilitation Sciences

Dear Olu

Submission No: PO6-07-01

Title: Pull/push off in Parkinson's Disease Gait

I am pleased to confirm **full approval** for your study has now been given. The approval has been granted by the School of Health Professions and Rehabilitation Sciences Ethics Committee.

You are required to complete a University Research Governance Form (enclosed) in order to receive insurance clearance before you begin data collection. You need to submit the following documentation in a plastic wallet to Dr Martina Dorward in the Research Support Office (RSO, University of Southampton, Highfield Campus, Bldg. 37, Southampton SO17 1BJ):

- Completed Research Governance form (**signed by both student and supervisor**)
- Copy of your research protocol (final and approved version)
- Copy of participant information sheet
- Copy of SoHPRS Risk Assessment form, **signed by yourself and supervisor** (original should be with Zena Galbraith)
- Copy of your information sheet and consent form
- Copy of this SoHPRS Ethical approval letter

Your project will be registered at the RSO, and then automatically transferred to the Finance Department for insurance cover. **You can not commence data collection until you have received a letter stating that you have received insurance clearance.**

Please note that you have ethics approval only for the project described in your submission. If you want to change any aspect of your project (e.g., recruitment or data collection) you must discuss this with your supervisor and you may need to request permission from the Ethics Committee.

Yours sincerely

Dr Emma Stack
Chair, SHPRS Ethics Committee

Appendix 6



**University
of Southampton**

**School of Health Professions and Rehabilitation
Sciences**

Mr Olumide Sofuwa
Rehabilitation Research
Mailpoint 886, Level E,
Southampton General Hospital,
SO16 6YD

**Rehabilitation Research Unit, Mailpoint 886
Southampton General Hospital
SO16 6YD United Kingdom
Tel +44 (0)23 8079 6469
Fax +44 (0)23 8079 4340
Web www.sohp.soton.ac.uk/sohD/**

02 October 2006

Dear Mr Sofuwa

Project Title: Ankle and Hip Function in Parkinson's Disease gait

I am writing to confirm that the University of Southampton is prepared to act as sponsor for this study under the terms of the Department of Health Research Governance Framework for Health and Social Care (2001).

The University of Southampton fulfils the role of research sponsor in ensuring management, monitoring and reporting arrangements for research.

I understand that you will be acting as the Principal Investigator responsible for the daily management for this study, and that you will be providing regular reports on the progress of the study to the School on this basis.

I would like to take this opportunity to remind you of your responsibilities under the terms of the Research Governance Framework for researchers, principal investigators and research sponsors. These are included with this letter for your reference. In this regard if your project involves NHS patients or resources please send us a copy of your NHS REC and Trust approval letters when available.

Please do not hesitate to contact me should you require any additional information or support. May I also take this opportunity to wish you every success with your research.

Yours sincerely

Dr Martina Dorward
Research Governance Manager

cc: File

Ruth McFayden, Insurance Services
Research Secretary/Manager, School Office, School of Health Professions and Rehabilitation Sciences

Supervisor:

Professor Ann Ashburn
School of Health Professions and Rehabilitation Sciences
University of Southampton
University Road
Highfield, Southampton, SO17 1BJ

:

Dr Emma Stack
School of Health Professions and Rehabilitation Sciences
University of Southampton
University Road
Highfield
Southampton SO17 1BJ

Appendix 7



From Ruth Mc Fayden

To Olumide Sofuwa

Ext 22417

Dept: School of Health Profession and
Rehabilitation Sciences

E-mail: firm@soton.ac.uk

Date: 27 September 2006

Reference: HRM/GFT/4SQ3

Professional Indemnity Insurance

Project No: P06-Q7-01

Ankle and Hip Function in Parkinson's Disease Gait

Thank you for forwarding the completed questionnaire and attached papers.

Having taken note of the information provided, I can confirm that this project will be covered under the terms and conditions of the above policy, subject to written consent being obtained from the participating volunteers.

Ruth McFayden
Insurance Services Manager

Appendix 8



**University
of Southampton**

**School of Health Professions and Rehabilitation
Sciences**

**Rehabilitation Research Unit, Mailpoint 886
Southampton General Hospital
SO16 6YD United Kingdom
Tel +44 (0)23 8079 6469
Fax +44 (0)23 8079 4340
Web www.sohp.soton.ac.uk/sohD/**

Hip and Ankle Function in Parkinson's Disease

Dear Sir/Madam,

I am a chartered Physiotherapist working with Professor Ann Ashburn and Dr Emma Stack at the University of Southampton. I am conducting research on the way people with Parkinson's disease (PD) walk, in particular, the way in which the hip and ankle joints function. Many people with PD walk with slow shuffling steps when compared with other people of their age category. I have collected data of how people without PD walk, therefore, I would like to investigate people with PD in detail.

I am writing to ask if you, as a person with PD, would like to participate in my study, which would involve you coming only once to our laboratory at Southampton General Hospital. I will ask you to change into shorts, and then I will stick some small markers on your legs and feet and ask you to walk up and down between our cameras. You would spend approximately two hours with us.

Please read the information sheet accompanying this letter. If you are interested in taking part, please complete and send back the return slip attached to this letter or contact me by telephone or e-mail. Thank you.

Olu Sofuwa,
oasl@soton.ac.uk, phone: 023 8079 6466,

Return slip Hip and Ankle Function in Walking

Dear Researcher,

I am interested in taking part in the above study and am happy for you to contact me using the details below to discuss it further.

Name

Address

Postcode.

Telephone.

Mobile

Email..

Post to the following address:

Olumide Sofuwa Rehabilitation Research
University of Southampton, Mailpoint 886,
Level E Centre Block Southampton General
Hospital, Tremona Road, Southampton,
SO166YD

Ethics number: PO6-07-01



**University
of Southampton**

**School of Health Professions and Rehabilitation
Sciences**

Rehabilitation Research Unit, Mailpoint 886
Southampton General Hospital
SO16 6YD United Kingdom
Tel +44 (0)23 8079 6469
Fax +44 (0)23 8079 4340
Web www.sohp.soton.ac.uk/sohD/

Participant Information Sheet

28/07/06

Study title

Hip and Ankle function in Parkinson's disease

What is the purpose of the study?

I am a chartered physiotherapist and my research interest at the University of Southampton is on walking problems in people with Parkinson's disease (PD). Problems with walking are common in PD. With the use of motion analysis camera system, problems with walking can be investigated and analyzed. On physical examination, it is observed that people with PD improved their walking when on medication. However, at the biomechanical level, they still generated a reduced power at the hip to lift the thigh (called pull-off power) and a reduced power at the ankle joint (called push-off power). In people without PD, pull-off and push-off power occur when walking, at the point when the foot is about to leave the ground. The inability to properly generate push-off by the muscle acting on the ankle joint has been associated with falls.

The purpose of this study is to investigate this problem and hopefully identify improved treatment.

To collect the information about how people with PD walk, I will need to measure what happens at the ankle, knee and hip during walking. This information will be collected using special equipment in the gait analysis laboratory of the Southampton General Hospital.

The equipment to be used is a motion analysis device that measures the angles at the joints and a force platform that measures the contact of your foot with the floor.

This is a student research project and the results of this research will contribute towards my PhD studies.

Why have I been chosen?

You were chosen because you are a member of the local Parkinson's disease society from which we received permission to contact the members for the purpose of this research.

I am aiming to recruit thirty individuals diagnosed with Idiopathic PD. To take part, you should be a person between the age of 50 and 80 years. You should be able to travel to Southampton General Hospital and be able to walk without assistance or using a walking aid.

You should not have any condition that affects your ability to walk such as acute illness, other neurological disorder, back and leg pain.

Do I have to take part?

It is up to you to decide whether or not to take part. If you do, you will be asked to sign a consent form. You are still free to withdraw at any anytime without giving any reason. A decision to withdraw at anytime, or a decision not to take part, will not affect the standard of any care you may be receiving elsewhere.

What will happen to me if I take part?

If you respond to this invitation to show that you would like to participate, I will either telephone or write to you to discuss the project further and answer any questions you may have.

For the first part of the study, a convenient time will be arranged to meet you to check if you meet with criteria and ask you questions about your mobility and medication. This part takes about 30 minutes and you may decide for it to take place at your home (or at the hospital just before the second part starts).

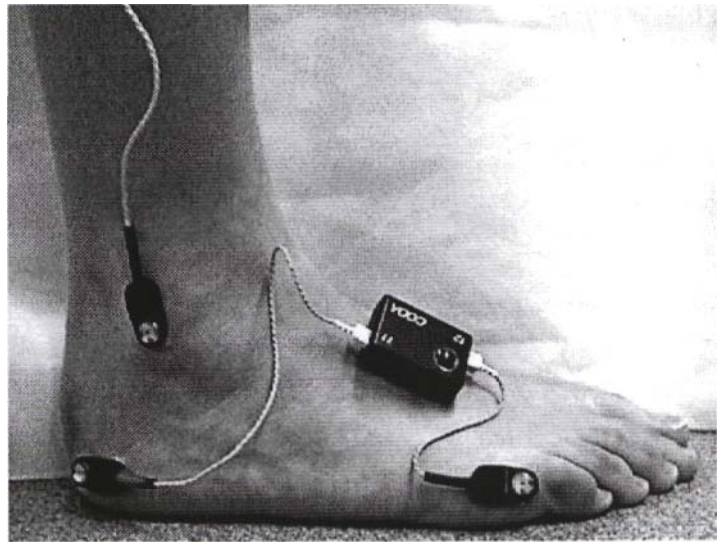
For the second part which is the walking test, you are required to visit the laboratory at Southampton General Hospital. You will only need to make one visit and this test should take no more than 1 hour 15minutes, (i.e. about 1 hour to set you up with the equipment and about 15 minutes (or less) for the actual walking).

The first part and the second part will last for a total of 2 hours.

When you arrive at the hospital, you will be met at reception. At the laboratory, a record will be taken of your weight and height and you will be asked to change into shorts. Two researchers will be present (Myself & Malcolm Burnett) and they will place reflective markers over your ankles, knees and hips on both legs.

Ethics number: PO6-07-01

The picture shows the type of markers we will need to attach. Once all the markers are in place, you will then be asked to walk (barefooted) as you usually do back and forth on an 8-metre walkway and then to walk faster than usual. Walking back and forth the walkway will be done about 8-10 times. Special cameras will film the markers on your legs whilst you are walking.



I will offer refreshment and you can rest at anytime during the session. If you like, you can bring along a friend or family member with you.

What do I have to do?

You just have to make yourself available for the researcher to answer questions about your mobility and medication and then to complete the walking test.

What are the side effects of the procedures when taken part?

There should be no side effects to this study, but please tell us if you are allergic to alcohol wipes or sticky plasters. You may become a little tired from the walking although you will be given sufficient time to rest. To reduce the risk of falling when walking, an overhead safety harness must be worn.

What are the possible benefits of taking part?

There is no clinical benefit to participants from taking part in this study but we hope you enjoy finding out about how we conduct research. The results will help us to improve treatment for people with PD who have difficulty walking.

What happens when the research study stops?

The results will contribute towards my PhD studies and will be presented in a scientific conference and published in a scientific journal. All data will be retained at the University of Southampton for fifteen years. If you would like to know the outcome of the study, you can let us know and we will provide you with a summary.

What if there is a problem?

In the unlikely event that you are harmed by taking part in this research project, or if you have any concerns about any aspect of the way you have been approached or treated during the course of this study, the normal University complaint mechanisms are available to you. If you have a complaint please contact the research supervisor who is Professor Ann Ashburn, on 023 8079 6469.

What will happen if I don't want to carry on with the study?

If you want to withdraw from the study you are free to do so and I will destroy all the documents you are identifiable with.

Will my taking part in the study be kept confidential?

All information that is collected will be kept strictly confidential. Any information about you will have your name and address removed so that you cannot be recognized from it. All information will be kept in a secure locked drawer and data will be stored in accordance with the University of Southampton's guidelines.

contact details

If you have any further queries, please contact either
Olu Sofuwa (Main researcher) 023 8079 6466, oasl@soton.ac.uk

Or the research supervisors

Dr Emma Stack (023 8079 8669, elsl@soton.ac.uk)

And Prof Ann Ashburn (023 8079 6469, ann@soton.ac.uk)

Ethics number: PO6-07-01



**University
of Southampton**

**School of Health Professions and Rehabilitation
Sciences**

Rehabilitation Research Unit, Mailpoint 886
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Web www.sohp.soton.ac.uk/sohD/

Hip and Ankle Function in Walking

Name of researcher: Olumide Sofuwa

1. I confirm that I have read and understand the information sheet dated 28/07/06 for the above study and have had the opportunity to ask questions. ☐
2. I understand that my participation is voluntary and that I am free to withdraw at any time, without giving any reason, and without my legal rights being affected. ☐
3. I understand that this is a student project. ☐
4. I understand that all information and data collected from me, as part of the project, will be retained by the University of Southampton for fifteen years in line with University policy ☐
5. I confirm that I have no condition that affects the ability to walk such as acute illness, other neurological disorder, spinal/lower limb pain or fracture. ☐
5. I understand that it is necessary that I wear a safety harness during the fast walk test. ☐
6. I confirm that I have no allergy to any of the materials to be used in this study (sticky tape, ethyl alcohol). Neither do I have any condition such as acute illness, body pain nor other neurological condition that can affect my walking. ☐
7. I agree to take part in the above study. ☐

Participant (Name and signature)

Researcher (Name and signature)

Appendix 9

UNIFIED PARKINSON'S DISEASE RATING SCALE

I. MENTATION, BEHAVIOR AND MOOD

1. Intellectual Impairment

- 0 = None.
- 1 = Mild. Consistent forgetfulness with partial recollection of events and no other difficulties.
- 2 = Moderate memory loss, with disorientation and moderate difficulty handling complex problems. Mild but definite impairment of function at home with need of occasional prompting.
- 3 = Severe memory loss with disorientation for time and often to place. Severe impairment in handling problems.
- 4 = Severe memory loss with orientation preserved to person only. Unable to make judgements or solve problems. Requires much help with personal care. Cannot be left alone at all.

2. Thought Disorder (Due to dementia or drug intoxication)

- 0 = None.
- 1 = Vivid dreaming.
- 2 = "Benign" hallucinations with insight retained.
- 3 = Occasional to frequent hallucinations or delusions; without insight; could interfere with daily activities.
- 4 = Persistent hallucinations, delusions, or florrid psychosis. Not able to care for self.

3. Depression

- 1 = Periods of sadness or guilt greater than normal, never sustained for days or weeks.
- 2 = Sustained depression (1 week or more).
- 3 = Sustained depression with vegetative symptoms (insomnia, anorexia, weight loss, loss of interest).
- 4 = Sustained depression with vegetative symptoms and suicidal thoughts or intent.

4. Motivation/Initiative

- 0 = Normal.
- 1 = Less assertive than usual; more passive.
- 2 = Loss of initiative or disinterest in elective (nonroutine) activities.
- 3 = Loss of initiative or disinterest in day to day (routine) activities.
- 4 = Withdrawn, complete loss of motivation.

II. ACTIVITIES OF DAILY LIVING (for both "on" and "off")

5. Speech

- 0 = Normal.
- 1 = Mildly affected. No difficulty being understood.
- 2 = Moderately affected. Sometimes asked to repeat statements.
- 3 = Severely affected. Frequently asked to repeat statements.
- 4 = Unintelligible most of the time.

6. Salivation

- 0 = Normal.
- 1 = Slight but definite excess of saliva in mouth; may have nighttime drooling.
- 2 = Moderately excessive saliva; may have minimal drooling.
- 3 = Marked excess of saliva with some drooling.
- 4 = Marked drooling, requires constant tissue or handkerchief.

7. Swallowing

- 0 = Normal.
- 1 = Rare choking.
- 2 = Occasional choking.
- 3 = Requires soft food.
- 4 = Requires NG tube or gastrotomy feeding.

8. Handwriting

- 0 = Normal.
- 1 = Slightly slow or small.
- 2 = Moderately slow or small; all words are legible.
- 3 = Severely affected; not all words are legible.
- 4 = The majority of words are not legible.

9. Cutting food and handling utensils

- 0 = Normal.
- 1 = Somewhat slow and clumsy, but no help needed.
- 2 = Can cut most foods, although clumsy and slow; some help needed.
- 3 = Food must be cut by someone, but can still feed slowly.
- 4 = Needs to be fed.

10. Dressing

- 0 = Normal.
- 1 = Somewhat slow, but no help needed.
- 2 = Occasional assistance with buttoning, getting arms in sleeves.
- 3 = Considerable help required, but can do some things alone.
- 4 = Helpless.

11. Hygiene

- 0 = Normal.
- 1 = Somewhat slow, but no help needed.
- 2 = Needs help to shower or bathe; or very slow in hygienic care.
- 3 = Requires assistance for washing, brushing teeth, combing hair, going to bathroom.
- 4 = Foley catheter or other mechanical aids.

12. Turning in bed and adjusting bed clothes

- 0 = Normal.
- 1 = Somewhat slow and clumsy, but no help needed.
- 2 = Can turn alone or adjust sheets, but with great difficulty.
- 3 = Can initiate, but not turn or adjust sheets alone.
- 4 = Helpless.

13. Falling (unrelated to freezing)

- 0 = None.
- 1 = Rare falling.
- 2 = Occasionally falls, less than once per day.
- 3 = Falls an average of once daily.
- 4 = Falls more than once daily.

14. Freezing when walking

- 0 = None.
- 1 = Rare freezing when walking; may have starthetisation.
- 2 = Occasional freezing when walking.
- 3 = Frequent freezing. Occasionally falls from freezing.
- 4 = Frequent falls from freezing.

15. Walking

- 0 = Normal.
- 1 = Mild difficulty. May not swing arms or may tend to drag leg.
- 2 = Moderate difficulty, but requires little or no assistance.
- 3 = Severe disturbance of walking, requiring assistance.
- 4 = Cannot walk at all, even with assistance.

16. Tremor (Symptomatic complaint of tremor in any part of body.)

- 0 = Absent.
- 1 = Slight and infrequently present.
- 2 = Moderate; bothersome to patient.
- 3 = Severe; interferes with many activities.
- 4 = Marked; interferes with most activities.

17. Sensory complaints related to parkinsonism

- 0 = None.
- 1 = Occasionally has numbness, tingling, or mild aching.
- 2 = Frequently has numbness, tingling, or aching; not distressing.
- 3 = Frequent painful sensations.
- 4 = Excruciating pain.

III. MOTOR EXAMINATION**18. Speech**

- 0 = Normal.
- 1 = Slight loss of expression, diction and/or volume.
- 2 = Monotone, slurred but understandable; moderately impaired.
- 3 = Marked impairment, difficult to understand.
- 4 = Unintelligible.

19. Facial Expression

- 0 = Normal.
- 1 = Minimal hypomimia, could be normal "Poker Face".
- 2 = Slight but definitely abnormal diminution of facial expression
- 3 = Moderate hypomimia; lips parted some of the time.
- 4 = Masked or fixed faces with severe or complete loss of facial expression; lips parted 1/4 inch or more.

20. Tremor at rest (head, upper and lower extremities)

- 0 = Absent.
- 1 = Slight and infrequently present.
- 2 = Mild in amplitude and persistent. Or moderate in amplitude, but only intermittently present.
- 3 = Moderate in amplitude and present most of the time.
- 4 = Marked in amplitude and present most of the time.

21. Action or Postural Tremor of hands

- 0 = Absent.
- 1 = Slight; present with action.
- 2 = Moderate in amplitude, present with action.
- 3 = Moderate in amplitude with posture holding as well as action.
- 4 = Marked in amplitude; interferes with feeding.

22. Rigidity (Judged on passive movement of major joints with patient relaxed in sitting position. Cogwheeling to be ignored.)

- 0 = Absent.
- 1 = Slight or detectable only when activated by mirror or other movements.
- 2 = Mild to moderate.
- 3 = Marked, but full range of motion easily achieved.
- 4 = Severe, range of motion achieved with difficulty.

23. Finger Taps (Patient taps thumb with index finger in rapid succession.)

- 0 = Normal.
- 1 = Mild slowing and/or reduction in amplitude.
- 2 = Moderately impaired. Definite and early fatiguing. May have occasional arrests in movement.
- 3 = Severely impaired. Frequent hesitation in initiating movements or arrests in ongoing movement.
- 4 = Can barely perform the task.

24. Hand Movements (Patient opens and closes hands in rapid succession.)

- 0 = Normal.
- 1 = Mild slowing and/or reduction in amplitude.
- 2 = Moderately impaired. Definite and early fatiguing. May have occasional arrests in movement.
- 3 = Severely impaired. Frequent hesitation in initiating movements or arrests in ongoing movement.
- 4 = Can barely perform the task.

25. Rapid Alternating Movements of Hands (Pronation-supination movements of hands, vertically and horizontally, with as large an amplitude as possible, both hands simultaneously.)

- 0 = Normal.
- 1 = Mild slowing and/or reduction in amplitude.
- 2 = Moderately impaired. Definite and early fatiguing. May have occasional arrests in movement.
- 3 = Severely impaired. Frequent hesitation in initiating movements or arrests in ongoing movement.
- 4 = Can barely perform the task.

26. Leg Agility (Patient taps heel on the ground in rapid succession picking up entire leg. Amplitude should be at least 3 inches.)

- 0 = Normal.
- 1 = Mild slowing and/or reduction in amplitude.
- 2 = Moderately impaired. Definite and early fatiguing. May have occasional arrests in movement.
- 3 = Severely impaired. Frequent hesitation in initiating movements or arrests in ongoing movement.
- 4 = Can barely perform the task.

27. Arising from Chair (Patient attempts to rise from a straightbacked chair, with arms folded across chest.)

- 0 = Normal.
- 1 = Slow; or may need more than one attempt.
- 2 = Pushes self up from arms of seat.
- 3 = Tends to fall back and may have to try more than one time, but can get up without help.
- 4 = Unable to arise without help.

28. Posture

- 0 = Normal erect.
- 1 = Not quite erect, slightly stooped posture; could be normal for older person.
- 2 = Moderately stooped posture, definitely abnormal; can be slightly leaning to one side.
- 3 = Severely stooped posture with kyphosis; can be moderately leaning to one side.
- 4 = Marked flexion with extreme abnormality of posture.

29. Gait

- 0 = Normal.
- 1 = Walks slowly, may shuffle with short steps, but no festination (hastening steps) or propulsion.
- 2 = Walks with difficulty, but requires little or no assistance; may have some festination, short steps, or propulsion.
- 3 = Severe disturbance of gait, requiring assistance.
- 4 = Cannot walk at all, even with assistance.

30. Postural Stability (Response to sudden, strong posterior displacement produced by pull on shoulders while patient erect with eyes open and feet slightly apart. Patient is prepared.)

- 0 = Normal.
- 1 = Retropulsion, but recovers unaided.
- 2 = Absence of postural response; would fall if not caught by examiner.
- 3 = Very unstable, tends to lose balance spontaneously.
- 4 = Unable to stand without assistance.

31. Body Bradykinesia and Hypokinesia (Combining slowness, hesitancy, decreased armswing, small amplitude, and poverty of movement in general.)

- 0 = None.
- 1 = Minimal slowness, giving movement a deliberate character; could be normal for some persons. Possibly reduced amplitude.
- 2 = Mild degree of slowness and poverty of movement which is definitely abnormal. Alternatively, some reduced amplitude.
- 3 = Moderate slowness, poverty or small amplitude of movement.
- 4 = Marked slowness, poverty or small amplitude of movement.

IV. COMPLICATIONS OF THERAPY fin the oast week)

A. DYSKINESIAS

32. Duration: What proportion of the waking day are dyskinesias present? (Historical information.)

- 0 = None
- 1 = 1-25% of day.
- 2 = 26-50% of day.
- 3 = 51-75% of day.
- 4 = 76-100% of day.

33. Disability: How disabling are the dyskinesias? (Historical information; may be modified by office examination.)

- 0 = Not disabling.
- 1 = Mildly disabling.
- 2 = Moderately disabling.
- 3 = Severely disabling.
- 4 = Completely disabled.

34. Painful Dyskinesias: How painful are the dyskinesias?

- 0 = No painful dyskinesias.
- 1 = Slight.
- 2 = Moderate.
- 3 = Severe.
- 4 = Marked.

35. Presence of Early Morning Dystonia (Historical information.)

- 0 = No
- 1 = Yes

B. CLINICAL FLUCTUATIONS

36. Are "off" periods predictable?

- 0 = No
- 1 = Yes

37. Are "off" periods unpredictable?

- 0 = No
- 1 = Yes

38. Do "off" periods come on suddenly, within a few seconds?

- 0 = No
- 1 = Yes

39. What proportion of the waking day is the patient "off" on average?

- 0 = None
- 1 = 1-25% of day.
- 2 = 26-50% of day.
- 3 = 51-75% of day.
- 4 = 76-100% of day.

C. OTHER COMPLICATIONS

40. Does the patient have anorexia, nausea, or vomiting?

- 0 = No= Yes

not functioning.

41. Any sleep disturbances, such as insomnia or hypersomnolence?

0 = No

1 = Yes

42. Does the patient have symptomatic orthostasis?

(Record the patient's blood pressure, height and weight on the scoring form)

0 = No

1 = Yes

V. MODIFIED HOEHNAND YAHR STAGING

STAGE 0 = No signs of disease.

STAGE 1 = Unilateral disease.

STAGE 1.5 = Unilateral plus axial involvement.

STAGE 2 = Bilateral disease, without impairment of balance.

STAGE 2.5 = Mild bilateral disease, with recovery on pull test.

STAGE 3 = Mild to moderate bilateral disease; some postural instability; physically independent.

STAGE 4 = Severe disability; still able to walk or stand unassisted.

STAGE 5 = Wheelchair bound or bedridden unless aided.

VI. SCHWAB AND ENGLAND ACTIVITIES OF DAILY LIVING SCALE

100% = Completely independent. Able to do all chores without slowness, difficulty or impairment. Essentially normal.

Unaware of any difficulty.

90% = Completely independent. Able to do all chores with some degree of slowness, difficulty and impairment.

Might

take twice as long. Beginning to be aware of difficulty.

80% = Completely independent in most chores. Takes twice as long. Conscious of difficulty and slowness.

70% = Not completely independent. More difficulty with some chores. Three to four times as long in some. Must spend a large part of the day with chores.

60% = Some dependency. Can do most chores, but exceedingly slowly and with much effort. Errors; some impossible.

50% = More dependent. Help with half, slower, etc. Difficulty with everything.

40% = Very dependent. Can assist with all chores, but few alone.

30% = With effort, now and then does a few chores alone or begins alone. Much help needed.

20% = Nothing alone. Can be a slight help with some chores. Severe invalid.

10% = Totally dependent, helpless. Complete invalid.

0% = Vegetative functions such as swallowing, bladder and bowel functions are Bedridden.

Appendix 10

MINI MENTAL STATE EXAMINATION

Client Name _____ Assessor _____ Date _____

Maximum Score

Score: Achieved:

Record client's answers in the spaces provided

5	()	Q1 <u>ORIENTATION:</u> What is the - Year _____ Season _____ Month _____ Day _____ Date _____
5	()	Q2 Where are we - Country _____ State _____ Town/City _____ Hospital/Street _____ Ward/House no. _____
3	()	Q3 <u>REGISTRATION:</u> Name these 3 objects - apple, penny, table. 1 second to say each. Then ask the person to repeat all three after you have said them. Score 1 point for each one correct on the first attempt. Repeat them (maximum 5 times) until he/she learns them. Count trials and record. Trials: _____
5	()	Q4 <u>ATTENTION AND CALCULATION:</u> Serial 7's: Count backwards from 100 by subtracting 7 (93 86 79 72 65) Score 1 point for each correct. (A correct response is one that is 7 less than the previous response even if the previous response is incorrect) Stop at 5 responses. - OR - Ask the person to spell the word "WORLD" forward and then backwards. Score 1 point for each letter in correct order. e.g. DLROW = 5, DLORW = 3
3	()	Q5 <u>RECALL:</u> Ask for the names of the three objects given to remember in Q3. Score 1 point for each correct answer irrespective of the order they are recalled in. apple penny table
2	()	Q6 <u>LANGUAGE:</u> Show the person a "PENCIL" and a "WATCH". Have the person name them as you point. Score 1 point for each correct answer.
1	()	Q7 Have the person repeat the phrase - "NO IFS, ANDS, OR BUTS". Score 1 point for a correct repetition.
3	()	Q8 Have the person follow a 3 stage command. Take the paper in your right/left hand. Fold it in half once with both hands. Put it on the floor. Score 1 point for each part correctly executed.
1	()	Q9 Read and obey the message. CLOSE YOUR EYES Score 1 point if the person closes their eyes. They do not have to read aloud.
1	()	Q10 Ask the person to write a sentence of his/her own choice. The sentence should contain a subject and an object and make sense. Ignore spelling errors.
1	()	Q11 Ask the person to copy the design. Score 1 point if all sides and angles are preserved and the intersecting sides form a quadrangle.
30	()	TOTAL SCORE



References

- Albanese, A. 2003, "Diagnostic criteria for Parkinson's disease", *Neurol Sci*, vol. 24, no. S23-S26.
- Allard, P., Lachance, R., Aissaoui, R., Duhaime, M. 1996. "Simultaneous bilateral able-bodied gait". *Hum Mov sci*, vol 15, no 3, pp 327-346.
- Almeida, Q. J., Frank, J. S., Roy, E. A., Patla, A. E., & Jog, M. S. 2007, "Dopaminergic modulation of timing control and variability in the gait of Parkinson's disease", *Mov Disord.*, vol. 22, no. 12, pp. 1735-1742.
- Arnadottir, S. A. & Mercer, V. S. 2000, "Effects of footwear on measurements of balance and gait in women between the ages of 65 and 93 years", *Phys Ther*, vol.80, no.1, pp. 17-27.
- Auvinet, B., Berrut, G., Touzat, C., Moutel, L., Collet, N., Chaleil, D and Barrey, E. 2002. "Reference data for normal subjects obtained with and accelerometric device". *Gait Posture*, vol. 16, no. 2, pp. 124-134.
- Baker, K., Rochester, L., Nieuwboer, A. 2007. "The immediate effect of attentional, auditory, and a combined cue strategy on gait during single and dual tasks in Parkinson's disease." *Arch Phys Med Rehabil*, vol 88, no 12, pp 1593-6000.
- Bassuk, S. S. & Murphy, J. M. 2003, "Characteristics of the Modified Mini-Mental State Exam among elderly persons", *J Clin Epidemiol*, vol 56, no. 7, pp. 622-628, 2003.
- Behrman, A.L., Teitelbaum, P., Cauraugh, J.H., 1998. "Verbal instructional sets to normalise the temporal and spatial gait variables in Parkinson's disease". *J Neurol Neurosurg Psychiatr*, vol 65, pp 580-582.
- Berardelli, A., Rothwell, J.C., Thompson, P.D., Hallet, M. 2001. "Pathophysiology of bradykinesia in Parkinson's disease". *Brain*, vol 124, pp 2131-2146.
- Blanc, Y., balmer, C., Landis, T. And Vingerhoets, F. 1999. "Temporal parameters and patterns of the foot roll over during walking: normative data for healthy adults". *Gait Posture*, vol. 10, no. 2, pp 124-134.
- Behl, C., Manthey, D. 2000. "Neuroprotective activities of estrogen: an update". *J Neurocytol*, vol. 29, no. 5-6, pp. 351-358.
- Bland, J., Altman, D. 1986, "Statistical methods for assessing agreement between two methods of clinincal measurement.", *Lancet*, vol. i, pp. 307-310.
- Blanke, D. J. & Hageman, P. A. 1989, "Comparison of gait of young men and elderly men", *Phys Ther*, vol 69, no.2, pp 144-148.
- Blin, O., Ferrandez, A. M., & Serratrice, G. 1990, "Quantitative analysis of gait in Parkinson patients: increased variability of stride length", *J Neurol Sci.*, vol. 98, no. 1, pp. 91-97.
- Blin, O., Ferrandez, A. M., Pailhous, J., & Serratrice, G. 1991, "Dopa-sensitive and dopa-resistant gait parameters in Parkinson's disease", *J Neurol Sci.*, vol. 103, no. 1, pp. 51-54.
- Bohannon RW.1997. "Comfortable and maximum walking speed of adults aged 20-79 years: reference values and determinants. *Age Ageing*, vol. 26, no. 1, pp. 15-19.

- Bohannon, R. W. E., Andrews, A. W. M., & Thomas, M. W. B. 1996, "Walking Speed: Reference Values and Correlates for Older Adults. [Miscellaneous]", *J Orthop Sports Phys Ther*, vol. 24, no. 2, pp. 86-90.
- Bowes, S. G., Clark, P. K., Leeman, A. L., O'Neill, C. J., Weller, C., Nicholson, P. W., Deshmukh, A. A., Dobbs, S. M., & Dobbs, R. J. 1990, "Determinants of gait in the elderly parkinsonian on maintenance levodopa/carbidopa therapy", *Br J Clin Pharmacol*, vol. 30, no. 1, pp. 13-24.
- Brach, J.S., Berthold, R., Craik, R., Van Swearingen, J.M., Newman, A.B. 2001. "Gait variability in community-dwelling older adults". *J Am Geriatr Soc*, vol. 49, no. 12, pp. 1646-1650.
- Burke, D., Hagbarth, K. E., & Wallin, B. G. 1977, "Reflex mechanisms in Parkinsonian rigidity", *Scand. J Rehabil Med.*, vol. 9, no. 1, pp. 15-23.
- Cakit, B. D., Saracoglu, M., Genc, H., Erdem, H. R., & Inan, L. 2007, "The effects of incremental speed-dependent treadmill training on postural instability and fear of falling in Parkinson's disease", *Clin Rehabil*, vol. 21, no. 8, pp. 698-705.
- Camicioli, R., Bouchard, T., & Lici, L. 2006, "Dual-tasks and walking fast: relationship to extra-pyramidal signs in advanced Alzheimer disease", *J Neurol Sci.*, vol. 248, no. 1-2, pp. 205-209.
- Campbell, F. M., Ashburn, A. M., Pickering, R. M., & Burnett, M. 2001, "Head and Pelvic Movements During a Dynamic Reaching Task in Sitting: Implication for Physical Therapists", *Arch Phys Med Rehabil*, vol. 82, pp. 1655-1660.
- Canning, C.G., Ada, L., Johnson, J.J., McWhirter, S. 2006. "Walking capacity in mild to moderate Parkinson's disease" *Arch Phys Med Rehabil*, vol 87, no 3, pp 371-375.
- Cersosimo, M. G. & Koller, W. C. 2006, "The diagnosis of manganese-induced parkinsonism", *Neurotoxicology*, vol. 27, no. 3, pp. 340-346.
- Chao, E. Y., Laughman, R. K., Schneider, E., & Stauffer, R. N. 1983, "Normative data of knee joint motion and ground reaction forces in adult level walking", *J Biomech*, vol. 16, no. 3, pp. 219-233.
- Chastan, N., Westby, G.W., Yelnik, J., Bardinet, E., Do, M.C., Agid, Y., Welter, M.L. 2008. "Effect of nigral stimulation on locomotion and postural stability in patients with Parkinson's disease". *Brain*, vol 132 (pt 1), pp 172-184.
- Cheng, P. L. & Pearcy, M. 1999, "A three-dimensional definition for the flexion/extension and abduction/adduction angles", *Med Bio Eng Comput*, vol.37, no.4, pp. 440-444.
- Clarke, C. E. 2001. "Clinical features," in *Parkinson's disease in practice*, Royal Society of Medicine Press Ltd, Plymouth, pp. 17-18.
- Craik, R. 1995. *Gait analysis : Theory and application*, 1st edn, St. Louis : Mosby.
- Cunningham, D. A., Rechnitzer, P. A., Pearce, M. E., & Donner, A. P. 1982, "Determinants of self-selected walking pace across ages 19 to 66", *J Gerontol.*, vol. 37, no. 5, pp. 560-564.
- Davis, R. B., Ounpuu, S. & Deluca P. A. 1997. "Gait data: Reporting, archiving and sharing,." In: *Three-dimensional analysis of human locomotion*, Allard P., Cappozzo A., Lundberg A., & Vaughan C., ed., Wiley, Chichester, pp. 389-406.

- DeVita, P. & Hortobagyi, T. 2000, "Age causes a redistribution of joint torques and powers during gait", *J Appl Physiol*, vol. 88, no. 5, pp. 1804-1811.
- Di Monte, D. A., Lavasani, M., & Manning-Bog, A. B. 2002, "Environmental factors in Parkinson's disease", *Neurotoxicology*, vol. 23, no. 4-5, pp. 487-502.
- Dodson, M. W. & Guo, M. 2007, "Pink1, Parkin, DJ-1 and mitochondrial dysfunction in Parkinson's disease", *Curr Opin Neurobiol*, vol. 17, no. 3, pp. 331-337.
- Dujardin, K., Sockeel, P., Devos, D., Delliaux, M., Krystkowiak, P., Destee, A., & Defebvre, L. 2007, "Characteristics of apathy in Parkinson's disease", *Mov Disord.*, vol. 22, no. 6, pp. 778-784.
- Ebersbach, G., Sojer, M., Valldeoriola, F., Wissel, J., Muller, J., Tolosa, E., & Poewe, W. 1999, "Comparative analysis of gait in Parkinson's disease, cerebellar ataxia and subcortical arteriosclerotic encephalopathy", *Brain*, vol. 122 (Pt 7), pp. 1349-1355.
- Eke-Okoro, S. T., Gregoric, M., & Larsson, L. E. 1997, "Alterations in gait resulting from deliberate changes of arm-swing amplitude and phase", *Clin Biomech*, vol. 12, no. 7-8, pp. 516-521.
- Eng, J. J. & Winter, D. A. 1995, "Kinetic analysis of the lower limbs during walking: What information can be gained from a three-dimensional model?", *J Biomech*, vol.28, no 6, pp 753-758.
- Ferrarin, M., Rizzone, M., Bergamasco, B., Lanotte, M., Recalcati, M., Pedotti, A., Lopiano, L. 2005. "Effects of bilateral subthalamic stimulation on gait kinematics and kinetics in Parkinson's disease". *Exp Brain Res*, vol 160, no 4, pp 517-527.
- Ferrarin, M., Rabuffetti, M., Tettamanti, M., Pignatti, R., Mauro, A., Albani G. 2008. "Effect of optical flow versus attentional strategy on gait in Parkinson's disease: a study with portable optical stimulating device" *J NeuroEng Rehabil*, vol 5, no 3.
- Frenkel-Toledo, S., Giladi, N., Peretz, C., Herman, T., Gruendlinger, L., & Hausdorff, J. M. 2005, "Effect of gait speed on gait rhythmicity in Parkinson's disease: variability of stride time and swing time respond differently", *J Neuroeng Rehabil*, vol. 2, pp. 23.
- Folstein, M.F., Folstein, S.E., McHugh, P.R. 1975. "Mini-mental state". A practical method for grading the cognitive state of patients for the clinician. *J Psychiatr Res*, vol. 12, no. 3, pp 189-198.
- Gage, R. 1991, "Gait analysis in cerebral palsy". London, Mac Keith press.
- Giladi N, Kao R, Fahn S. 1997, "Freezing phenomenon in patients with parkinsonian syndromes", *Mov Disord* no. 12, pp. 302-305.
- Goetz CG, Poewe W, Rascol O, Sampaio C, Stebbins GT, Counsell C, Giladi N, Holloway RG, Moore CG, Wenning GK, Yahr MD, Seidl L; Movement Disorder Society Task Force on Rating Scales for Parkinson's Disease.2004 "Movement Disorder Society Task Force report on the Hoehn and Yahr staging scale: status and recommendations". *Mov Disord*. vol 19, no.9, pp 1020-1028.

- Goodwin, V.A, Richards, S.H., Taylor, R.S., Taylor, A.H., Campbell, J.L. 2008. "The effectiveness off exercise interventions for people with Parkinson's disease : a systematic and meta analysis". *Mov Disord*, vol 23, no 5, pp 631-640.
- Gorton, G.E. 3rd, Hebert, D.A., Gannotti, M.E. 2009. "Assessment of the kinematic variability among 12 motion analysis laboratories". *Gait Posture*, vol 29, no 3, pp 398-402.
- Grabiner, M. D., Feuerbach, J. W., Lundin, T. M., & Davis, B. L. 1995, "Visual guidance to force plates does not influence ground reaction force variability", *J Biomech*, vol. 28, no. 9, pp. 1115-1117.
- Graf, A., Judge, J. O., Ounpuu, S., & Thelen, D. G. 2005, "The effect of walking speed on lower-extremity joint powers among elderly adults who exhibit low physical performance", *Arch.Phys Med.Rehabil*, vol. 86, no. 11, pp. 2177-2183.
- Gray, P., Hildebrand, K. 2000. "Fall risk factors in Parkinson's disease". *J Neurosci Nurs*. Vol 32, no 4, pp 222-228
- Haaxma, C.A., Bloem, B.R., Borm, G.F., Oyen, W.J., Leenders, K.L., Eshuis, S., Booi, J., Dluzen, D.E., Horstink, M.W. 2007. "Gender differences in Parkinson's disease". *J Neurol Neurosurg Psychiatr*, vol 78, no 8, pp 819-824.
- Haber, S.N., Calzavara, R., 2009. "The cortico-basal ganglia integrative network: the role of the thalamus". *Brain Res Bull*. vol 78, no 2-3, pp 69-74.
- Hageman, P.A., Blanke, D.J. 1986. "Comparison of gait of young women and elderly women" *Phys Ther*, vol 66, no 9, pp 1382-1387.
- Hase, K. & Stein, R. B. 1998, "Analysis of rapid stopping during human walking", *J Neurophysiol* vol.80, no. 1, pp 255-261.
- Hausdorff JM, Zemani L, Peng C, Goldberger AL. 1999. "Maturation of gait dynamics: stride-to-stride variability and its temporal organization in children." *J Appl Physiol*, vol. 86, no. 3, pp. 1040-1047.
- Hesse, S. A., Jahnke, M. T., Bertelt, C. M., Schreiner, C., Lucke, D., & Mauritz, K. H. 1994, "Gait outcome in ambulatory hemiparetic patients after a 4-week comprehensive rehabilitation program and prognostic factors", *Stroke*, vol. 25, no. 10, pp. 1999-2004.
- Hof, A.L. 2003. "Muscle mechanics and neuromuscular control". *J Biomech*, vol. 36, no. 7, pp. 1031-1038.
- Jankowski, J., Scheef, L., Hüppe, C., Boecker, H. 2009. Distinct striatal regions for planning and executing novel and automated movement sequences. *Neuroimage*, vol. 44, no. 4, pp. 1369-1379.
- Jansen, E. C., Vittas, D., Hellberg, S., & Hansen, J. 1982, "Normal gait of young and old men and women. Ground reaction force measurement on a treadmill", *Acta Orthop Scand*, vol. 53, vol. 2, no. 2, pp. 193-196.
- Jöbges, M., Heuschkel, G., Pretzel, C., Illhardt, C., Renner, C., Hummelsheim, H. 2004. "Repetitive training of compensatory steps: a therapeutic approach for postural instability in Parkinson's disease". *J Neurol Neurosurg Psychiatr*, vol. 75, no. 12, 1682-1687.

- Jones, D., Playfer, J. "Motor Control" In: Stokes M. 2007. "Physical management in neurological rehabilitation". 2nd ed, Elsevier, Mosby.
- Judge, J.O., Davis, R.B. III and Ounpuu, S., 1996. "Step length reductions in advanced age: the role of ankle and hip kinetics". *J Gerontol A Biol Sci Med Sci*, vol. 51, no. 6, M303-312.
- Kadaba, M. P., Ramakrishnan, H. K., Wootten, M. E., Gainey, J., Gorton, G., & Cochran, G. V. 1989, "Repeatability of kinematic, kinetic, and electromyographic data in normal adult gait", *J Orthop Res*, vol 7, no. 6, pp. 849-860.
- Kahle-Wroblewski K, Corrada MM, Li B, Kawas CH. 2007. "Sensitivity and specificity of the mini-mental state examination for identifying dementia in the oldest-old: the 90+ study". *J Am Geriatr Soc*, vol.55, no. 2, pp 284-289.
- Katzen, H.L., Levin, B.E., Weiner, W. 2006. "Side and symptom influence cognition in Parkinson's disease". *Mov Disord*, vol 21, no 11, pp. 1947-1953.
- Kempster, P. A., Frankel, J. P., Bovingdon, M., Webster, R., Lees, A. J., & Stern, G. M. 1989, "Levodopa peripheral pharmacokinetics and duration of motor response in Parkinson's disease", *J Neurol Neurosurg Psychiatry*, vol. 52, no. 6, pp. 718-723.
- Kerrigan, D.C., Lee, L.W., Nieto, T.J., Markman, J.D., Collins, J.J., Riley, P.O. 2000 "Kinetic alterations independent of walking speed in elderly fallers" *Arch Phys Med Rehabil*, vol. 81, no. 6, pp. 730-735.
- Kerrigan, D. C., Todd, M. K., Della, C. U., Lipsitz, L. A., & Collins, J. J. 1998b, "Biomechanical gait alterations independent of speed in the healthy elderly: evidence for specific limiting impairments", *Arch Phys Med Rehabil*, vol. 79, no. 3, pp. 317-322.
- Kerrigan, D. C., Xenopoulos-Oddsson, A., Sullivan, M. J., Lelas, J. J., & Riley, P. O. 2003, "Effect of a hip flexor-stretching program on gait in the elderly", *Arch Phys Med Rehabil*, vol. 84, no. 1, pp. 1-6.
- Kerrigan, D.C., Todd, M.K. and Della, C.U. 1998. "Gender differences in joint biomechanics during walkng: normative study in young adults", *Am J Phys Med Rehabil*, vol. 77, no. 1, pp. 2-7.
- Kirtley, C. 2005. *Clinical Gait Analysis: Theory and Practice*. 1st edn, Churchill Livingstone.
- Koozekanani, S. H., Balmaseda, M. T. Jr., Fatehi, M. T., & Lowney, E. D. 1987, "Ground reaction forces during ambulation in parkinsonism: pilot study", *Arch Phys Med Rehabil*, vol. 68, no. 1, pp. 28-30.
- Kottke, F. J., Halpern, D., Easton, J. K., Ozel, A. T., & Burrill, C. A. 1978, "The training of coordination", *Arch Phys Med Rehabil*, vol. 59, no. 12, pp. 567-72.
- Krack, P., Poepping, M., Weinert, D., Schrader, B., Deuschl, G. 2000. "Thalamic, Pallidal, or Subthalamic surgery for Parkinson's disease?". *J Neurol*, vol. 247, suppl 2, no. 2, pp. 22-34.
- Laufer, Y. 2003, "Age- and gender-related changes in the temporal-spatial characteristics of forwards and backwards gaits", *Phys Res Int*, vol. 8, no. 3, pp. 131-42.
- Lehmann, J. F. 1993, "Push-off and propulsion of the body in normal and abnormal gait", *Clinical Orthopaedics and Related Research*, vol. 288, pp. 97-108.

- Lelas, J. L., Merriman, G. J., Riley, P. O., & Kerrigan, D. C. 2003, "Predicting peak kinematic and kinetic parameters from gait speed", *Gait Posture*, vol. 17, no. 2, pp. 106-112.
- Levy, R. & Czernecki, V. 2006, "Apathy and the basal ganglia", *J Neurol*, vol. 253 Suppl 7, p. VII54-VII61.
- Lewis, G. N., Byblow, W. D., & Walt, S. E. 2000, "Stride length regulation in Parkinson's disease: the use of extrinsic, visual cues", *Brain*, vol. 123 (Pt 10), pp. 2077-2090.
- Lin, R. S. & Gage, J. R. 1989, "The neurological control system for normal gait", *Journal of Prosthetics and Orthotics*, vol. 2, no. 1, pp. 1-13.
- Lord, S. R. & Bashford, G. M. 1996, "Shoe characteristics and balance in older women", *J Am Geriatr Soc*, vol. 44, no. 4, pp. 429-433.
- Lozano, A.M., Lang, E., Galvez-Jimenez, M. et al. 1995 "Effect of GPi pallidotomy on motor function in parkinson's disease". *Lancet*, vol. 346, pp. 1383-1387.
- Lubik, S., Fogel, W., Tronnier, V., Krause, M., König, J., Jost, W.H. 2006. "Gait analysis in patients with advanced Parkinson disease: different or additive effects on gait induced by levodopa and chronic STN stimulation. *J Neural Transm*, vol. 113, no2, pp163-173.
- Macphee, G. J. A. 2001, "Diagnosis and differential diagnosis," In: Playfer, J.R., Hindle, J., eds. *Parkinson's disease in the older patient*, J. R. H. J. V. Playfer, ed., London, Arnold, pp. 43-70.
- Maki, B.E. 1997. "Gait changes in older adults: predictors of falls or indicators of fear". *J Am Geriatr Soc*. vol. 45, no. 3, pp. 313-320.
- Mannion AF, Dolan P. 1996. "Relationship between myoelectric and mechanical manifestations of fatigue in the quadriceps femoris muscle group. *Eur J Appl Physiol Occup Physiol*.vol. 74, no. 5, pp. 411-419.
- Marks, R. 1997, "The effect of restricting arm swing during normal locomotion", *Biomed Sci Instrum*, vol.33, pp. 209-215.
- Martinez-Martin, P., Gil-Nagel, A., Gracia, L. M., Gomez, J. B., Martinez-Sarries, J., & Bermejo, F. 1994, "Unified Parkinson's Disease Rating Scale characteristics and structure. The Cooperative Multicentric Group", *Mov Disord.*, vol. 9, no. 1, pp. 76-83.
- Maynard, V., Bakheit, A. M. O., Oldham, J., & Freeman, J. 2003, "Intra-rater and inter-rater reliability of gait measurements with CODA mpx30 motion analysis system", *Gait Posture*, vol. 17, no. 1, pp. 59-67.
- McGibbon, C. A. & Krebs, D. E. 1999, "Effects of age and functional limitation on leg joint power and work during stance phase of gait", *J Rehabil Res.Dev.*, vol. 36, no. 3, pp. 173-182.
- McGinley, J. L., Goldie, P. A., Greenwood, K. M., & Olney, S. J. 2003, "Accuracy and reliability of observational gait analysis data: judgments of push-off in gait after stroke", *Phys Ther*, vol. 83, no. 2, pp. 146-60.
- Meara J, Hobson P. 2000 "Epidemiology of Parkinson's disease and Parkinsonism in elderly subjects". In Meara J, Koller W, eds. *Parkinson's disease and Parkinsonism in the elderly*. Cambridge: Cambridge University Press, pp. 111-122.

- Menz, H. B., Lord, S. R., & Fitzpatrick, R. C. 2003, "Age-related differences in walking stability", *Age Ageing*, vol. 32, no. 2, pp. 137-142.
- Metman, L. V., Myre, B., Verwey, N., Hassin-Baer, S., Arzbaecher, J., Sierens, D., & Bakay, R. 2004, "Test-retest reliability of UPDRS-III, dyskinesia scales, and timed motor tests in patients with advanced Parkinson's disease: an argument against multiple baseline assessments", *Mov Disord.*, vol. 19, no. 9, pp. 1079-1084.
- Mills, P. M. & Barrett, R. S. 2001, "Swing phase mechanics of healthy young and elderly men", *Hum Mov Sci.*, vol. 20, no. 4-5, pp. 427-446.
- Monaghan, K., Delahunt, E., & Caulfield, B. 2007. "Increasing the number of gait trial recordings maximises intra-rater reliability of the CODA motion analysis system", *Gait Posture*, vol. 25, no. 2, pp. 303-315.
- Morris, M.E., Iannsek, R., Matyas, T.A., Summers, J.J. 1996 "Stride length regulation in Parkinson's disease. Normalization strategies and underlying mechanisms". *Brain*, vol. 119 (Pt2), pp. 551-568.
- Morris, M. E., Iannsek, R., Matyas, T. A., & Summers, J. J. 1994a, "Ability to modulate walking cadence remains intact in Parkinson's disease", *J Neurol Neurosurg Psychiatr*, vol. 57, no. 12, pp. 1532-1534.
- Morris, M. E., Iannsek, R., Matyas, T. A., & Summers, J. J. 1994b, "The pathogenesis of gait hypokinesia in Parkinson's disease", *Brain*, vol. 117, no. 5, pp. 1169-1181.
- Morris, M. E., Matyas, T. A., Iannsek, R., & Summers, J. J. 1996, "Temporal stability of gait in Parkinson's disease", *Phys Ther*, vol 76, no. 7, pp. 763-777; discussion 778-80.
- Morris, M. E., McGinley, J., Huxham, F., Collier, J., & Iannsek, R. 1999, "Constraints on the kinetic, kinematic and spatiotemporal parameters of gait in Parkinson's disease", *Hum Mov Sci*, vol. 18, no. 2-3, pp. 461-483.
- Morris, M., Iannsek, R., Matyas, T., & Summers, J. 1998, "Abnormalities in the stride length-cadence relation in parkinsonian gait", *Mov Disord*, vol. 13, no. 1, pp. 61-69.
- Morris, M., Iannsek, R., McGinley, J., Matyas, T., & Huxham, F. 2005, "Three-dimensional gait biomechanics in Parkinson's disease: evidence for a centrally mediated amplitude regulation disorder", *Mov Disord*. vol. 20, no. 1, pp. 40-50.
- Morris, M.E. 2000. "Movement Disorders in people with Parkinson's disease: A model for physical therapy". *Phys Ther*, vol. 80, pp. 578-597.
- Murray, M.P., Septic, S.B., Gardner, G.M., & Downess, W.J 1978, "Walking patterns of men with Parkinsonism", *Amer J Phys Med*, pp. 278-294.
- Murray, M. P., Kory, R. C., & Clarkson, B. H. 1969, "Walking patterns in healthy old men", *J Gerontol*, vol. 24, no. 2, pp. 169-178.
- Murray, M. P., Kory, R. C., & Sepic, S. B. 1970, "Walking patterns of normal women", *Arch Phys Med Rehabil*, vol. 51, no. 11, pp. 637-650.
- Nadeau, S., Gravel, D., Arsenault, A. B., & Bourbonnais, D. 1999, "Plantarflexor weakness as a limiting factor of gait speed in stroke subjects and the compensating role of hip flexors", *Clin Biomech*, vol. 14, no. 2, pp. 125-135.

- Nieuwboer, A., Feys, P., De Weerd, W., Dom R. 1997. "Is using a cue the clue to the treatment of freezing in Parkinson's disease?" *Phys ther Res Int*, vol 2, no 3, pp 125-132.
- Nieuwboer, A., De, W. W., Dom, R., & Lesaffre, E. 1998, "A frequency and correlation analysis of motor deficits in Parkinson patients", *Disabil Rehabil*, no. 4, pp. 142-150.
- Nieuwboer, A., Dom, R., De Weerd, W., Desloovere, K., Janssens, L., Stijn, V. 2004. "Electromyographic profiles of gait prior to onset of freezing episodes in patients with Parkinson's disease." *Brain*, vol. 127, pt 7, pp. 1650-1660.
- Nurse, M. A., Hulliger, M., Wakeling, J. M., Nigg, B. M., & Stefanyshyn, D. J. 2005, "Changing the texture of footwear can alter gait patterns", *J Electromyogr Kinesiol*, vol. 15, no. 5, pp. 496-506.
- Nutt, J.G. 2008. "Pharmacokinetics and pharmacodynamics of levodopa". *Mov Disord*, vol. 23, suppl 3, S580-584.
- Oberg, T., Karsznia, A., & Oberg, K. 1993, "Basic gait parameters: reference data for normal subjects, 10-79 years of age", *J Rehabil Res Dev*, vol. 30, no. 2, pp. 210-223.
- Oberg, T., Karsznia, A., & Oberg, K. 1994, "Joint angle parameters in gait: reference data for normal subjects, 10-79 years of age", *J Rehabil Res Dev*, vol. 31, no. 3, pp. 199-213.
- Obeso, J.A., Olanow, C.W., Roriguez-Oroz, M.C., Krack, P., Kumar, R., Lang, A.E. 2001. "Deep-brain stimulation of the subthalamic nucleus or pars interna of the globus pallidus In: Parkinson's disease; Deep-brain stimulation of the Parkinson's disease study group". *N Engl J Med*, vol. 345, no. 13, pp. 956-963.
- Obeso, J.A., Rodriguez-Oroz, M.C., Chana, P., Lera, G., Rodriguez, M., Olanow, C.W. 2000. "The evolution and origin of motor complications in Parkinson's disease". *Neurol*, vol. 55, no 11 suppl 4, S13-S20.
- Oeffinger, D., Brauch, B., Cranfill, S., Hisle, C., Wynn, C., Hicks, R., & Augsburger, S. 1999, "Comparison of gait with and without shoes in children", *Gait Posture*, vol. 9, no. 2, pp. 95-100.
- Ostrosky, K. M., VanSwearingen, J. M., Burdett, R. G., & Gee, Z. 1994, "A comparison of gait characteristics in young and old subjects", *Phys Ther*, vol. 74, no. 7, pp. 637-644.
- Paul, J. P. 1996, "Visual guidance to forceplates does not influence ground reaction force variability", *J Biomech*, vol. 29, no. 6, p. 833.
- Parkinson's disease Society, UK. 2008 (<http://www.parkinsons.org.uk/>)
- Pellicano, C., Benincasa, D., Giovannelli, M., Buttarelli, F.R., Ruggieri, S., Pontieri, F.E. 2009. "Entacapone in elderly Parkinsonian patients experiencing levodopa-related wearing-off: a pilot study". *Neurol Res*, vol, 31, no1, pp 74-76.
- Peppe, A., Chiavalon, C., Pasqualetti, P., Crovato, D., & Caltagirone, C. 2007, "Does gait analysis quantify motor rehabilitation efficacy in Parkinson's disease patients?", *Gait Posture*, vol. 26, no. 3, pp. 452-462.
- Pereira, E. A. C. & Aziz, T. Z. 2006, "Surgical insights into Parkinson's disease", *J R Soc Med*, vol. 99, no. 5, pp. 238-244.
- Perry, J. 1992, "Gait analysis-normal and pathological gait," Slack Inc, USA.

- Perttunen, J., Anttila, E., Sodergard, J., Merikanto, J., & Komi, P. 2004, "Gait asymmetry in patients with limb length discrepancy", *Scand J Med and Sci Sports*, vol. 14, no. 1, pp. 49-56.
- Pijnappels, M., Bobbert, M. F., & Dieen, J. H. 2005a, "Push-off reactions in recovery after tripping discriminate young subjects, older non-fallers and older fallers", *Gait Posture*, vol. 21, no. 4, pp. 388-394.
- Poewe, W. 2006. "The natural history of Parkinson's disease". *J Neurol*, vol 253 (suppl 7), pp. 72-76.
- Prince, F., Corriveau, H., Hebert, R., & Winter, D. A. 1997, "Gait in the elderly", *Gait Posture*, vol.5, no 2, pp 128-135, 1997. no. 2, pp. 128-135.
- Rezak, M. 2007. "Current pharmacotherapeutic treatment options in parkinson's disease". *Dis Mon*, vol. 53, no. 4, pp. 214-222.
- Rocchi, L., Chiari, L., Mancini, M., Carlson-Kuhta, P., Gross, A., Horak, F.B., 2006. "Step initiation in Parkinson's disease: influence of initial stance conditions" *Neurosci Lett*, vol. 406, no. 1-2, pp. 128-132.
- Rochester, L., Nieuwboer, A., Baker, K., Hetherington, V., Willems, A.M., Kwakkel, G., Van Wegen, E., Lim, I., Jones, D. 2008. "Walking speed during single and dual tasks in Parkinson's disease which characteristics are important?" *Mov Disord*, vol. 23, no. 16, pp. 2312-2318.
- Rochester, L., Hetherington, V., Jones, D., Nieuwboer, A., Willems, A.M., Kwakkel, G., Van Wegen, E. 2004. "Attending to the task: Interference effects of functional task on walking in Parkinson's disease and the roles of cognition, depression, fatigue and balance". *Arch Phys Med Rehabil*, vol. 85, pp. 1578-1585.
- Rochester, L., Nieuwboer, A., Baker, K., Hetherington, V., Willems, A.M., Chavret, F., Kwakkel, G., Van Wegen, E., Jones, D. 2007. "The attentional cost of external rhythmical cues and their impact on gait in Parkinson's disease: effect of cue modality and task complexity". *J Neural Transm*, vol. 114, pp. 1243-1248.
- Roland, P. E., Skinhoj, E., Lassen, N. A., & Larsen, B. 1980, "Different cortical areas in man in organization of voluntary movements in extrapersonal space", *J Neurophysiol*, vol. 43, no. 1, pp. 137-50.
- Rothwell, J.C. "Motor Control" In: Stokes M. 2007. "Physical management in neurological rehabilitation". 2nd ed. Elsevier, Mosby.
- Sadeghi, H. 2000, "Contributions of lower limb power in gait of people without impairments", *Phys ther*, vol. 80, no. 12, pp. 1188-1196.
- Sadeghi, H., Sadeghi, S., Allard, P., Labelle, H., & Duhaime, M. 2001, "Lower limb muscle power relationships in bilateral able-bodied gait", *Am J Phys Med Rehabil*, vol. 80, no. 11, pp. 821-830.
- Sanderson, D. J., Franks, I. M., & Elliott, D. 1993, "The effects of targeting on the ground reaction forces during level walking", *Hum Mov Sci*, vol. 12, no. 3, pp. 327-337.
- Saunders, J. B., Inman, V. T., & Eberhart, H. D. 1953, "The major determinants in normal and pathological gait", *J Bone Joint Surg*, vol. 35A, pp. 543-558.

Saunders, J.B., Inman, V.T., & Eberhart, H.D. 1953. "The major determinants in normal and pathological gait". *J Bone Joint Surg*, vol. 35A, pp. 543-558.

Scott, B., Borgman, A., Engler, H., Johnels, B., Aquilonius, SM. 2000. "Gender differences in Parkinson's disease symptom profile". *Acta Neurol Scand*, vol. 102, no. 1, pp. 37-43.

Selby, G. 1975, "Parkinson's disease," in *Handbook of Clinical Neurology*, 2nd edn, Amsterdam, Elsevier, pp. 173-211.

Shaw, J. E., van Schie, C. H., Carrington, A. L., Abbott, C. A., & Boulton, A. J. 1998, "An analysis of dynamic forces transmitted through the foot in diabetic neuropathy", *Diabetes Care*, vol. 21, no. 11, pp. 1955-1959.

Shkuratova, N., Morris, M. E., & Huxham, F. 2004, "Effects of age on balance control during walking", *Arc Phys Med Rehabil*, vol. 85, no. 4, pp. 582-588.

Shulman, L.M, Gruber-Baldini, A.L., Anderson, K.E., Vaughan, C.G., Reich, S.G., Fishman, P.S., Weiner, W.J. 2008. "The evolution of disability in Parkinson's disease". *Mov Disord*, vol. 23, no. 6, pp. 790-796.

Shulman, L.M. 2007. "Gender differences in Parkinson's disease". *Gend Med*, vol. 4, no. 1, pp. 8-18.

Simuni T, Sethi K. 2008. Nonmotor manifestations of Parkinson's disease. *Ann Neurol*, vol. 64 Suppl 2, pp. S65-80.

Sofuwa, O., Nieuwboer, A., Desloovere, K., Willems, A. M., Chavret, F., & Jonkers, I. 2005, "Quantitative gait analysis in Parkinson's disease: comparison with a healthy control group", *Arch Phys Med Rehabil*, vol. 86, no. 5, pp. 1007-1013.

Solano, S. M., Miller, D. W., Augood, S. J., Young, A. B., & Penney, J. B., Jr. 2000, "Expression of alpha-synuclein, parkin, and ubiquitin carboxy-terminal hydrolase L1 mRNA in human brain: genes associated with familial Parkinson's disease", *Ann Neurol*, vol. 47, no. 2, pp. 201-210.

Stolze, H., Friedrich, H. J., Steinauer, K., & Vieregge, P. 2000, "Stride parameters in healthy young and old women--measurement variability on a simple walkway", *Exp.Aging Res.*, vol. 26, no. 2, pp. 159-168.

Sturman, M.M., Vaillancourt, D.E., Metman, L.V., Bakay, R.A, Corcos, D.M. 2004. Effects of subthalamic nucleus stimulation and medication on resting and postural tremor in Parkinson's disease. *Brain*, vol. 127, no. 9, pp. 2131-43.

Sutherland, D. H. 1998, "The development of mature walking", Mackeith Press London.

Toth, C., Raiput, M., Raiput, A.H. (2004). "Anomalies of asymmetry of clinical signs in Parkinsonism". *Mov Disord* vol. 19, no. 2, pp. 151-151.

Twelves, D., Perkins, K.S., Counsell, C. 2003. "Systematic review of incidence studies of Parkinson's disease". *Mov Disord*, vol. 18, no. 1, pp. 19-31.

- Van Den Eeden, S. K., Tanner, C. M., Bernstein, A. L., Fross, R. D., Leimpeter, A., Bloch, D. A., & Nelson, L. M. 2003, "Incidence of Parkinson's Disease: Variation by Age, Gender, and Race/Ethnicity", *American Journal of Epidemiology*, vol. 157, no. 11, pp. 1015-1022.
- Van Emmerik, R.E., Wagenaar, R.C., Winogrodzka, A., Wolters, E.C. 1999. Identification of axial rigidity during locomotion in Parkinson disease. *Arch Phys Med Rehabil*, vol. 80., no. 2, pp. 186-191.
- Van Wegen, E., Lim, I., De Goede, C., Nieuwboer, A., Willems, A., Jones, D., Rochester, L., Hetherington, V., Berendse, H., Zijlman, J., Wolters, E., Kwakkel, G. 2006. "The effects of visual rhythms and optic flow on stride patterns of patients with Parkinson's disease. *Parkinsonism Relat Disord*, vol. 12, pp. 21-27.
- Vardaxis, V. G., Allard, P., Lachance, R., & Duhaime, M. 1998, "Classification of able-bodied gait using 3-D muscle powers", *Hum Mov Sci*, vol. 17, no. 1, pp. 121-136.
- Watelain, E., Barbier, F., Allard, P., Thevenon, A., & Angue, J. C. 2000, "Gait pattern classification of healthy elderly men based on biomechanical data", *Arch Phys Med Rehabil*, vol. 81, no. 5, pp. 579-586.
- Waters, R.L., Lunsford, B.R., Perry, J., Byrd, R. 1988. "Energy-speed relationship of walking: standard tables." *J Orthop Res*, vol. 6, no. 2, pp. 215-222.
- Wearing, S. C., Urry, S. R., & Smeathers, J. E. 2000, "The effect of visual targeting on ground reaction force and temporospatial parameters of gait", *Clin Biomech*, vol. 15, no. 8, pp. 583-591.
- Weintraub, D., Moberg, P.J., Duda, J.E., Katz, I.R., Stern, M.B. 2004. Effect of psychiatric and other nonmotor symptoms on disability in Parkinson's disease. *J Am Geriatr Soc*, vol. 52, no. 5, pp. 784-788.
- Whittle, M. W. 2006. "Gait analysis: an introduction". 4th edn, Butterworth-Heinemann.
- Winter DA. 1991 "The biomechanics and motor control of human gait : normal, elderly and pathological", Ont, Waterloo Biomechanics.
- Winter, D. A., Patla, A. E., Frank, J. S., & Walt, S. E. 1990, "Biomechanical walking pattern changes in the fit and healthy elderly", *Phys.Ther.*, vol. 70, no. 6, pp. 340-347.
- Wooten, G. F., Currie, L. J., Bovbjerg, V. E., Lee, J. K., & Patrie, J. 2004, "Are men at greater risk for Parkinson's disease than women?", *Journal of Neurology, Neurosurgery, and Psychiatry*, vol. 75, no. 4, pp. 637-639.
- Zijlstra, W. & Hof, A. L. 2003, "Assessment of spatio-temporal gait parameters from trunk accelerations during human walking", *Gait Posture*, vol. 18, no. 2, pp. 1-10.
- Zijlstra, W. 2004, "Assessment of spatio-temporal parameters during unconstrained walking", *Eur J Appl Physiol*, vol.92, no. 1-2, pp. 39-44.

References

- Albanese, A. 2003, "Diagnostic criteria for Parkinson's disease", *Neurol Sci*, vol. 24, no. S23-S26.

Allard, P., Lachance, R., Aissaoui, R., Duhaime, M. 1996. "Simultaneous bilateral able-bodied gait". *Hum Mov sci*, vol 15, no 3, pp 327-346.

Almeida, Q. J., Frank, J. S., Roy, E. A., Patla, A. E., & Jog, M. S. 2007, "Dopaminergic modulation of timing control and variability in the gait of Parkinson's disease", *Mov Disord.*, vol. 22, no. 12, pp. 1735-1742.

Arnadottir, S. A. & Mercer, V. S. 2000, "Effects of footwear on measurements of balance and gait in women between the ages of 65 and 93 years", *Phys Ther*, vol.80, no.1, pp. 17-27.

Auvinet, B., Berrut, G., Touzat, C., Moutel, L., Collet, N., Chaleil, D and Barrey, E. 2002. "Reference data for normal subjects obtained with and accelerometric device". *Gait Posture*, vol. 16, no. 2, pp. 124-134.

Baker, K., Rochester, L., Nieuwboer, A. 2007. "The immediate effect of attentional, auditory, and a combined cue strategy on gait during single and dual tasks in Parkinson's disease." *Arch Phys Med Rehabil*, vol 88, no 12, pp 1593-6000.

Bassuk, S. S. & Murphy, J. M. 2003, "Characteristics of the Modified Mini-Mental State Exam among elderly persons", *J Clin Epidemiol*, vol 56, no. 7, pp. 622-628, 2003.

Behrman, A.L., Teitelbaum, P., Cauraugh, J.H., 1998. "Verbal instructional sets to normalise the temporal and spatial gait variables in Parkinson's disease". *J Neurol Neurosurg Psychiatr*, vol 65, pp 580-582.

Berardelli, A., Rothwell, J.C., Thompson, P.D., Hallet, M. 2001. "Pathophysiology of bradykinesia in Parkinson's disease". *Brain*, vol 124, pp 2131-2146.

Blanc, Y., balmer, C., Landis, T. And Vingerhoets, F. 1999. "Temporal parameters and patterns of the foot roll over during walkng: normative data for healthy adults". *Gait Posture*, vol. 10, no. 2, pp 124-134.

Behl, C., Manthey, D. 2000. "Neuroprotective activities of estrogen: an update". *J Neurocytol*, vol. 29, no. 5-6, pp. 351-358.

Bland, J., Altman, D. 1986, "Statistical methods for assessing agreement between two methods of clinincal measurement.", *Lancet*, vol. i, pp. 307-310.

Blanke, D. J. & Hageman, P. A. 1989, "Comparison of gait of young men and elderly men", *Phys Ther*, vol 69, no.2, pp 144-148.

Blin, O., Ferrandez, A. M., & Serratrice, G. 1990, "Quantitative analysis of gait in Parkinson patients: increased variability of stride length", *J Neurol Sci.*, vol. 98, no. 1, pp. 91-97.

Blin, O., Ferrandez, A. M., Pailhous, J., & Serratrice, G. 1991, "Dopa-sensitive and dopa-resistant gait parameters in Parkinson's disease", *J Neurol Sci.*, vol. 103, no. 1, pp. 51-54.

Bohannon RW.1997. "Comfortable and maximum walking speed of adults aged 20-79 years: reference values and determinants. *Age Ageing*, vol. 26, no. 1, pp. 15-19.

Bohannon, R. W. E., Andrews, A. W. M., & Thomas, M. W. B. 1996, "Walking Speed: Reference Values and Correlates for Older Adults. [Miscellaneous]", *J Orthop Sports Phys Ther*, vol. 24, no. 2, pp. 86-90.

- Bowes, S. G., Clark, P. K., Leeman, A. L., O'Neill, C. J., Weller, C., Nicholson, P. W., Deshmukh, A. A., Dobbs, S. M., & Dobbs, R. J. 1990, "Determinants of gait in the elderly parkinsonian on maintenance levodopa/carbidopa therapy", *Br J Clin Pharmacol*, vol. 30, no. 1, pp. 13-24.
- Brach, J.S., Berthold, R., Craik, R., Van Swearingen, J.M., Newman, A.B. 2001. "Gait variability in community-dwelling older adults". *J Am Geriatr Soc*, vol. 49, no. 12, pp. 1646-1650.
- Burke, D., Hagbarth, K. E., & Wallin, B. G. 1977, "Reflex mechanisms in Parkinsonian rigidity", *Scand. J Rehabil Med.*, vol. 9, no. 1, pp. 15-23.
- Cakit, B. D., Saracoglu, M., Genc, H., Erdem, H. R., & Inan, L. 2007, "The effects of incremental speed-dependent treadmill training on postural instability and fear of falling in Parkinson's disease", *Clin Rehabil*, vol. 21, no. 8, pp. 698-705.
- Camicioli, R., Bouchard, T., & Lici, L. 2006, "Dual-tasks and walking fast: relationship to extra-pyramidal signs in advanced Alzheimer disease", *J Neurol Sci.*, vol. 248, no. 1-2, pp. 205-209.
- Campbell, F. M., Ashburn, A. M., Pickering, R. M., & Burnett, M. 2001, "Head and Pelvic Movements During a Dynamic Reaching Task in Sitting: Implication for Physical Therapists", *Arch Phys Med Rehabil*, vol. 82, pp. 1655-1660.
- Canning, C.G., Ada, L., Johnson, J.J., McWhirter, S. 2006. "Walking capacity in mild to moderate Parkinson's disease" *Arch Phys Med Rehabil*, vol 87, no 3, pp 371-375.
- Cersosimo, M. G. & Koller, W. C. 2006, "The diagnosis of manganese-induced parkinsonism", *Neurotoxicology*, vol. 27, no. 3, pp. 340-346.
- Chao, E. Y., Laughman, R. K., Schneider, E., & Stauffer, R. N. 1983, "Normative data of knee joint motion and ground reaction forces in adult level walking", *J Biomech*, vol. 16, no. 3, pp. 219-233.
- Chastan, N., Westby, G.W., Yelnik, J., Bardinet, E., Do, M.C., Agid, Y., Welter, M.L. 2008. "Effect of nigral stimulation on locomotion and postural stability in patients with Parkinson's disease". *Brain*, vol 132 (pt 1), pp 172-184.
- Cheng, P. L. & Percy, M. 1999, "A three-dimensional definition for the flexion/extension and abduction/adduction angles", *Med Bio Eng Comput*, vol.37, no.4, pp. 440-444.
- Clarke, C. E. 2001. "Clinical features," in *Parkinson's disease in practice*, Royal Society of Medicine Press Ltd, Plymouth, pp. 17-18.
- Craik, R. 1995. *Gait analysis : Theory and application*, 1st edn, St. Louis : Mosby.
- Cunningham, D. A., Rechnitzer, P. A., Pearce, M. E., & Donner, A. P. 1982, "Determinants of self-selected walking pace across ages 19 to 66", *J Gerontol.*, vol. 37, no. 5, pp. 560-564.
- Davis, R. B., Ounpuu, S. & Deluca P. A. 1997. "Gait data: Reporting, archiving and sharing.," In: *Three-dimensional analysis of human locomotion*, Allard P., Cappozzo A., Lundberg A., & Vaughan C., ed., Wiley, Chichester, pp. 389-406.
- DeVita, P. & Hortobagyi, T. 2000, "Age causes a redistribution of joint torques and powers during gait", *J Appl Physiol*, vol. 88, no. 5, pp. 1804-1811.

- Di Monte, D. A., Lavasani, M., & Manning-Bog, A. B. 2002, "Environmental factors in Parkinson's disease", *Neurotoxicology*, vol. 23, no. 4-5, pp. 487-502.
- Dodson, M. W. & Guo, M. 2007, "Pink1, Parkin, DJ-1 and mitochondrial dysfunction in Parkinson's disease", *Curr Opin Neurobiol*, vol. 17, no. 3, pp. 331-337.
- Dujardin, K., Sockeel, P., Devos, D., Delliaux, M., Krystkowiak, P., Destee, A., & Defebvre, L. 2007, "Characteristics of apathy in Parkinson's disease", *Mov Disord.*, vol. 22, no. 6, pp. 778-784.
- Ebersbach, G., Sojer, M., Valldeoriola, F., Wissel, J., Muller, J., Tolosa, E., & Poewe, W. 1999, "Comparative analysis of gait in Parkinson's disease, cerebellar ataxia and subcortical arteriosclerotic encephalopathy", *Brain*, vol. 122 (Pt 7), pp. 1349-1355.
- Eke-Okoro, S. T., Gregoric, M., & Larsson, L. E. 1997, "Alterations in gait resulting from deliberate changes of arm-swing amplitude and phase", *Clin Biomech*, vol. 12, no. 7-8, pp. 516-521.
- Eng, J. J. & Winter, D. A. 1995, "Kinetic analysis of the lower limbs during walking: What information can be gained from a three-dimensional model?", *J Biomech*, vol.28, no 6, pp 753-758.
- Ferrarin, M., Rizzone, M., Bergamasco, B., Lanotte, M., Recalcati, M., Pedotti, A., Lopiano, L. 2005. "Effects of bilateral subthalamic stimulation on gait kinematics and kinetics in Parkinson's disease". *Exp Brain Res*, vol 160, no 4, pp 517-527.
- Ferrarin, M., Rabuffetti, M., Tettamanti, M., Pignatti, R., Mauro, A., Albani G. 2008. "Effect of optical flow versus attentional strategy on gait in Parkinson's disease: a study with portable optical stimulating device" *J NeuroEng Rehabil*, vol 5, no 3.
- Frenkel-Toledo, S., Giladi, N., Peretz, C., Herman, T., Gruendlinger, L., & Hausdorff, J. M. 2005, "Effect of gait speed on gait rhythmicity in Parkinson's disease: variability of stride time and swing time respond differently", *J Neuroeng Rehabil*, vol. 2, pp. 23.
- Folstein, M.F., Folstein, S.E., McHugh, P.R. 1975. "Mini-mental state". A practical method for grading the cognitive state of patients for the clinician. *J Psychiatr Res*, vol. 12, no. 3, pp 189-198.
- Gage, R. 1991, "Gait analysis in cerebral palsy". London, Mac Keith press.
- Giladi N, Kao R, Fahn S. 1997, "Freezing phenomenon in patients with parkinsonian syndromes", *Mov Disord* no. 12, pp. 302-305.
- Goetz CG, Poewe W, Rascol O, Sampaio C, Stebbins GT, Counsell C, Giladi N, Holloway RG, Moore CG, Wenning GK, Yahr MD, Seidl L; Movement Disorder Society Task Force on Rating Scales for Parkinson's Disease.2004 "Movement Disorder Society Task Force report on the Hoehn and Yahr staging scale: status and recommendations". *Mov Disord*. vol 19, no.9, pp 1020-1028.

- Goodwin, V.A, Richards, S.H., Taylor, R.S., Taylor, A.H., Campbell, J.L. 2008. "The effectiveness off exercise interventions for people with Parkinson's disease : a systematic and meta analysis". *Mov Disord*, vol 23, no 5, pp 631-640.
- Gorton, G.E. 3rd, Hebert, D.A., Gannotti, M.E. 2009. "Assessment of the kinematic variability among 12 motion analysis laboratories". *Gait Posture*, vol 29, no 3, pp 398-402.
- Grabiner, M. D., Feuerbach, J. W., Lundin, T. M., & Davis, B. L. 1995, "Visual guidance to force plates does not influence ground reaction force variability", *J Biomech*, vol. 28, no. 9, pp. 1115-1117.
- Graf, A., Judge, J. O., Ounpuu, S., & Thelen, D. G. 2005, "The effect of walking speed on lower-extremity joint powers among elderly adults who exhibit low physical performance", *Arch.Phys Med.Rehabil*, vol. 86, no. 11, pp. 2177-2183.
- Gray, P., Hildebrand, K. 2000. "Fall risk factors in Parkinson's disease". *J Neurosci Nurs*. Vol 32, no 4, pp 222-228
- Haaxma, C.A., Bloem, B.R., Borm, G.F., Oyen, W.J., Leenders, K.L., Eshuis, S., Booi, J., Dluzen, D.E., Horstink, M.W. 2007. "Gender differences in Parkinson's disease". *J Neurol Neurosurg Psychiatr*, vol 78, no 8, pp 819-824.
- Haber, S.N., Calzavara, R., 2009. "The cortico-basal ganglia integrative network: the role of the thalamus". *Brain Res Bull*. vol 78, no 2-3, pp 69-74.
- Hageman, P.A., Blanke, D.J. 1986. "Comparison of gait of young women and elderly women" *Phys Ther*, vol 66, no 9, pp 1382-1387.
- Hase, K. & Stein, R. B. 1998, "Analysis of rapid stopping during human walking", *J Neurophysiol* vol.80, no. 1, pp 255-261.
- Hausdorff JM, Zemaný L, Peng C, Goldberger AL. 1999. "Maturation of gait dynamics: stride-to-stride variability and its temporal organization in children." *J Appl Physiol*, vol. 86, no. 3, pp. 1040-1047.
- Hesse, S. A., Jahnke, M. T., Bertelt, C. M., Schreiner, C., Lucke, D., & Mauritz, K. H. 1994, "Gait outcome in ambulatory hemiparetic patients after a 4-week comprehensive rehabilitation program and prognostic factors", *Stroke*, vol. 25, no. 10, pp. 1999-2004.
- Hof, A.L. 2003. "Muscle mechanics and neuromuscular control". *J Biomech*, vol. 36, no. 7, pp. 1031-1038.
- Jankowski, J., Scheef, L., Hüppe, C., Boecker, H. 2009. Distinct striatal regions for planning and executing novel and automated movement sequences. *Neuroimage*, vol. 44, no. 4, pp. 1369-1379.
- Jansen, E. C., Vitas, D., Hellberg, S., & Hansen, J. 1982, "Normal gait of young and old men and women. Ground reaction force measurement on a treadmill", *Acta Orthop Scand*, vol. 53, no. 2, pp. 193-196.
- Jöbges, M., Heuschkel, G., Pretzel, C., Illhardt, C., Renner, C., Hummelsheim, H. 2004. "Repetitive training of compensatory steps: a therapeutic approach for postural instability in Parkinson's disease". *J Neurol Neurosurg Psychiatr*, vol. 75, no. 12, 1682-1687.
- Jones, D., Playfer, J. "Motor Control" In: Stokes M. 2007. "Physical management in neurological rehabilitation". 2nd ed, Elsevier, Mosby.

Judge, J.O., Davis, R.B. III and Ounpuu, S., 1996. "Step length reductions in advanced age: the role of ankle and hip kinetics". *J Gerontol A Biol Sci Med Sci*, vol. 51, no. 6, M303-312.

Kadaba, M. P., Ramakrishnan, H. K., Wootten, M. E., Gaine, J., Gorton, G., & Cochran, G. V. 1989, "Repeatability of kinematic, kinetic, and electromyographic data in normal adult gait", *J Orthop Res*, vol 7, no. 6, pp. 849-860.

Kahle-Wroblewski K, Corrada MM, Li B, Kawas CH. 2007. "Sensitivity and specificity of the mini-mental state examination for identifying dementia in the oldest-old: the 90+ study". *J Am Geriatr Soc*, vol.55, no. 2, pp 284-289.

Katzen, H.L., Levin, B.E., Weiner, W. 2006. "Side and symptom influence cognition in Parkinson's disease". *Mov Disord*, vol 21, no 11, pp. 1947-1953.

Kempster, P. A., Frankel, J. P., Bovingdon, M., Webster, R., Lees, A. J., & Stern, G. M. 1989, "Levodopa peripheral pharmacokinetics and duration of motor response in Parkinson's disease", *J Neurol Neurosurg Psychiatry*, vol. 52, no. 6, pp. 718-723.

Kerrigan, D.C., Lee, L.W., Nieto, T.J., Markman, J.D., Collins, J.J., Riley, P.O. 2000 "Kinetic alterations independent of walking speed in elderly fallers" *Arch Phys Med Rehabil*, vol. 81, no. 6, pp. 730-735.

Kerrigan, D. C., Todd, M. K., Della, C. U., Lipsitz, L. A., & Collins, J. J. 1998b, "Biomechanical gait alterations independent of speed in the healthy elderly: evidence for specific limiting impairments", *Arch Phys Med Rehabil*, vol. 79, no. 3, pp. 317-322.

Kerrigan, D. C., Xenopoulos-Oddsson, A., Sullivan, M. J., Lelas, J. J., & Riley, P. O. 2003, "Effect of a hip flexor-stretching program on gait in the elderly", *Arch Phys Med Rehabil*, vol. 84, no. 1, pp. 1-6.

Kerrigan, D.C., Todd, M.K. and Della, C.U. 1998. "Gender differences in joint biomechanics during walking: normative study in young adults", *Am J Phys Med Rehabil*, vol. 77, no. 1, pp. 2-7.

Kirtley, C. 2005. *Clinical Gait Analysis: Theory and Practice*. 1st edn, Churchill Livingstone.

Koozekanani, S. H., Balmaseda, M. T. Jr., Fatehi, M. T., & Lowney, E. D. 1987, "Ground reaction forces during ambulation in parkinsonism: pilot study", *Arch Phys Med Rehabil*, vol. 68, no. 1, pp. 28-30.

Kottke, F. J., Halpern, D., Easton, J. K., Ozel, A. T., & Burrill, C. A. 1978, "The training of coordination", *Arch Phys Med Rehabil*, vol. 59, no. 12, pp. 567-72.

Krack, P., Poepping, M., Weinert, D., Schrader, B., Deuschl, G. 2000. "Thalamic, Pallidal, or Subthalamic surgery for Parkinson's disease?". *J Neurol*, vol. 247, suppl 2, no. 2, pp. 22-34.

Laufer, Y. 2003, "Age- and gender-related changes in the temporal-spatial characteristics of forwards and backwards gaits", *Phys Res Int*, vol. 8, no. 3, pp. 131-42.

Lehmann, J. F. 1993, "Push-off and propulsion of the body in normal and abnormal gait", *Clinical Orthopaedics and Related Research*, vol. 288, pp. 97-108.

Lelas, J. L., Merriman, G. J., Riley, P. O., & Kerrigan, D. C. 2003, "Predicting peak kinematic and kinetic parameters from gait speed", *Gait Posture*, vol. 17, no. 2, pp. 106-112.

- Levy, R. & Czernecki, V. 2006, "Apathy and the basal ganglia", *J Neurol*, vol. 253 Suppl 7, p. VII54-VII61.
- Lewis, G. N., Byblow, W. D., & Walt, S. E. 2000, "Stride length regulation in Parkinson's disease: the use of extrinsic, visual cues", *Brain*, vol. 123 (Pt 10), pp. 2077-2090.
- Lin, R. S. & Gage, J. R. 1989, "The neurological control system for normal gait", *Journal of Prosthetics and Orthotics*, vol. 2, no. 1, pp. 1-13.
- Lord, S. R. & Bashford, G. M. 1996, "Shoe characteristics and balance in older women", *J Am Geriatr Soc*, vol. 44, no. 4, pp. 429-433.
- Lozano, A.M., Lang, E., Galvez-Jimenez, M. et al. 1995 "Effect of GPi pallidotomy on motor function in parkinson's disease". *Lancet*, vol. 346, pp. 1383-1387.
- Lubik, S., Fogel, W., Tronnier, V., Krause, M., König, J., Jost, W.H. 2006. "Gait analysis in patients with advanced Parkinson disease: different or additive effects on gait induced by levodopa and chronic STN stimulation. *J Neural Transm*, vol. 113, no2, pp163-173.
- Macphee, G. J. A. 2001, "Diagnosis and differential diagnosis," In: Playfer, J.R., Hindle, J., eds. *Parkinson's disease in the older patient*, J. R. H. J. V. Playfer, ed., London, Arnold, pp. 43-70.
- Maki, B.E. 1997. "Gait changes in older adults: predictors of falls or indicators of fear". *J Am Geriatr Soc*. vol. 45, no. 3, pp. 313-320.
- Mannion AF, Dolan P. 1996. "Relationship between myoelectric and mechanical manifestations of fatigue in the quadriceps femoris muscle group. *Eur J Appl Physiol Occup Physiol*.vol. 74, no. 5, pp. 411-419.
- Marks, R. 1997, "The effect of restricting arm swing during normal locomotion", *Biomed Sci Instrum*, vol.33, pp. 209-215.
- Martinez-Martin, P., Gil-Nagel, A., Gracia, L. M., Gomez, J. B., Martinez-Sarries, J., & Bermejo, F. 1994, "Unified Parkinson's Disease Rating Scale characteristics and structure. The Cooperative Multicentric Group", *Mov Disord.*, vol. 9, no. 1, pp. 76-83.
- Maynard, V., Bakheit, A. M. O., Oldham, J., & Freeman, J. 2003, "Intra-rater and inter-rater reliability of gait measurements with CODA mpx30 motion analysis system", *Gait Posture*, vol. 17, no. 1, pp. 59-67.
- McGibbon, C. A. & Krebs, D. E. 1999, "Effects of age and functional limitation on leg joint power and work during stance phase of gait", *J Rehabil Res.Dev.*, vol. 36, no. 3, pp. 173-182.
- McGinley, J. L., Goldie, P. A., Greenwood, K. M., & Olney, S. J. 2003, "Accuracy and reliability of observational gait analysis data: judgments of push-off in gait after stroke", *Phys Ther*, vol. 83, no. 2, pp. 146-60.
- Meara J, Hobson P. 2000 "Epidemiology of Parkinson's disease and Parkinsonism in elderly subjects". In Meara J, Koller W, eds. *Parkinson's disease and Parkinsonism in the elderly*. Cambridge: Cambridge University Press, pp. 111-122.
- Menz, H. B., Lord, S. R., & Fitzpatrick, R. C. 2003, "Age-related differences in walking stability", *Age Ageing*, vol. 32, no. 2, pp. 137-142.

- Metman, L. V., Myre, B., Verwey, N., Hassin-Baer, S., Arzbaeher, J., Sierens, D., & Bakay, R. 2004, "Test-retest reliability of UPDRS-III, dyskinesia scales, and timed motor tests in patients with advanced Parkinson's disease: an argument against multiple baseline assessments", *Mov Disord.*, vol. 19, no. 9, pp. 1079-1084.
- Mills, P. M. & Barrett, R. S. 2001, "Swing phase mechanics of healthy young and elderly men", *Hum Mov Sci.*, vol. 20, no. 4-5, pp. 427-446.
- Monaghan, K., Delahunt, E., & Caulfield, B. 2007. "Increasing the number of gait trial recordings maximises intra-rater reliability of the CODA motion analysis system", *Gait Posture*, vol. 25, no. 2, pp. 303-315.
- Morris, M.E., Iansek, R., Matyas, T.A., Summers, J.J. 1996 "Stride length regulation in Parkinson's disease. Normalization strategies and underlying mechanisms". *Brain*, vol. 119 (Pt2), pp. 551-568.
- Morris, M. E., Iansek, R., Matyas, T. A., & Summers, J. J. 1994a, "Ability to modulate walking cadence remains intact in Parkinson's disease", *J Neurol Neurosurg Psychiatr*, vol. 57, no. 12, pp. 1532-1534.
- Morris, M. E., Iansek, R., Matyas, T. A., & Summers, J. J. 1994b, "The pathogenesis of gait hypokinesia in Parkinson's disease", *Brain*, vol. 117, no. 5, pp. 1169-1181.
- Morris, M. E., Matyas, T. A., Iansek, R., & Summers, J. J. 1996, "Temporal stability of gait in Parkinson's disease", *Phys Ther*, vol 76, no. 7, pp. 763-777; discussion 778-80.
- Morris, M. E., McGinley, J., Huxham, F., Collier, J., & Iansek, R. 1999, "Constraints on the kinetic, kinematic and spatiotemporal parameters of gait in Parkinson's disease", *Hum Mov Sci*, vol. 18, no. 2-3, pp. 461-483.
- Morris, M., Iansek, R., Matyas, T., & Summers, J. 1998, "Abnormalities in the stride length-cadence relation in parkinsonian gait", *Mov Disord*, vol. 13, no. 1, pp. 61-69.
- Morris, M., Iansek, R., McGinley, J., Matyas, T., & Huxham, F. 2005, "Three-dimensional gait biomechanics in Parkinson's disease: evidence for a centrally mediated amplitude regulation disorder", *Mov Disord.* vol. 20, no. 1, pp. 40-50.
- Morris, M.E. 2000. "Movement Disorders in people with Parkinson's disease: A model for physical therapy". *Phys Ther*, vol. 80, pp. 578-597.
- Murray, M.P., Septic, S.B., Gardner, G.M., & Downess, W.J 1978, "Walking patterns of men with Parkinsonism", *Amer J Phys Med*, pp. 278-294.
- Murray, M. P., Kory, R. C., & Clarkson, B. H. 1969, "Walking patterns in healthy old men", *J Gerontol*, vol. 24, no. 2, pp. 169-178.
- Murray, M. P., Kory, R. C., & Sepic, S. B. 1970, "Walking patterns of normal women", *Arch Phys Med Rehabil*, vol. 51, no. 11, pp. 637-650.
- Nadeau, S., Gravel, D., Arsenault, A. B., & Bourbonnais, D. 1999, "Plantarflexor weakness as a limiting factor of gait speed in stroke subjects and the compensating role of hip flexors", *Clin Biomech*, vol. 14, no. 2, pp. 125-135.
- Nieuwboer, A., Feys, P., De Weerd, W., Dom R. 1997. "Is using a cue the clue to the treatment of freezing in Parkinson's disease?" *Phys ther Res Int*, vol 2, no 3, pp 125-132.

- Nieuwboer, A., De, W. W., Dom, R., & Lesaffre, E. 1998, "A frequency and correlation analysis of motor deficits in Parkinson patients", *Disabil Rehabil*, no. 4, pp. 142-150.
- Nieuwboer, A., Dom, R., De Weerd, W., Desloovere, K., Janssens, L., Stijn, V. 2004. "Electromyographic profiles of gait prior to onset of freezing episodes in patients with Parkinson's disease." *Brain*, vol. 127, pt 7, pp. 1650-1660.
- Nurse, M. A., Hulliger, M., Wakeling, J. M., Nigg, B. M., & Stefanyshyn, D. J. 2005, "Changing the texture of footwear can alter gait patterns", *J Electromyogr Kinesiol*, vol. 15, no. 5, pp. 496-506.
- Nutt, J.G. 2008. "Pharmacokinetics and pharmacodynamics of levodopa". *Mov Disord*, vol. 23, suppl 3, S580-584.
- Oberg, T., Karsznia, A., & Oberg, K. 1993, "Basic gait parameters: reference data for normal subjects, 10-79 years of age", *J Rehabil Res Dev*, vol. 30, no. 2, pp. 210-223.
- Oberg, T., Karsznia, A., & Oberg, K. 1994, "Joint angle parameters in gait: reference data for normal subjects, 10-79 years of age", *J Rehabil Res Dev*, vol. 31, no. 3, pp. 199-213.
- Obeso, J.A., Olanow, C.W., Roriguez-Oroz, M.C., Krack, P., Kumar, R., Lang, A.E. 2001. "Deep-brain stimulation of the subthalamic nucleus or pars interna of the globus pallidus In: Parkinson's disease; Deep-brain stimulation of the Parkinson's disease study group". *N Engl J Med*, vol. 345, no. 13, pp. 956-963.
- Obeso, J.A., Rodriguez-Oroz, M.C., Chana, P., Lera, G., Rodriguez, M., Olanow, C.W. 2000. "The evolution and origin of motor complications in Parkinson's disease". *Neurol*, vol. 55, no 11 suppl 4, S13-S20.
- Oeffinger, D., Brauch, B., Cranfill, S., Hisle, C., Wynn, C., Hicks, R., & Augsburger, S. 1999, "Comparison of gait with and without shoes in children", *Gait Posture*, vol. 9, no. 2, pp. 95-100.
- Ostrosky, K. M., VanSwearingen, J. M., Burdett, R. G., & Gee, Z. 1994, "A comparison of gait characteristics in young and old subjects", *Phys Ther*, vol. 74, no. 7, pp. 637-644.
- Paul, J. P. 1996, "Visual guidance to forceplates does not influence ground reaction force variability", *J Biomech*, vol. 29, no. 6, p. 833.
- Parkinson's disease Society, UK. 2008 (<http://www.parkinsons.org.uk/>)
- Pellicano, C., Benincasa, D., Giovannelli, M., Buttarelli, F.R., Ruggieri, S., Pontieri, F.E. 2009. "Entacapone in elderly Parkinsonian patients experiencing levodopa-related wearing-off: a pilot study". *Neurol Res*, vol, 31, no1, pp 74-76.
- Peppe, A., Chiavalon, C., Pasqualetti, P., Crovato, D., & Caltagirone, C. 2007, "Does gait analysis quantify motor rehabilitation efficacy in Parkinson's disease patients?", *Gait Posture*, vol. 26, no. 3, pp. 452-462.
- Pereira, E. A. C. & Aziz, T. Z. 2006, "Surgical insights into Parkinson's disease", *J R Soc Med*, vol. 99, no. 5, pp. 238-244.
- Perry, J. 1992, "Gait analysis-normal and patholocival gait," Slack Inc, USA.
- Perttunen, J., Anttila, E., Sodergard, J., Merikanto, J., & Komi, P. 2004, "Gait asymmetry in patients with limb length discrepancy", *Scand J Med and Sci Sports*, vol. 14, no. 1, pp. 49-56.

Pijnappels, M., Bobbert, M. F., & Dieen, J. H. 2005a, "Push-off reactions in recovery after tripping discriminate young subjects, older non-fallers and older fallers", *Gait Posture*, vol. 21, no. 4, pp. 388-394.

Poewe, W. 2006. "The natural history of Parkinson's disease". *J Neurol*, vol 253 (suppl 7), pp. 72-76.

Prince, F., Corriveau, H., Hebert, R., & Winter, D. A. 1997, "Gait in the elderly", *Gait Posture*, vol.5, no 2, pp 128-135, 1997. no. 2, pp. 128-135.

Rezak, M. 2007. "Current pharmacotherapeutic treatment options in parkinson's disease". *Dis Mon*, vol. 53, no. 4, pp. 214-222.

Rocchi, L., Chiari, L., Mancini, M., Carlson-Kuhta, P., Gross, A., Horak, F.B., 2006. "Step initiation in Parkinson's disease: influence of initial stance conditions" *Neurosci Lett*, vol. 406, no. 1-2, pp. 128-132.

Rochester, L., Nieuwboer, A., Baker, K., Hetherington, V., Willems, A.M., Kwakkel, G., Van Wegen, E., Lim, I., Jones, D. 2008. "Walking speed during single and dual tasks in Parkinson's disease which characteristics are important?" *Mov Disord*, vol. 23, no. 16, pp. 2312-2318.

Rochester, L., Hetherington, V., Jones, D., Nieuwboer, A., Willems, A.M., Kwakkel, G., Van Wegen, E. 2004. "Attending to the task: Interference effects of functional task on walking in Parkinson's disease and the roles of cognition, depression, fatigue and balance". *Arch Phys Med Rehabil*, vol. 85, pp. 1578-1585.

Rochester, L., Nieuwboer, A., Baker, K., Hetherington, V., Willems, A.M., Chavret, F., Kwakkel, G., Van Wegen, E., Jones, D. 2007. "The attentional cost of external rhythmical cues and their impact on gait in Parkinson's disease: effect of cue modality and task complexity". *J Neural Transm*, vol. 114, pp. 1243-1248.

Roland, P. E., Skinhoj, E., Lassen, N. A., & Larsen, B. 1980, "Different cortical areas in man in organization of voluntary movements in extrapersonal space", *J Neurophysiol*, vol. 43, no. 1, pp. 137-50.

Rothwell, J.C. "Motor Control" In: Stokes M. 2007. "Physical management in neurological rehabilitation". 2nd ed. Elsevier, Mosby.

Sadeghi, H. 2000, "Contributions of lower limb power in gait of people without impairments", *Phys ther*, vol. 80, no. 12, pp. 1188-1196.

Sadeghi, H., Sadeghi, S., Allard, P., Labelle, H., & Duhaime, M. 2001, "Lower limb muscle power relationships in bilateral able-bodied gait", *Am J Phys Med Rehabil*, vol. 80, no. 11, pp. 821-830.

Sanderson, D. J., Franks, I. M., & Elliott, D. 1993, "The effects of targeting on the ground reaction forces during level walking", *Hum Mov Sci*, vol. 12, no. 3, pp. 327-337.

Saunders, J. B., Inman, V. T., & Eberhart, H. D. 1953, "The major determinants in normal and pathological gait", *J Bone Joint Surg*, vol. 35A, pp. 543-558.

Saunders, J.B., Inman, V.T., & Eberhart, H.D. 1953. "The major determinants in normal and pathological gait". *J Bone Joint Surg*, vol. 35A, pp. 543-558.

- Scott, B., Borgman, A., Engler, H., Johnels, B., Aquilonius, SM. 2000. "Gender differences in Parkinson's disease symptom profile". *Acta Neurol Scand*, vol. 102, no. 1, pp. 37-43.
- Selby, G. 1975, "Parkinson's disease," in *Handbook of Clinical Neurology*, 2nd edn, Amsterdam, Elsevier, pp. 173-211.
- Shaw, J. E., van Schie, C. H., Carrington, A. L., Abbott, C. A., & Boulton, A. J. 1998, "An analysis of dynamic forces transmitted through the foot in diabetic neuropathy", *Diabetes Care*, vol. 21, no. 11, pp. 1955-1959.
- Shkuratova, N., Morris, M. E., & Huxham, F. 2004, "Effects of age on balance control during walking", *Arc Phys Med Rehabil*, vol. 85, no. 4, pp. 582-588.
- Shulman, L.M, Gruber-Baldini, A.L., Anderson, K.E., Vaughan, C.G., Reich, S.G., Fishman, P.S., Weiner, W.J. 2008. "The evolution of disability in Parkinson's disease". *Mov Disord*, vol. 23, no. 6, pp. 790-796.
- Shulman, L.M. 2007. "Gender differences in Parkinson's disease". *Gend Med*, vol. 4, no. 1, pp. 8-18.
- Simuni T, Sethi K. 2008. Nonmotor manifestations of Parkinson's disease. *Ann Neurol*, vol. 64 Suppl 2, pp. S65-80.
- Sofuwa, O., Nieuwboer, A., Desloovere, K., Willems, A. M., Chavret, F., & Jonkers, I. 2005, "Quantitative gait analysis in Parkinson's disease: comparison with a healthy control group", *Arch Phys Med Rehabil*, vol. 86, no. 5, pp. 1007-1013.
- Solano, S. M., Miller, D. W., Augood, S. J., Young, A. B., & Penney, J. B., Jr. 2000, "Expression of alpha-synuclein, parkin, and ubiquitin carboxy-terminal hydrolase L1 mRNA in human brain: genes associated with familial Parkinson's disease", *Ann Neurol*, vol. 47, no. 2, pp. 201-210.
- Stolze, H., Friedrich, H. J., Steinauer, K., & Vieregge, P. 2000, "Stride parameters in healthy young and old women--measurement variability on a simple walkway", *Exp.Aging Res.*, vol. 26, no. 2, pp. 159-168.
- Sturman, M.M., Vaillancourt, D.E., Metman, L.V., Bakay, R.A, Corcos, D.M. 2004. Effects of subthalamic nucleus stimulation and medication on resting and postural tremor in Parkinson's disease. *Brain*, vol. 127, no. 9, pp. 2131-43.
- Sutherland, D. H. 1998, "The development of mature walking", Mackeith Press London.
- Toth, C., Raiput, M., Raiput, A.H. (2004). "Anomalies of asymmetry of clinical signs in Parkinsonism". *Mov Disord* vol. 19, no. 2, pp. 151-151.
- Twelves, D., Perkins, K.S., Counsell, C. 2003. "Systematic review of incidence studies of Parkinson's disease". *Mov Disord*, vol. 18, no. 1, pp. 19-31.
- Van Den Eeden, S. K., Tanner, C. M., Bernstein, A. L., Fross, R. D., Leimpeter, A., Bloch, D. A., & Nelson, L. M. 2003, "Incidence of Parkinson's Disease: Variation by Age, Gender, and Race/Ethnicity", *American Journal of Epidemiology*, vol. 157, no. 11, pp. 1015-1022.

- Van Emmerik, R.E., Wagenaar, R.C., Winogrodzka, A., Wolters, E.C. 1999. Identification of axial rigidity during locomotion in Parkinson disease. *Arch Phys Med Rehabil*, vol. 80., no. 2, pp. 186-191.
- Van Wegen, E., Lim, I., De Goede, C., Nieuwboer, A., Willems, A., Jones, D., Rochester, L., Hetherington, V., Berendse, H., Zijlman, J., Wolters, E., Kwakkel, G. 2006. "The effects of visual rhythms and optic flow on stride patterns of patients with Parkinson's disease. *Parkinsonism Relat Disord*, vol. 12, pp. 21-27.
- Vardaxis, V. G., Allard, P., Lachance, R., & Duhaime, M. 1998, "Classification of able-bodied gait using 3-D muscle powers", *Hum Mov Sci*, vol. 17, no. 1, pp. 121-136.
- Watelain, E., Barbier, F., Allard, P., Thevenon, A., & Angue, J. C. 2000, "Gait pattern classification of healthy elderly men based on biomechanical data", *Arch Phys Med Rehabil*, vol. 81, no. 5, pp. 579-586.
- Waters, R.L., Lunsford, B.R., Perry, J., Byrd, R. 1988. "Energy-speed relationship of walking: standard tables." *J Orthop Res*, vol. 6, no. 2, pp. 215-222.
- Wearing, S. C., Urry, S. R., & Smeathers, J. E. 2000, "The effect of visual targeting on ground reaction force and temporospatial parameters of gait", *Clin Biomech*, vol. 15, no. 8, pp. 583-591.
- Weintraub, D., Moberg, P.J., Duda, J.E., Katz, I.R., Stern, M.B. 2004. Effect of psychiatric and other nonmotor symptoms on disability in Parkinson's disease. *J Am Geriatr Soc*, vol. 52, no. 5, pp. 784-788.
- Whittle, M. W. 2006. "Gait analysis: an introduction". 4th edn, Butterworth-Heinemann.
- Winter DA. 1991 "The biomechanics and motor control of human gait : normal, elderly and pathological", Ont, Waterloo Biomechanics.
- Winter, D. A., Patla, A. E., Frank, J. S., & Walt, S. E. 1990, "Biomechanical walking pattern changes in the fit and healthy elderly", *Phys.Ther.*, vol. 70, no. 6, pp. 340-347.
- Wooten, G. F., Currie, L. J., Bovbjerg, V. E., Lee, J. K., & Patrie, J. 2004, "Are men at greater risk for Parkinson's disease than women?", *Journal of Neurology, Neurosurgery, and Psychiatry*, vol. 75, no. 4, pp. 637-639.
- Zijlstra, W. & Hof, A. L. 2003, "Assessment of spatio-temporal gait parameters from trunk accelerations during human walking", *Gait Posture*, vol. 18, no. 2, pp. 1-10.
- Zijlstra, W. 2004, "Assessment of spatio-temporal parameters during unconstrained walking", *Eur J Appl Physiol*, vol.92, no. 1-2, pp. 39-44.

The following references are AUTOMATICALLY generated and INCOMPLETE please refer to the above for a complete list of references

References

- Arnadottir, S. A. & Mercer, V. S. 2000, "Effects of footwear on measurements of balance and gait in women between the ages of 65 and 93 years", *Phys Ther*, Vol.80, no.1, pp17-27.
- Bassuk, S. S. & Murphy, J. M. 2003, "Characteristics of the Modified Mini-Mental State Exam among elderly persons", *Journal of Clinical Epidemiology*.56(7)(pp 622-628), 2003.Date of Publication: 01 Jul 2003. no. 7, pp. 622-628.
- Blanke, D. J. & Hageman, P. A. 1989, "Comparison of gait of young men and elderly men", *Phys Ther*, vol 69, no.2, pp 144-148.
- Burke, D., Hagbarth, K. E., & Wallin, B. G. 1977, "Reflex mechanisms in Parkinsonian rigidity", *Scand.J Rehabil Med.*, vol. 9, no. 1, pp. 15-23.
- Cheng, P. L. & Pearcy, M. 1999, "A three-dimensional definition for the flexion/extension and abduction/adduction angles", *Med Bio Eng Comput*, vol.37, no.4, pp 440-444.
- Clarke, C. E. 2001, "Clinical features," in *Parkinson's disease in practice*, Royal Society of Medicine Press Ltd, Plymouth, pp. 17-18.
- Craik, R. 1995, *Gait analysis : Theory and application*, 1st edn, St. Louis : Mosby.
- Dodson, M. W. & Guo, M. 2007, "Pink1, Parkin, DJ-1 and mitochondrial dysfunction in Parkinson's disease", *Current Opinion in Neurobiology*, vol. 17, no. 3, pp. 331-337.
- Eke-Okoro, S. T., Gregoric, M., & Larsson, L. E. 1997, "Alterations in gait resulting from deliberate changes of arm-swing amplitude and phase", *Clinical Biomechanics*, vol. 12, no. 7-8, pp. 516-521.
- Eng, J. J. & Winter, D. A. 1995, "Kinetic analysis of the lower limbs during walking: What information can be gained from a three-dimensional model?", *Journal of Biomechanics*.Vol.28(6)(pp 753-758), 1995. no. 6, pp. 753-758.
- Gage, J. R. 1991, *Gait analysis in Cerebral Palsy* MacKeith Press, New York.
- Kadaba, M. P., Ramakrishnan, H. K., Wootten, M. E., Gainey, J., Gorton, G., & Cochran, G. V. 1989, "Repeatability of kinematic, kinetic, and electromyographic data in normal adult gait", *Journal of Orthopaedic Research*.7(6):849-60.

- Kempster, P. A., Frankel, J. P., Bovingdon, M., Webster, R., Lees, A. J., & Stern, G. M. 1989, "Levodopa peripheral pharmacokinetics and duration of motor response in Parkinson's disease", *J Neurol Neurosurg Psychiatry*, vol. 52, no. 6, pp. 718-723.
- Kerrigan, D. C., Todd, M. K., Della, C. U., Lipsitz, L. A., & Collins, J. J. 1998, "Biomechanical gait alterations independent of speed in the healthy elderly: evidence for specific limiting impairments", *Arch Phys Med Rehabil*, vol. 79, no. 3, pp. 317-322.
- Kottke, F. J., Halpern, D., Easton, J. K., Ozel, A. T., & Burrill, C. A. 1978, "The training of coordination", *Archives of Physical Medicine & Rehabilitation*.59(12):567-72.
- Lelas, J. L., Merriman, G. J., Riley, P. O., & Kerrigan, D. C. 2003, "Predicting peak kinematic and kinetic parameters from gait speed", *Gait Posture*, vol. 17, no. 2, pp. 106-112.
- Lewis, G. N., Byblow, W. D., & Walt, S. E. 2000, "Stride length regulation in Parkinson's disease: the use of extrinsic, visual cues", *Brain*, vol. 123 (Pt 10), pp. 2077-2090.
- Lin, R. S. & Gage, J. R. 1989, "The neurological control system for normal gait", *Journal of Prosthetics and Orthotics*, vol. 2, no. 1, pp. 1-13.
- Lord, S. R. & Bashford, G. M. 1996, "Shoe characteristics and balance in older women", *J Am Geriatr Soc*, vol. 44, no. 4, pp. 429-433.
- Macphee, G. J. A. 2001, "Diagnosis and differential diagnosis," in *Parkinson's disease in the older patient*, J. R. H. J. V. Playfer, ed., Arnold, London, pp. 43-70.
- Marks, R. 1997, "The effect of restricting arm swing during normal locomotion", *Biomedical Sciences Instrumentation*.33:209-15.
- Martinez-Martin, P., Gil-Nagel, A., Gracia, L. M., Gomez, J. B., Martinez-Sarries, J., & Bermejo, F. 1994, "Unified Parkinson's Disease Rating Scale characteristics and structure. The Cooperative Multicentric Group", *Mov Disord.*, vol. 9, no. 1, pp. 76-83.
- Maynard, V., Bakheit, A. M. O., Oldham, J., & Freeman, J. 2003, "Intra-rater and inter-rater reliability of gait measurements with CODA mpx30 motion analysis system", *Gait & Posture*, vol. 17, no. 1, pp. 59-67.
- Metman, L. V., Myre, B., Verwey, N., Hassin-Baer, S., Arzbaecher, J., Sierens, D., & Bakay, R. 2004, "Test-retest reliability of UPDRS-III, dyskinesia scales, and timed motor tests in patients with advanced Parkinson's disease: an argument against multiple baseline assessments", *Mov Disord.*, vol. 19, no. 9, pp. 1079-1084.
- Morris, M. E., Iansek, R., Matyas, T. A., & Summers, J. J. 1994, "Ability to modulate walking cadence remains intact in Parkinson's disease", *Journal of Neurology, Neurosurgery & Psychiatry*.57(12):1532-4.
- Morris, M. E., Matyas, T. A., Iansek, R., & Summers, J. J. 1996, "Temporal stability of gait in Parkinson's disease", *Physical Therapy*.76(7):763-77; discussion 778-80.
- Morris, M. E., McGinley, J., Huxham, F., Collier, J., & Iansek, R. 1999, "Constraints on the kinetic, kinematic and spatiotemporal parameters of gait in Parkinson's disease", *Hum Mov Sci*, vol. 18, no. 2-3, pp. 461-483.

Nadeau, S., Gravel, D., Arsenault, A. B., & Bourbonnais, D. 1999, "Plantarflexor weakness as a limiting factor of gait speed in stroke subjects and the compensating role of hip flexors", *Clinical Biomechanics*, vol. 14, no. 2, pp. 125-135.

Nieuwboer, A., De, W. W., Dom, R., & Lesaffre, E. 1998, "A frequency and correlation analysis of motor deficits in Parkinson patients", *Disability & Rehabilitation*. no. 4, pp. 142-150.

Oeffinger, D., Brauch, B., Cranfill, S., Hisle, C., Wynn, C., Hicks, R., & Augsburger, S. 1999, "Comparison of gait with and without shoes in children", *Gait & Posture*.9(2):95-100.

Ostrosky, K. M., VanSwearingen, J. M., Burdett, R. G., & Gee, Z. 1994, "A comparison of gait characteristics in young and old subjects", *Phys Ther*, vol. 74, no. 7, pp. 637-644.

Pereira, E. A. C. & Aziz, T. Z. 2006, "Surgical insights into Parkinson's disease", *Journal of the Royal Society of Medicine*, vol. 99, no. 5, pp. 238-244.

Perttunen, J., Anttila, E., Sodergard, J., Merikanto, J., & Komi, P. 2004, "Gait asymmetry in patients with limb length discrepancy", *Scandinavian Journal of Medicine and Science in Sports*, vol. 14, no. 1, pp. 49-56.

Roland, P. E., Skinhoj, E., Lassen, N. A., & Larsen, B. 1980, "Different cortical areas in man in organization of voluntary movements in extrapersonal space", *Journal of Neurophysiology*.43(1):137-50.

Sadeghi, H., Sadeghi, S., Allard, P., Labelle, H., & Duhaime, M. 2001, "Lower limb muscle power relationships in bilateral able-bodied gait", *American Journal of Physical Medicine & Rehabilitation*.80(11):821-30.

Saunders, J. B., Inman, V. T., & Eberhart, H. D. 1953, "The major determinants in normal and pathological gait", *J Bone Joint Surg*, vol. 35A, pp. 543-558.

Selby G 1975, "Parkinson's disease," in *Handbook of Clinical Neurology*, 2nd edn, Elsevier, Amsterdam, pp. 173-211.

Shaw, J. E., van Schie, C. H., Carrington, A. L., Abbott, C. A., & Boulton, A. J. 1998, "An analysis of dynamic forces transmitted through the foot in diabetic neuropathy", *Diabetes Care*, vol. 21, no. 11, pp. 1955-1959.

Solano, S. M., Miller, D. W., Augood, S. J., Young, A. B., & Penney, J. B., Jr. 2000, "Expression of alpha-synuclein, parkin, and ubiquitin carboxy-terminal hydrolase L1 mRNA in human brain: genes associated with familial Parkinson's disease", *Ann.Neurol*, vol. 47, no. 2, pp. 201-210.

Van Den Eeden, S. K., Tanner, C. M., Bernstein, A. L., Fross, R. D., Leimpeter, A., Bloch, D. A., & Nelson, L. M. 2003, "Incidence of Parkinson's Disease: Variation by Age, Gender, and Race/Ethnicity", *American Journal of Epidemiology*, vol. 157, no. 11, pp. 1015-1022.

Winter, D. A., Patla, A. E., Frank, J. S., & Walt, S. E. 1990, "Biomechanical walking pattern changes in the fit and healthy elderly", *Phys.Ther.*, vol. 70, no. 6, pp. 340-347.

Wooten, G. F., Currie, L. J., Bovbjerg, V. E., Lee, J. K., & Patrie, J. 2004, "Are men at greater risk for Parkinson's disease than women?", *Journal of Neurology, Neurosurgery, and Psychiatry*, vol. 75, no. 4, pp. 637-639.

Zijlstra, W. 2004, "Assessment of spatio-temporal parameters during unconstrained walking", *European Journal of Applied Physiology*.92(1-2):39-44.