

**THE EFFECT OF FUNCTIONAL ELECTRICAL
STIMULATION ON AKINETIC GAIT IN PATIENTS
WITH PARKINSON'S DISEASE**

By

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STATEMENT

I Nicole Ashleigh Uys, declare that the dissertation which I hereby submit for the degree M-PhysT at the University of Pretoria, is my own work and has not been previously submitted by me for a degree at another tertiary institution.

Where secondary material has been used, this has been carefully acknowledged and referenced in accordance with the university requirements. I am ware of university policies and implications regarding plagiarism.

Nicole Ashleigh Uys

Date

LANGUAGE EDITORS LETTER

DEDICATION

This dissertation is dedicated to Dr. Carina Eksteen. This is for her dedication to assisting me in completing this clinical trial. For her words of motivation, assurance and continued support throughout the clinical trial. It has been an honour to work with such a talented, amazing person. Without Dr. Eksteen behind me every step of the way, this clinical trial may not have been completed.

EXPRESSION OF THANKS

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ABSTRACT

Impaired gait and postural instability in patients living with Parkinson's disease (PD) are regarded as the main aspects of the disease that causes disability in their home and work environment. As a progressive neurological movement disorder due to degeneration in the basal ganglia, PD is the second most common neurological disease after stroke and the fourth most common neuro-degenerative disease in the elderly.

Functional Electrical Sensory Stimulation (FESS) (Group 1) and Functional Electrical Sensory and Motor Stimulation (FES&MS) (Group 2) was administered to the common peroneal nerve as external cues to facilitate the initiation of taking a step in patients with akinesia.

The hypotheses that were tested were:

Hypothesis 1 (H1)

FESS and FES&MS decrease freezing/akinesia and bradykinesia/hypokinesia during gait in patients suffering from PD.

Null Hypothesis (H0)

FESS and FES&MS do not decrease freezing/akinesia and bradykinesia/hypokinesia during gait in patients suffering from PD.

Hypothesis 2 (H2)

FESS and FES&MS decrease freezing/akinesia and bradykinesia/hypokinesia during gait in patients suffering from PD to such an extent that it improves the quality of life of patients.

Null Hypothesis 2 (H02)

FESS and FES&MS do not decrease freezing/akinesia and bradykinesia/hypokinesia during gait in patients suffering from PD to such an extent that it improves the quality of life of the patients.

Hypothesis 3 (H3)

FESS decreases freezing/akinesia and bradykinesia/hypokinesia during gait in patients suffering from PD more than FES&MS.

Null Hypothesis 3 (H03)

FESS does not decrease freezing/akinesia and bradykinesia/hypokinesia during gait in patients suffering from PD more than FES&MS.

A single blind, randomized active controlled clinical trial was conducted.

Patients with PD who experienced freezing/akinesia and bradykinesia / hypokinesia and met the inclusion and exclusion criteria of the study were allocated randomly into two groups. Ten (10) patients were randomly allocated to each group.

The baseline measurement was determined by calculating the average of the measurements at week zero (0) and week two (2). Results of the participants in each group at week fourteen (14) (after twelve (12) weeks of FESS and FES&MS respectively) were compared to their baseline measurement, as well as between Group 1 and Group 2. The FESS and FES&MS was removed at week fourteen (14) and measurements were repeated at week twenty four (24).

Parameters of gait that were used in the trial included; the time, speed, number of steps, average step length, Freezing of gait scale and PCI to complete a walking task. The QoI was determined by using the PDQ-39 and the motor part of the UPDRS. From the results obtained the alternative hypotheses H1, H2 and H3 was accepted for Group 1.

The null hypothesis H0 and the alternative hypothesis H3 was accepted for Group 2.

It can be concluded from the results of this clinical trial that FESS decreases akinetic episodes in patients with PD statistically significantly and clinically improves their QoI. QoI was statistically significantly improved in Group 2 although hypothesis H1 was not accepted for Group 2.

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ABBREVIATIONS

ADL	Activities of Daily Living
ANS	Autonomis Nervous System
DBS	Deep Brain Stimulation
DLB	Dementia with Lewy Bodies
EMF's	Electromagnetic Fields
EMG	Electromyographic Activity
FES	Functional Electrical Stimulation
FES&MS	Functional Electrical Sensory Stimulation and Motor Stimulation
FESS	Functional Electrical Sensory Stimulation
FOG	Freezing of Gait
GPe	Globus Pallidus External
GPi	Globus Pallidus Internal
GS	Gastrocnemius
ICD-10	International Statistical Classification of Disease Related Health Problems
ICF	International Classification of Function and Disability
ICIDH	International Classification of Disability and Handicap
MMSE	Mini-Mental State Examination
M-W	Mann-Whitney

NCMRR	The National Centre for Medical Rehabilitation Research
ODFS	Odstock Dropped Foot Stimulator
PCI	Physiological Cost Index
PD	Parkinson's Disease
PDD	Parkinson's Disease Dementia
PDQ-39	Parkinson's Disease Questionnaire
QoL	Quality of Life
ROM	Range of Movement
ROMS	Rehabilitation Outcomes Measure System
SA	South Africa
SD	Standard Deviation
SNpc	Substantia Nigra Pars Compacta
SNpr	Substantia Nigra Reticulata
STN	Subthalamic Nucleus
TA	Tibialis Anterior
TES	Therapeutic Electrical Stimulation
UCT	University of Cape Town
UCTPAH	University of Cape Town Private Academic Hospital
UP	University of Pretoria
UPDRS	Unified Parkinson's Disease Rating Scale
WHO	World Health organization

CHAPTER 1

INTRODUCTION AND PROBLEM IDENTIFICATION

1.1 Introduction

Impaired gait and postural instability in patients living with Parkinson's disease (PD) are regarded as the two main aspects of the disease that cause disability in patients within their home and work environment. Postural instability is thought to be the main cause of falling in these patients and this occurs in two thirds of all patients diagnosed with PD. Melnick (in Umphred, 2007) states that "*People with Parkinson's disease have a nine fold risk of falls compared with age-matched control subjects.*" Such a high prevalence of falling amongst patients with PD has a detrimental effect on their productivity and quality of life.

PD is a progressive neurological movement disorder due to degeneration mainly in the basal ganglia (Whittle, 2007). It is the second most common neurological disease after stroke and the fourth most common neuro-degenerative disease in the elderly.

The chief clinical features of PD are: rigidity, bradykinesia and akinesia, and a resting tremor. Postural instability is thought to: *result from inflexibility in response repertoire, an inability to inhibit unwanted programmes as well as the interaction of akinesia, bradykinesia and rigidity with some disturbance in central sensory processing (Melnick in Umphred 2007)*

The secondary clinical characteristics that are present in patients with PD in varying degrees include: A change in the tone of voice, speech difficulties, masked facial expression, cognitive changes, incontinence of bowel and bladder, psychiatric disorders such as dementia, autonomic nervous system impairments, muscle weakness, dyskinesia and bradyphrenia. Fatigue and muscular aches experienced by the patient are probably due to the increased rigidity and hypokinesia. The patient's posture becomes stooped and, due to the decrease in the amplitude of their physical movement, walking becomes progressively more difficult. The patient begins to take short shuffling steps and the normal arm swing associated with gait ceases to happen. Steps may quicken and the patient may even break into a run (propulsion) or festinating gait to prevent falling forwards (Marsden, 1994; Oliverira, Gurd and Nixon, 1997; Melnick in Umphred, 2007). (Retropulsion is festinating backwards.)

Muscle strength may appear normal on testing but functional muscle power may be diminished and the ability to perform rapid, successive movements is impaired. It is a combination of these physical disabilities and the decrease in cognitive abilities (dementia is present in fifty percent (50%) of these patients) that cause patients with PD to begin to withdraw from community life. They avoid or are not able to mobilise with ease in public. It is especially the effect of the freezing/akinesia and bradykinesia/hypokinesia on the patient's gait that results in a decrease in his/her ambulation in the community and, as such, their quality of life. This ultimately leads to further immobilisation and a worsening of the level of functional activity of the patients.

Due to the fact that PD is a progressive neurological movement disorder, patients are treated from different perspectives i.e. medication, surgery and physiotherapy. The main aims of physiotherapy are to optimise and maintain the patient's functional ability, which will specifically include his/her functional gait, and to prevent secondary complications, due to a progressive decrease in the functional ability. The treatment of patients with PD is discussed extensively in Chapter 2.

1.2 Limitation (in literature) in the medical treatment of Parkinson's disease

From the early stages of the onset of PD treatment of the presenting signs and symptoms of PD includes pharmacological intervention with leva-dopa drugs ([www.dartmouth.edu/~dons/part 2/chapter 18.html](http://www.dartmouth.edu/~dons/part%20chapter%2018.html) 18/11/2006). The effect of these drugs does, however, tend to diminish with time and the doses have to be progressively increased, thereby also increasing the side effect of the drugs. The side effect of these drugs may include a decrease in the patient's cognitive ability, drowsiness and fatigue. Freezing/akinesia and/or bradykinesia/hypokinesia have also been found to be among the long term side effects of leva-dopa drugs (Nieuwboer, Feys and De Weerd, 1997).

Surgical intervention in patients with PD is indicated to alleviate signs and symptoms such as the resting tremor, and bradykinesia/hypokinesia; although the signs and symptoms have been noted to return within approximately five (5) years after surgery (Krause, Fogal, Mayer, Kloss and Tronnier, 2004).

None of the surgical interventions (or medications) has a positive effect on freezing/akinesia and/or bradykinesia/hypokinesia during gait.

1.3 Physiotherapy for patients with PD

1.3.1 A model for intervention for patients with PD

The all-encompassing goal of physiotherapy in the treatment of patients with PD is to maximize function and minimize disability. Various disability models (Shumway-Cook and Woollacott, 2001) or disablement/enablement models (Melnick in Umphred 2007) have been described to enable therapists to understand the interaction between disease (pathology) and disability, as well as the impact of disease on the different levels of human functioning. These models include, amongst others, the International Classification of Disability and Handicap (ICIDH), the Nagi model of disablement, the National Centre for Medical Rehabilitation Research (NCMRR) and the enablement model of the WHO (2001) namely the International Classification of Function and Disability (ICF) (Melnick in Umphred, 2007). The ICF (WHO, 2001) is widely used in the rehabilitation of patients as a simple yet holistic model of enablement and is the model on which the assessment and intervention by means of Functional Electrical Stimulation (FES) in this study is based. The International Statistical Classification of Disease Related Health Problems (ICD-10) and the ICF are encouraged by WHO to be used together to ensure a meaningful and complete picture of the health of a population. The ICF relates data about functioning that is important for the efficacy, effectiveness and cost effectiveness of health services. The clinical intervention of the health care professional is determined by the functional ability of the patient. The ICF makes it possible to identify and monitor the effectiveness of clinical interventions. The

ICF is easily communicated between health care professions (Ustun, Chatter, Bickenback, Kostanjsek and Schneider, 2003).

Assessment of the patient on impairment level of the ICF entails determining the body structures and functions that are affected (impaired), and the extent of the impairment (muscle tone, shortened muscles, sensory impairments, stiff joints, etcetera) that causes or contributes to the patient's dysfunction (Ashburn, Stack and Jupp, 2001). Examples of such outcomes measures on impairment level are the range of movement of the joints, the Modified Ashworth Scale to assess muscle tone, muscle strength using the Oxford grading scale and discrimination of sensation according to the ASIA scale. It would also include an assessment of the functioning of the cardiovascular and neuro-muscular systems.

Assessment of the patient on the level of functional activity entails assessment of the functional activities that the patient is able to perform such as grooming, dressing, eating, walking etcetera. Outcomes measures that are used to assess the patient's level of functional activity include assessment of gait by making use of the parameters of gait such as the time to walk a specific distance such as ten (10) or twenty (20) meters, the number of steps to walk the distance, the average step/stride length and the effort to walk the distance. It can also include assessment of the use of walking aids and the tendency to fall or number of falls that the patient experiences. The assessment of the functional activity of turning around while standing can also be assessed by quantifying the time, and number of steps that the patient takes to turn around 360 degrees.

Assessment of the level of participation of the patient with PD within their home, recreational and work environments gives an indication of the extent that the patients' quality of life is affected by this disease.

Such an assessment includes the implementing of the Unified Parkinson's Disease Rating Scale (UPDRS), freezing of gait (FOG) scale, Parkinson's disease Questionnaire-39 (PDQ39). The assessment outcomes indicate how the patient with PD participates in their home, recreational, and work environments and the effect that PD has on these aspects of their life.

Although assessment procedures for patients with PD are discussed in the literature, the effect of therapeutic intervention on impairment, functional and participation levels of the ICF is not discussed in depth. Such a discussion on the effect of a particular type of intervention on the different levels of functioning would be important to improve the understanding of the effect /consequences of intervention (physiotherapy) on the disabling /enabling process of patients with PD.

Assessment and intervention based on the ICF enables therapists to formulate holistic goals for the physiotherapy sessions for each patient.

Implementing outcomes measures on all three levels of the ICF at regular intervals enables physiotherapists to optimize evidence-based physiotherapy practice.

1.3.2 Forms of Physiotherapy intervention for patients with PD

Morris (2005) describes a few types of interventions that may be effective in the treatment of a movement disorder such as PD. These aims and interventions would include the:

- The prevention of secondary complications due to the non use of the affected body parts; i.e. maintenance of the range of movement of the ankles, knees, and hips.
- Assist in the reduction of the swelling of the lower limbs due to the limited active movement by encouraging circulation exercises.
- Optimising the patient's functional ability by maintaining or optimising biomechanical structures (joints, muscle length, muscle strength etcetera) and the patients physiological functioning of systems such as the cardio-vascular-respiratory systems.
- External visual and auditory cues probably turn the locus of control of movement away from the basal ganglia to the in-tact frontal regions of the brain (Morris, 2005; O'Shea, Morris and Iansak, 2002; Perry, Morris, Unsworth, Dodd, Taylor and Skeat, 2004). The use of the cues enables the patient to maintain gait performance while he performs complex tasks. Research by Suteerawattananon, Morris, Etnyre, Jankovic, and Protas, (2004), indicated that auditory cues improved the cadence, whereas visual cues improved stride length.
- Gait timing can be improved with rhythmic auditory facilitation in patients with PD (Del Olmo and Cudeiro, 2005).

- Strategies to enhance cognitive attention include concentrating on taking long steps (Morris, Iansak, Matyas and Summers, 1994), maintaining postural stability while turning (Morris, 2001), and breaking long and complex activities/tasks up into components of functional movements. By using such cognitive strategies the frontal cortical regions of the brain are utilized to conduct the desired movement to compensate for the dysfunctional basal ganglia (O'Shea, et al, 2002).

Fatigue could influence the cognitive attention on the functional activity and increase the gait interference during complex tasks and this could have a negative effect on the safety and functional gait of the patient (Rochester, Hetherington, Jones, Nieuwboer, Willems, Kwakkel and Van Wegen, 2005).

- Multiple task performance is a further possible form of training patients with PD to complete more than one task at a time (Brauer and Morris, 2004)
- Multi-Component Programmes entail a multi disciplinary team approach to treatment of patients with PD resulting in positive effects on the movement difficulties experienced by patients with PD (Morris, 2005).

Morris evaluated many randomised control trials on the intervention and external cueing effects on the mobility of patients with PD. From the evaluation of the trials it can be concluded that there is not sufficient available research in the field to determine the effectiveness of cognitive or exercise input for patients that suffer from PD (Morris, 2005).

As stated before, the use of external cues tend to wear off and patients with PD are continuously looking for new methods to overcome their difficulties with gait (Morris, 2005).

Bilney, Morris, Georgiou, Churchard and Chiu (2005), and Polgar, Morris, Reilly, Bilney and Sanberg (2003) in two different studies found evidence that physiotherapy, in the form of offering an external stimulus in the form of audio, sensory or tactile cueing, stretching of shortened muscles and maintenance of muscle strength, encouraged patients with PD to ambulant for longer periods. However their results have shown that there is no evidence to favor a particular type of physiotherapy intervention in PD.

1.3.3 Practical experience

I was introduced to Functional Electrical Stimulation (FES) in 2004 by a physician who had read an article about FES on the website www.salisburyfes.com. FES is defined as the implementation of an electrical current to cause an active muscle contraction in order to assist a patient in performing a functional activity such as walking. FES aims to optimize a patient's functional ability while performing the functional activity as part of performing activities of daily living.

Therapeutic electrical stimulation (TES) is based on the same principle as FES, namely using an electrical current to improve the muscle strength of either one, or a group of muscles with the aim to optimize a patient's functional ability. However, TES

is only used in a therapeutic environment or therapy session, to achieve the aim of muscle strengthening of a muscle or a muscle group.

On this website it was stated that FES could improve functional gait pattern in patients with any type of upper motor neuron disease. I contacted a physiotherapist from South Africa (SA) who had completed training in Salisbury using both FES and TES. The apparatus used to administer the electrical stimulation is the Odstock Dropped Foot Stimulator (ODFS), for FES, and the Microstim, for TES. I subsequently attended a course on "Introduction to FES" in Cape Town during August 2004 (Buhrs, 2004). During that course it was hypothesized that FES could benefit patients with PD who suffer from akinesia/freezing. However, to date no research has been conducted to determine the effect of FES on patients with PD. Only a small pilot study was conducted by Mann (2006) in the United Kingdom. This pilot study is still to be published. During this pilot study FES was applied on the lower limb of patients suffering from PD, to assist patients to overcome the lack of initiation of gait (freezing or akinesia) and, as such, improve their functional gait. From this small study it was assumed that FES could improve the gait pattern of patients with PD, but the questions of 'Why?' and 'How?' have not yet been answered.

The fact that freezing/akinesia and/or bradykinesia/hypokinesia are/is one of the most devastating aspects of PD and that medication and surgery have little/no effect on it lead to the idea that a clinical trial need be conducted in which the effect of FES on patients with PD's gait could be determined.

During the administering of an electrical stimulus with the Odstock Dropped Foot Stimulator (ODFS) patients first become aware of a sensory sensation. As the current's intensity is increased a motor stimulus in the form of a muscle contraction of the muscle over which the active electrode is placed is elicited. This results from the fact that FES can be administered in two ways, namely a sensory stimulus only or a sensory and motor stimulus combined.

In this clinical trial the distinction between the two forms of FES stimulation is indicated as a functional electrical sensory stimulation (FESS) and functional electrical sensory stimulation and motor stimulation (FES&MS).

In an attempt to solve the problem with akinesia/freezing in patients with PD, I decided to implement the Odstock Dropped Foot Stimulator (ODFS) in a few patients who were diagnosed with PD and who suffered from akinesia/freezing and/or bradykinesia/hypokinesia. All members of the rehabilitation team, which also included the patients' neurosurgeon and neurologist, found the initial results of this procedure to be very positive.

1.4 Problem Statement

Bradykinesia is defined as extremely slow movement (Evarts, Tervainen, and Calne, 1981). The term bradykinesia is used synonymously with akinesia and hypokinesia in the literature. Bradykinesia is the term used to describe the slowness of movement, whereas akinesia refers to the lack of spontaneous or associated movement or the prolonged time to initiate the movement (Evarts, et al, 1981). Freezing is also used synonymously with akinesia and occurs once the patient is stationary and can not

initiate movement, or if the bradykinesia causes a decrease in the mobility causing the patient to move ever slower, and eventually become stationary.

Physiotherapy for patients with PD who suffer from freezing/akinesia or bradykinesia/hypokinesia during gait is presently treated by:

1. A detailed assessment on participation, functional assessment and impairment level.
2. Prevention of secondary complications.
3. Increasing the mobility of the patient with PD.
4. Increasing the endurance of the patient with PD.
5. Improving the musculo-skeletal and neuro-muscular functioning.
6. Introducing compensatory mechanisms such as the use of external visual and auditory cues to assist the patient with mobility.
7. Training of multiple task performance strategies.
8. Offering advice and education to the patient and to the care-givers on the management of difficult situation that they may encounter.
9. Assisting patients in acquiring assistive devices (Morris, 2005).

The limitations of these treatment approaches include:

1. ICF implementation as mentioned in paragraph 1.3.1.
2. The effect of external and internal cueing to overcome freezing/akinesia tends to wear off and new cueing strategies or techniques need to be investigated.

3. There is no comparison between recognized treatment methods for gait disturbances and that of FES (FESS and FES&MS) on gait in patients with PD.
4. The effectiveness of levo-dopa drugs tends to wear off over time and the corresponding increase in the dosages increase the side effects of the drugs. This also leads to drowsiness and a decrease in the cognitive ability of the patient with PD (Morris, 2005). An alternative way to overcome freezing/akinesia and/or bradykinesia/hypokinesia without the pharmacological effect of drugs is therefore indicated.
5. A literature search of the use of FES in PD revealed no present publications in English. The shortcomings of the unpublished pilot study conducted by Mann (2006) include:
 - Only 20 patients were involved in the research pilot study that was conducted (Mann, 2006). No statistical significance effect on the participants gait was published.
 - The patients who participated in the pilot study were from the United Kingdom only.

1.5 Motivation for the study

FESS or FES&MS administered to the common peroneal nerve of the most affected lower limb can be administered to serve as an external cue that facilitates the initiation of taking a step in patients with akinesia/freezing and/or bradykinesia/hypokinesia due to PD. If this would be consistent for all patients with PD who suffer from

akinesia/freezing and/or bradykinesia/hypokinesia episodes, another method of intervention in the form of an external sensory or external sensory-and-motor cue may have been identified to use in isolation or in combination with the treatment techniques already described in the literature to optimize the gait of patients who suffer from PD and, as such, their functioning in their home, recreational and work environments, along with their quality of life.

If it can be indicated that the ODFS has a consistent effect on the akinesia / freezing and/or bradykinesia/hypokinesia episodes this type of treatment modality may improve the quality of life and health belief of patients living with PD. A few of the expected benefits include:

- Assist these patients to live a better and more productive life for a longer period of time. The patient will be able to continue with social and vocational activities for longer, before the freezing effects become so debilitating that the patient is forced to retire from the workforce or to withdraw from the community.
- Improve their social interaction, as people with freezing episodes tend to withdraw from society just because of this debilitating aspect of the movement disorder (Bloem, B. R., Hausdorff, J. M., Visser, J. E., and Giladi, N (2004).
- Offer a treatment modality that does not require constant purposeful attention of the patient to voluntarily adapt/initiate step taking during gait. FESS and/or FES&MS may therefore make it easier for patients to multi

task during walking; i.e. walking and picking up something from the floor, or manipulating objects during walking.

Based on the initial clinical implementation of the ODFS on patients with PD who experienced akinesia/freezing and/or bradykinesia/hypokinesia during gait, resulted in the arising of the research questions discussed in the following paragraph.

1.6 Research questions

1. Does FESS and FES&MS, administered to patients as separate interventions, decrease akinesia/freezing during gait in patients suffering from PD?
2. Is there a statistical and/or clinical difference between the effect of FESS and FES&MS on the quality of life of patients suffering from akinesia/freezing?
3. Is there a statistical and/or clinical difference between FESS and FES&MS on freezing/akinesia in patients suffering from PD?

1.7 Hypotheses

1.7.1 Hypothesis 1 (H1)

FESS and FES&MS decrease freezing/akinesia and bradykinesia/hypokinesia during gait in patients suffering from PD.

1.7.2 Null Hypothesis (H0)

FESS and FES&MS do not decrease freezing/akinesia and bradykinesia/hypokinesia during gait in patients suffering from PD.

1.7.3 Hypothesis 2 (H2)

FESS and FES&MS decrease freezing/akinesia and bradykinesia/hypokinesia during gait in patients suffering from PD to such an extent that it improves the quality of life of patients.

1.7.4 Null Hypothesis 2 (H02)

FESS and FES&MS do not decrease freezing/akinesia and bradykinesia/hypokinesia during gait in patients suffering from PD to such an extent that it improves the quality of life of the patients.

1.7.5 Hypothesis 3 (H3)

FESS decreases freezing/akinesia and bradykinesia/hypokinesia during gait in patients suffering from PD more than FES&MS.

1.7.6 Null Hypothesis 3 (H03)

FESS does not decrease freezing/akinesia and bradykinesia/hypokinesia during gait in patients suffering from PD more than FES&MS.

1.8 Aims and objectives

1.8.1 The aims of this study are to determine:

1. The effect of an external cue in the form of FESS and FES&MS (on the common peroneal nerve of the more affected leg) in patients with PD to

overcome episodes of akinesia /freezing and other parameters of gait i.e. time to walk a measured distance including a 180 degree turn, speed, step length, number of steps and the Physiological Cost Index (PCI).

2. To determine the effect of FESS and FES&MS on the quality of life of patients with Parkinson's disease who suffer from akinesia/freezing.
3. To determine whether there is a difference between FESS and FES&MS on akinesia/freezing in patients with PD.

1.8.2 Objectives of the study

The objectives of the study are to determine whether:

1. FESS and FES&MS has a significant effect on the following parameters of gait:
 - time to walk ten (10) meters, turn 180 degrees and walk ten (10) meters back to the starting point.
 - the speed with which patients walk ten (10) meters, turn 180 degrees and walk ten (10) meters back to the starting point.
 - the number of steps (half gait cycles) patients give to walk ten (10) meters, turn 180 degrees and walk ten (10) meters back to the starting point. This does include the number of steps to turn 180 degrees.
 - the average step length (distance of a half stride length).
2. The time and number of steps patients will take to turn 360 degrees to the right and to the left

3. The Physiological Cost Index (PCI) of the patients to walk ten (10) meters, turn 180 degrees and walk ten (10) meters back to the starting point.
4. FESS and/or FES&MS have an effect on the patients quality of life as measured on the Parkinson's disease Questionnaire 39 (PDQ-39)
5. The motor changes that patients living with PD experience as measured on the motor part of the PDQ-39 and Parkinson's disease Rating Scale (PDRS).
6. The patient's episodes of akinesia / freezing as measured on the Freezing of Gait Scale (FOG) decrease significantly due to the administering of FESS or FES&MS.
7. The number of falls patients experience in comparison with the time before they use the FESS and FES&MS decrease significantly due to the administering of FESS or FES&MS.
8. The patient's dependence on a walking aid decrease significantly due to the administering of FESS or FES&MS.
9. The degree of rigidity (increased tone) the patients may have before and after intervention with either FESS or FES&MS as graded on the Modified Ashworth Scale.
10. Sensory level impairment is present in the patients with PD that may have an effect on the patient's movement.

1.9 Research approach

1.9.1 Research setting

The primary study will be conducted at the University of Cape Town Private Academic Hospitals' (UCTPAH) rehabilitation unit. This unit was established in October 2001 and functions at present in full capacity as a rehabilitation unit. It is a private hospital setting, but is also an academic facility where research can and is being conducted.

There is a close working relationship between Groote Schuur Hospital (a tertiary hospital), University of Cape Town (UCT) and the UCTPAH.

In the UCTPAH, rehabilitation is taking place in a trans- disciplinary approach. The rehabilitation team consists of medical doctors, nursing staff, physiotherapists, occupational therapists, speech therapists and social workers.

The 45-bed unit caters for all rehabilitation candidates, which includes patients with neurological diseases, spinal cord injuries, head injuries and other complicated neuromusculo-skeletal conditions. Patients are also treated on an out-patient basis for which-ever condition they may have that requires rehabilitation.

In-patients receive therapy on a daily basis and out-patients are treated according to their need. Clinical notes are recorded after each contact or consultation with the patient. These are recorded on the (Rehabilitation Outcomes Measure System) ROMS database.

Patients are referred to the unit by medical practitioners from around South Africa (SA) as well as from outside the borders of SA. The patient has to be referred from doctors or therapists that were involved in the initial treatment of the patient. The rehabilitation team then assesses the patient and they determine whether the patient is a suitable

candidate for the rehabilitation facility or not. After this decision the patient is then either admitted to the UCTPAH or put onto an out-patient program.

1.9.2 Apparatus

The apparatus that will be used to administer the FESS and FES&MS is the ODFS. The ODFS is a neuro-stimulator that is powered by a 9 Volt battery that produces pulses of 300 microsecond's duration at a frequency of 40Hz. Adhesive, external electrodes will be utilized to deliver the current to the skin surface over the common peroneal nerve, just behind the head of the fibula and the other electrode over the muscle belly of the tibialis anterior muscle (TA). A foot switch that is placed under the heel of the more affected side on an inner sole that is inside the patient's shoe triggers the stimulation of dorsi-flexion during the swing phase of gait. Stimulation of the common peroneal nerve will give dorsi-flexion and inversion (tibialis anterior) and eversion (peronei). During gait one wants dorsi-flexion with the foot in the mid position and not in inversion, hence the additional stimulation of the peronei. The amount of dorsi-flexion in the mid position will depend on the intensity of the current that is set on the ODFS. Enough dorsi-flexion in the mid position needs to be obtained in order to ensure toe clearance during the swing phase of gait. Either a sensory or a sensory and motor stimulus will be administered to the common peroneal nerve as the heel of the affected side is lifted. With heel strike pressure on the foot switch under the heel occurs and the stimulation of the common peroneal nerve, and therefore the stimulation of the tibialis anterior and the peronei muscles will cease and the patient can lower their forefoot.

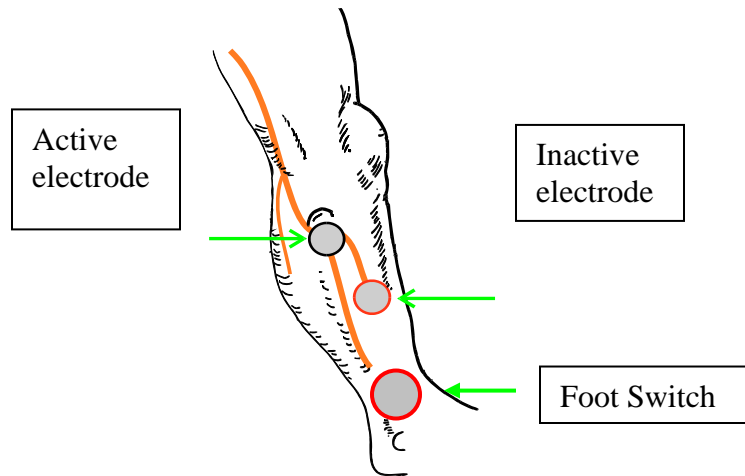


Figure 1.1: Electrode placement

1.9.3 Study Design

In this clinical trial each patient's response to the FESS of FES&MS will be compared to his/her own baseline measurement that will be calculated and determined before FESS or FES&MS will be administered. Thereafter the effect of FESS and FES&MS on patients suffering from akinesia/freezing and bradykinesia/hypokinesia will be determined by comparing the responses of the two groups of patients against pre-determined outcomes measures. The patient's response to FESS or FES&MS will be compared to their baseline measurements twelve (12) weeks after the ODFS has been administered (week fourteen (14) of the trial). ODFS will then be withdrawn and ten (10) weeks later (week twenty four (24) of the trial) the patient's response will again be tested. The differences between the two groups will then be determined.

A randomised clinical trial will be conducted to determine the effect of FESS and FES&MS as external cues, as well as to compare the effect of the two types of stimuli on freezing/akinesia and bradykinesia/hypokinesia in patients suffering from PD. The two patient groups will be randomly selected. A comparison will be made between baseline measurements and measurements at week fourteen (14) and week twenty four (24).

1.9.4 Study Population

All patients with PD who experience problems with freezing/akinesia and/or bradykinesia /hypokinesia are eligible to participate in the trial

1.9.5 Selection of sample group

All patients with PD who experience freezing/akinesia and bradykinesia/ hypokinesia that meet the inclusion and exclusion criteria of the study will be considered as being part of the sample group. Based on the consultation with a statistician it was determined that twenty five (25) patients should be recruited for the two groups. This number makes provision for the possibility of five (5) patients within each group withdrawing from the study for some reason.

A sample size of 20 in each group will have 90% power to detect a difference in mean time of 1.500 (the difference between Group 1 mean of 3.0 and a Group 2 mean of 1.5) assuming that the common standard deviation is 1.250 using a two group t-test with a 0.050 two-sided significance level.

Neurologists, neurosurgeons and neuro-rehabilitation physiotherapists, who will be approached with the study protocol as well as inclusion and exclusion criteria for the study, will be requested to refer patients for participation in the study. Patients that voluntarily request to participate in the study will also be considered for the investigation if they meet the inclusion and exclusion criteria.

The Parkinson's Association of SA and PD support groups will be contacted in order to ensure that all patients with PD are aware of the study and that they can have the opportunity to volunteer to participate in this study. All volunteer patients will be evaluated individually in order to determine whether they meet the inclusion and exclusion criteria of the clinical trial.

1.9.6 Randomisation of the sample group

The names of the selected patients will be entered into a data base in an excel programme. The patients will be numbered and the computer will do a random selection of the patients. E.g. Number 1: Mr. X, number 2: Mr. Y etcetera. Two groups of 25 patients each will be randomly selected, into Group 1 and Group 2. The final study population will be determined by the data of the first 20 patients to complete the 24 weeks duration of the study in each group. Group 1 will receive only sensory input from the ODFS (FESS), while Group 2 will have a sensory and a motor stimulation (FES&MS) of increased dorsi-flexion added to the treatment.

1.9.7 Control Group

Each patient will act as his/her own control in this clinical trial. A baseline measurement for each patient will be obtained by evaluating the patient twice (at week zero (0) and at week two (2)) and calculating the average score between the two (2) measurements. No intervention will be administered to patients during these two (2) weeks. Starting from week two (2) of the trial each patient will be given the relevant intervention for twelve (12) weeks. The intervention will be withdrawn at week twelve (12) (week fourteen (14) of the trial) and patients will be re-assessed after ten (10) weeks (week twenty four (24) of the trial) in order to determine the difference in effect of FESS and FES&MS on freezing/akinesia and bradykinesia/ hypokinesia. It will also be determined whether there are differences in the gait parameters and indicators of the patient's quality of life, at week fourteen (14) and whether these are sustained once the intervention has been withdrawn.

The results of the tests for the two groups will be compared and discussed in Chapter 4. The average of the numerical measurements will be calculated and used as the baseline measurement, with which the results of all further assessments on weeks fourteen (14) and week twenty four (24) will be compared. In this way each patient's results at week fourteen (14) and week twenty four (24) will be compared with his/her own baseline measurement, to determine any change in functional activity and activities on participation level.

1.9.8 Inclusion Criteria

Patients will be included into the study if they comply with the following inclusion criteria:

1. Idiopathic PD, responsive to medication. (Patients with non- idiopathic PD will be accepted if they fit all the other criteria).
2. Patients must be stable on current medication.
3. Patients must have difficulty with gait (reduced stride length, cadence, and decreased heel strike).
4. Patients must experience occurrence of tripping or falling during gait.
5. Patients must experience akinesia (freezing) during walking.
6. Patients must be able to walk 100 meters with or without a walking aid.
7. Patients must be able to comply with assessment procedures.
8. Patients must be able to give informed consent.
9. Patients must be eligible for treatment as an out-patient at the UCTPAH.

1.9.9 Exclusion Criteria

1. Patients with any other neurological condition in addition to the PD.
2. Patients with any cardio-vascular condition affecting endurance during gait.
3. Patients who have a cardiac pace maker.
4. Patients with any other musculo-skeletal condition that affects gait.
5. Patients with any cognitive or psychological aspects that would affect the ability to comply with the study protocol as determined from the Mini Mental State Examination (MMES).

6. Patients who are in another clinical trial i.e. to determine the effects of medication on the signs and symptoms of PD.

1.9.10 Outcomes Measures that will be used in this study

The ICF has been explained in paragraph 1.3. The outcome measures that will be used in this clinical trial are internationally recognized scales and are used to assess the patient on a participation, functional activity and impairment level.

Patients in both Group 1 and Group 2 will be assessed by using the following assessment instruments and procedures before the study commences.

1) Motor examination of the Unified Parkinson's Disease Rating Scale (UPDRS). This is an internationally recognized validated and reliable rating scale that is used as an outcome measurement tool that measures the various facets of impairment that is affected by the course of PD. It includes sections on mental behavior, and mood, activities of daily living (ADL) and motor sections. A total score of 199 points is possible, where 199 points represents total disability and 0 represents no disability (www.query.fcgi?cmd=retrieve&db=PubMed&lists_uids=12815652&dopt=Abstract) (8 February 2006) (See Addendum 1a). Only the motor part of this scale will be utilized (See Addendum 1b).

2) The Mini-Mental State Examination (MMSE) is an internationally recognized standardized validated rating scale utilized to determine the cognitive impairment of a patient. The test includes memory, attention and use of language. A score of 24 is the

maximum score indicating no cognitive difficulties and 0 indicating marked cognitive fall out (Folstein, Folstein and McHugh, 1975). (See Addendum 2a and 2b)

3) The Parkinson's Disease Questionnaire (PDQ 39) quality of life questionnaire. This is an internationally recognized and validated questionnaire to determine the quality of life of the patient living with PD (Jenkinson, Fitzpatrick, Peto, Greenhall and Hyman, 1997). (See Addendum 3a and 3b).

4) Freezing of gait scale (FOG). This is an internationally recognized and validated scale that will be used to determine the extent and severity of the freezing episodes of the patient (See Addendum 4).

5) Gait analysis to determine any asymmetry in gait and the use of a walking aid. This will be used to determine which side will be stimulated (the more severely affected side will be stimulated) (Mann, 2006). (See Addendum 5).

6) The Range of Movement (ROM) of the upper and the lower limbs will be determined and the end feel of any restricted movement will be clearly described. Shortening of any muscle and/or soft tissue will be noted. (See Addendum 6).

7) The Modified Ashworth Scale for Spasticity. This is an internationally validated scale to grade increased tone in the upper and the lower limbs on the scale from zero to five. Zero being no spasticity and five being maximal spasticity (increased tone) (Gregson, Leathley, Moore, Sharma, Smith and Watkins 1999). (See Addendum 7).

8) The Oxford grading scale is an internationally recognized scale for the grading of muscle power; zero being no muscle power and five full muscle power. All muscle strengths will be graded according the scale. (See Addendum 8).

9) The discrimination of sensation of the patient with regards to light touch, pinprick, and hot and cold sensation will be determined. This will be rated as normal (2), impaired (1) and absent (0) as used on the ASIA scale. (See Addendum 9).

10) A timed ten (10) meter walk, with a 180 degree turn and a 10 meter walk back to the start, that presents no obstacles, will be conducted. From this activity the time in seconds, the number of steps, the average step length in millimeters and the change in heart rate in beats per second will be measured. The heart rate of the patient will be taken at the beginning of the task and at the end to determine the Physiological Cost Index (PCI) of the patient to complete the walking activity.

This walking activity will be performed by two colleagues, who will do the test independently of each other to determine the inter-rater reliability and to eliminate any researcher bias. This measurement of time to walk the 10 meters, turn 180° and walk 10 meters again, as well as the change in heart rate will be conducted three times at each assessment to determine an average time and average change in heart rate over the distance. The average reading will be noted in the data sheet (Mann, 2006). (See Addendum 4).

11) A diary that will document the number of falls the patients experienced before intervention, during and after intervention will be kept. In the diary the date and time of each fall will be documented. It will also describe the situation in which the fall occurred. A fall will be considered a fall when the patient falls to the ground. The diary will be checked at every assessment and re-assessment of the patients (Mann, 2006). At week zero (0) there will be no entry of falls (See Addendum 10).

1.10 Intervention Procedure

1.10.1 Data Collection

I will be the primary investigator, and will collect all the data from the initial interview that will be conducted with the patient and from all the assessments carried out over the twenty four (24) week trial period.

The timeline of the study will be as follows:

Week zero (0): Initial assessment on full battery of outcomes measures

Week two (2): Second assessment on full battery of outcomes measures.

The average of the measurements based on the outcomes measures at week zero (0) and week two (2) will form the baseline measurement.

Week eight (8): Monitoring of electrodes and skin reaction to stimulation and patients diaries on falling incidents

Week fourteen (14): Assessment of patients with the FESS and FES&MS switched on. The full battery of outcomes measures will be conducted. The FESS and FES&MS will then be removed.

Week eighteen (18): Monitoring of the patients and their diaries on falling incidents

Week twenty four (24) Assessment of patients on the full battery of outcomes measures

It was decided that the initial two weeks in which an assessment will be performed and repeated, will serve as a control for all measurements on patients regardless whether they will be allocated to Group 1 or Group 2, to ensure that a Hawthorne effect is not created by the initial assessment and the expectation of participating in this study.

The patients will then be randomly divided into one of the two groups by a computer. After the re-assessment on the outcomes measures at week 2, Group 1 will receive FESS and Group 2 will receive FES&MS by means of the ODFS positioned as described in paragraph 1.9.2 The output of the FESS in Group 1 will be set to only give a sensory stimulus with no stimulation of the common peroneal nerve to cause a muscle contraction of the tibialis anterior and peronei brevis and tertius muscles, causing dorsi-flexion of the foot in the mid-position (not in inversion or eversion), during the swing phase of the gait cycle. The intensity will be kept at the level that gives a good sensory input, but with no motor effect.

The patients in Group 2 will receive FES&MS to the common peroneal nerve of the more affected lower limb. The output will be set to cause a sensory as well as a motor effect, to give a muscle contraction of the tibialis anterior and peronei brevis and tertius muscles, resulting in dorsi flexion and in the mid-position between inversion and eversion

From week two (2) until week fourteen (14), patients in Group 1 will use FESS and Group 2 will use FES&MS when they walk during the day. The ODFS will be switched off by pressing the pause button when they sit down or lie down to take a rest. At week eight (8) of the trial the patients of both groups will be monitored to make sure that the electrodes are still in good working order and are not irritating their skin.

Based on the principles of motor learning twelve (12) weeks was thought to be sufficient time for plasticity to have taken place in the brain. At week fourteen (14) the FESS and FES&MS will be stopped in both the groups after a full assessment on all the outcomes measures.

The reasoning behind this is to determine if a significant change in the patients' functional gait has taken place and whether the change could be maintained without using the stimulus. If a significant change has occurred after the first twelve (12) weeks and could be maintained for another ten (10) weeks, functional carry over due to motor learning and brain plasticity would have taken place. If the patients regress back to the baseline measurement when assessed on the outcomes measures at week twenty four (24), functional carry over would not have taken place.

The information will be entered into a data sheet on an excel programme and comparisons will be made. At each assessment it was ensured that the patient had the correct positioning of the electrodes. The patient and the primary care giver were instructed on the positioning of the electrodes as well as the exact setting on the ODFS.

In Table 1.1 a summary of the timeline of the clinical trial and when each outcome measure will be conducted is presented.

Table 1.1 Summary of the implementation of the outcomes measures

Tests/ Outcomes measures	Week 0	Week 2	Week 8	Week 14	Week 18 (without ODFS)	Week 24 (without ODFS)
UPDRS	X	X		X		X
PDG 39	X	X		X		X
Freezing of Gait	X	X		X		X
ROM	X	X		X		X
Ashworth scale	X	X		X		X
Oxford Muscle strength	X	X		X		X
Sensation: somato- sensory & proprioception	X	X		X		X
10 meter walking test with 180 degree turn and 10 meters walk back to start (*Parameters of gait)	X	X		X		X
360 Degree turn	X	X		X		X
Use of walking aid and posture assessment	X	X		X		X
Falls diary	X	X	X	X	X	X

***Parameters of gait include: Time, Speed, number of steps, step length to complete the task**

1.10.2 Statistical Analysis

I will capture all the data, which will then be analysed by a statistician, who I have already approached in this regard using the Wilcoxon and Anova programme on Statistica. The results are described in Chapter 4. A comparative analysis will be conducted on all the variables (dependent as well as independent) to determine the relationship between them. A p-value of 0.05 will be utilized. Variables that will be compared include the effect of FESS and FES&MS on time, speed, number of steps, step length, heart rate and PCI on the 10 meter walk with 180 degree turn and 10 meter walk back to the start and the time and number of steps to turn 360 degrees on the spot to the left and to the right. The results of all the questionnaires will also be compared.

1.10.3 Reliability and validity of results

In order to ensure reliability of the research project, the same therapist will do all the evaluations of the patients. That therapist will be the primary investigator. Videos of the assessments will be taken and assessed by an independent therapist. This will ensure the inter-rater reliability. All the questionnaires that are to be utilized are internationally recognized. This will ensure the validity of the questions asked and the results obtained.

The patients will be asked not to change the dosage of their medication before or during the trial. They should continue taking their medication as normal, to ensure the reliability of the end result of the trial. As medication plays a major role in the physical performance of a patient with PD, all assessments for each particular participant will be carried out with the same time-span after the participants have taken their medication.

The environment in which the evaluations are to be carried out will remain unchanged for all patients. There will be no background noise. I will measure and mark the distances, which will then be double-checked by two colleagues. I will use a stopwatch for the timing and will round off each timed period to the nearest split second. There will also not be large crowds of people, or people approaching from different directions, that may alarm or confuse the patient and add to an increase in freezing episodes in the area where the testing will be done.

1.10.4 Informed Consent and ethical considerations

The protocol has been submitted to both the Ethics Committee at the University of Pretoria (UP) as well as University of Cape Town, who will be overseeing the trial conducted at Groote Schuur Hospital and UCT private Hospital, both in the Western Cape. No formal letter was issued to provide an approval number from UCT, as that institution oversees much research that is conducted at UCTPAH. Ethical approval to conduct this study was granted by the Ethics Committee of the Faculty of Health Sciences at the University of Pretoria (S159/2005).

An information sheet and informed consent form (See Addendum 10) with the aims and general details of the study will be given to all the prospective patients prior to the commencement of this study. On the consent form it is clearly explained that the participation in the study is voluntary and that they are free to withdraw from this study at any time; that their withdrawal will, however, not affect their future treatment within the UCT private rehabilitation unit; and that all aspects of their medical information will be kept confidential. The patients are informed that the information obtained from this trial will only be used for research purposes and that their identity will under no circumstances be disclosed to any member other than the investigator and the clinical supervisor. The consent form will also include the gaining of patient consent to have a video taken of their gait and the dynamic motor assessment procedures. The face of the patient will be included in the video, but the identity of the subject in the video will not be released under any circumstances.

All the risks of the ODFS are explicitly listed. This included the chance of burns on the skin under the electrodes, may increase the chance of falling, and may increase tone if tone is present and that it may cease to work without any notice.

Patients will sign the consent form (or a thumb print will be accepted if the subject is unable to write) in the presence of two witnesses and me. The two witnesses will co-sign the document.

1.11 Course of the study

In Chapter 2 a literature study focussing on the medical diagnosis, pathophysiology, signs and symptoms and multidisciplinary intervention for patients with PD is

discussed in depth. The discussion of the intervention specifically focuses on the physiotherapy assessment and approach to intervention. The outcome measures for determining the progression or the regression of the patients are also discussed to enhance evidence-based physiotherapy.

In Chapter 3 a detailed account is given on how the research was performed. This includes the research setting, the recruitment of patients, the randomisation procedure, the intervention apparatus, the research process, and the assessment procedure. The data analysis is also discussed.

In Chapter 4 the results of the research methodology is presented visually by means of tables, graphs and figures as well as a discussion of each aspect that was measured.

In Chapter 5 the results of the trial will be discussed against the background of the relevant literature.

In Chapter 6 the conclusion of the effect of FESS and FES&MS on patients with PD's gait and FOG will be discussed. The limitations of the study and suggestions for further studies will also be discussed.

CHAPTER 2

LITERATURE REVIEW

2.1 Introduction

My aim in Chapter 2 is to describe and discuss the epidemiology, etiology, pathophysiology of PD, the clinical signs and symptoms and the functional problems that patients with PD experience. I will also discuss the various forms of the treatment of patients with PD by the different members of the rehabilitation team with the emphasis on physiotherapy.

It is important to discuss the pathophysiology of PD as well as the various forms of therapeutic intervention for patients with PD, to understand the effects that FESS and FES&MS may have on the signs and symptoms, functional activities, on the participation level and on the impairment level of these patients (WHO, 2001).

As FESS and/or FES&MS may have an effect on the impairment, functional activity and participation levels of patients with PD I also discuss outcomes measures that can identify and quantify any effect of the FESS and FES&MS on the impairment, functional activity and participation levels of these patients.

Extensive research on the medical treatment and physiotherapy for patients with PD has been done in the past. This includes pharmacological, surgical and external

cueing, to optimize patient's functional ability in spite of the signs and symptoms of PD.

Parkinson's disease is a chronic progressive neurodegenerative movement disorder. It is the second most common cause of neurological disability in the UK (Schoenberg, 1987; Whittle, 2007). There is evidence that the diagnosis of PD dates back to 5000 B.C. Doctor James Parkinson first described it in 1817 as the "Shaking Palsy". It is due to his work in the identifying some of the signs and symptoms that the disease got its name (www.parkinsons.org/ 23/09/2005).

The characteristic signs and symptoms of the disease include bradykinesia and akinesia, rigidity, tremor and postural instability (Calne, Snow and Lee, 1992). These signs and symptoms are often misleading to observers and patients with PD are stigmatized as being nervous, lazy, emotionally cold, angry, incompetent or intoxicated (Frazier, 2000; Karlsen, Tandberg, Arslan, and Larsen, 2000; Nijhof, 1995).

The signs and symptoms of PD arise due to the loss of dopamine producing neurons in the substantia nigra in the brainstem. Imbalances in neurotransmitters occur and the functions of the basal ganglia are compromised, resulting in the movement disorder.

The basal ganglia are responsible for the planning of movements and the internal cueing of motor skills (Morris, 2005).

2.2 Literature search strategy

Various Internet search engines were used to obtain relevant literature for this clinical trial. These included:

- Google
- Pubmed
- Electronic library of University of Pretoria
- Electronic search of the library of the University of Cape Town

Key words and phrases that were used to search for articles include:

Parkinson's disease, Signs and symptoms of Parkinson's disease, diagnosis of Parkinson's disease, epidemiology of Parkinson's disease, etiology of Parkinson's disease, pathophysiology of Parkinson's disease, medical treatment of Parkinson's disease, surgical treatment of Parkinson's disease, physical therapy treatment of Parkinson's disease, physiotherapy treatment of Parkinson's disease, incidence of Parkinson's disease, electrical stimulation and Parkinson's disease, functional electrical stimulation and Parkinson's disease, bradykinesia in Parkinson's disease, akinesia in Parkinson's disease, freezing in Parkinson's disease, rigidity in Parkinson's disease, tremor in Parkinson's disease, festinating gait in Parkinson's disease, levodopa, surgical intervention in Parkinson's disease, deep brain stimulation, Unified Parkinson's disease rating scale, freezing of gait scale, Parkinson's disease questionnaire-39, quality of life and Parkinson's disease, international classification of diseases, Odstock Dropped Foot Stimulator. Literature searches were conducted regularly from September 2005 until July 2008.

2.3 Epidemiology

Due to the misdiagnosis of PD, figures for the prevalence and incidence of this disease need to be accepted with caution (Hughes, Ben-Shlomo, Daniel, and Lees, 1992). Idiopathic PD accounts for over seventy per cent (70%) of patients with PD (Macphee, 2001) and up to ten per cent (10%) of idiopathic PD occurs in these patients by the age of forty (40).

PD usually occurs in people in their late fifth or early sixth decade of life. The median age of the onset of PD is 62.4 years.

In the UK, the incidence of PD is 18 per 100 000 per population per year. This means there are approximately 10 000 new cases per year in the UK. The prevalence is, however, 164 cases per 100 000 of population. This is due to the patients having a normal life expectancy (Maera and Hobson, 2000). In the United States, the incidence of PD is 10.9 cases per 100 000 persons and 49.7 cases per 100 000 persons over the age of 50. The incidence is growing as the average age of the general population increases. The prevalence is estimated at 300 cases per 100 000 in the United States and Canada (Bower, Maraganore, McDonnell, and Rocca, 1999). In Europe the prevalence of PD is 1.8 cases per 100 inhabitants over the age of 50. For the ages between 65 and 69 the prevalence is 2.4 cases per 100 and for the ages between 85 and 89 years, 2.6 cases per 100 inhabitants (de Rijk, Launer, and Berger, 2000).

The estimated prevalence of PD in South Africa (SA) is 163,413 persons for a population of about 50.28 million or 325 cases per 100 000 persons.

(www.health.iafrica.com/doconline/neurological/parkinsons.htm 2007).

This is an extrapolated statistic and is not based on a data source from the country.

Statistics used for the prevalence and incidence of PD in SA are typically based on the US, UK, Canadian and Australian prevalence and incidence statistics, that have been extrapolated using only the population of SA. This figure does not take into account the genetic, cultural, environmental, social, racial or other differences between the named countries. The extrapolation is therefore inaccurate and only gives a general indication of the incidence and the prevalence of PD in SA.

(www.wrongdiagnosis.com/p/parkinson's_disease/stats-country.htm).

2.4 Etiology

The etiology of PD is still not fully understood; it is however believed that pathological processes, ageing, environmental factors and genetic factors are contributing factors towards the development of PD. The younger the age of onset of PD the more likely it is that genetic factors play a role in the etiology. Findings on the genetic factors of PD include, amongst other results, that eight genes are associated with rare forms of PD (Burn, Mark, and Playford 1992). Three of these genes have been identified and many different loci are associated with the inherited forms of levo-dopa responsive PD. The analysis of the functions and structure of these three genes has confirmed the role of protein aggregation in the dopaminergic neurons of the substantia nigra as the cause

for the degeneration of the neurons in the substantia nigra in all the known forms of PD.

The biochemical and molecular cascades from these genetic studies in PD can provide targets for curative therapies (Mouradian, 2002). All these factors that play a role in the development of PD indicate that PD does not have a single cause.

2.5 Pathophysiology

There is no consensus between experts on the pathogenesis of PD, probably due to the different causes and contributing factors. However, it is believed that these causes / contributing factors lead to a dopamine deficiency in the basal ganglia of the brain.

Dopamine is one of the neurotransmitters in the nervous system and it is important for the optimal functioning of the basal ganglia. In PD the neural cells that produce dopamine degenerate. When the dopamine levels reach abnormally low levels, the patient starts to present with signs and symptoms of PD. These symptoms include bradykinesia, rigidity and a resting tremor. Differences in the signs and the symptoms that patients present with occur at different ages when PD is diagnosed. The reason for this is not known (<http://tcw2.ppsw.rug.nl/~vdbosch/pd.html>).

The basal ganglia form part of the extra-pyramidal system which controls movement and is interconnected with the pyramidal and corticospinal systems and, as such, influences the motor control centers within the brain (Cote and Crutcher, 1991).

The following discussion is based on the work published by Cote and Crutcher, 1991.

The basal ganglia consist of four (4) interconnected nuclei, namely:

- The globus pallidus internal (GPi) and external (GPe) segments.

Both these segments receive information from the caudate nucleus and putamen.

The GPi sends major inhibitory output from the basal ganglia to the thalamus. The inhibitory output is also sent to the midbrain and this presumably assists in postural control.

- The subthalamic nucleus
- The substantia nigra pars compacta (SNpc) and substantia nigra reticulata (SNpr).

The SNpc receives input from the caudate nucleus and putamen and transmits the information back along the same route. The SNpr also receives information from the caudate nucleus and putamen, and then transmits it outside the basal ganglia to control head and eye movements. The SNpc produces dopamine.

- The Striatum.

This consists of the caudate nucleus and the putamen.

The basal ganglia control the initiation and the maintenance of movement. They are involved in the decision making of what the body is going to do next and fulfill the role of maintenance of normal motor activity and the continuous control and co-ordination thereof. This decision-making takes place consciously as well as sub-consciously and automatically.

The basal ganglia fulfill the role of maintenance of normal motor activity and the continuous control and co-ordination through four circuit loops that start and end in the cortex of the brain:

- A motor loop, that is associated with learned movements and is involved in the correct sequencing of actions during the execution of learned motor programmes.
- A cognitive loop is associated with motor intentions and advanced planning for later movements.
- A limbic loop is associated with the emotional aspects of movement.
- An occulo-motor loop is associated with voluntary saccadic eye movements.

The striatum is connected to the globus pallidus and the substantia nigra through two neural pathways, namely the direct and indirect pathways. The projection of the striatum to the globus pallidus internal and substantia nigra pars reticulata is known as the direct pathway. The indirect pathway runs from the striatum to the globus pallidus external. Through these pathways the cortex of the brain can either increase the excitatory thalamo-cortical projections through the direct pathway or decrease the excitatory thalamo-cortical projections through the indirect pathway. In other words, the direct pathway facilitates movement and indirect pathway inhibits movement.

The substantia nigra contains neurons that produce the neurotransmitter dopamine. Dopamine appears to facilitate the activity of the direct neural pathways and inhibit the indirect neural pathways.

Circuits that use different neuro-transmitters maintain these direct and indirect neural pathways from the cortex through the basal ganglia. There is a balance between the

two pathways that is partly maintained by the release of dopamine from the substantia nigra to the striatum (Cote and Crutcher, 1991). When there is a decrease in dopamine, the motor and pre-motor cortex are less excitable and the patient is ultimately less mobile, due to slower responses and diminished spontaneous movement. (www.dartmouth.edu/~dons/part_2/chapter_18.html, 18 November 2006).

The basal ganglia are important in the process of initiation and automatic maintenance of movements once they have been initiated. This includes postural control, resting muscle tone, automatic associated movements such as arm swing during gait, pointing a finger, dressing action, walking and writing. It is also believed to control automatic emotional facial expression e.g. smiling, frowning, crying, etcetera (Marsden and Fahn, 1994).

In Figure 2.1 the difference in the neural pathways of normal functioning basal ganglia versus the pathways and functioning of the basal ganglia in PD are depicted.

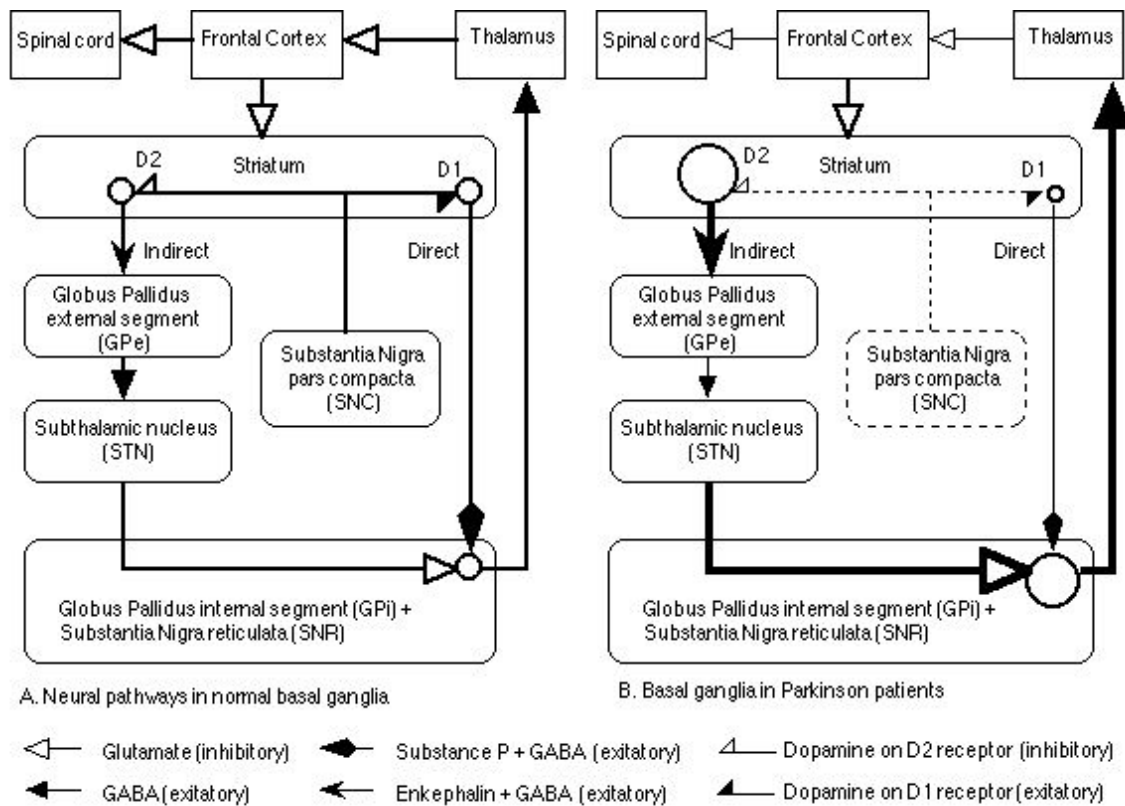


Figure 2.1 Neural Pathways in normal basal ganglia and basal ganglia in Parkinson's patients (Vermeulen, 1994)

The basal ganglia link the thalamus and the cerebral cortex. Although there is still debate about the actual functioning of the basal ganglia, the classic model described for the functioning of the basal ganglia proposes two principle neural pathways that involve the planning, initiation and co-ordination of movement and the direct and the indirect neural pathways as discussed previously in this paragraph (Flaterty and

Gabriel, 1994). The neural pathways have opposite effects on the output of the basal ganglia to the thalamus. In other words, in PD, the decreased dopamine levels lead to an inhibitory output from the globus pallidus to the thalamus, causing a suppression of movement that leads to the bradykinesia (Figure 2.1).

The degeneration of the nigro-striatal dopaminergic pathway is involved in the main change in postural control in patients with PD. Post mortem studies reveal that the substantia nigra loses pigment in PD (Cote and Crutcher, 1991). The degeneration of the dopaminergic nigro-striatal neural pathways lead to an imbalance in the output of the striatal neural pathways. The loss of dopamine leads to an increase in activity of the striatal neurons in the indirect pathway and a decrease in the activity of the striatal neurons in the direct pathway. The decreased inhibition through the direct pathways results in less activation of the motor cortex. The bradykinesia and akinesia are thought to be caused by this decreased inhibition of the motor cortex (Cote and Crutcher, 1991).

The increased activity in the indirect pathway has a similar effect through the dysinhibition of the subthalamic nucleus. Therefore in PD the facilitatory input from the basal ganglia to the cortical areas that should have a controlling inhibitory effect on the motor cortex is reduced.

Dopamine levels do decrease with age, but have to fall to one fifth of the normal level before signs and symptoms of PD develop

(www.familydoctor.co.uk/htdocs/PARKINSONS/PARKINSONS_specimen.html, 17

November 2006).

To summarise: It is believed that a decrease in the grey matter structures in the sub-cortical region of the cerebrum, the mid-brain and the basal ganglia are responsible for the pathology of PD. The main area of neuro-degeneration occurs in the pars compacta of the substantia nigra. This is where degeneration of the neuro-melanin containing cells occurs. Apoptosis of these cells follows the process in which the cell destroys itself enzymatically. The destruction of these cells results in neuro-chemical changes and dopamine depletion in the striatum. As the dopamine levels deplete, compensatory changes occur in the inter-connections of the basal ganglia with motor control centers in the brain. It is these changes that are involved in the development of the clinical features typical of patients with PD.

Lewy bodies are intracellular inclusions that occur in the neurons that undergo apoptosis in the substantia nigra. This can be seen as depigmentation of the substantia nigra in the mid-brain on CT scans of the brain. It is estimated that a patient with PD has already lost 60-70% of the substantia nigra in the mid-brain, resulting in dopamine levels falling to near one fifth of the normal levels by the time these patients first present themselves for a clinical evaluation, diagnosis and treatment.

(www.mdvu.org/library/disease/pd/par_path.html, 08 February 2006).

The activity of the post-synaptic cells in the basal ganglia depends on the interaction of the dopamine with the dopamine receptors in the post-synaptic cells. There are at least six different types of dopamine receptors in the post-synaptic cells, that consist of two families; namely, the D-1 like and D-2 like dopamine receptors (Strange, 1992).

The D2 receptors are particularly important in PD as they are involved with the planning, initiation and performance of the motor actions of the individual.

Although the above-mentioned model of the basal ganglia is successful in the explanation of the symptoms of the basal ganglia disease, there are still many unanswered questions. An example of this is that the model (see Figure 2.1) predicts that any uni- or bi-lateral lesion of the globus pallidus in subjects with normal values of dopamine should produce a syndrome that is characterised by excessive involuntary movements. In practice however, bi-lateral lesions of the globus pallidus cause a condition that resembles mild signs and symptoms of PD. It could then be predicted that lesions of the thalamus would also produce a PD state, but this is not the case. Lesions in the thalamus usually produce a dystonic syndrome (Alexander and Crutcher, 1990). From this it can be seen that the pathophysiology of PD is still poorly understood and requires more clarification and research.

Apart from the genetic theories, other theories for the cause of PD symptoms include that environmental factors may play a role in causing the signs and symptoms of PD. In PD, only selected populations of neurons die off. This may suggest the involvement of a toxin affecting the dopamine producing cells. Epidemiological and toxicological studies have failed to support any theory of an environmental toxin causing the degeneration and or death of the neurons. However, other possibilities of endogenous production of a substance are still being actively explored

(www.acnp.org/g4/GN401000142/CH139.html, 13 February 2006).

2.6 Clinical Diagnosis

According to Lennox and Lowe, 1997, the diagnosis of PD is based on the patient's medical history and a neurological examination that determines the presence of characteristic symptoms of the disease. Although there are no specific tests on which the diagnosis of PD is based, at least two of the three characteristic signs and symptoms, namely, rigidity, bradykinesia or a resting tremor, need to be present to diagnose this condition. A variety of laboratory tests can be done to exclude other factors that may lead to a Parkinsonian type syndrome. PD can be confirmed by a dopamine challenge test, where the patient is given a trial of levo-dopa drugs and the effects of the drugs are measured as to whether they have an effect on the patient's signs and symptoms or not. The other investigations include CT or MRI brain scans, PET scanning to determine areas of dopamine cell degeneration, and the measurement of ceruloplasmin levels through a lumbar puncture. The various scans will identify areas of hypertrophy within the brain or other associated lesions of PD.

PD presents three stages of development:

- 1) Clinical Stage: From onset of symptoms to appearance of motor fluctuations.
- 2) Early Stage: A decrease in the motor activities and/or movement due to the motor fluctuations affecting the patient's movement in varying degrees. This leads to moderate to severe disability and thereby limits the patient's participation in his/her self care activities, ambulation, recreational activities and other activities of daily living.

- 3) Late Complex Stage: Moderate to severe disability is present in all activities of daily living.

Impairment of memory and a decrease in cognitive functioning is rarely encountered in the early stages of PD. Depression is a common feature in approximately 30 per cent of patients. In the later stages of PD the patients may present symptoms of Alzheimer's disease or other forms of dementia

(www.therubins.com/illnessz/Parkinson.htm, 17 November 2006).

A twenty five per cent (25%) diagnostic error rate in the diagnosis of PD was found in 1992, when the accuracy of the clinical diagnosis of PD was compared to the postmortem findings in PD brain bank studies (Hughes, et al., 1992).

This research was repeated ten years later and revealed that the error rate had decreased to ten per cent (10%) (Hughes, Ben-Shlomo, Daniel and Lees, 2001).

The most common differential diagnoses that are reported by Quinn (1998), Lennox and Lowe (1997), and Litvan (1997) are essential tremor, drug induced PD, Parkinson's-plus syndromes that include multiple system atrophy, progressive supranuclear palsy and Shy-Drager syndrome, arteriosclerotic parkinsonism, viral infections such as post-encephalic parkinsonism, repeated head trauma, Wilson's disease, rare genetic disorders such as Hallervorden-Spatz syndrome, normal pressure hydrocephalus, diffuse Lewy body disease, cortical basal ganglionic degeneration and tumors (Quinn, 1998; Lennox and Lowe, 1997; Litvan, 1997).

2.7 Signs, Symptoms and other/secondary clinical features diagnosed in patients with PD

Apart from the motor symptoms of PD, namely: rigidity, bradykinesia and akinesia, and a resting tremor, other secondary clinical characteristics that patients present in varying degrees include:

- A change in the tone of voice – monotone voice,
- Speech difficulties - such as slurring and excessive salivation,
- Masked facial expression,
- Cognitive changes - such as slowing of thought processes and short and long term memory loss.
- Bowel and bladder incontinence,
- Psychiatric disorders - such as dementia,
- Autonomic nervous system impairments - such as urinary incontinence, constipation, hypostatic hypotension and increased salivation,
- Secondary muscle weakness due to decreased functionality of the limbs,
- Dyskinesias and bradyphrenia.

2.7.1 Rigidity and postural instability

The rigidity that is observed in patients with PD is ascribed to co-activation of antagonistic muscles, causing the appearance of stiffness in the muscles. The co-activation of the antagonistic muscles, results in a lack of efficient segmental movement in the body and an inefficient movement strategy that is needed for efficient balance. The pattern of muscle activity that they use is ineffective to recover balance

when the patient's centre of gravity is displaced. This explains why patients with PD present with poor balance (Shumway-Cook and Woollacott, 2001).

Rigidity can be detected by slow passive movement of the limb in both agonist and antagonist muscles. This is classified as either 'lead pipe' (slow and sustained) or 'cogwheel' rigidity (where a tremor is superimposed on the rigidity). Usually PD is characterised by cogwheel rigidity. In the latter case the patient's resistance to passive movement has a 'ratchet' quality. This is usually seen in the lower limbs of the patient with PD and can be felt when moving the limb passively through the full passive range of movement and when allowing the patient to conduct the movements actively through the range of movement.

The change in the mechanical properties of the muscle, due to disuse and stiffness, adds to the rigid feeling during passive movements (Burke, Hagbart, and Wallin, 1977). Rigidity is described by the patients as stiffness or as muscle pain that is evident in the upper and the lower limbs, as well as in the trunk.

Deep tendon reflexes appear enhanced in PD and these could potentially contribute to the bradykinesia. If the stretch reflex is elicited in an antagonist muscle during an active isotonic contraction of the agonist bradykinesia may result (Berardelli, Sabra, and Hallett, 1983; Rothwell, Obeso, Traub, and Marsden, 1983). A later study by Johnson, Cunnington, Bradshaw, Phillips, Iansek, and Rogers, (1998) also revealed that reflexes elicited in the antagonist muscle were enhanced when compared to that in healthy subjects. The degree of decrease in abnormality of enhanced reflexes was

related to the degree of clinical bradykinesia. However, the amount of activity in the antagonistic muscle measured by EMG during the movements was no greater than that of the healthy subjects. Thus there is no evidence in the movements tested that antagonist co-contraction could have caused rigidity in the muscles and thereby add to the bradykinesia. There is no evidence that rigidity plays a role in bradykinesia.

In paragraph 2.5 it is stated that the basal ganglia are involved with the initiation and maintaining of movement patterns. According to Shumway-Cook and Woollacott (2001), the delayed activation of postural responses, and the amplitude of those responses are responsible for the poor balance seen in patients with PD. This is confirmed by Burleigh-Jacobs, Horak, Nutt, and Obeso (1997) who found an inability to modify or adapt movement strategies to recover balance during changes in the support surface in sitting or standing in patients with PD.

Postural instability results in a stooped posture with increased flexor tone and a decrease in the stride length that results in a shuffling gait. Short shuffling steps are taken and there is no normal associated arm swing during gait. Steps may quicken and the patient may break into a run (propulsion) or festinating gait to prevent falling forwards (Marsden, 1994; Oliverira, Gurd, and Nixon, 1997; Melnick in Umphred, 2007). (Retropulsion is festinating backwards.)

This forms part of a compensatory mechanism as a shorter stride length reduces the postural instability, but slowness of movement results from the compensatory movement as well as from the increased inhibition in the indirect neural pathways, as

discussed in paragraph 2.5. The rapid shuffling gait is also believed to be a compensatory mechanism to prevent falling forwards (Shumway-Cook and Woollacott, 2001).

The amplitude of movements in patients with PD is less relative to the movement parameters in normal subjects. When a patient with PD conducts a task there is over-activity in the lateral pre-motor areas in the cortex (Shumway-Cook and Woollacott, 2001).

The patient with PD probably compensates with a flexion position of the trunk, hips and knees in the attempt to lower the center of gravity of the body and to contribute in improving his/her stability. This flexion position lowers the center of gravity and results in a stooped posture. When the patient steps forward, a shuffle gait occurs, probably due to the protective steps, which are a compensatory postural mechanism to aid in preventing falling forwards. The shuffling gait is probably the result of the decreased amplitude in repetitive movements as well as the bradykinesia; a combination of the stooped posture and bradykinesia result in a forward inclination. These inefficient movement strategies and poor recovery of balance probably result in a festinating gait (Oliverira, Gurd, and Nixon, 1997)

2.7.2 Akinesia and Bradykinesia

Akinesia is defined as 'a loss of the ability to move'. It is agreed upon by patients with PD that akinesia or the sudden unpredictable and total inability to initiate a step or turning is the most debilitating effect of PD. (Lamberti, Armenise, Castaldo, de Mari, Iliceto, Tronci, and Serlenga, 1997)

Akinesia is also known as 'freezing' and the terms freezing and akinesia are used interchangeably in the literature. Akinesia/freezing occurs when the patient is stationary and can not initiate movement, or if the bradykinesia causes a decrease in the mobility, so that the patient moves slower and slower and eventually becomes stationary. Examples of functional activities that are most affected by this slowing down of movement includes walking, talking, writing and eating.

Akinesia occurs in the advanced stages of PD, usually ten (10) or more years after the onset of this disease (Morris, 2000). Freezing of gait (FOG) occurs with the initiation of gait, turning on the spot, walking through narrow doorways and narrow passages (Lamberti et al 1997). The movement disorder in patients is progressive and occurs due to the fact that Parkinson's disease is primarily a degenerative disease in nature (Hallett, 1990; Morris, et al, 1994). About eighty per cent (80%) of patients with PD will never experience freezing at any stage of the disease. This fact may therefore suggest that freezing may be associated with pathophysiology in the brain that is not present in all patients with PD. Freezing is not unique to PD. It is also described in other hypokinetic, extrapyramidal movement disorders such as Huntington's disease (Giladi, Kao, and Fahn, 1997).

Three different types of freezing can be distinguished. The first is that a patient, for no reason, cannot initiate any form of gait from a standing position or when attempting to turn on the spot. It may also occur when she/he is already walking and experiences an inability to continue ambulation. Such a complete absence of movement is not the

most common presentation of freezing in PD (Giladi, McMahon, and Przedborski, 1992).

Freezing is further frequently associated with an effort to overcome an inability to step over an obstacle. It can also occur when a person is about to walk over a change in the texture of the floor surface or enter a doorway or is about to climb stairs. The akinetic or freezing episodes that a patient may experience under these environmental circumstances are often characterised by a stepping activity on one place that has the appearance of the legs trembling in one place. The movement disorder appears to be worse in situations where the patient experiences increased sensory (including visual) stimuli from or within a complex environment, such as narrow doorways, crowds, and crossing surfaces with different textures (Giladi, et al, 1992; Shkuratova, Morris, and Huxham, 2004).

The third type of akinesia is the performing of small shuffling steps during gait. In this case it is theorised that the patient is trying to adapt to the environment or change in environment that appears to cause the shuffling or freezing and is continuing the movement in an attempt to solve the sudden inability to move the lower limbs. Patients that freeze completely with no shuffling steps are less likely to fall as they freeze in one place and do not have small shuffling steps on the spot to try and compensate for the lack of movement (Schaafsma, Balash, Gurevich, Bartels, Hausdorff, and Giladi, 2003).

Readings of electromyographic (EMG) activity of the tibialis anterior and the gastrocnemius muscles have been recorded during circumstances that provoke freezing. The EMG analysis revealed significant abnormal timing in the tibialis anterior and gastrocnemius muscles, but usually with retained reciprocity between the tibialis anterior and gastrocnemius muscles. Just before freezing, tibialis anterior swing activity was prematurely evident in the pre-swing gait phase and was shortened significantly during the swing phase, causing a decrease in the extent of dorsi-flexion. Premature activation and termination of the contraction in the stance phase prior to a freezing episode was recorded in the gastrocnemius muscle. The gastrocnemius muscle also showed bursts of activity in the swing phase (while the leg is non-weight bearing) that is not evident in a normal gait pattern. The total EMG activity in both the tibialis anterior and gastrocnemius muscles was reduced in a pre-freezing gait episode (Nieuwboer, Dom, De Weerd, Desloovere, Janssens, and Stijn, 2004).

Electrophysiological studies have shown abnormalities in the size of the movements and the response of the movements in every phase and component of the postural reaction in patients with PD. These include:

- Abnormally sized autonomic postural responses, with enlarged destabilising responses and decreased stabilising responses;
- The inflexibility of autonomic postural responses, i.e. inability to modulate responses according to the postural demand;
- Delayed initiation or reduced scaling of the voluntary postural response;
- An inadequate anticipatory postural response (Bloem and Bhatia, 2003).

Bradykinesia is defined as 'slowness of movement'. The term bradykinesia is used synonymously with akinesia and hypokinesia in the literature. While Bradykinesia is the term used to describe the slowness of movement, akinesia refers to the lack of spontaneous or associated movement or the prolonged time to initiate the movement (Evarts,et al, 1981).

Akinesia and/or bradykinesia rank among the diagnostic signs of PD and should be present before the diagnosis of PD is made. Bradykinesia results from a disruption in the neurotransmitters used in the neural projections from the internal segment of the globus pallidus to the cortical regions of the supplementary motor area and the primary motor cortex (Alexander and Crutcher, 1990). Normal functioning of the basal ganglia is critical for the regulation of an activity before a movement is even executed. It also ensures that the movement is terminated at the correct time. The correct timing of each component of gait are important, to ensure that a flowing coordinated movement is conducted in the execution of the task at hand. If there is a disruption in the functioning of the basal ganglia, bradykinesia may result. If there is no activity in the primary motor cortex or in the supplementary motor area, no movement will occur which will result in freezing or the lack of the initiation of gait (Bronchie, lansek, and Horne, 1991; Cunnington, lansek, Bradshaw, and Phillips, 1995). Episodes of akinesia usually occur later on in the disease and are related to "off" periods of medication (Evarts,et al, 1981).

The presence of bradykinesia is evident in the stride length of a patient with PD, as their stride length becomes shorter the further they walk. The evidence of bradykinesia is also evident in the patient's handwriting, in that the size of the letters decreases as

the length of a sentence progresses. If the patient stops the task they are busy with to take a rest, to later re-continue, the amplitude of the movements are close to normal and start reducing once again as the task continues (Marsden, 1994; Oliveira, Gurd, and Nixon, 1997).

Secondary muscle weakness occurs in patients with PD due to the decreased activation of muscles in functional movement patterns and in the speed of the movement, resulting in a decrease in general mobility. The immobility of the patients is a main contributing factor causing the patients to become bedridden, which leads to further muscle weakness. (Berardelli, Accornero, Argenta, Meco, and Manfredi, 1996a). Muscle weakness, tremor and rigidity contribute to (but do not fully explain) the cause of the bradykinesia. In turn, muscle weakness can contribute to a decrease in the amplitude and velocity of movement, which contributes to the bradykinesia (Berardelli, Dick, Rothwel, Day and Marsden, 1996b).

Some patients with PD have difficulty in ceasing motor tasks. It is presumed that this is due to the sustained electrical discharge from the supplementary motor areas. This is a large predisposing factor to falls, tripping and slipping (Elble, 1998; Morris and Iansek, 1997).

It is thought that bradykinesia results from the failure of the output of the basal ganglia which reinforces the cortical mechanisms that prepare to perform the components of specific movements. Movement difficulty is also experienced with self-paced

movements, increased reaction times and abnormal pre-movement EEG activity. The use of compensatory mechanisms to conduct more than one task at a time, such as sensory, cognitive or auditory input, however, leads to decreased quality of movement as the patient becomes cognitively fatigued and can only focus on one task at hand at a time (Berardelli, Rothwell, Thompson, and Hallett, 2001).

There is clear evidence that the slowness in the programming of motor stimuli in the pre-motor cortex is an important factor in prolonging reaction times to particular stimuli.

Brain activity reflects both outgoing motor stimuli and the sensory input that results from the initiation and movement planning. Studies of motor activity in the brain by Ikeda, Luders, Burgess, and Shibasaki (1992) have focused on the period just before the onset of movement, when changes in sensory input are at a minimum. In general it was found that there is under-activity of the cortical motor areas in patients with PD. This under-activity may be related to the delay in the pre-planning of movement instructions that occurs in the pre-motor cortex. The active process of compensation that may be related to the improvement in performance observed when external cues are given to guide the movements in the patients with PD. Pre-movement EEG potential is a negativity that occurs over widespread areas of the brain before the start of a self paced voluntary movement. Results from studies done by Ikeda et al, (1992), show that a pre-movement EEG potential begins 1-2 seconds before the movement is observed and is bi-laterally symmetrical. This is followed by the second movement component that begins 650 micro seconds before the onset of the movement and is

assisted from the contra-lateral motor and pre-motor cortex (Ikeda et al, 1992). It has now been shown that levo-dopa could affect the amplitude of the EEG potential in both healthy individuals and PD subjects. This leads to smoother more co-ordinated movements. The difference in amplitude between healthy subjects and patients with PD depends on the level of dopaminergic function prior to the levo-dopa dose (Dick, Rothwell, Day, Cantello, Buruma, and Gioux, 1989). It has also been suggested that under-activity in the supplementary motor areas is responsible for the decrease in the early initiation of movement. Over- activity in the motor cortex compensates for the decreased timing during the onset of movement. It has been noted that pre-movement EEG is normal in patients with PD that perform an externally triggered task compared to the results in the self-paced movements (Jahanshahi, Jenkins, Brown, Marsden, Passingham, and Brooks, 1995). Under-activation of the supplementary motor areas in patients with PD makes them more reliant on external cues to initiate movement (Cunnington, et al, 1995).

Studies by Soliveri, Brown, Jahanshahi, and Marsden, (1992) and Morris, Iansek, and Matyas, (1996), have shown that the performance of single tasks in PD are normal, until the single movement has to be carried out and incorporated into a more complex task or a multiple task situation. The complex task is then carried out at a slower rate and with more errors within the task e.g. freezing or shuffle gait (Soliveri, et al, 1992; Morris, et al, 1996).

The basal ganglia play a role in controlling the learned skilled movements. These skilled movements become automatic movements that are performed with little

attention to how the task at hand should be performed. Patients with PD need to rely on frontal cortical regions to consciously control and guide the individual movements to bypass the defective basal ganglia (Morris and Iansek, 1996). Contrary to the above-mentioned concepts, a study by Bond and Morris (2000) has shown that the increased complexity of a secondary task, or a task that is being performed simultaneously with another task, did not produce a significant decrease in the gait speed, stride length or cadence (Bond and Morris, 2000). They found that it is the habitual movements, like arm swing, that is mostly affected in patients with PD. It is suggested by the authors that newly diagnosed patients should be taught to practice multiple tasking from the early stages of PD, to encourage better functional carry over. This specific study was limited in that it was only applied to a small sample group of patients with PD and who were only moderately affected by the PD.

Oliveira, Gurd, Nixon, Marshall, and Passingham (1998) found that bradykinesia increases when adding complexity to the movement, by either repeating the movement or combining two or more tasks. The result of their study showed that patients seem to alternate tasks rather than perform them simultaneously.

2.7.3 Falling

People with basal ganglia impairment are prone to tripping and falling, due to the decrease in stride length and non toe-clearance of the ground during the initial and early swing phase of gait (Morris, 2005). The reduction in stride length, that is characteristic of akinesia/bradykinesia, adds to the increased risk of falling because of

the risk of tripping over obstacles. It has been suggested that bilateral uncoordinated gait and asymmetry of gait due to the signs and symptoms of PD, such as bradykinesia, rigidity and poor posture, are associated with freezing episodes (Plotnik, Giladi, Balash, Peretz, and Hausdorff, 2005).

Patients with PD have a characteristic stooped posture resulting in the anterior displacement of the centre of gravity. This is also a typical posture of elderly people with a fear of falling (Maki et al, 1991 in Shumway-Cook and Woollacott, 2001). This change in postural alignment can either be the result of musculo-skeletal impairment, or be a compensatory mechanism for basal ganglia impairment such as found in PD. Because patients diagnosed with PD are often elderly, causes for the stooped posture may be due to the compensatory strategy for the postural instability as well as due to the musculo-skeletal impairment (Playfer, 2001; Bloem, Grimbergen, Cramer, Willemsen, and Zwinderman, 2001).

Treating the injuries as a result of falling is costly to society and to the health care funders, and can leave older patients more incapacitated after they have fallen (Bloem, et al, 2004). The combination of the risk of falling and the decrease in speed of ambulation contributes to the limited community ambulation of patients with PD, resulting in a decrease in their quality of life (Morris, 2000).

Falling and freezing are thought to be closely linked for various factors:

- 1) Both symptoms are common in the advanced stages of PD.

- 2) Freezing is likely to disturb balance and be the main cause of falling.
- 3) Recent studies have revealed that the pathophysiology of falls and of freezing are poorly understood.
- 4) Falls and freezing often respond poorly to treatment with dopaminergic medication.
- 5) The episodic and unpredictability of falls and of freezing underscores the possible connections between both falls and freezing in PD.
- 6) Falls and freezing are threats to both the well being of patient and in terms of the high cost of healthcare in the case of injuries due to falling.

According to Bloem et al (2004), it is the above mentioned points that indicate common underlying contributing factors to falling and freezing. The authors of most studies have found no relation between the disease duration and the incidence of falls (Bloem et al, 2004).

Stiffness in the trunk and in the limbs caused by disease related rigidity in antagonistic muscles, results in secondary changes in intrinsic muscle properties, and an active co-contraction in the trunk and limbs due to fear of the instability or of falling. Stiffness in the antagonistic muscles results in rigidity of the muscles and either the loss or impairment of the postural reactions due to changes that occur in the posture or that occur during normal movement. This may lead to postural fixation or an active co-contraction in the trunk and in the lower limbs. The fear of falling may contribute to this.

When losing their balance, patients with PD always fall sideways or backwards, while holding themselves rigid like a log. A fear of falling results in the restriction of activities of daily living. Normal subjects react to falling by flexion of various body parts (ankles, hips, knees and trunk as part of the postural reaction). This lack of natural postural reaction that contributes to the lateral falls may explain the high occurrence of hip fractures in patients with PD. In these patients, the tendency to fall backwards is counteracted by the stooped posture they assume (Bloem, van Dijk, and Beckley, 1999). It is assumed that if the gait is improved, the incidence of falls will decrease. Sensory processing abnormalities could explain the observation that patients with PD are unaware of their stooped posture. Protective stepping and associated arm movements during gait are abnormal. Corrective steps are slow and small and protective arm movements are not used to counteract fall direction or to break the fall. The arms in patients with PD are adducted and this could explain the low incidence of arm and wrist fractures, as they do not put their arms out as a protective reaction when they lose their balance (Visser, Allum, and Carpenter, 2002). Hip fractures occur in about twenty five (25%) percent of patients with PD per year, and tend to occur within ten (10) years after the diagnosis of PD has been made. Fractures occur more regularly when the patient with PD also suffers from osteoporosis, which may be caused by immobilization and associated endocrine disorders that are related not only to PD, but also to the aging process. From the time that patients with PD start to suffer from recurrent falls, the number of years of survival is reduced by about six years (Wenning, Ebersbach, and Verny, 1999). This can be

explained by the occurrence of lethal falls, a decrease in fitness level and/or an increased risk of cardiovascular diseases due to immobilisation following the signs and symptoms of PD. The decrease in mobilisation results in an increase of dependence on others and ultimately a withdrawal from society.

The incidence of falls eventually decreases again as the patients become progressively more immobile with the worsening of their condition (Bloem, van Vugt, and Beckley, 2001). Immobility is also associated with constipation, sleep disorders and pressure sores. Falls in patients with PD are associated with the advanced stages of PD, as already mentioned, patients with a fear of falling, the use of alcohol and concurrent use of Benzodiazepines (Bloem, Steijns, and Smits-Engelsman, 2003). It is estimated that at least sixty percent (60%) of patients with PD fall, compared to the estimated twenty nine percent (29%) of patients that report falling in the elderly population (Shkuratova, et al, 2004). This usually leads to the incapacitating fear of further falls. One hundred and eighteen (118) patients with PD were assessed to determine their risk of falling. It was concluded that freezing was closely linked to the eighty percent (80%) of the reported falls amongst patients with PD. The clinical impact of falls in PD patients is considerable and treatment strategies are developed to assist the patient in reducing or preventing falls (Grimbergen, Munneke, and Bloem, 2004).

An analysis of falls revealed that most falls in patients with PD occur indoors. This is probably due to patients minimizing walking out of doors, due to the mobility issues

already discussed. It has also been found that falls usually occur when the patient is in a good physical condition i.e. during the “on stage” of medication because they are immobile during the “off period”. Most falls occur under harmless circumstances such as turning around (Bloem, et al, 2003). Dual or multi-tasking while walking or standing also commonly contributes to the incidence of falling among patients with PD. This is due to patients not being able to prioritise the movement patterns automatically, so as to maintain balance under complex circumstances. Bloem, Grimbergen, and Cramer (2000) state that maintaining balance while ambulating is an example of a dual task.

When patients perform dual or multi-tasks, they try to perform all the tasks equally well, but usually they find that only one of the tasks can be completed properly at a time. The other ‘tasks’ usually have poor coordinated movements or the patient is not able to initiate more than one activity at a time. (Bloem, Valkenberg, Slabberkoorn, and van Dijk, 2001).

Figure 2.2 presents a summary of the clinical impact of falling in patients with PD. It has been used to indicate how certain characteristics of PD contribute to other impairments and cause a vicious circle of ailments that all tend to decrease the quality of life of the patient living with PD.

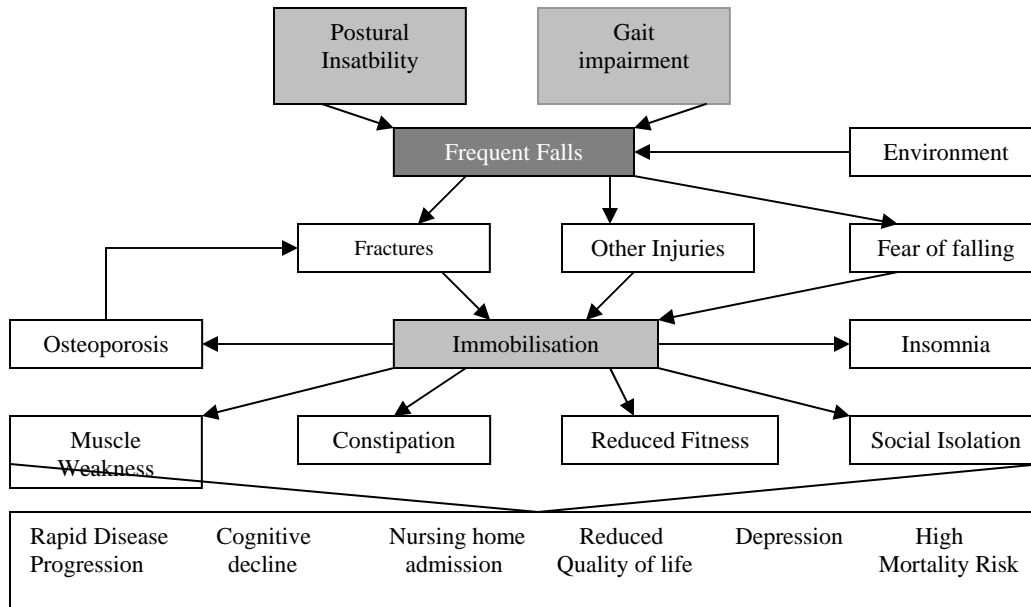


Figure 2.2. Clinical impact of falls and freezing of gait in PD (Bloem et al 2004).

In summary: akinesia and bradykinesia are related to dysfunctional basal ganglia, but there are secondary factors related to this impaired movement manifestation that need to be considered. Akinesia and bradykinesia can lead to an increase in the number of falls experienced by patients with PD. However there are five factors that can potentially contribute to bradykinesia and akinesia, namely, tremor, muscle weakness, rigidity, movement variability and slower thought processes (Berardelli, et al 2001). These factors and other symptoms of PD will be discussed in the following paragraphs.

2.7.4 Tremor

Tremor is defined as a rhythmic, involuntary, oscillating movement of a body part, occurring in isolation or as part of a clinical syndrome

(www.clevelandclinicmeded.com/diseasemanagement/neurology/tremor/tremor.htm 18 November 2006). Five types of tremor are described in the literature. It is necessary to consider each one individually, as patients with PD can present with more than one type of tremor. The common types of tremor include:

Resting tremor is present when the body part is at rest and gravity has no effect on it. The amplitude of a resting tremor decreases with voluntary activity. Resting tremors often begin in one of the hands. The characteristic resting tremor in PD is referred to as “pill rolling” as the tremor resembles a pill being rolled between the thumb and the forefinger. The tremor can contribute to bradykinesia and has been discussed as causing fatigue due to the increased muscle contractions involved in the resting tremor.

A postural tremor on the other hand is present during the maintenance of a position and increases with activity.

An action or kinetic tremor is a tremor that occurs during voluntary movement.

A task-specific tremor emerges during a specific activity.

An intention or terminal tremor is characterised by a marked increase in the amplitude of the movement during the terminal part of the voluntary movement. (Elble, 1996; www.clevelandclinicmeded.com/diseasemanagement/neurology/tremor/tremor.htm, 18 November 2006).

Four basic mechanisms have been identified that may cause a tremor. All these mechanisms may play a roll in PD.

In any type of tremor, mechanical oscillations of the limb can occur at any joint causing the appearance of a tremor in that limb.

Reflex oscillation is elicited by afferent muscle spindle pathways, and causes stronger oscillations than the mechanical oscillations. Abnormal functioning of the cerebellum may also produce a postural tremor (Deuschl, Krack, Lauk, and Timmer, 1996; Elble, 1996). However there is a difference between the postural tremor found in PD and the tremor caused by cerebellar damage. The tremor due to a cerebellar lesion is an intention tremor and is present when the patient uses the limbs during a purposeful activity.

“Central oscillators” are groups of cells in the central nervous system that are present in the thalamus and the basal ganglia that fire repetitively and uncontrollably with an oscillatory effect on the muscle spindles, resulting in a tremor of the limb or part of the limb.

It is thought that the tremor evident in PD originates in the latter way, and is referred to as the Parkinsonian tremor. The Parkinsonian tremor is a rhythmical tremor with a frequency of four (4) to eight (8) oscillations per second caused by alternating contractions of opposing muscle groups. It is more prominent in the distal extremities and usually presents in the upper extremities first. The tremor may begin in only one extremity, usually in the left or the right hand before it is noticed in the rest of the extremity. When a patient is entirely at rest or sleeping the tremor usually disappears. The tremor in PD is suppressed by the initiation of voluntary movement. As the disease progresses, the patients with PD may develop a co-existing intention tremor which supports the idea of cerebellar involvement in the pathogenesis of PD tremor.

(www.dartmouth.edu/~dons/part_2/chapter_18.html, 18 November 2006). This intention tremor is probably due to an altered firing rate of thalamic neurons and is evident in seventy percent (70%) of patients with PD. It has been noted that an action or intention tremor can be a contributing factor to muscle weakness in PD. The weakness is ascribed to the early fatiguing of the muscle due to the increased oscillations and contractions of the muscle groups. The tremor could also slow down the initiation of movement and as such, contribute to the impaired initiation of movement.

Action tremor can be observed during active purposeful movements. A postural tremor can be observed during weight bearing through a limb or during resistance to a movement of the head, or one of the limbs (Wierzbicka, Staude, Wolf, and Dengler, 1993).

2.7.5 Muscle Weakness

The muscle strength of normal subjects compared to that of patients with PD has been compared in many studies that concluded there is a decrease in muscle strength in patients with PD (Jordan, Sagar, and Cooper, 1992). In a study by Corcos, Chen, Quinn, McAuley, and Rothwell, (1996), the researchers compared the strength of the elbow flexors and extensors during the “on” and “off” periods of medication. In the “off” period there was a thirty percent (30%) decrease in muscle power in the elbow extensors and a ten percent (10%) decrease in elbow flexion in patients with PD. This decrease could not be attributed to a decrease in intrinsic muscle power and the researchers therefore concluded that it was related to an inability of the patients to

actively contract the muscle to the maximum. A later study by Brown, Corcos, and Rothwell (1997), showed a decrease in muscle strength and the physiological reason for this was found to be the persistence of the tremor during maximal muscle contraction. The tremor leads to early fatigue of the muscle and therefore a decrease in muscle strength. A tremor of ten (10) Hertz (Hz) prevents maximal fusion of the motor unit during contraction and therefore can contribute to muscle weakness in the “off” phase. However it cannot account for the decrease in muscle power in other antagonistic muscle groups, leading the researchers to presume that central factors within the brain are involved in muscle weakness. Muscle weakness and fatigue will inevitably contribute to the slowness and poor initiation of movement.

2.7.6 Movement Accuracy and Dyskinesia

Patients with PD portray movements that are less accurate than those of normal subjects. Especially if the patient is expected to move at normal speed there is a decrease in the accuracy of movements performed with speed (Sheridan and Flowers, 1990; Phillips, Martin, Bradshaw, and Iansek, 1994). It is suggested by Sheridan and Flowers (1990) that patients with PD use slower and smaller movements as a strategy to increase the accuracy of their movements and decrease the chances of falling (Sheridan and Flowers, 1990). Patients with PD also portray a decreased accuracy in simple ballistic tasks such as pointing towards something in space or catching a ball (Majasak, Kaminski, Gentile, and Flanagan, 1998). The decreased accuracy is obvious in movements with a complex sequence (Benecke, Rothwell, and Dick, 1987). This is probably due to the fact that the primary motor cortex, brain stem and spinal

cord are the main control centers for the simple ballistic movements, whereas the cerebellar circuits control the more complex sequences. It is the cerebral circuits that are defective in patients with PD (Iansek, Bradshaw, and Phillips, 1995). People with PD benefit functionally by breaking the sequences of complex movements down into simple movement sequences and avoid performing dual task or multi tasks (Morris, Bruce, and Smithson, 1997; Bond and Morris, 2000).

It has also been determined from studies on animals as well as on humans that interval timing processes associated with voluntary behaviors, such as hand grip and when taking a glass, are dependent on intact dopaminergic pathways. If the pathways are not completely intact, only one movement at a time will be carried out at a normal velocity and timing as in healthy subjects. The reaching towards the glass may be normal, but the grip and withdrawal may then be delayed or have decreased amplitude of movement. Other associated movements will be slow and rigid. The person with PD therefore has difficulty with multi-tasking or performing two or more movements simultaneously. Movements associated with lower reflexive behaviors (such as the startle eye blink) are spared (Jurkowski, 2005).

Dyskinesia is a term used for involuntary, irregular, twisting movements of the extremities, which may involve the head and neck in patients with PD. These movements occur when the dose of leva-dopa is at high levels in the brain in patients with advanced PD. Dyskinesia is present in fifty percent (50 %) of patients with PD

after using leva-dopa drugs for approximately 5 years

(www.neuro.jhmi.edu/hopkinspdmd/symptoms/dyskinesias.htm, 18 November 2006).

2.7.7 Communication Difficulties

A variety of speech difficulties may occur in patients with PD. If patients are not encouraged to communicate with carer(s) and/or in other special environments, they lose their desire to communicate and socialise. They also lose their non-verbal communication skills, due to the decrease in spontaneous facial expression. Their facial expression becomes mask-like with decreased eye blinking. This lack of body language and facial expression is often interpreted as a lack of understanding or disinterest in their environment (www.pdcaregiver.org, 17 November 2006).

Another feature of speech deficits in patients with PD is a monotonous voice with reduced volume and lack of rhythm of speech. This could be due to the rigidity in the muscles of the diaphragm and muscles of the soft palate as well as those involved with articulation (Hindle, 2001). Weak abdominal muscles may also contribute to the loss of volume in speech.

Patients with PD may also experience problems with swallowing and drooling due to the muscle weakness in the oral area. Speech may become rapid and slurred, due to the muscle weakness and fatigue (Hindle, 2001).

2.7.8 Cognitive Changes

Cognitive changes that occur affect the frontal lobe executive dysfunction, causing patients with PD to become confused, have slower thought processes and lose short-

term memory capacity. Patients with PD often experience emotions such as fear, irritability and insecurity in the advanced stages of PD.

Dementia refers to the cognitive impairment that affects activities of daily living. The cognitive impairment in PD affects the following aspects:

- Impairment of executive functions and goal directed behavior.
- Visio-spatial dysfunction.
- Instrumental functions such as language and praxis.
- Long term memory functions are usually well spared in PD. There are deficits in the learning of new information or in the short term memory (Pillon, 2001).

Dementia affects up to forty percent (40%) of patients with PD during the course of the disease. Parkinson's disease dementia (PDD) and dementia with Lewy bodies (DLB) are two common syndromes with similar clinical symptoms found in patients with PD. Clinically it is useful to distinguish between those disorders, but it has limited value when discussing the neurobiology and physiology as that is so similar (McKeith and Mosimann, 2004). If patients with PD are diagnosed with dementia in the first 12 months after the initial motor symptoms, they should be diagnosed with DLB. If the dementia develops later in the course of the disease, the diagnosis should be PDD (McKeith, 1996). The deficits in dopaminergic, cholinergic and noradrenergic mechanisms discussed in paragraph 2.5 are proposed as the basis of cognitive impairment in PD (Pillon, Czerneck, and, Dubois, 2003).

Cognitive impairment manifests as bradyphrenia and word finding difficulties. This does not usually hinder activities of daily living.

Naville described bradyphrenia, or slowness of thought processes, in 1922. Bradyphrenia is the slowing of cognitive processes related to PD. Wilson (1947) described this “lethargy of the mind” as “distinguished by a lack of interest, initiative, attention, concentration, fatigue and slow reactions: uncommunicative and wishing to be left alone.” In this case the patient sits and does nothing. There is, however, still controversy as to whether cognitive impairment exists as a separate entity in PD, or whether it can be explained by dementia, age and/or depression (Brown and Marsden, 1990). Bradyphrenia will only be involved in the slowing of the thought processes when dementia is present or when drugs interfere with the cognitive processes (Cooper, Sagar, Tidswell, and Jordan, 1994; Pate and Margolin, 1994). Bradyphrenia could also lead to bradykinesia by interfering with the planning of movement and the increase in reaction time. It is suggested that many patients with PD develop dementia, which has an influence on the speed of thought processes. The slowing of thought processes also occurs during the aging process and in patients with depression which is associated with PD (Cooper et al 1994). Thinking is however not just one thought process, it is a physiological process, and whether it is slower than expected normal values, will depend on the task at hand and the time duration required to complete the task effectively and efficiently. Some studies report the slowing of thought processes as having an effect on the movement quality in patients with PD (Cooper et al 1994; Pate and Margolin, 1994), while others do not (Duncombe, Bradshaw, Iansek and Phillips, 1994; Spicer, Brown, and Gorell, 1994).

2.7.9 Autonomic Nervous System Impairments

Patients with PD also reveal autonomic nervous system impairments. That will also have an effect on the daily functioning of the patient with PD on all three levels of disability i.e. on the impairment, functional activity level, and participation level. The most common of these autonomic impairments are the urinary dysfunction and constipation. Incontinence is one of the main reasons that patients with PD withdraw from the community, to save themselves from embarrassment in public. The hypostatic hypotension contributes to the number of falls, while excessive salivation and difficulties with swallowing contribute to the speech difficulties experienced by patients with PD.

- 1) Constipation is common in the elderly, but in patients with PD there is a higher incidence of this condition, due to the slow transit of food through the gastrointestinal tract (GIT). Patients with PD are relatively immobile, as already discussed, and this adds to the slowing of the GIT (www.neuro.jhmi.edu/hopkinspdmd/symptoms/constipation.htm, 18 November 2006).
- 2) Urinary dysfunction includes difficulty with urination and the urgency to urinate. Incontinence of both the bladder and the bowel has been reported in patients with PD (Hindle, 2001).

Incontinence may develop due to the decrease in physical activity and the lack of being able to 'hold their urine' due to the decrease in the voluntary external sphincter control. Another problem is the inability to empty the bladder completely causing an increase in the residual urine volume which

can lead to urinary tract infections (www.neuro.jhmi.edu/hopkinspdmd/symptoms/constipation.htm, 18 November 2006).

- 3) Orthostatic Hypotension is a sudden decrease in blood pressure upon sitting or standing up. This may occur due to PD and/or to the drugs used to treat the patient, as it is a known side effect of the drugs used to treat PD. This is more evident in the morning following a prolonged time of rest in a supine position (www.neuro.jhmi.edu/hopkinspdmd/symptoms/hypotension.htm, 18 November 2006).
- 4) Sexual dysfunction may arise due to the decreased mobility of the patient with PD. The resting tremor can worsen in an excited emotional state. Some men have difficulty obtaining and then maintaining an erection. It has also been found that in rare instances, both men and woman may develop an increase in sexual drive, which may cause social problems. It is usually related to their medication. An alteration of medication could improve the condition (www.neuro.jhmi.edu/hopkinspdmd/symptoms/sexual.htm, 18 November 2006).
- 5) Excessive salivation, rather than the excessive production of saliva, is ascribed to the accumulation of saliva in the mouth due to less frequent swallowing (www.neuro.jhmi.edu/hopkinspdmd/symptoms/salivation.htm , 18 November 2006).
- 6) Sleep dysfunctions are common, often accompanied by nightmares and restlessness.

The intervention of the multidisciplinary team will be discussed in the following paragraphs.

2.8 Treatment for Patients with PD

A wide variety of treatment modalities that are used to treat patients with PD are described in literature. And they reflect a variety of approaches to intervention. These are summarised in table format. The treatment modalities as well as the approaches to intervention reveal that patients with PD are treated by a multidisciplinary team that consists of the doctor (which includes various medical specialists) nurse, physiotherapist, occupational therapist, speech therapist, psychologist and social worker. Despite this team approach to intervention, the discussion on the intervention for these patients is focused on physiotherapy and related intervention by other team members specifically drug therapy and surgical interventions. The roles of the other team members are not discussed in this paper.

It is important that an extensive assessment of the patient with PD is done to determine the stage of the disease, and to plan and implement the relevant physiotherapy. Continual assessment and adaptation of the treatment plan will be necessary, due to the fact that PD is a progressive disease. Physiotherapy should be administered in close relationship with the other team members, to ensure that treatment approaches compliment each other at each stage of the disease. The effect of the patient's medication, the dose and timing of the medication with physiotherapy assessment as well as intervention is of utmost importance, due to the effect that the

medication has on the patient's functional ability. Clear outcome measures should be implemented during the assessment of patients to:

- Plan the desired individualised intervention that will accommodate the changing needs of the patient's motor performance over time (Morris, Iansek, and Churchard, 1998).
- Monitor the patient's progress or regression.
- Contribute to evidence based physiotherapy practice.

PD is one of the two most common conditions of the basal ganglia that are treated by physiotherapists (Morris, 2000). Physiotherapy is reported to be a low cost treatment and useful addition to the standard pharmacological interventions (Keller, Kessler, Meuser, Fogel, Bremen, and Jost, 2003; de Goede, Keus, Kwakkel, and Wagenaar, 2001; Gage and Storey, 2004; Morris, 2000).

The main aim of the physiotherapy programme is to ensure that the patient with PD remains as independent as possible, for as long as possible, with the use of the exercises and external cues. It is of vital importance that the patient with PD remains active within society and the community, to prevent withdrawal and immobility issues. All the exercises should be integrated into the home or the work environment, to ensure the patient remains active in that environment. The multidisciplinary team's responsibility is to assess the condition of the patient on a regular basis and to adapt or adjust the exercises/home programme as necessary, to maintain the level of independence.

Despite data obtained from several studies, there is still little evidence to support the efficacy of sustained physiotherapy on the motor performance in PD (Deane, Jones, and Playford, 2001). The reason for the lack of evidence for the efficacy of physiotherapy is due to the methodological weaknesses and the small number of randomized controlled trials that have been conducted to date (Dean, Jones, Ellis-Hill, Clarke, Playford, and Ben-Shlomo, 2001). There is also no evidence to favor a particular type of physiotherapy intervention in PD. (Bilney, et al, 2005; Polgar, et al, 2003). The principles of physiotherapy in the management of PD include (Plant, Jones, and Ashburn, 2000; Morris, 2000):

- 1) Physical management within a multidisciplinary team to coordinate patient centered goals on impairment, functional and participation levels. This should be regarded as 'the gold standard'.
- 2) Early referral to physiotherapy, to encourage participation in regular physical activity. This will assist in optimising the muscle strength, endurance, range of movement of the stiff joints and encouragement to socially interact.
- 3) Ongoing assessment and review. Patient goals and meaningful outcome measures should be continuously considered and the progress should be monitored.
- 4) The intervention should focus on addressing movement difficulties and enhancing the functional abilities of the patient.
- 5) Intervention should be done in the home and community setting, to ensure a safe and optimally functional participation outcome for the patient with PD.

Certain adjustments can be made to ensure easier mobility of the patient; for example, a higher, firmer bed, raised toilet seat etcetera. A simple home exercise programme should be given to the patient to optimise his/her functional ability. The intervention and the home programme should be closely monitored.

- 6) The forming of special care groups for both patients and care-givers, where mutual problems and difficulties that include issues of mobility, accessibility to services, medication etcetera and practical solutions to problems can be discussed. This can be in a formal setting of a special PD interest group, or smaller meetings at homes. Care-givers can also seek guidance and support from other care givers in the same situation.

Physiotherapy in the early stage (or in the case of milder disease) potentially aids at encouraging the patient to practice multi/dual tasks, improve general physical endurance and improve general muscle strength, while maintaining length (Nieuwboer, De Weerd, and Dom, 2002).

In the clinical stage of diagnosis and maintenance of functional ability, these patients will benefit from physiotherapy intervention by:

- 1) Addressing concerns of the differential diagnosis. The acceptance of being diagnosed with PD is difficult and many of the patients that are diagnosed with PD will initially have been diagnosed with other conditions and these may still be of concern to the patient.

- 2) Assess and monitor the patient's gait and movement patterns for early identification of movement problems.
- 3) Encourage participation in programmes to optimise cardiovascular output, muscular skeletal and neuromuscular systems and improve general fitness levels.
- 4) Prevent secondary complications due to immobility.
- 5) Introduce coping mechanisms during the progression of the disease to maintain functional mobility.
- 6) Education about the disease and the management of PD. Monitor drug efficacy.

In the early stage, specific physiotherapy aims should focus on maintenance of the patient with PD by:

- 1) Preventing musculoskeletal impairment through regular exercise, stretching of shortened muscles and soft tissue, and strengthening of functionally weak muscles
- 2) Maintain/optimize the patient's cardiovascular endurance
- 3) Minimise the difficulty that the patient may experience with gait, falling and transfers.
- 4) Provision of assistive devices and equipment to optimise the patient's functional ability.

Advice should be given to the care-givers and to the patient to ensure that the patient remains active in his/her home and work environment (Exercises/activities should be task specific and integrated into everyday life).

In the late stage of PD, patients who are no longer ambulant, benefit from a specific home programme that addresses and manages the impaired motor action of gait, the shift from sitting to standing, getting out of bed, etcetera (Nieuwboer et al 2002). The aim of physiotherapy should also be to emphasise the training of the care-givers to handle and care for the patient with PD.

Physiotherapy in the advanced stage of PD will focus on the:

- 1) Prevention of falls during transfers.
- 2) Promote cardio-respiratory system functioning and prevent circulatory complications such as pressure ulcers.
- 3) Alternative mobility issues that may include the issue of an assistive walking device or a wheelchair.
- 4) Assist the patient to function optimally in the position that he/she is in; i.e. in bed, positioning is of vital importance or to assist him/her with transfers into and out of bed.

According to the American Physical Therapy Association's *Guide to Physical Therapy Practice*, (2001), the main role of the physiotherapist within the multi disciplinary team is to teach people with PD coping strategies to manage impairments of gait and

movement on their own. The care-givers and/or close relatives should also be involved in this process.

Physiotherapists with a special interest in PD should be able to assess and monitor changes in functional ability, activity and participation, and the control of the patient's environment, such as in their responses to medication, surgery and the natural progression of the disease (Morris, et al, 1998; Bagley, Kelly Turncliffe, and Walker, 1991).

Table 2.1 summarises the assessment by the physiotherapist as a member of the multidisciplinary team. Assessment is essential in designing a relevant treatment programme for the individual patient. The assessment procedures are categorized according to the International Classification of Functioning Disability and Health (ICF) (WHO, 2002) to indicate the relationship between the different levels of assessment.

Table 2.1: Classification of assessment procedures according to the principles of the ICF

Impairment level	Functional Activity Level	Participation Level
<ol style="list-style-type: none"> 1) Range of Movement <ul style="list-style-type: none"> • Posture • Joint range • Muscle length 2) Muscle strength <ul style="list-style-type: none"> • Posture 3) Cardiovascular endurance 4) Sequence of movement during the task performance Shift to impairment level 5) Bradykinesia 6) Movement accuracy 7) Dyskinesia 8) Akinesia 9) Tremor 10) Lead pipe and/or cog wheel rigidity 11) Speech difficulties e.g. articulation. 12) Tone of voice 13) Bradyphrenia 14) Cognitive changes 15) Psychiatric disorders 16) Autonomic dysfunction <ul style="list-style-type: none"> • Orthostatic hypotension • Sexual dysfunction • Incontinence • Constipation 	<ol style="list-style-type: none"> 1) Gait <ul style="list-style-type: none"> • Use of walking aid • Stride length • Number of steps over distance • Stair climbing ability • Walking through doorways • Time to walk distance • Turning on the spot 2) Complexity of task performance 3) Balance during functional activities 4) Change in position <ul style="list-style-type: none"> • Getting out of bed • Sitting to standing • Turning in standing • Bending down in standing 5) Transfers <ul style="list-style-type: none"> • From bed to wheelchair • From bed to arm chair • From the ground in kneeling to standing 6) Speech and communication difficulties 	<ol style="list-style-type: none"> 1) ADL (self care) <ul style="list-style-type: none"> • Dressing • Grooming • Eating 2) Community ambulation <ul style="list-style-type: none"> • Freezing • Falls / Falls diary • Immobility 3) Community integration in terms of participating in meetings and other community activities. 4) Performing functional activities in context of lifestyle <ul style="list-style-type: none"> • Family role • Ability to cope in the work environment • Hobbies • Sport 5) Cognitive changes – having a conversation with peer group.(includes cognitive functioning and ability to communicate effectively) 6) Getting in and out of a car

(Pullman, Watts, Juncus, Chase, and Sanes, 1988; Reid, Broe, Hely, Morris, Williamson, and O'Sullivan, 1989; Harrison, Goodrich, Kennard, and Henderson, 1993; Wierzbika, Staude, Wolf, and Dengler, 1993; Marsden, 1994; Morris and Iansek, 1996; Cunnington, et al, 1995; Klockgether, Borutta, Rapp, Spieker, and Dichgans, 1995; Berardelli, et al, 1996b; Morris and Iansek, 1997; Oliveira, et al, 1998; Viliani, Pasquetti, and Magnolfi, 1999; Ashburn, et al. 2001; Shumway-Cook and Woollacot, 2001; Morris, 2005.)

Table 2.2 summarises the outcome measures that are used by the physiotherapist during the assessment of the patient with PD. All the outcome measures are evidenced based and internationally recognised scales. The use of these outcome measures is essential for applying evidenced based physiotherapy practice.

Table 2.2: Classification of outcome measures according to the principles of the ICF

Impairment level	Functional Activity Level	Participation Level
<ol style="list-style-type: none"> 1) Range of Movement: Measurement with goniometer. 2) Muscle strength: Oxford grading scale 3) Cardiovascular endurance: PCI 4) Bradykinesia is measured by: the time it takes for the patient to make a 360 degree turn and number of steps to turn 5) Movement accuracy 6) Sequence of movement during the task performance 7) Akinesia: Freezing of gait scale 8) Tremor: 9) Rigidity and cog wheel rigidity: Modified Ashworth scale 10) Speech difficulties: By speech therapist 11) Tone of voice: By speech therapist 12) Bradyphrenia 13) Cognitive changes: Mini mental assessment scale 14) Psychiatric disorders: Psychological assessment Autonomic dysfunction: By Medical Practitioner. 	<ol style="list-style-type: none"> 1) Gait: Full clinical gait assessment <ul style="list-style-type: none"> • Use of walking aid • Stride length • Number of steps over distance • Stair climbing ability • Movement through doorways • 10 metre timed walk • Turning • PCI • Berg Balance scale 2) Complexity of task performance: observation and description 3) Falls diary 4) Change in position: Parkinson's disease rating scale and assessment 5) Transfers : Parkinson's disease rating scale and assessment 	<ol style="list-style-type: none"> 1) ADL (self care): Parkinson's disease rating scale and Parkinson's Disease quality of life 39 scale 2) Community ambulation: Parkinson's disease rating scale and Parkinson's disease quality of life 39 scale. Rivermead Mobility Index is also used, but was not used in this clinical trial 3) Community integration: Freezing of gait scale, Parkinson's disease rating scale, Parkinson's Disease quality of life scale 4) Performing functional activities in context of lifestyle: Freezing of gait scale, Parkinson's disease rating scale, Parkinson's Disease quality of life scale 5) Cognitive changes: Mini mental assessment scale 6) Bradykinesia: Freezing of gait scale during community ambulation

(Folstein, et al, 1975; Bohannon, 1989; Duncan, Horner, and Reker, 1990; Lennon and Hastings, 1996; Jenkinson, et al, 1997;

Katz, Mills, and Cassidy, 1997; Gregson, et al, 1999; Fuller, 1999; Guidelines Group, 2001;

www.query.fcgi?cmd=retrieve&db=PubMed&lists_uids=12815652&dopt=Abstract, 8 February 2006)

Table 2.3 summarises the intervention of the physiotherapist. The medical and pharmacological intervention is mentioned under Impairment Level because it plays a role in the patient's performance during physiotherapy and therefore should be considered during treatment. The treatment of patients with PD should be based on a multidisciplinary team approach.

Emphasis should be placed on the early intervention once PD has been diagnosed, to ensure the full potential of the patient is reached despite the stage of PD that the patient is in. This will also ensure that the prevention of mental and physical fatigue of the patient with PD and could aid in preventing attention problems, falls and other injuries. This will also ensure the optimisation of the functional ability and independence of the patient with PD. The ongoing assessment of the patient's reaction to the treatment using valid outcome measures is essential.

Table 2.3: Classification of physiotherapy according to the principles of the ICF

Impairment Level	Functional Activity Level	Participation Level
<ol style="list-style-type: none"> 1) Regular free active large range of movement activities. <ul style="list-style-type: none"> • Active and passive stretching of shortened muscles and soft tissue to assist in improving posture and mobility of the patient • Regular strengthening exercises of all large groups of muscles to maintain mobility 2) Optimise cardiovascular endurance to maintain endurance during mobility or walking 3) The above will assist in reducing the bradykinesia and the rigidity in the early stages of PD. <ul style="list-style-type: none"> • Use of internal and external cues to overcome episodes of bradykinesia and akinesia. • Breaking multi-tasks up into smaller components of movement and practice separately before practising it together again. 4) Improve balance reactions <ul style="list-style-type: none"> • Voluntary lifting of the feet • External and internal cues • Weight transfers • Use of a walking aid 5) Use of external and internal cueing to assist in turning 6) Conduct the movement at a slower pace to improve movement accuracy 	<ol style="list-style-type: none"> 1) Maintain and improve gait and ambulation of the patient through <ul style="list-style-type: none"> • External cues, i.e. audio, visual, sensory and verbal cues • Internal cues, i.e. cognitive attention to the task at hand • Maintain physical endurance • Regular strengthening activities and stretching of shortened muscles and soft tissue • Use of walking aids as required • Constant optimizing of posture 2&3) Facilitate smoother movements by changing position from sitting to standing by <ul style="list-style-type: none"> • Using external cueing • Using internal cueing • Using supportive chairs with arm rests or chairs with higher seats that require less effort to stand up. • Breaking up of the functional task at hand into simple movements, i.e. shifting forward in the chair, look upwards, press on the arm rests, lean forward and then extend the hips and the knees. ii) Getting up from lying to sitting/ standing up and walking <ul style="list-style-type: none"> • Breaking the task into simple components, i.e. roll onto side, swing legs down, and lift body with arms. 4) Falls diary to be kept in order to determine in 	<ol style="list-style-type: none"> 1) Advice must be given on mechanisms to assist the patient in carrying out all ADL's independently. These can then be integrated and treated on the impairment level. The patient must take responsibility and practice these strategies independently. <ul style="list-style-type: none"> • Break the tasks up into simple movement components • Use of external cues • Use of internal cues • Maintain joint mobility to be as independent as possible 2) Practice complex/ simultaneous tasks by walking outdoors while <ul style="list-style-type: none"> • Talking or carrying something, • Stopping and picking something up • Changing direction from walking forward to walking sideways. • Walk specific functional distances in the community, i.e. to shops, cinema, church, over normal variations in surface texture and obstacles with the assistance of a care giver, without the care giver and with a variation in the walking speed. 3) Remain active in community sports and social clubs for as long as possible 4) Adaptation of the home environment as

<p>7) Control dyskinesia with medication or surgical intervention</p> <p>8) Tremor treated by</p> <ul style="list-style-type: none"> • Surgery • Habitual tasks such as putting the hands in the pockets when the tremor gets too much <p>9) Stretching</p> <ul style="list-style-type: none"> • Maintain joint mobility • Maintain physical fitness and activity level • Weight bearing activities <p>10) Consult a speech therapist for relevant exercises for articulation difficulties</p> <p>11) Social integration</p> <ul style="list-style-type: none"> • Remain involved in social activities • Remain as active as possible • Continue with activities such as crosswords puzzles, etcetera to remain cognitively active <p>12) Orientate the patient on a daily basis to time, place and person</p> <ul style="list-style-type: none"> • Maintain a diary to assist with short term memory loss • Continue with cognitively stimulating exercises <p>13) Psycho-social and emotional status interventions for psychiatric disorders</p> <p>14) Get up slower than usual and take deep breaths to avoid hypostatic hypotension. Other measures to treat ANS dysfunctions include:</p> <ul style="list-style-type: none"> • Wear tension stockings to prevent a sudden drop in the blood pressure 	<p>what situation the falls occur and this can be addressed with the</p> <ul style="list-style-type: none"> • Use of internal and external cueing. • The timing of medication may need to be adjusted. <p>5) Turning around Lift the feet one at a time or turn with a rhythm in the head. The use of external and internal cues can be used to prevent freezing during the activity.</p> <p>6) Patient and care giver education on the effect and progression of PD and how to manage the progression of the disease.</p> <p>7) Communication and articulation exercises by the speech therapist</p>	<p>the patient regresses, i.e. introduce a high bed with a firm mattress. The use of a raised toilet seat, fixed rails and/or bars in the bathroom for the patient to push up on.</p> <p>5) Join a support group.</p> <ul style="list-style-type: none"> • Discussion with the physiotherapist or the team to enhance self management by the patient. • Encourage the patient to do problem solving in difficult situations. • Assess problems with medication • Accessibility of rehabilitation services • Acquiring of assistive/supportive devices. • Patients goals in life in terms of managing himself with his condition <p>6) Managing and monitoring own medication</p> <ul style="list-style-type: none"> • Social interaction • Participation in care group activities • Follow home exercise programme <p>7) Balance reactions</p> <ul style="list-style-type: none"> • External and internal cueing • Avoid / manage environment with increased sensory input. • Narrow doorways • Changes in surface • Increase stride length • Multi tasking
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<ul style="list-style-type: none"> • Medication • Diet • Increase water intake • Use of catheters or other external devices for incontinence 		
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(Evarts, et al, 1981; Gibberd, Page, and Spencer, 1981; Stelmach, Worringham, and Strand, 1986; Sheridan, Flowers, and Hurrell, 1987; Yekutieli, Pinhasov, Shahar, and Sroka, 1991; Fertl, Droppelbauer, and Auff, 1993; Morris, et al, 1994; Kamsma, Brouer, and Lakke, 1995; Morris and Iansek, 1997; Demirci, Grill, McShane, and Hallett, 1997; Morris, Collier, and Matyas, 1998; Berardelli, et al 2001; Morris, 2000; Morris, 2001; Nieuwboer, et al, 2002; Del Olmo and Cudeiro, 2005; Rochester, et al, 2005; Maurer, Mergner, Xie, Faist, Pollak, and Luching, 2003; Suteerawattananon et al, 2004; Fernandez, Lannon, Treichmann, and Friedman, 2004; Cubo, Leurgans, and Goetz, 2004; Mak and Hui-Chan, 2004; Thanvi and Lo, 2004; Gage and Storey, 2004; Brauer and Morris, 2004; <http://org/library/disease/pd/par-sur.html>, 8 February 2006; www.neuro.jhmi.edu/hopkinspdmd/symptoms/sexual.htm, 18 November 2006.)

The approach to rehabilitation for patients with PD is one of compensation and not restoration of movement impairment. Restoration occurs due to plasticity of the brain and function can often be restored. If there is no improvement, as in the progressive nature of PD, restoration is limited and the rehabilitation approach is on of compensation to over come difficulties that are experienced.

As can be concluded from the Table 2.1, 2.2 and 2.3, internal and external cueing play a large role in the treatment and rehabilitation of patients with PD. They are compensatory mechanisms and the use of these in the treatment of PD will therefore be further discussed.

2.9 External and Internal cueing in the treatment of PD

Patients with PD appear to be reliant on cortical control mechanisms to initiate movement because they do not loose the ability to move, but rather the ability to activate the movement (Ivansek et al 1995; Cunnington, et al, 1995). External cueing has been used to enhance the patient with PD's motor performance since 1942 (Gage and Storey, 2004). External cues can compensate for the defective basal ganglia by utilising nerve pathways that bypass the defective basal ganglia and the supplementary motor area (Jahanshani, Jenkins, Brown, Marsden, Passingham, and Brooks, 1995; Morris, 2000; O'Shea, et al, 2002; Perry, et al, 2004; Morris, 2005).

The rehabilitation of gross motor skills require compensatory, as opposed to normal, movement strategies (Kamsma, et al, 1995). The compensatory movement strategies

are approaches that meet both the sensory and the motor requirements of a task, to ensure that the task is completed correctly. Compensatory movement strategies include processing sequential tasks and undertaking motor and cognitive tasks concurrently to ensure that the task is carried out correctly. The principles of the underlying development of these compensatory mechanisms include the break down of the movement into simple components, the rearranging of the components into a logical order, the application of prior mental rehearsal of the movement before performing it, the performance of each component of the movement separately, the avoidance of simultaneous motor or cognitive tasks and the application of the appropriate external cues to initiate and maintain the movement (Morris and Iansek, 1997; Morris, 2000).

A model for physiotherapy intervention in PD described by Morris (2000) is based on the assumption that normal movement can be obtained by teaching the patient strategies to compensate for the lack of ability to conduct a task due to the basal ganglia pathology.

External cues that are used to initiate gait include visual, auditory or somato-sensory information. In combination, these aid to improve the quality of the movement (Suteerawattananon, et al, 2004; Del Olmo and Cudeiro, 2005; Dibble, Nicholson, Shultz, Mac Williams, Marcus, and Moncur, 2004).

Internal cues can also be utilised to improve the quality of the movement of the patient with PD. These internal cues utilize vestibular stimuli with mental rehearsal (repetition

of the thought of movement components) to improve awareness of the movement components. The effectiveness of the internal cues may relate to how much cognitive attention is used to effectively perform the task at hand (Morris, Iansek and Matyas, 1996). However Marchese, Diverio, and Zucchi (2000) found that by teaching patients to use compensatory movement strategies and sensory cues (a combination of internal and external cueing) have a longer lasting positive effect on gait, when compared to those programmes that focus on exercises and functional activities alone.

Morris (2005a) describes various interventions that may be effective in the treatment of a movement disorder such as PD:

- External visual and auditory cues probably serve the purpose to turn the locus of control of movement away from the basal ganglia to the intact frontal regions of the brain. This enables the patient to maintain a functional gait performance while performing complex tasks.
- Auditory cues improved the cadence, whereas visual cues improved stride length (Suteerawattananon, et al, 2004). The application of rhythmic auditory stimuli to facilitate gait improves the time it takes patients to walk a pre-determined distance (Del Olmo and Cudeiro, 2005).

From the randomized control trials evaluated by Morris (2005) it can be concluded that there is not sufficient research available in the field of physiotherapy that determines the effectiveness of cognitive input to improve the functional ability of patients with PD.

Neither is there sufficient research to determine the effectiveness of the breaking down of movement components into simple components to improve their functional ability.

Cubo, et al (2004) did, however, conclude that rhythmic auditory cueing actually slows the walking pace of the patient with PD and is not beneficial in overcoming their freezing episodes (Cubo et al 2004). As only twelve (12) patients with PD participated in this research the statistical power of the study is questionable.

With appropriate external visual and auditory cues patients with PD reveal a gait pattern of normal velocity, cadence and stride length. This indicates that the deficiency experienced in patients with PD involves the internal regulation of stride length (Lewis, Byblow, and Walt, 2000). The precise mechanism behind the reduced stride length is still poorly understood. As yet there is no treatment method for assisting patients with PD to regulate their stride length throughout the course of this progressive neurological disease (Morris, et al, 1996). The increased cadence is a compensation for the decrease in stride length (Morris, et al, 1994). A common finding in these research articles is that normal range movement can be achieved with some form of external cue in patients with PD under the correct conditions.

In a randomized control study, Mak and Hui-Chan (2004) explained the positive effect of audio-visual cues on the process of sitting to standing in patients with PD. It was theorised that the audio-visual cues are probably integrated by the sensory-motor system via different nerve pathways to bypass the affected pathways in the defective basal ganglia in the altered initiation of movement, sequence of movement and speed during the movement from sitting to standing.

Due to the limited research on the role of sensory cueing in the gait of patients with PD, Dibble, et al (2004) conducted a study to determine the effect of sensory cueing on the maximal gait speed that patients with PD could achieve during gait. The patients with PD were compared to a healthy group of the same age with regards to gait and the analysis of movement components during gait. The variables administered to the patients were, no cue, single auditory cue, repetitive auditory cues and repetitive sensory cutaneous cues. Patients with PD reacted more slowly on all the cues than the matched healthy age group. The outcome of this trial suggests that when the speed of gait is the primary goal of the participant, sensory cues may interfere with body movement outcomes and the lower limb, causing a slower swing phase and gait speed over the specific distance, probably due to rigidity in patients with PD. The trunk movements were less and the arms appeared to be more adducted and braced to the side of the body giving a rigid appearance of the posture during gait (Dibble et al, 2004).

Meiran, Friedman, and Yehene (2004) discovered that patient's with PD experience problems with prioritizing goals when they are confronted with a choice between tasks. They had difficulty in determining the most relevant task at hand when given a sequence of consecutive tasks to perform. This indicates that their motor planning or executive motor performance is affected.

In a systematic review by Lim, van Wegen, de Goede, Deutekom, Nieuwboer, Williems, Jones, Rochester, and Kwakkel (2005) it was found that only one study evaluated the effects of tactile cueing on gait. The results of the trial on the visual and

the auditory cueing were reported to be invalid due to the poor methodology of the trials. The external cues assist patients with PD to move within the correct movement components via the intact pre-motor cortex, instead of the defective basal ganglia (Goldberg, 1985).

Kinesia Paradoxa is a phenomenon exhibited by some patients with PD that suffer from akinesia. Kinesia Paradoxa occurs when the patient with PD is able to step over obstacles in their path, easily climb up or down stairs, all with a significant decrease in the shuffling and freezing gait patterns evident in PD. Bagley, et al (1991) and Lewis, et al (2000) concluded that these situations provide visual cues that result in near normal gait patterns of patients with PD, including normal stride length, cadence and velocity (Bagley, et al, 1991; Lewis, et al 2000). The authors developed a portable gait-enabling device which looks like a pair of 'non-correctional' spectacles and give different and intermittent optic stimuli during gait. The continuous flow of optic stimuli increased the gait velocity and resulted in increased stride length in patients with PD (Ferrarin, Brambilla, Gravello, Di Candida, Pedotti, and Rabuffetti, 2004). No mention was made of the effect of the non-correctional spectacles on the incidence of falls in this particular study. Neither is there evidence of the effect of the 'non-correctional' spectacles on freezing (Urquart, Morris, and Iansek, 1999).

In a study done by Weghurst and Kaminsky (1999), it was concluded that the stride length was increased when walking with physical cueing as well as with virtual cueing spectacles. Virtual cueing spectacles gave the image of virtual lines that are scrolling down, giving the illusory effect of movement. Patients reported that they felt walking

was easier with the external cues than without. The authors also found that the more realistic the visual cue was the more patient stride length increased (Weghurst and Kaminsky, 1999).

The patient's speed of reaction to either a visual or a virtual cue depends largely on the preceding cue of the command that is given, according to Williams, Kuhn, Kupsch, Tisjssen, van Bruggen, Speelman, Hotton, Yarrow, and Brown (2003). Shorter reaction times to external cues occur when reliable verbal warning cues are given and the patient has pre-prepared responses to the external cues that are to be given. Williams et al (2003) are of the opinion that local field potential activity in the subthalamic nucleus is modulated by the behavioral relevance of the external cue. It implies that the subthalamic nucleus is involved in facilitating the response from the external cue. This finding is coherent with the earlier discussion on reaction times in bradykinesia. In patients with no cognitive deficits, the focus of attention on the task and the critical movement component will result in the accurate amplitude and speed of the movement (Morris, et al, 1998). Coping strategies that rely on the focus of attention on the task at hand will however not be effective in people with severe cognitive impairments in the advanced stage of PD (Greenfield and Bosanquet, 1953). Physiotherapy that focuses on cortically mediated learning processes and cognitive strategies i.e. the learning of new movement strategies will depend on the patient's ability for learning new strategies (Morris, et al, 1998). Patients in the final stages of PD will benefit from external cues and other strategies that rely on less complex information processing.

2.10 General aspects to be considered in planning a physiotherapy intervention programme for patients with PD

- The effects of aging and adaptive changes in the musculoskeletal, cardiovascular and neuromuscular systems must be considered when assessing the patient's performance of a functional task (Morris, 2000). By using task specific training principles, the patient could be taught to perform functional tasks of everyday life using coping strategies such as: control of dystonia, how to compensate for the dystonia in walking or the functional tasks that are important to the patient and in which he/she experiences functional limitations. The task specific training should be focused on the most problematic environment to ensure the patient is independent in all areas of life (Morris, 2000).
- Strategies to enhance cognitive attention to the task that is being performed at the time includes that of concentrating on taking long steps (Morris, et al 1994), maintaining postural stability while turning (Morris, 2001), and breaking long and complex movements up into simple movement components of functional activities. By using such cognitive strategies the frontal cortical regions are utilised to compensate for the dysfunctional basal ganglia via the external locus of control (O'Shea, et al, 2002).
- Multiple task performance is a further possible form of training patients with PD to complete more than one task at a time (Brauer and Morris, 2004).

- Multi-Component Programmes entail a multi disciplinary team approach to treatment of patients with PD, that result in positive effects on the movement difficulties experienced by PD patients (Morris, 2005).
- The therapist should take into consideration, the response of the patient to the external cues, knowledge of how interventions can be adapted according to the severity of the cognitive impairment, the ability to analyse functional task performance and the effects of medication on the movement, when designing the intervention programme for an individual with PD.
- Both mental and physical fatigue could influence the patient's attention on the functional activity and increase the interference of gait during complex or multi-tasks and this could have a negative effect on the safety and functioning of gait (Rochester, et al, 2005).

2.11 Improvement and maintenance of the functional ability in patients with PD

The improvement and maintenance of a patients' functional ability entail that the deterioration in the patients physical ability due to secondary muscle weakness, a decrease in their joint range of movement, and cardiovascular endurance and the development of spinal deformities (Morris, et al, 1997) should be prevented. The aims of most of the general exercise programmes for PD patients promote function through improvements in strength, flexibility, co-ordination, balance and relaxation (Viliani, et al, 1999). Through a well planned exercise programme, that takes factors such as aging and other medical conditions into consideration in the planning of the patients' treatment programme, exercise endurance and general functional mobility on

participation level is maintained / optimised. This enhances activities such as continued ambulation within the community (walking to the shops, to church or to visit friends, etcetera).

Pellecchia, Grasso, Biancardia, Squillante, Bonavita, and Barone (2004), have shown that there is functional improvement as well as sustained improvement in the motor skills of PD patients that follow a long term comprehensive rehabilitation programme that is designed by a physiotherapist. The comprehensive programme that resulted in the sustained functional improvement addressed the patients' problems on the impairment, functional activity and the participation levels.

A study on hypokinetic patients with PD revealed that there is consistency in the components of gait, such as speed and stride length, provided that the subjects were measured in the "on" state of medication, i.e. at least 30 minutes post drug administration. The same subjects were assessed during the peak phase of the medication and then again 30 minutes prior to the next administration of medication. Marked differences in the gait pattern of these patients could be observed. The change in the gait performance strongly suggests that medication has a marked impact on the gait pattern in patients with PD (Urquhart, Morris, and Iansek, 1999). Physiotherapists, should therefore teach patients with PD to cope with the movement disorder in both the "on" and the "off" periods of medication (Morris and Iansek, 1997).

Morris (2000) explains strategies that can be used to optimise the performance of functional motor tasks in these patients in a number of functional activities. The

problems that patients with PD experience in performing these activities and the strategy that used to cope with the situation are discussed in the following paragraphs.

Walking: The main problem with gait for these patients is the deficit in regulation of their stride length. There is a relationship between the step length and ground clearance, and this is why patients with hypokinesia are at risk of tripping and falling. Patients with a step length of less than one meter have a ground clearance of 0.8cm compared to the normal value of 1 to 1.3cm (Winter, 1991). The use of external cues and cognitive strategies are the focus of the physiotherapist when trying to improve the functional gait of a patient with PD.

Visual cues have been shown to normalize the spatial and temporal variables of gait. Avoiding dual tasks has proven to assist in normalising the stride length of patients with PD in the early stages of the disease (Bond and Morris, 2000). If the attention of these patients is diverted to a second task away from the one they are busy performing, their stride length and gait speed decreased immediately. There has been no research in the field of long-term effects of external cues and cognitive strategies on gait in PD. Burleigh-Jacobs, et al (1997) have shown that auditory cues can assist in avoiding episodes of freezing. Teaching patients to concentrate on the heel strike and push off phase of gait (i.e. the normal sequence of movement components during gait) also aided in increasing the stride length and decreased the episodes of freezing (Morris, 2000).

Turning (as part of functional gait): Patients with PD find turning especially difficult when they experience episodes of freezing. Yekutieli et al (1991) found that these patients can take up to twenty (20) steps to turn 360 degrees. During the turning each step gets smaller until all movement ceases. There is also little normal associative movement in the head, arms and trunk while turning. To overcome this, patients with PD are taught to turn in large arcs instead of on one place (Yekutieli et al, 1991). In preparing patients to turn in small/narrow places, they can also be taught to use the clock method that entails the patient to consciously step with each foot to a relevant position of a number on an imaginary clock while turning, though there is no research data to back this intervention (Kirkwood, Cattermole, Winkler, and Shears, 1997).

Preventing falls: Falls probably occur due to dystonia that manifests in the plantar flexors and invertors of the foot or both the feet. This increases the chances of tripping and falling. A 'falls diary' can be used to help determine the circumstances under which falls most often occur (Morris, 2000). The patient needs to be taught prevention strategies based on the contributing factors that may include cognitive impairment, the way in which tasks are performed, environmental factors, medication and age related factors.

Standing up and sitting down as a functional limitation: The common problem in patients with PD is that they do not lean forward enough (hip flexion and forward weight transfer) to get to a standing position. The line of gravity (center of mass) is then maintained to the posterior of the center of gravity of the body, between the feet

during standing up. It is also possible due to the impaired sequence of movement components that is characteristic of patients with PD. Akinesia also plays a role in the difficulty patients experience to get from sitting to standing (Carr and Shepherd, 1998). Getting from sitting to standing is a typical example of the breakdown in the sequence of movement that patients need to acquire to stand up. Using verbal cues to complete the task is usually essential. Proprioceptive cues and vestibular stimulation, such as rocking forward and backwards, assists patients with akinesia to accomplish the task (Morris and Iansek, 1997). Chairs with armrests are also beneficial for propelling oneself up into the standing position by using the arms.

Maintaining General fitness: Physiotherapy needs to focus on muscle strengthening, range of movement and endurance from the beginning of intervention, to ensure that the patient will be able to remain as mobile as possible for as long as possible. This will also ensure that the patient with PD will remain socially active for a longer period of time. The patient should be encouraged to participate in some form of recreational sport, such as bowls, swimming, yoga, walking, etcetera (Fertl, et al, 1993) to optimise cardiovascular endurance. The older population enjoys these types of sporting activities and the patient with PD is assisted to remain socially active in the community if their level of fitness is maintained. They should also be encouraged to go shopping, visit friends, go to church and participate in other social activities. This will also ensure that they maintain general fitness, while improving on the participation level in the community. The maintenance of their endurance will also ensure the maintenance of motor function and help to reduce stiffness and other musculo-skeletal impairments.

2.12 Rehabilitation team

The core rehabilitation team that consists of the doctor, nurse, physiotherapist, occupational therapist, speech therapist, social worker and the dietitian, should offer health education and give the necessary treatment required at the relevant stage. Neuropsychologists and neurosurgeons are role players in the later stages of the disease when neuro-surgery becomes one of the intervention options (Guidelines group, 2001).

Morris (2000) states that it is necessary to determine the core elements of that will form the basis of physiotherapy input in the patients as early as possible after the diagnosis, to ensure that a relevant treatment programme is designed for each patient. As the disease progresses it is necessary to adjust the intervention according to the stage of PD. Care-givers also need to be taught additional strategies to cope with their spouses'/family member's movement disorder.

Physiotherapy intervention cannot cure PD, but offers symptomatic relief by teaching movement strategies to overcome the influence of the degenerating basal ganglia on movement and function of the patient. Together with medication and other members of the rehabilitation team, physiotherapists can assist the patient in decreasing the effect of this disability and improve the quality of life of those living with PD.

Further research is however needed to enhance the understanding of the pathology of PD and to determine the most effective intervention or combination of intervention

strategies that will optimize the functional ability and the quality of life of patients suffering from PD.

2.13 Medical treatment of PD

Medication is used to treat bradykinesia, tremor, dyskinesia, bradyphrenia and dementia in PD. These will be discussed in the following paragraphs. The pharmacological intervention of the secondary signs and symptoms of PD, namely, constipation and urinary dysfunction, orthostatic hypotension, sexual dysfunction and salivation, will also be discussed.

2.13.1 Pharmacological treatment in PD

The treatment of PD is usually initiated with a combination of levo-dopa, (a dopamine precursor that crosses the blood-brain barrier) and carbi-dopa (an inhibitor of dopa decarboxylase that does not cross the blood-brain barrier). The carbi-dopa prevents the conversion of levo-dopa to dopamine outside the brain. This assists in decreasing the dopaminergic side effects and assures the optimum delivery of dopamine to the brain (www.dartmouth.edu/~dons/part_2/chapter_18.html, 18 November 2006). A prospective study revealed that PD is symptomatically controlled with the standard treatment of levo-dopa, but this effect decreases after a few years. The anticholinergic drugs are used only in younger patients with tremor, due to the adverse effect it has on cognitive functioning in the older patient (Playfer, 2001). Levo-dopa is the drug most widely used to improve most of the PD signs and symptoms. It prevents the peripheral metabolic breakdown of levo-dopa to dopamine. However, the effect wears off rapidly

and increasing the dose treats this rapid decline in effect but the increase in the dose is associated with rapid switches from the “on” to the “off” periods. Increasing the dose of leva-dopa has not proven to be effective in preventing falls (Bloem, et al, 2001).

A study was conducted to evaluate the evolution and the motor complication during the first five years after diagnosis in 59 patients with PD. Those patients showed a significant improvement on the UPDRS in the first year but revealed progressively worsening scores thereafter - especially after the third year. Motor fluctuations, dyskinesias and freezing episodes were found to have increased, following year three (3) from ten percent (10%), sixteen percent (16%) and thirty five percent (35%) respectively to thirty five percent (35%), thirty two percent (32%) and twenty seven percent (27%). It was found that after five (5) years, fifty percent (50%) of the subjects still had UPDRS scores equal to or better than the initial baseline, and forty four percent (44%) had no motor complications. Thirty eight percent (38%) of the patients had initially been treated with leva-dopa and the other fifty two percent (52%) with other agents (Garcia Ruiz, Meseguer, Del Var, Vazquezz, and Sanchez Bernardos, 2004). Freezing has also been found to be one of the long-term side effects of leva-dopa, and is evident in both the “on” and the “off” periods of medication (Nieuwboer, Feys, and De Weerd, 1997). The extent to which these patients were affected fluctuated from normal function to severe hypokinesia and rigidity typical of PD. This could be due to erratic absorption or distribution of the drug, or transport across the blood brain barrier. Mechanisms employed in the attempt to overcome these fluctuations included a low protein diet, the gastric administration of leva-dopa at a

constant rate, by duodenal infusion, addition of vitamin C and the administration of water-soluble dopamine agonists (www.acnp.org/g4/GN401000142/CH139.html, 13 February 2006). In cases where the dose of leva-dopa was increased to 600mg with no corresponding effect on the patient, it became evident the diagnosis of PD needed to be questioned. Only about twenty percent (20%) of patients with symptoms of PD have a poor response to dopaminergic treatment and it was found that those patients usually had a different underlying process, with a more severe prognosis (www.patient.co.uk/showdoc/40002307, 22 February 2006).

Holloway et al (2004) found that leva-dopa assists in decreasing freezing episodes in the early stages of PD. It has been proven that treatment of PD with pramipexole decreases the effects of dyskinesia to a greater extent than treatment with leva-dopa. Pharmacological treatments tend to lose their effectiveness, while surgical intervention has no effect on freezing; therefore it becomes necessary for non-pharmacological treatment to be explored for this serious problem (Gray and Hildebrand, 2000; Morris, 2000; Schrag, Ben-Shlomo, Brown, Marsden, and Quinn, 1998). To optimise these pharmacological interventions a better understanding of the pathogenic mechanisms is required (Thanvi and Lo, 2004). The basal ganglia are involved in the sequencing of movement components and ensuring that the execution of movement takes place smoothly with little conscious attention to each movement component (Brauer and Morris, 2004).

Dopaminergic medication can cause orthostatic hypotension and therefore contribute to the occurrence of falls (van Dijk, Haan, Zwinderman, Kremer, van Hilton, and Roos, 1993). Other side effects of medications include confusion, hallucinations and psychosis that could also contribute to falls.

Deep brain stimulation (DBS) can partially alleviate gait impairment and postural instability. Other means to aid in preventing falls include the use of elastic compression stockings to prevent orthostatic hypotension. Using anti-orthostatic maneuvers such as squatting or standing with crossed legs can also aid in preventing orthostatic hypotension. Orthostatic hypotension can also be treated pharmacologically using fludrocortisone or sympathicomimetics (Bloem, Overeem, and van Dijk, 2004).

Osteoporosis can be treated with oral provitamin or menatetrenone to decrease the incidence of fractures.

There is no preventative measure for the essential tremor in PD.

A mild resting tremor does not require treatment. Pharmacological treatment of tremor usually begins with primidone or propranolol monotherapy. The initial dosages are gradually increased, until a required effect is reached. Propranolol is usually started at a dose of 40mg twice daily. This may be increased to 120-320mg per day. Primidone is found to reduce the amplitude of tremors by up to 60 to 70 per cent. The starting dose of this is 12.5-25mg per day and can be increased to 750mg daily. If one of these pharmacological agents is found to be not effective on its own, it may have an effect

by being used in combination with other drugs. Other pharmacological interventions include the use of botulinum toxin in the form of localized injections in the motor end plate, to reduce muscle activity. Botulinum Toxin type A has been reported to help in overcoming freezing episodes. Fernandez, et al (2004) found that the use of the toxin type B injected into the most affected soleus or gastrocnemius muscle complex did not significantly improve the freezing of gait. This study only had a few participants presenting with freezing episodes (n=17), so there is a chance that a small beneficial effect may have been missed (Fernandez et al, 2004).

Benzodiazepines may reduce the tremor, but this however has side effects of severe sedation.

Treatment for essential tremors includes pharmacological approaches and surgical management. If the tremor is severe and is not responding to the intervention, surgery may need to be considered (Wissel, Masuhr, Schelosky, Ebersbach, and Poewe, 1997). Physical techniques and psychological measures, such as biofeedback and relaxation techniques, may prove to be helpful in managing mild tremor.

(www.clevelandclinicmeded.com/diseasemanagement/neurology/tremor/tremor.htm, 18 November 2006).

Decreasing the dose of the dopamine agonist can alleviate dyskinesia. Amantadine has recently been shown to have an effect in reducing dyskinesia. If the dyskinesia is incapacitating and cannot be treated with medication adjustments, deep brain stimulation is an alternative intervention

(www.neuro.jhmi.edu/hopkinspdmd/symptoms/dyskinesias.htm,
18 November 2006).

Dementia poses a problem in patients with PD, as the patients are sensitive to dopaminergic drugs, which can precipitate confusion and hallucinations. Anti-parkinsonian medication should be reviewed and gradually withdrawn in the following order if dementia is a problem: anticholinergics, amantadine, selegiline and dopamine agonists. If the withdrawal of the drugs offers no improvement, an antipsychotic agent can be given as an alternative (Friedman and Factor, 2000).

Constipation can be controlled naturally by a high fibre diet, drinking plenty of fluids and increasing physical activity. Stool softeners can be used and if required, laxatives. Protective garments may be required for the bowel and bladder incontinence. Depending on the severity of the problem, medications may be helpful (www.neuro.jhmi.edu/hopkinspdmd/symptoms/constipation.htm, 18 November 2006).

As already discussed under the treatment of falls, elastic compression stockings may aid in decreasing the incidence of hypotension. The patient should be taught to stand up slowly in the morning, allowing the blood pressure to regulate before he/she gets up to a full standing position. Medications that could help this condition include fludrocortisone, ProAmatine and Yohimbine (www.neuro.jhmi.edu/hopkinspdmd/symptoms/hypotension.htm, 18 November 2006).

Viagra has proven to be effective in aiding males to obtain and sustain an erection (www.neuro.jhmi.edu/hopkinspdmd/symptoms/sexual.htm, 18 November 2006).

There are few patients with PD that seek advice and treatment from a speech therapist throughout the course of the disease. The speech therapist addresses problems such as slurring, drooling and unintelligible speech. Coping mechanisms are taught to both the patient and carer. If drooling becomes severe, ophthalmic solutions of atropine can be used under the tongue (Schneider, Diamond, and Markham, 1986; www.neuro.jhmi.edu/hopkinspdmd/symptoms/salivation.htm, 18 November 2006).

The administration of functional electrical stimulation (FES) has limited reported findings in Parkinson's disease. The primary observed effect of FES is an improved gait pattern. The secondary effect of an improved gait pattern will be an increase in general functional muscle strength and fitness level. This will assist the patient to overcome other symptoms such as rigidity, stiffness of joints and muscles, constipation, urinary incontinence and general immobility (www.salisburyfes.com).

While this aspect has not yet been clinically tested, it is the focus of this clinical trial. It has been incorporated into the treatment of patients with PD by Mann (2006) and by me in a FES clinic that operates in Cape Town, South Africa.

2.13.2 Surgical intervention

There are three target locations for surgery to address the signs and symptoms of a patient with PD, namely the thalamus, globus pallidum internus and subthalamic

nucleus. Surgery includes a pallidotomy, thaladotomy and DBS. These are all discussed in the following paragraphs. Cell transplants, gene therapy and neurotrophic factor delivery are all still in the experimental stages (<http://.org/library/disease/pd/par-sur.html> , 08 February 2006).

2.13.2.1 Pallidotomy

Pallidotomy was the most common type of surgery performed in PD until the late 1990s. This entails the stimulation of the globus pallidus. The effects of a pallidotomy are almost immediate. Improvements of a pallidotomy range from seventy per cent (70%) to ninety per cent (90%) for dyskinesia and dystonia, and twenty five per cent (25%) to fifty percent (50%) for the tremor, rigidity, bradykinesia and gait. Surgery is usually performed bilaterally. There is an increased risk of cognitive and bulbar effects post surgery, which includes dementia, facial weakness and loss of sight. Adverse effects of the pallidotomy include, hemorrhage in the brain (2%-6%), general muscle weakness (2%-6%), visual field deficits (0%-12%), cognitive confusion (0%-12%), and weight gain (50% -70%) (<http://.org/library/disease/pd/par-sur.html>, 08 February 2006).

2.13.2.2 Thaladotomy

Thaladotomy surgery is effective in the treatment of tremor and entails the stimulation of the thalamus. Bilateral surgery is not indicated, due to the increased risks and complications especially of damage to the visual field

(<http://.org/library/disease/pd/par-sur.html> 08 February 2006).

2.13.2.3 Deep Brain Stimulation (DBS)

DBS entails the electromagnetic stimulation of the internal globus pallidus and the subthalamic nucleus (STN). Stimulation of these structures bilaterally has proven more effective than the unilateral DBS (Maurer, et al, 2003; Yokoyama, Sugiyama, Nishizawa, Yokota, Ohta, and Uemura, 1999). Both uni- and bilateral stimulation alleviate freezing in the “off” phase of medication, but have no effect on freezing in the “on” phase of medication (Stolze, Klebe, and Poepping, 2001). Deep brain stimulation (DBS) of the subthalamic nucleus (STN) has now become a popular treatment option for patients with PD and dyskinestic movements. After a 30-month follow-up of bilateral DBS it was found that there was significant “off” medication improvement in motor function. The outcome of the DBS was monitored by using the UPDRS. An increase of forty per cent (40%) to forty four (44%) in the patient’s UPDRS motor function measured in the “off” medication phase was documented at the thirty (30)-month follow-up. There was no improvement in the “on” medication state. As the follow-up period continued the UPDRS scores worsened from the initial improvement score, but they remained better than the initial scores on the UPDRS, probably due to the natural progression of the disease (Krack, Batir, Van Blercom, Chabardes, Fraix, Ardouin, Koudsie, Limousin, Benazzouz, LeBas, Benabidm, and Pollak, 2003). Thirty (30) months post operative there was still an improvement of seventeen percent (17%) on the activity of daily living (ADL) scale on the UPDRS compared to the initial scores recorded. A lasting improvement of seventy percent (70%) in the dyskinesias was recorded. Freezing was measured using the UPDRS. The freezing decreased and the dosage of leva-dopa was reduced by thirty nine percent (39%) at twelve (12) months

and thirty percent (30%) at thirty (30) months post surgery. It can be concluded that DBS of the STN improves motor function and leads to a lasting decrease in medication (Krause, et al, 2004).

A five-year prospective study of DBS was conducted to determine the long-term effects of that treatment. The results were similar to the above-mentioned study. There was a significant improvement of fifty four percent (54%) in the patient's motor function while they were in the "off" medication state five (5) years after the DBS had been inserted. The ADL's improved by forty nine percent (49%).

The scores on the UPDRS for the motor function during the "on" period did not improve in the first year. Akinesia, speech and freezing was found to worsen between year one (1) and year five (5) when the patients were on their medication. The use of medication for akinesia and freezing was still however decreased at year five (5) post surgery. These results are consistent with the progressive nature of the disease, where one expects the symptoms of PD to worsen as time continues (Krack, et al, 2003). Freezing and balance did not show improvement with DBS intervention.

Other types of surgery include, thalamic DBS, subthalamic DBS and globus pallidus internal DBS. Patients with bilateral DBS show significant improvement on physical activity as part of the quality of life questionnaires, but no improvement on the mental and emotional impairment, including cognition and communication ability (Drapier, Raoul, Drapier, Leray, Lallement, Rivier, Sauleau, Lajat, Edan, and Verin, 2005). Although surgery does have positive outcomes, there are complications that may occur. Major surgical complications include haemorrhaging, resulting in transient

hemiparesis, infection and pulmonary embolisation (Landi, Parolin, Piolti, Antonini, Grimaldi, Crespi, Lurlaro, Aliprandi, Pezzoli, Ferrarese, and Gaini, 2003).

2.13.2.4 Electromagnetic fields

A study by Sandyk (1996) investigated the effect of weak electromagnetic fields (EMF's) on freezing in patients PD. The effect of the EMF's decreased freezing episodes initially, but the episodes gradually re-appeared. It was therefore suggested that EMF's have an effect on the neuro-chemical mechanisms of freezing in patients with PD, probably due to the facilitation of serotonin neurotransmission at both the synaptic and non synaptic neuronal target sites (Sandyk, 1996). However the effect was not lasting.

2.14 Outcome Measures

Evidence based physiotherapy requires the assessment of patients by using recognised, valid and reliable outcomes measures to determine their problems on the levels of impairment, functional activity and participation (WHO, 2001), not only to determine the degree of impairment but also to monitor their progress or regression and the effect of drug intervention. Outcome measures not only assist the physiotherapists in identifying and quantifying the level of the patients impairment and functional ability on activity and participation levels, but it also provides a tool for the multidisciplinary health care team to continually, plan and implement and assess the rehabilitation programme based on evidence. It is of vital importance that the physiotherapist actively involves the patient, the spouse/ family/carer in the decision

making and setting of goals for the outcome of treatment. General guidelines for goal setting include,

- 1) Involve the patient early in the goal setting.
- 2) Respect the patient's preferences.
- 3) Inform the patient of possible anticipated needs.
- 4) Negotiate goals with the patient and re-structure them when necessary.
- 5) Communicate clearly.
- 6) Let the patient consider his social support framework and the impact of the goals on family members (Haas, 1993).

Long-term goals need to consider the etiology and clinical manifestations of the condition. The likely prognosis has to be considered, as well as the patient's personal circumstances (Ward and McIntosh, 1993). The outcomes measures are used to describe the results of the health care interventions expressed in a relative value. If the results are not recorded with numerical values, descriptive scales are used to indicate the change in the patients condition (Ross, 2001). Results of outcome measures are further used to assist the health care team in predicting the service needs of a patient with respect to level of care, hospitalization, work performance and social integration (WHO, 2001). Outcomes measures need to be valid, reliable, and responsive to change (sensitive) to be of any value.

Outcomes measures on impairment level include the assessment of muscle strength, tone and endurance, joint range. Appropriate scales to determine the degree (level) of

impairment are the Oxford muscle grading scale for the assessment of muscle strength, the Modified Ashworth Scale and Australian Spasticity Assessment for the assessment of muscle tone. Sensory functions that need to be assessed include proprioception, discrimination between light touch and pain as well as temperature discrimination (Fuller, 1999).

The Unified Parkinson's Disease rating scale (UPDRS), the Berg Balance Scale, the Functional Reach Test, Gait assessment, are all relevant outcomes measures to determine the patients' problems on the level of functional activity (Bohannon and Smith, 1987; Lang and Fahn, 1989; Berg, Maki, Williams, Holliday, and Wood-Dauphinee, 1992; Duncan, et al, 1990).

(<http://online.unn.ac.uk/facilities/hsw/research/Rehab/Guidelines/assessmentoutcomes.htm>, 22 February 2006).

Relevant outcomes measures for patients with PD on participation level include the Freezing of gait questionnaire, a Falls Diary and the Parkinson's Disease Questionnaire-39 to determine how the patient functions in his immediate surroundings and within the community.

2.15 Summary

In South Africa PD is the second most common neurological disorder and the prevalence is estimated at 163 413 persons. It is a degenerating disease of the basal ganglia which causes the characteristic signs and symptoms of bradykinesia, rigidity

and a resting tremor. Secondary signs and symptoms include communication difficulties, cognitive changes, ANS impairments, muscle weakness and dyskinesia.

There is no consensus of the pathogenesis of PD. The cause of PD is due to a dopamine deficiency in the basal ganglia of the brain. The signs and symptoms occur when the level of dopamine drops abnormally low.

Patients with PD are treated by a multi-disciplinary team. Ongoing assessment of the patient on impairment, functional ability and participation level is necessary to ensure that the physiotherapy intervention addresses the clinical problems of the patient regardless the developmental stage of PD. The physiotherapy intervention that addresses the primary and secondary characteristics of PD on the impairment, functional ability and participation levels according to the ICF are summarised in Tables 2.1 and 2.2. The outcome measures relevant to the impairment, functional activity and participation levels that can be implemented as part of the evidence based physiotherapy practice are summarised in Table 2.3. The medical treatment of patients with PD is also briefly discussed because especially the pharmacological treatment influences the physiotherapy intervention directly.

Further, it is clear from some of the studies discussed in this chapter that the effectiveness of the current methods of intervention tends to wear off or deteriorate as the disease progresses. From the recent literature it is clear that the focus of physiotherapy falls on the use of internal and external cues to assist patients to overcome the bradykinesia/akinesia and tripping and falling during ambulation.

Randomised clinical trials are required to determine the effect of physiotherapy in patients with PD and to validate a model for physiotherapy intervention in PD.

In Chapter 3 a description of the research methodology of a clinical trial to determine the effect of FESS and FES&MS as external cues on the freezing of gait in patients with PD will be provided.

CHAPTER 3

STUDY DESIGN AND METHODOLOGY

3.1 Introduction

From the literature reviewed in Chapter 2 it became clear that freezing/akinesia is a major problem for all patients with PD who suffer from freezing/akinetetic episodes. Pharmacological intervention does help initially with overcoming the freezing episodes but the effectiveness of this treatment soon wears off and freezing becomes a problem that eventually leads to the patient withdrawing from the life of the community. These days most of the symptoms of PD can be corrected with surgical intervention, including pallidotomies and deep brain stimulation. Although these interventions have excellent results on improving tremor and posture, the surgical intervention has no impact on freezing. It is the freezing episodes that patients with PD find most debilitating and it is due to this phenomenon that they withdraw from participation in society, inevitably having to eventually rely on a walking aid or a wheelchair for mobilisation. Freezing also leads to falling and this is one of the greatest fears of patients with PD. Falls are not only costly to the health care providers, but the patients become less mobile, to avoid falling and further withdraw from the community. And after a fall such a patient may then become confined to a wheelchair.

Akinesia/freezing and bradykinesia/hypokinesia are addressed by the rehabilitation team, which includes the physiotherapist, who teach these patients to utilise auditory/cognitive/sensory and/or visual cues to overcome freezing/akinesia and/or bradykinesia/hypokinesia. General cardio-vascular endurance programmes also contribute to improving the general mobility of the patient (Morris, 2005).

From the literature study conducted for this clinical trial that views this problem from a physiotherapy perspective, it is clear that further research is needed to investigate additional possibilities of an external cue, to aid patients with PD to overcome their difficulties with gait. None of the present day solutions have a lasting effect and these patients all continually look for new methods to aid them in overcoming their difficulties with gait that includes the problem of freezing.

In this chapter the research methodology to investigate FESS and FES&MS as two forms of external cueing on akinesia/freezing and bradykinesia/hypokinesia in patients with PD is described.

3.2 Ethical Approval

Ethical approval to conduct this study was granted by the Ethics Committee of the Faculty of Health Sciences at the University of Pretoria (S159/2005).

Permission for me to conduct this study at the University Of Cape Town Private Academic Hospital (UCTPAH) was also granted by the University of Cape Town.

3.3 Aim and objective of this study

3.3.1 Aim of this study

The aim of this study was to determine:

1. The effect of an external cue in the form of FESS and FES&MS in people with PD to aid them in overcoming episodes of freezing/akinesia and bradykinesia/hypokinesia and assist in improving these patients' Physiological Cost Index (PCI), step length, number of steps and time to walk ten (10) meters, turn 180 degrees and walk ten (10) meters back to the starting point.
2. To determine the effect of FESS and FES&MS on the quality of life of the patient with Parkinson's disease.
3. To determine whether there is a difference between FESS and FES&MS on freezing/akinesia and bradykinesia/hypokinesia in patients with PD.

3.3.2 Objective of this study

The objective of this study was to determine whether FESS and FES&MS had an effect on:

1. The following parameters of gait:
 - Time to walk ten (10) meters, turn 180 degrees and walk ten (10) meters back to the starting point.
 - The speed with which patients walk ten (10) meters, turn 180 degrees and walk ten (10) meters back to the starting point.

- The number of steps (half gait cycles) patients give to walk ten (10) meters, turn 180 degrees and walk ten (10) meters back to the starting point.
 - The average step length (distance of a half gait cycle).
2. The time and number of steps patients will take to turn 360 degrees, to the right and to the left
 3. The Physiological Cost Index (PCI) of the patients to walk ten (10) meters, turn 180 degrees and walk ten (10) meters back to the starting point.
 4. The degree of rigidity the patients may have had at the time of this study.
 5. Whether a sensory impairment was present that may have had an effect on the patients' gait
 6. The patients' quality of life as measured on the Parkinson's Disease Questionnaire 39 (PDQ-39)
 7. The motor changes that patients who participated in the clinical trial might have experienced based on the motor part of the PDQ-39 and Parkinson's Disease Rating Scale (PDRS).
 8. The patient's episodes of freezing/akinesia as measured on the Freezing of Gait Scale (FOG).
 9. The number of falls patients experienced before and after they started using the FESS and FES&MS would be compared.
 10. The patient's dependence on a walking aid.

The ROM and the Oxford grading scale were measured at the assessments, but these variables were not used in the conclusion of the clinical trial. They can perhaps add value to clinical trials that will be conducted in the future.

3.3.3 Hypotheses

The hypotheses that were tested in the clinical trial were the following:

Hypothesis 1 (H1)

FESS and FES&MS decrease freezing/akinesia and bradykinesia/hypokinesia during gait in patients suffering from PD.

Null Hypothesis (H0)

FESS and FES&MS do not decrease freezing/akinesia and bradykinesia/hypokinesia during gait in patients suffering from PD.

Hypothesis 2 (H2)

FESS and FES&MS decrease freezing/akinesia and bradykinesia/hypokinesia during gait in patients suffering from PD to such an extent that it improves the quality of life of patients.

Null Hypothesis 2 (H02)

FESS and FES&MS do not decrease freezing/akinesia and bradykinesia/hypokinesia during gait in patients suffering from PD to such an extent that it improves the quality of life of the patients.

Hypothesis 3 (H3)

FESS decreases freezing/akinesia and bradykinesia/hypokinesia during gait in patients suffering from PD more than FES&MS.

Null Hypothesis 3 (H03)

FESS does not decrease freezing/akinesia and bradykinesia/hypokinesia during gait in patients suffering from PD more than FES&MS.

3.4 Research approach

The research approach that was chosen to conduct the research in this study was a single blind randomly selected controlled clinical trial, to determine the effect of FESS and FES&MS on freezing/akinesia and bradykinesia/hypokinesia in patients with PD.

The effect of FESS and FES&MS was tested on two patients who were randomly allocated to two groups.

Each patient knew that he/she was receiving FES input, but they were not aware that there was a difference between FESS and FES&MS. They were informed only of the intervention that they were allocated to. The patients did not have contact with each other and neither did they have access to information to be aware of which other

patients at the clinic was participating in this trial. The participants were requested not to discuss the trial in the support groups until the relevant stimulation was withdrawn.

3.4.1 Study Design

The study design chosen for this research was a two-group comparative interrupted time-series design.

The study design can be illustrated as follows:

Group 1

A1 A2 (Baseline measurement) $\frac{(A1+A2)}{2}$ X1 M1 A3 X2 M2 A4

Group 2

B1 B2 (Baseline measurement) $\frac{(B1+B2)}{2}$ X3 M1 B3 X4 M2 B4

The assessment procedures of groups 1 (A1, A2, A3 and A4) and group 2 (B1, B2, B3, B4) were identical throughout this study.

A1 and B1 were performed at the start of the study at week zero (0).

A2 and B2 were performed at week two (2) of the study.

The average of measurements at A1 and A2 $\frac{(A1+A2)}{2}$ as well as B1 and B2

$\frac{(B1+B2)}{2}$ were calculated and used as the baseline measurement for each

patient individually as well as for group1 and group 2 respectively. Each patient acted as his/her own control.

The intervention of group 1 (X1) at week two (2) entailed the application of FESS on group 1 and (X2) indicates removal of the FESS (X2) at week fourteen (14).

The intervention of group 2 (X3) at week two (2) entailed the application of FESS&MS on group 2 and (X4) indicates the removal of the FESS&MS (X4) at week fourteen (14).

M1 and M2 represent monitoring of the use of the equipment and patient diaries in both group 1 and group 2 at week eight (8) and week eighteen (18).

A4 and B4 represent the final assessments for group 1 and group 2 respectively at week twenty four (24) of the study. At week twenty four (24), patients of both groups had been without any intervention for ten (10) weeks.

Table 3.1 Graphic summary of the study design

Sample group selection			No Treatment (Baseline assessment)		Intervention: Week 2-14			No Intervention: Week 14-24		
Assessment for inclusion in the trial. Decision as to which leg the FES will be administered	Random assignment to group 1 or group 2	A	Week 0	Week 2	FESS	Monitoring week 8	Assessment week 14	No FESS	Monitoring week 18	Assessment week 24
		B	Week 0	Week 2	FES& MS	Monitoring week 8	Assessment week 14	No FES& MS	Monitoring week 18	Assessment week 24

3.4.2 Research Methodology

3.4.2.1 Research setting

The primary study was conducted at the rehabilitation unit of UCT Private Academic Hospitals' (UCTPAH). The research setting is clearly described in Chapter 1, paragraph 1.9.1.

3.4.2.2 Apparatus

The apparatus that was used to apply the FESS and FES&MS was the ODFS. This is described in Chapter 1, paragraph 1.9.2.

3.4.2.3 Outcomes Measures used in this clinical trial

The outcomes measures were chosen to determine the effect of FESS and FES&MS on the impairment gait, functional activity and participation of patients with PD. An improvement of these aspects should result in a change in their quality of life and participation in their community, such as doing shopping, visiting friends, attending activities at church, etcetera. It is therefore important to determine whether the intervention has an effect on all three levels of the International Classification of Function and Disability (ICF) (Melnick in Umphred, 2007).

The outcomes measures chosen for this study include:

1. Impairment level: The range of movement of the lower limbs, the modified Ashworth Scale, somato-sensory impairment and Physiological cost index (PCI).

2. Functional activity level: Gait analysis that included the time it took to walk ten (10) meters, turn 180 degrees and walk the ten (10) meters again (walking activity). Assessment of gait entailed measurement of the following parameters of gait: The time it took the patient to complete the selected walking activity in seconds, the speed to complete the walking activity in meters per second, the number of steps to complete the walking activity, the average step length and the possible difference between the resting heart rate and the heart rate directly after completion of the walking activity. The change in heart rate is necessary to determine the patients PCI, which gives an indication of the effort to perform the walking activity.

The patients were also assessed on the number of steps and time to turn a 360 degree turn on the spot,

3. Assessment on the level of participation level included: The Unified Parkinson's Disease Rating Scale (UPDRS), the Mini mental assessment scale (MMES), Parkinson's Disease Quality of Life scale (PDQ 39) and the Freezing of gait scale (FOG).

The scales are discussed in detail:

1. The Mini-Mental State Examination (MMSE) is an internationally recognized standardised and validated rating scale utilised to determine whether the patient had cognitive impairment to the extent that it would exclude the patient for this clinical trial, as they would not be able to comply with the assessment procedures. The reliability and validity of the clinical tests of the MMSE was

tested and documented by Folstein, et al, (1975). This test includes memory, attention and use of language, with a score of 29 being the maximum score, indicating no cognitive difficulties, and a score of 0 indicating marked cognitive fall out (see Appendix 1a and 1b).

2. Motor examination of the Unified Parkinson's Disease Rating Scale (UPDRS) is also an internationally recognised, validated and reliable rating scale that is used as a tool to assess the functional level of patients with PD. It measures the various facets of impairment that is affected by the course of the disease. This scale includes a comprehensive assessment of the motor skills of PD. It has good clinometric properties that include good reliability and validity. It includes sections on mental behavior, and mood, activities of daily living (ADL) and motor activity. A total score of 199 points is possible, where 199 points represents total disability and 0 represents no disability. In this study, only the motor assessment and scoring aspects were used in the assessment of these patients. The maximum score for the motor part of the test is 56 and the minimum score is 0. The higher the score the more severely the individual is affected by PD. (see Addendum 1a and 1b). The motor aspect of the UPDRS was used in most of the literature that was assessed for this clinical trial, as described in Chapter 2.
3. Freezing of Gait Scale. This is an internationally recognised and validated scale which was used to determine the extent and severity of the akinesia/ freezing episodes of the patient on a functional activity level. A maximum score of 24

indicates severe freezing episodes and a minimum of 0 indicates no problem with freezing (Lamberti, et al, 1997) (see Addendum 4).

4. The Parkinson's Disease Questionnaire (PDQ 39). This is an internationally recognised and validated questionnaire, used to determine the quality of life of the patient living with the disease. This questionnaire is shown to be valuable in studies that aim at determining the impact of treatment regimes in Parkinson's disease (Jenkinson, et al, 1997). The maximum score is 100 and the minimum score is 0. The higher the score the more PD has an impact on the quality of life of the patient living with PD. (see Addendum 3a and 3b).
5. The gait analysis used in this clinical trial was based on the parameters of gait that is used in the clinics where the ODFS was developed in Salisbury, UK. Mann et al (2006). This is called the FES Clinical Assessment (see Addendum 5). The assessment of the specific parameters of gait was used to determine whether the patient was more affected on the one side of the body than the other. The ODFS was administered to the more affected side. The gait assessment included:
 - Timed ten (10) meter, a 180 degree turn and ten (10) meter walk back to the start.
 - Speed to complete the walking activity in m/s
 - The average step length during the walking activity was measured from the video that was taken; this is described in paragraph 3.4.4.
 - The number of steps taken during the walking activity

- The resting heart rate of the patient was taken before and again on completion of the walking activity, to determine the change in heart rate.
6. General Observations were made of the patient's cadence, use of a walking aid and special note was taken of the main problem that the patient experienced during gait.
 7. The Range of Movement (ROM) of the lower limbs was measured by using a goniometer (see Addendum 6). Shortening of any muscle and/or soft tissue was noted.
 8. The Modified Ashworth Scale for grading spasticity was used to determine abnormally increased tone in the lower limbs. This is an internationally validated scale to grade increased tone in the upper and the lower limbs on a scale from zero to five. Zero being no spasticity (increased tone) and five being maximal spasticity (increased tone). (see Addendum 7).
 9. The Oxford grading scale is an internationally recognized scale for the grading of muscle power, with zero being no muscle power and five normal muscle power. The strength of all muscles in the lower limbs was graded according the Oxford grading scale (see Addendum 8).
 10. The discrimination of sensation of the patient, with regards to light touch, and pain (pin prick), was determined. This was rated as normal (2), impaired (1) and absent (0) according to the ASIA scale and documented on the body chart. (see Addendum 9).

11. Patients in both groups were asked to keep a diary to document the number of falls they experienced before the intervention of the FESS and FES&MS, during the use of FESS and FES&MS, and after the intervention with the FESS and FES&MS was removed (on week 14 of the clinical trial). In the diary the date, time, place and whether the ODFS was switched 'on' or 'off' when each fall happened were documented (see Addendum 10).

At the end of the trial patients were asked to write down their own observations, subjective feelings and experiences while using the ODFS. This data would then be coded, analysed and interpreted in conjunction with the PDQ 39 to answer the research question: Did FESS and FES&MS have an effect on the quality of life of the patient living with Parkinson's disease?

3.4.3 Study Population

The study population included all patients who were diagnosed by a neurologist as having PD and who experienced problems with freezing/akinesia and/or bradykinesia/hypokinesia with problems with the initiation of gait or at any point during the gait cycle.

3.4.3.1 Sample group

All patients with PD who experienced, freezing episodes/akinesia and bradykinesia/hypokinesia in the Western Cape, South Africa, who met the inclusion

and exclusion criteria of this study, were considered for participation in this sample group.

3.4.3.2 Recruitment of patients

I approached neurologists, neurosurgeons and physiotherapists with neuro-rehabilitation practices in the Western Cape area and gave each of them a protocol in which the inclusion and exclusion criteria were explained. They were requested to refer patients with PD that were suitable for participation in the study to me. I made a follow-up telephone call and faxed a letter to all the medical specialists two months after I had approached them, to remind them of this study and of the need that they identify patients for this study.

The Parkinson's Association of SA and all support groups for patients living with Parkinson's disease in the Western Cape were contacted to ensure that all people with PD were aware of this study. This action gave each patient the opportunity to decide voluntarily whether to participate in this study.

After a patient with PD was referred by either the medical practitioner or their support group, or they had contacted me of their own initiative, a time was set to conduct an initial assessment, to determine whether the patient was a suitable candidate for the study based on the afore-mentioned criteria.

During the recruitment process the potential participants were informed what this study entailed and their role and responsibility within the study would be. Upon deciding to participate in this study, each patient was required to give voluntary

written consent in the presence of a witness, who co-signed the voluntary consent form.

(i) Inclusion Criteria

Patients whose PD condition complied with the following criteria were included in this study:

1. Idiopathic PD, that is responsive to medication. (Patients with non idiopathic PD will be accepted if they fit all the other criteria).
2. Patients PD condition must be stable on current medication.
3. Patients must experience difficulty with their gait (reduced stride length, cadence, and decreased heel strike).
4. Patients must experience occurrence of tripping or falling during gait.
5. Patients must experience akinesia (freezing) during walking.
6. Patients must be able to walk 100 meters with or without a walking aid.
7. Patients must be able to comply with assessment procedures.
8. Patients must be able to give informed consent.
9. Patients must be eligible for treatment as an out-patient at the UCTPAH.

(ii) Exclusion Criteria

Patients whose PD condition revealed the following criteria were excluded from this study:

1. Patients with any other neurological condition in addition to the PD.
2. Patients with any cardio-vascular condition that affects endurance during gait.

3. Patients who have a cardiac pace maker.
4. Patients with any other musculo-skeletal condition that affects gait.
5. Patients with any cognitive or psychological aspects that would affect the ability to comply with the study protocol.
6. Patients who are currently participating in any other clinical trial to determine the effect of medication on the signs and symptoms of PD.

Each patient knew that he/she was receiving functional electrical stimulation (FES), but they were not made aware that there is a difference between FESS and FES&MS. They were informed only of the intervention relevant to the group that they were allocated to. The patients did not have social contact with each other and neither did they have access to information that revealed who else was participating in the trial. They were requested not to discuss the trial at support groups until the relevant stimulation was withdrawn.

3.4.3.3 Randomization of the sample group

As the patients who met the inclusion criteria were identified and accepted to participate in this study, their details were entered into an excel programme to form a data base for this study. The excel programme was designed by Mr. M.J. Uys on Microsoft Excel 2003, specifically and only for this clinical trial, to randomly allocate patients to either Group 1 or Group 2. The patients were also allocated an individual number. Those patients in Group 1 received FESS, while those patients in Group 2 received FES&MS.

The end result of the selection process was that two groups of eleven (11) patients (n=11) were randomly selected and divided into Group 1 and Group 2. One (1) patient from each group did not complete the trial, due to personal circumstances. The patient from Group 1 joined a pharmacological study that was to be conducted in George, in the Western Cape in South Africa; this therefore excluded him from further participation in this trial. The patient from Group 2 became ill and was admitted to hospital for a prolonged period of time and on discharge, he was weak and tired and did not see his way open to continue with the clinical trial. So ultimately the study population included only the data of ten (10) patients within each group. While developing the protocol for this study during discussions with the statistician it was calculated that there had to be minimum twenty (20) patients in each group of this study to effect a ninety percent (90%) power to reflect a difference in mean time of 1.500, on the assumption that the common two standard deviation is 1.250 when using a two group t-test with a 0.050 two-sided significance level.

However, after accepting the twenty two (22) patients who participated in this clinical trial, no more patients who met the inclusion criteria could be found in that part of the country. Through subsequent discussion with numerous neurologists and neurosurgeons in the area it became evident that there were a number of pharmacological clinical trials being conducted on patients with PD at that time, which meant that most of the patients with PD were already participating on one or other trial in and around South Africa (SA). When a patient is actively involved in a clinical trial he/she may not participate simultaneously in another trial, as their

response to the other trial could influence the effect of their response to this trial.

This factor excluded a large percentage of the patients I intended to have participate in this clinical trial and this is the reason the size of the study sample group was adjusted to only eleven (11) patients being allocated to each of the two groups. As already stated one (1) patient from each group later withdrew from the study.

3.4.3.4 Control Group

Based on the study design of this clinical trial each patient was his/her own control. A baseline measurement on all the outcomes measures used in this trial for each patient was obtained by evaluating each patient at week zero (0) and again at week two (2), and then calculating the average between the measurements of each outcomes measure. The effect of the relevant intervention on each individual patient in each group was determined by calculating the difference between the baseline measurements and the measurements taken at week fourteen (14) (i.e. after twelve (12) weeks of intervention with either FESS or FES&MS). The average change of the patients as a group was calculated at week fourteen (14) and compared with the baseline measurements. At week fourteen (14), after the assessment had been performed with the FESS or the FES&MS being active, the intervention in both groups was removed. At week twenty four (24) (i.e. ten 10 weeks after the removal of the intervention) another assessment of each patients' performance was made using the same outcomes measures and compared with the two measurements recorded at baseline and week fourteen (14).

3.4.4 Data gathering procedure

The data gathering procedure has been implied in the previous paragraphs and will be summarised below.

After the patients made contact with me (based on the information they obtained from their support groups) or were referred to me by a medical professional and/or physiotherapist, and it had been determined that they complied with all the inclusion and the exclusion criteria, I sent them a letter, wherein I explained the aims and objectives of the trial and their role in the trial. This explanation of the trial included a description of the apparatus that was to be used and an explanation of how they would be expected to manage and care for the ODFS for the duration of the study. The MMES was conducted prior to the clinical trial to ensure that the patient did comply with all the inclusion criteria and, especially, to ensure that they were cognitively able to comply with all requirements during the course of this trial.

Each potential patient had to give his/her written informed consent before they were finally accepted into the study. This consent form also made provision for each patient to give consent for a video to be taken of them during the assessment sessions. The video would show the patients face but would only be viewed by the primary researcher and her colleague, who was confirming measurements from the video for inter-rater reliability purposes (see Addendum 11).

Each patient was required to keep a 'falls diary', to be updated on a daily basis. This was to monitor any falls that the patient had, where they occurred, what the patients were busy doing when it occurred, whether the ODFS was switched on or not at the

time of the fall and during which week of the trial the fall occurred. The falls diaries were entered as part of the research documentation on each patient.

Upon arrival at the testing venue, each patient was fitted with a Polar heart rate monitor strap around the chest area that was to be used later in the assessment.

At each assessment session all the outcomes measures (UPDRS, FOG and PDQ-39) were first completed (i.e. week zero (0), week two (2), week fourteen (14) and week twenty four (24)). Each patient in turn sat at a table and the questions were read to them while they were reading their own copy of the questions. Patient responses were recorded immediately and each patient was not permitted to view the responses offered by the patients that were assessed before them, to ensure that each individual's response was not influenced by previous responses. The UPDRS was first dealt with, followed by the FOG and then the PDQ-39.

The patients were then assessed according to the Modified Ashworth Scale. The range of movement of the lower limbs was measured using a goniometer; starting at the hips, then the knees and then the feet. Their sensation was tested using both light touch and a pin prick on the lower limbs and, finally, the muscle strength of the lower limbs was measured in accordance with the Oxford muscle grading system.

The patients were then given fifteen (15) minutes to rest. During this time they had to sit quietly and relax.

The resting heart rate of the patients was then taken using a Polar heart rate monitor. As suggested in the user manual, the strap of the monitor was fastened around the patient's chest and the wrist monitor was put on the right wrist of the

patient upon arrival at the assessment venue. I took the reading after the fifteen (15) minute rest period and this was recorded as the resting heart rate.

The fifteen (15) minute rest period was done with the patient sitting in a chair one (1) meter from the start of the walking activity. This was to ensure that no effort was made to get to the starting point, as that may then have had an effect on the difference between the resting heart rate and the heart rate after the walking activity. After the rest period, the patient had to complete the ten (10) meter walk, 180 degree turn and walk back ten (10) meters to the starting point, and walk back to the chair that was one (1) meter from the start line (There were no obstacles in the course). Immediately after completing the walking activity the heart rate was taken on the heart rate monitor and the reading was recorded. The change in heart rate was then determined. The patient then had to sit down on the chair until their heart rate returned to the resting heart rate level. The patient started the walking activity one (1) meter from the start and had to continue walking for one meter on completion of the distance. The time taken and all the other measurements were recorded from the moment of crossing the start line to the crossing of the finish line, and excluded the one (1) meter from the chair to the start/finish line.

This protocol for the walking activity was performed four times; at week zero (0), at week two (2), at week fourteen (14) and at week twenty four (24). The average was determined for the parameters of gait (time, speed, average step length, number of steps, resting heart rate and the change in heart rate) at each assessment. The average was calculated on the FES clinical assessment Excel programme.

At week fourteen (14) the walking activity was conducted with the relevant stimuli (FESS or FES&MS) in working. The baseline measurement and the assessment at week twenty four (24) was conducted with no stimulation.

The falls diary was assessed and discussed at week fourteen (14) and again at week twenty four (24).

The time to walk the distance of ten (10) meters, turn 180 degrees and walk back was measured using a stop watch. The reading was rounded off to the nearest split second. The time was, for all three 'walking sessions', later confirmed by the second physiotherapist from the video that was taken.

The speed was calculated on the Excel programme, by dividing the distance by the time to complete the walking activity.

The number of steps that were taken was counted as the activity was being performed. This was later confirmed by the video, where the number of steps each patient had taken was counted again.

As the patients' stride length differed quite a bit between strides, I measured the step length rather than the stride length. The average step length was measured from the video that was taken. This was done using ten (10) centimeter markings along the skirting board on the floor of the assessment area. On the video, the step length was measured against the markings in the background. It was ensured that these markings could not be seen by the patient during the walking activity, as this may then have assisted in encouraging them to consciously improve their gait. This aspect was also then checked by the second physiotherapist.

Then each patient was required to first turn 360 degrees to the right and then 360 degrees to the left. The 360 degree turn was to be performed on the spot - no walking was involved. This task was recorded on video and was also completed three times, at each assessment session, to obtain the average number of steps as well as the time in seconds that they took to complete the 360 degree turn. This too was then checked by the second physiotherapist from the video of the assessment session.

The patients in both Group 1 and Group 2 used the stimulator through the course of the whole day; however the device was only switched on each time the patient walked. The stimulator was switched off while they were sitting or lying down and not used at night or while driving. The patient was asked to wear a closed shoe when using the ODFS to ensure good contact with foot switch.

Each ODFS stimulator that was issued to the patients at week two (2) of the clinical trial was issued together with a new battery. I tested the working condition of each battery at the start of week eight (8), using a battery tester. The same battery tester was used each time.

PALS 3.2 cm round adhesive, external electrodes were used to deliver the current to the skin surface and a pack of four new electrodes was issued to each patient at the start of week two (2). The patients received clear instructions to alternate using these electrodes from day to day, i.e. they had to use the first pair of electrodes on day one and the second pair of electrodes on day two, the first pair of electrodes on day three, and so on. The electrodes were clearly marked with a "1" and a "2". I

examined and revised the care of the electrodes at week eight (8) and made sure that they were still in good working condition. If the adhesive substance had worn off or the electrodes were torn, new electrodes were issued to that patient. Stimulation of the ODFS is triggered by a foot switch that is placed under the heel of the foot on the more affected side of the patient, on an inner sole inside the shoe. Each foot switch was checked to ensure it was still in correct working order at each assessment session.

The active electrode (black) was placed over the common peroneal nerve, at the point where it divides posterior to the head of the fibula into the deep and superficial peroneal nerves. The inactive electrode (red) was placed over the muscle body of the tibialis anterior muscle. The stimulation of the common peroneal nerve activated the tibialis anterior for dorsi-flexion and the peronei to add the eversion component in the FES&MS intervention (see Chapter 1, paragraph 1.9.2, diagram 1). The cables from the electrodes and the foot switch were held in place with a light fitting stocking (Tubigrip) that was placed around the patient's lower leg under his/her trousers. They were then plugged into the ODFS machine. The machine was worn either on the patients waist - belt or in the pocket of the patient's trousers or skirt. The stimulation current of the ODFS was activated with heel rise of the foot under which the foot switch is placed and de-activated on heel strike of the same limb.

The assessment conducted at week eight (8) was simply to determine whether the patient was still using the FESS or the FES&MS correctly and according to the protocol. The patient was requested to demonstrate to me their use of the ODFS and

I was then able to determine if they were administering the ODFS and the desired output correctly. At the same time, the battery was tested by using a battery tester supplied by Proffessa Health Services in Johannesburg, South Africa. This was to ensure that the battery still had a good power output. If a battery was running low, a new one was issued to that patient.

I examined all four (4) electrodes to ensure that they were still in good working condition (to ensure they still had sufficient adhesive gel and that they did not have dirt or hair etcetera stuck to the adhesive gel). If the electrodes looked worn, or were not in good working condition, new electrodes were issued to the patient. The foot switch was also tested to ensure that it was still in good working order. This was done manually, using the indicator lights on the ODFS machine.

The skin condition was also assessed to ensure that there was no burning of the skin from the electrodes or any other skin reaction or irritation.

The patients were also required to bring their falls diary to this assessment. The falls diary was read and any entries were discussed, to ensure that the information written by the patient was true and stated correctly.

It was also confirmed at this assessment that there had been no change in the medication dose or timing of the medication.

At week eighteen (18) the falls diary was again monitored. It was also ensured that the patient was able to walk safely without the input of the ODFS. It was also confirmed at this assessment that there had been no change in the medication dose or timing of the medication.

3.4.4.1 Setting of the ODFS

At week two (2) the ODFS was issued to the patients. They were instructed how to fit the ODFS and how to get the desired stimulation (FESS for Group 1 and FES&MS for Group 2). In Group 1 a sensory input with no stimulation of a muscle contraction from the common peroneal nerve stimulation was required. The intensity was to be kept at the level that gives a good sensory stimulus but no muscle contraction had to be observed by the physiotherapist or experienced by the patient. The increase in the intensity was stopped just before the muscle contraction was elicited. This was achieved by getting a slight dorsi-flexion (the first muscle contraction to be elicited from the stimulation of the common peroneal nerve) movement and then turning the intensity down very slightly, that no muscle contraction could be observed.

The patients in Group 2 received FES&MS by means of the ODFS, to the common peroneal nerve of the more affected lower limb. The placement of the electrodes for both the groups was exactly the same. The intensity of the stimulation in Group 2 was set to give a sensory stimulus as well as a motor stimulus of dorsi-flexion, with eversion following the stimulation of the common peroneal nerve.

The patients went home and had to use the desired stimulation for a period of twelve weeks. The use of the ODFS was monitored at the follow up assessments at week eight (8). At week fourteen (14) the ODFS was removed in both groups. At week eighteen (18) all the patients were monitored and at week twenty four (24) they were all re-assessed. At the week fourteen (14) assessment the ODFS was returned to

the investigator and the patient went home to continue on the clinical trial, but with no stimulation from the ODFS for a period of 10 weeks.

A period of twelve (12) weeks of intervention (from week 2 to week 14) was chosen to allow adequate time for central nervous system changes due to plasticity that might have taken place.

Table 3.2: Summary of the assessments done during the clinical trial

Tests/ Outcomes measures	Week 0	Week 2	Week 8	Week 14 (With FESS and FES&MS)	Week 18 (without)	Week 24 (without FESS and FES&MS)
UPDRS	X	X		X		X
Freezing of Gait	X	X		X		X
PDQ-39	X	X		X		X
FES clinical examination of gait parameters (*) during walking activity	X	X		X		X
ROM	X	X		X		X
Modified Ashworth scale	X	X		X		X
Oxford Muscle strength	X	X		X		X
Sensation: somato- sensory & proprioception	X	X		X		X

(*) Time, speed, number of steps, average step length, resting heart rate, change in heart rate, PCI to complete the walking activity

3.5 Validity and reliability of the clinical trial

- a. The patients who participated in this trial were aware that they received an active intervention during the trial because they had to set the intensity of the ODFS on a daily basis to the criteria either of FESS or FES&MS. They were however unaware that there was

another form of stimulation given except for the form of stimulation than they were instructed to use.

- b. The primary investigator (researcher) was aware which patient was in which group, as she issued the ODFS to the patients and instructed them on how to obtain the desired stimulus. I personally conducted all the assessments.
- c. Video recordings of all the assessments were made to optimise the objectivity (internal validity and reliability) of the results of subjective observations and comparison of the patient's scores on the tests at each date of assessment. These videos were co-evaluated by another physiotherapist not involved in the trial, to ensure that the observations by the researcher were objective.
- d. I also conducted the selection process of all the patients who participated in this clinical trial, based on the pre-determined inclusion and exclusion criteria. The allocation of patients to Group 1 or Group 2 was done by a computer programme and was not dependant on my decision or preference.
- e. The conditions of all the assessments were kept constant for each patient and for each assessment session, i.e. the conditions for the 20 meter walk together with the 180 degree turn were identical for each patient, at each assessment session. The same stop watch was used, as well as the same heart rate monitor and the same

goniometer. The questions in all the questionnaires were read word for word off the paper with no extra explanations given.

- f. Random sampling of the patients was conducted by a computer programme.
- g. All the outcomes measures used for this study are internationally recognised and validated and this ensured internal validity of the data capturing process and the data obtained. The results of this study can therefore be compared to similar studies on national and international level that have implemented the same outcome measures.
- h. The patients were asked not to change their medication dose or the time for taking their medication for the duration of the trial, unless prescribed by their medical doctor.
- i. Because medication plays a large role in the performance of a patient living with PD, patients had continue taking their medication as usual and all assessments for that patient were carried out at the same time of day / the same number of hours after taking their last dose of medication.

3.6 Data-analysis

I captured all the data, which was entered onto a data spreadsheet in an excel programme. This was sent via E-mail to the statistician for analysis. The data analysis is discussed in depth in Chapter 4.

3.7 Conclusion

In conclusion, Chapter 3 describes the research methodology used in this clinical trial. All the patients who were included in the clinical trial underwent a twenty four (24) week trial period. They were assessed on internationally recognized assessment scales. Monitoring of the patients was conducted at week eight (8) and again at week eighteen (18). All data gathered was sent to a statistician for analysis.

At this point I would like to thank all the patients who participated in this clinical trial for their time and input. Each patient was given the option to purchase an ODFS for their own use after completion of this clinical trial, to be able to continue using the FESS or FES&MS. The alternate option of FESS or FES&MS was also described to the patients and they were told that they would have access to results of the clinical trial to aid them in their decision making.

In Chapter 4 a detailed account of the analysis of the data gathered in the manner described in Chapter 3 will be discussed.

CHAPTER 4

RESULTS OF THE STUDY

4.1 Introduction

The main aim of this study was to determine the effect of FESS and FES&MS with an Odstock Dropped Foot Stimulator (ODFS) on the akinetic gait of patients living with Parkinson's disease. The clinical trial was planned to gather data pertaining to participating patients' gait according to and based on the principles of the ICF, namely the participation, activity and impairment levels of dysfunction (WHO, 2001). In evaluating the level of functional activity an assessment of various parameters of gait and turning around on the spot was conducted. The assessment of gait required the patients to walk a distance of ten (10) meters, turn 180 degrees and walk ten (10) meters back to the starting point. In the second functional activity, patients were asked to turn 360 degrees on the spot that they were standing on.

Assessment on participation level entailed the implementation of the UPDRS, QoL questionnaire and the PDQ-39, to determine how PD affects each patient's life.

On the impairment level of disability the patients muscle tone was assessed according to the Modified Ashworth Scale, their muscle strength was assessed according to the Oxford grading of muscle strength and the possible somato-sensory fall-out was tested according to the ASIA scale. Due to the close interaction and homeostasis of the neuromusculo-skeletal and the cardio-vascular-respiratory

systems when walking, the patients change in heart rate and the physiological cost index (PCI) was determined, to get an indication of the patients' cardio-vascular endurance and the effort of walking.

In this chapter the biographical data of the patients who participated in this clinical trial, as well as the results of the outcomes measures at the pre determined times are described.

4.2 Biographical data of the patients in the clinical trial

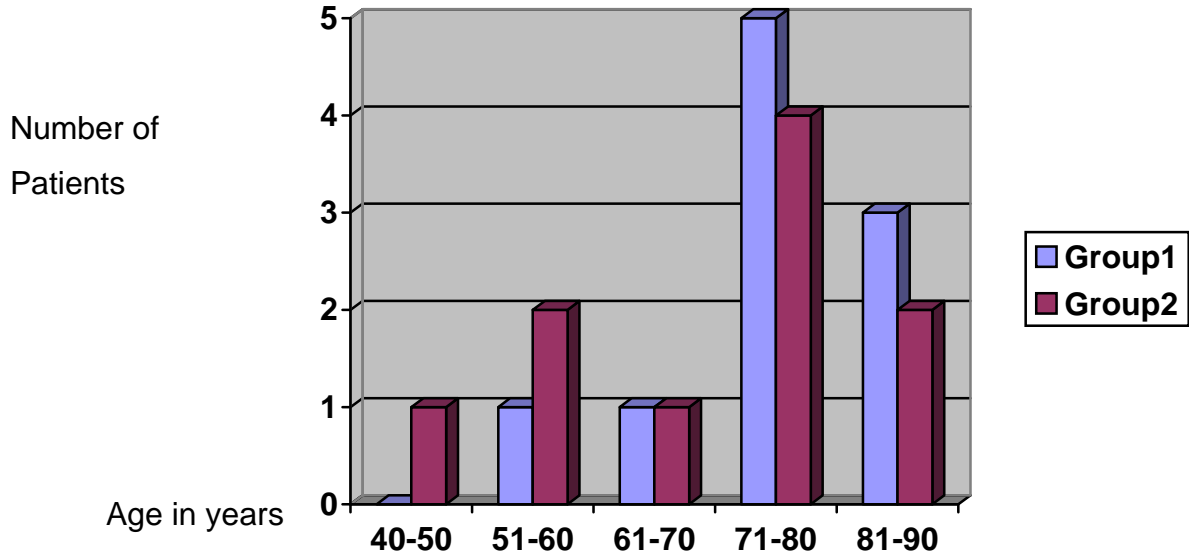
In this clinical trial seven (7) of the patients in Group 1 live in semi-urban regions in the Western Cape, while the remaining three (3) patients lived in urban areas. In Group 2, five (5) patients lived in semi urban areas, while the remaining five (5) patients lived in urban areas of the Western Cape.

All these patients were screened by means of the MMES before being allocated to one of the two groups. In Group 1 the lowest score was eighteen (18) and the highest twenty nine (29), while in Group 2 the lowest score was twenty two (22) and the highest twenty nine (29) - out of a possible maximum score of twenty nine (29).

Although the score of one specific patient in Group 1 was particularly low, he understood what was required of him and was able to manage the ODFS and participate in the clinical trial.

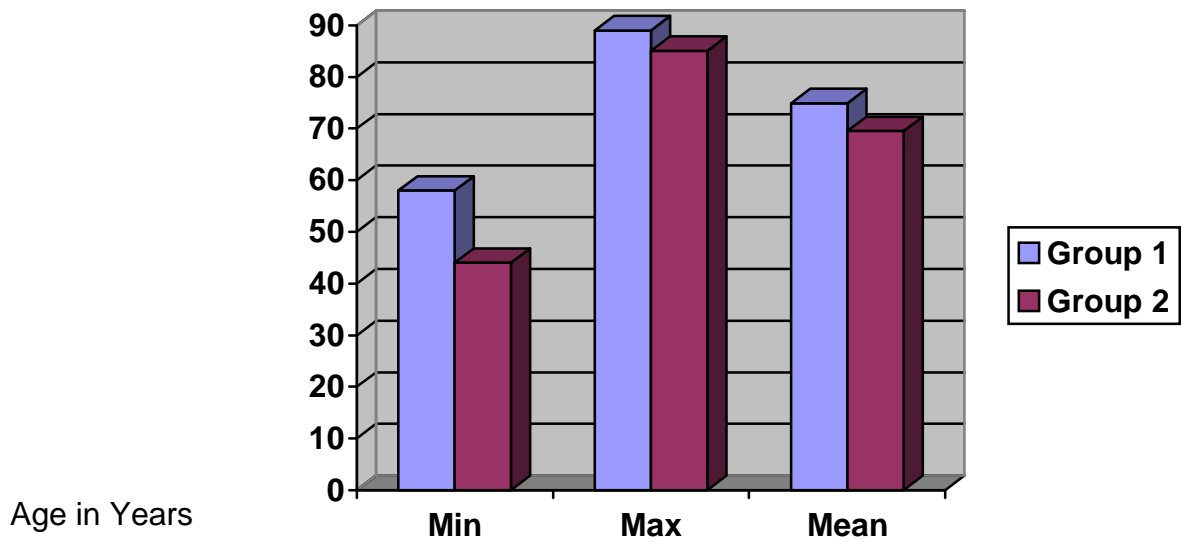
4.2.1 Age of the patients

The age distribution of the patients in Group 1 and Group 2 are illustrated in Graph 4.1



Graph 4.1: Age distribution in years

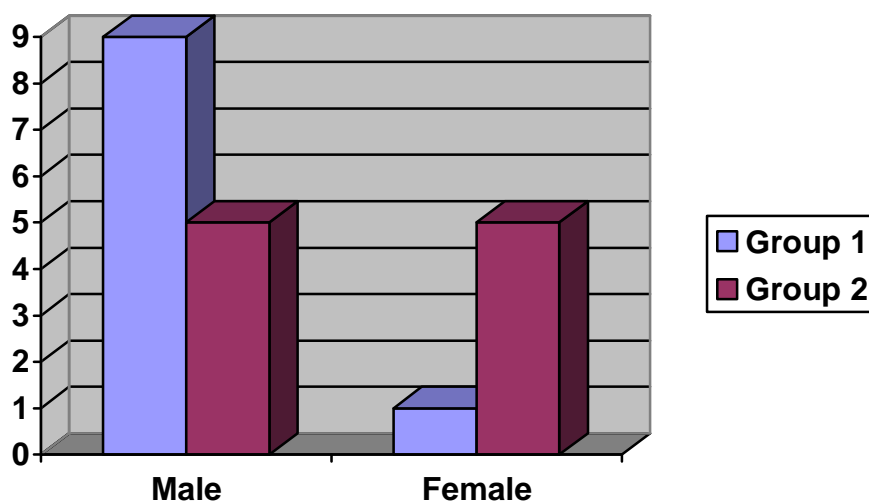
The maximum and minimum ages of the patients within each group are given in years and as illustrated in Graph 4.2.



Graph 4.2: Max, Min and Mean ages in years

The two groups had an equal number of patients assigned to them and the age distribution was fairly evenly distributed, even after the random division of the patients into the two groups. The age difference of the youngest patient and the oldest patient in each group was: Group 1, 58 years and 89 years, and Group 2, 44 years and 85 years. The mean ages of Group 1 and Group 2 were 74.9 years and 69.5 years respectively. It was therefore expected that the level of physical ability in the two groups would be similar. The effect of PD on the patient's functional ability would depend on the severity of the signs and symptoms and the time since the onset and diagnosis of PD.

4.2.2 Gender of patients who participated in this clinical trial.



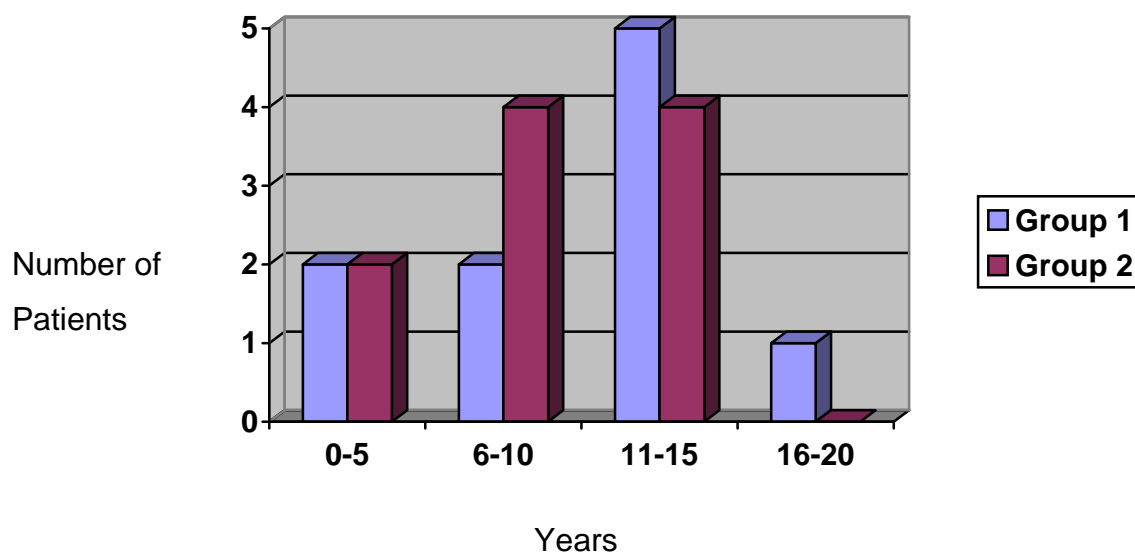
Graph 4.3: Gender of patients who participated in this clinical trial

Following the random allocation of patients into the two groups, Group 2 was allocated a higher number of female patients than Group 1. All research conducted

thus far did not report a difference in onset in the age of PD in these females and males. The diagnostic signs and symptoms, as well as common reactions to pharmacological and physical interventions for PD, were also similar for both genders. More males appear to be affected by PD than females, but there is no evidence to show that the signs and symptoms of PD are different in males and females. In this trial, of the total sample group of twenty (20) patients there were fourteen (14) male and six (6) female participants.

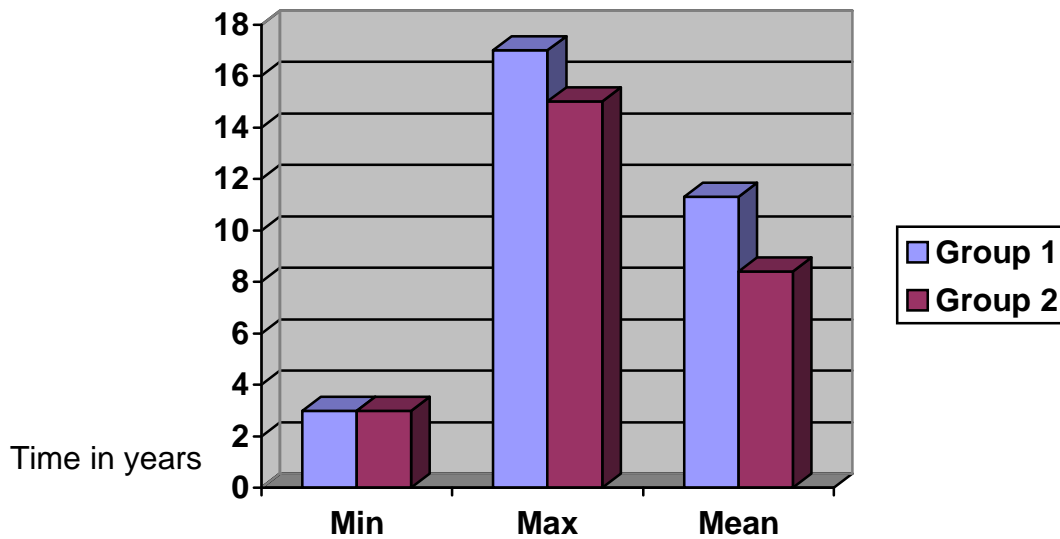
4.2.3 Time period since these patients were diagnosed with Parkinson’s disease (in years)

The time period since these patients were diagnosed with PD is given in Graph 4.4.



Graph 4.4: Time since diagnosis of PD in years

The minimum, maximum and mean years that indicate the duration that patients had been diagnosed with PD is indicated in Graph 4.5



Graph 4.5: Minimum, Maximum and Mean time in years since patients had been diagnosed with PD

This study revealed a great difference in the number of years since these patients were diagnosed with PD, which within Group 1 ranged from three (3) years to seventeen (17) years, and within Group 2, ranged from three (3) years and fifteen (15) years.

The equal distribution of the time since PD was diagnosed in these patients is not likely to result in false negative or false positive results, because these patient's functional ability is assessed on different levels, using a variety of assessment procedures.

4.3 Results from the empirical measurement

After the patients were accepted to participate in this trial, the following was assessed and documented:

- The time since they last took the medication

- Their resting heart rate
- General observations were made regarding the patients
 - posture
 - the use of a walking aid
 - the number of falls they experienced prior to the trial

The questionnaires were completed and the Ashworth Scale for spasticity, sensory testing and the grading of muscle power was performed.

The following parameters of gait while walking were assessed:

- time to complete the distance
- the speed to complete the distance
- the number of steps
- the average step length
- resting heart rate
- change in heart rate
- PCI
- time and number of steps taken to turn 360 degrees on the spot

The walking activity consisted of walking ten (10) metres, turning 180 degrees and returning to the starting point. Patients started walking one (1) metre before the starting point, to ensure that the parameters of gait that were being assessed were optimally performed over the pre-determined distance, which included the 180 degree turn.

The results at baseline (the average of the results of the measurements at week zero (0) and week two (2)), compared to the assessment at week fourteen (14) (with FESS and FES&MS), and week twenty four (24) (without FESS and FES&MS) are discussed comparatively in the following paragraphs and tables to indicate the effect of FESS and FES&MS on the parameters as they were measured during the clinical trial.

4.3.1 Statistical analysis used throughout the study

A comparison of the differences between the two groups was made at baseline with the Mann-Whitney U-test (M-W test), to determine whether there was a difference between the groups before the intervention was administered. After that the non parametric ANCOVA using ranks was used for comparisons for change between the two groups over time.

4.3.2 Walking aid

Patients with PD often depend on a walking aid, not only to cope with a decrease in mobility, but also to assist them in preventing falls. Certain patients use the walking aid as an external cue to aid them to overcome episodes of freezing/akinesia and bradykinesia/hypokinesia. At week zero (0) and at week two (2) of the trial, ten (10) patients used a walking aid on a permanent basis as an aid to assist them to walk as functionally as possible. In Group 1, seven (7) of the patients used a walking aid. Two (2) used a walking frame to initiate gait and then held it in the air as they walked. They also used the frame to assist them to stop walking by putting it down and picked it up again to initiate walking. One (1) of the patients used two walking

sticks, one in each hand, with which he followed a four (4) point gait pattern (I.e. the stick in the left hand, right foot forward, the stick in the right hand and then left foot forward). This particular patient's caregiver reported that on a very good day, he could manage with one walking stick, but he felt safer while using both. Four (4) other patients used a walking stick in their dominant hand. Only one (1) patient put a lot of weight on the walking stick on the right, which was due to a scoliosis to the right (his dominant side).

In Group 2, three (3) of the patients used a walking aid. One (1) was completely dependent on the aid of a walking frame, one (1) used two walking sticks, one in each hand, and the third patient used a single walking stick in the dominant hand. Two (2) patients in each group also had a wheelchair, which they used for long distances, as their gait was too slow to cover the longer distances, i.e. during their visits to UCTPAH and to go to a shopping centre. They did not, however, use the wheelchair in their home environment.

4.3.3 Time to complete the walking activity

Table 4.1: The comparison of times taken for the walking activity (measured in seconds)

Week	Group 1 (N=10) Seconds	Group 2 (N=10) Seconds	P-Value*		
			Between groups	Within group1	Within group 2
Baseline	111.743 (84.19481)	63.1145 (74.28019)	0.0588 (#)		
Week 14	33.439 (13.59857)	41.328 (41.1825)			
Week 24	83.334 (57.96066)	62.955 (80.19312)			
Change from baseline to week 14	78.304 (76.0117)	21.7865 (66.89272)	0.0257 (*)	0.0051 (*)	0.0593 (*)
Change from baseline to week 24	28.409 (63.0979)	0.1595011 (14.70724)	0.5452	0.3863	0.7213
Change from 14 to 24	-49.895 (47.06844)	-21.627 (78.43593)	0.0098 (*)	0.0051 (*)	0.2411

- Values indicated between brackets show the standard deviation

- p-values from ANCOVA for ranks between groups and from Wilcoxon's matched pairs signed ranks test for within groups
- (*) Statistical significant difference in the time in seconds to walk the distance of 20 metres.
- (#)P-value from the M-W test compares between groups at baseline

A positive result indicates an improvement in time (i.e. it took the patients a shorter time in seconds to complete the walking activity). A negative result indicates that the time in seconds to walk the distance increased (i.e. it took the patients longer to complete the walking activity).

In Table 4.1 the actual average time (in seconds) for the patients in Group 1 and Group 2 to complete the walking activity at week fourteen (14) and week twenty four (24) is illustrated, together with the change in time compared to the baseline measurement. It was found that there was a difference in the average time it took the patients in Group 1 to complete the walking activity, when compared to the patients in Group 2. Group 1 completed the activity 48.6 seconds (almost one (1) minute) slower than Group 2.

The standard deviation (SD) in both Group 1 and 2 was high, 84.19481 seconds and 74.28019 seconds respectively.

The M-W test ($p= 0.0588$) shows a marginally significant difference in the baseline time it took patients in Group 1 and Group 2 to complete the walking activity before implementing the intervention programme.

After patients in Group 1 received the intervention with FESS, their average time decreased significantly ($p=0.0051$).

Patients in Group 2 decreased their average time marginally more significantly ($p=0.0593$). The change in time in Group 1 (using FESS) was significantly better (shorter) than in Group 2 (using FES&MS) ($p= 0.0257$). The SD in both groups also decreased in comparison with the baseline measurement; giving readings of 13.59857 for Group 1 and 41.1825 for Group 2.

Although the time taken to perform the walking activity increased in both groups from week fourteen (14) to week twenty four (24), the patients in Group 1 still walked an average of half a minute faster than their baseline time, while the patients in Group 2 returned to their baseline time. The SD in both groups decreased.

The increase in the time it took for the patients in both groups to perform the same activity after the stimuli was removed at week 14 was statistically significant ($p=0.0051$) for Group 1 but not for Group 2 ($p= 0.2411$). Although Group 1's time during the walking activity increased significantly more than that of Group 2, they still walked an average of half a minute faster than their baseline time, which was not the case with Group 2.

The results, ten (10) weeks after the stimuli in both groups was removed (week twenty four) indicate that the effect of both the FESS and FES&MS wears off with the passage of time.

It would however appear that no carry over effect of either of the two forms of stimulation has taken place after it has been removed.

4.3.4 Comparison of the speed with which patients performed the walking activity

The speed with which the patients walked was calculated by dividing the time measured in seconds to complete the walking activity by the distance walked, and is expressed in metres per second (m/sec).

Table 4.2: The comparison of speed over 20 metres measured in metres per second

Week	Group 1 (N=10) m/sec	Group 2 (N=10) m/sec	P-Value*		
			Between groups	Within group1	Within group 2
Baseline	0.312 (0.1769683)	0.637 (0.3662588)	0.0694 (#)		
Week 14	0.809 (0.519689)	0.74 (0.3724692)			
Week 24	0.344 (0.1545028)	0.663 (0.4204772)			
Change from baseline to week 14	-0.497 (0.5362431)	-1.03 (0.1271962)	0.0315 (*)	0.0093 (*)	0.0367 (*)
Change from baseline to week 24	-0.032 (0.1098787)	-0.026 (0.0859845)	0.8296	0.3860	0.3860
Change from week 14 to week 24	-0.465 (0.519689)	-0.097 (0.3724692)	0.0170 (*)	0.0093(*)	0.1394

- Values between brackets indicate the standard deviation

- p-values from ANCOVA for ranks between groups and from Wilcoxon's matched pairs signed ranks test for within groups
- (*) Statistical significant difference in the speed measured in m/s to walk the distance of 20 metres with a 180-degree turn.
- A negative result indicates that there has been an increase in the speed when compared to the baseline speed to perform the walking activity.
- (#)P-value from the M-W test compares between groups at baseline

In Table 4.2 the change in the speed (expressed in m/sec) of the two groups to complete the walking activity is illustrated.

Although the baseline speed with which Group 1 and Group 2 walked differed, the difference is marginally significant (M-W 0.0694). The speed at which both groups performed the walking activity at week fourteen (14) (with the FESS and FES&MS switched on), increased significantly ($p=0.0093$) in both Group 1 and in Group 2 ($p=0.0369$). The change in the speed from baseline to week fourteen (14) in Group 1 was significantly faster than the change in speed that was measured in Group 2 ($p=0.0315$).

After recording the measurements at week fourteen (14), the FESS and FES&MS were removed and it was found that the walking speed of all these patients decreased again. In Group 1 the decrease in the walking speed was significant ($p=0.0093$). At week twenty four (24) there was no significant change in the walking speed compared to baseline ($p=0.3860$)

In Group 2 the change in walking speed between week fourteen (14) and week twenty four (24) was also not significantly different ($p=0.1394$). The change in

patients walking speed at week twenty four (24) compared to baseline values was also not significant ($p=0.3860$). The change in speed from week fourteen (14) to week twenty four (24) in Group 1 was significantly bigger ($p=0.0170$) than in Group 2, which indicates that the walking speed of the patients in group 1 decreased more than that of the patients in Group 2. The decrease in the walking speed observed at week fourteen (14) in Group 1 and in Group 2 with FESS and FES&MS was not sustained at week twenty four (24) in either group.

These results indicate that there is a significant improvement in the walking speed with the relevant stimulus in both groups, but it is not sustained when the FESS and the FES&MS were removed.

4.3.5 Number of steps taken during the walking activity

Table 4.3: Number of steps taken over 20 metres

Week	Group 1 (N=10)	Group 2 (N=10)	P-Value*		
			Between groups	Within group1	Within group 2
Baseline	78.919 (36.02249)	74.0785 (52.42286)	0.4057 (#)		
Week 14	45.966 (14.54497)	56.643 (36.62384)			
Week 24	78.34 (32.82084)	75.519 (72.70484)			
Change from baseline to week 14	32.953 (29.32383)	17.4355 (34.35716)	0.1463	0.0051 (*)	0.1688
Change from baseline to week 24	0.5789993 (9.824968)	-1.440501 (39.44443)	0.6536	0.7989	0.7989
Change from week 14 to week 24	-32.374 (26.74177)	-18.876 (67.47562)	0.0366(*)	0.0050(*)	0.3329

- Values between brackets indicate the standard deviation

- p-values from ANCOVA for ranks between groups and from Wilcoxon's matched pairs signed ranks test for within groups
- (*) Statistical significant difference in the number of steps to perform the walking activity.
- A negative result indicates that there has been an increase in the number of steps when compared to the baseline number of steps
- (#)P-value from the M-W test compares between groups at baseline

Table 4.3 illustrates that the average baseline number of steps taken to complete the walking activity by the patients in Group 1 (78.919) is not significantly different from the number of steps taken by the patients in Group 2 (74.0785) (M-W test $p=0.4057$). The number of steps taken by the patients in Group 1 and Group 2 after 14 weeks (of receiving the relevant stimuli) was statistically significant in Group 1 ($p= 0.0051$), but not for Group 2 ($p=0.1688$), when compared with the baseline figure. The decrease in the number of steps to perform the walking activity after the fourteen (14) weeks of intervention is higher in Group 1 than in Group 2, but is not statistically more significant ($p=0.1463$) than in Group 2.

The difference in the change (decrease) in the number of steps by Group 1 at week fourteen (14) is not significantly different from the decrease in the number of steps in Group 2.

However, after the stimuli was removed at week fourteen (14), there was a statistically significant difference in the increase in the number of steps between Group 1 and Group 2 ($p=0.0366$). The number of steps taken by the patients in

Group 1 increased significantly from week fourteen (14) to week twenty four (24) ($p=0.0050$), but this was not so in Group 2.

There is a non-significant difference between the number of steps to perform the walking activity at week twenty four (24), when compared to the number of steps measured at baseline, which for Group 1 was $p=0.7989$ and for Group 2 was $p=0.7989$.

The improvement (decrease in the number of steps) that was measured at week fourteen (14) was not sustained at week twenty four (24), after the external cue was removed.

4.3.6 Average step length (in millimetres) to complete the walking activity.

As each patient participated in the walking activity, a video recording of their efforts was made. The step length recorded for each patient was later calculated by taking the sum of the measurements of the step lengths as observed from the video; the total distance was then divided by the number of steps taken by the particular patient to complete the walking activity. The resulting figure was then recorded as each patient's individual step length.

Table 4.4: Average Step length over 20 metres (measured in millimetres)

Week	Group 1 (N=10) (mm)	Group 2 (N=10) (mm)	P-Value*		
			Between groups	Within group1	Within group 2
Baseline	332.79 (168.8869)	374.405 (178.4335)	0.7055 (#)		
Week 14	472.78 (138.6825)	437.28 (167.2676)			
Week 24	304.38 (138.9115)	393.85 (186.3784)			
Change from baseline to week 14	-139.99 (98.50434)	-62.875 (132.8711)	0.1463	0.0051(*)	0.1688
Change from baseline to week 24	28.41 (79.72665)	-19.445 (74.7814)	0.3855	0.5076	0.7213
Change from week 14 to week 24	168.4 (93.6497)	43.43 (102.6319)	0.0258(*)	0.0051(*)	0.3329

- Values between brackets indicate the standard deviation
- p-values from ANCOVA for ranks between groups and from Wilcoxon's matched pairs signed ranks test for within groups

- (*) Statistical significant difference in the average step length in mm to walk the distance of 20 metres.
- A negative result indicates that there has been an increase in the step length when compared to the time before, to walk the 20 metres with a 180-degree turn.
- (#)P-value from the M-W test compares between groups at baseline

Although the step length differed in Group 1 and Group 2 at the baseline measurement, the difference is not statistically significant (M-W= 0.7055). The SD in both groups is high (168.8869 and 178.4335, respectively).

The improvement in the average step length increased with statistical significance in Group 1 ($p=0.0051$), but not in Group 2 ($p=0.1688$) at week fourteen (14). The difference in the improvement of the step length between the two groups from baseline to week fourteen is not statistically significant ($p=0.1463$).

After the FESS and the FES&MS were removed at week fourteen (14), the average step length decreased in both Group 1 and Group 2. In Group 1, the decrease in the average step length was statistically significant ($p= 0.0051$) at week fourteen (14), but at week twenty four (24) there was no statistical significant change in the average step length when compared to baseline measurement ($p=0.5076$). And these patients' step length returned almost to the baseline measurement by week twenty four (24).

In Group 2 the change in the average step length between week fourteen (14) and week twenty four (24) was not statistically significant ($p=0.3329$). There was also not

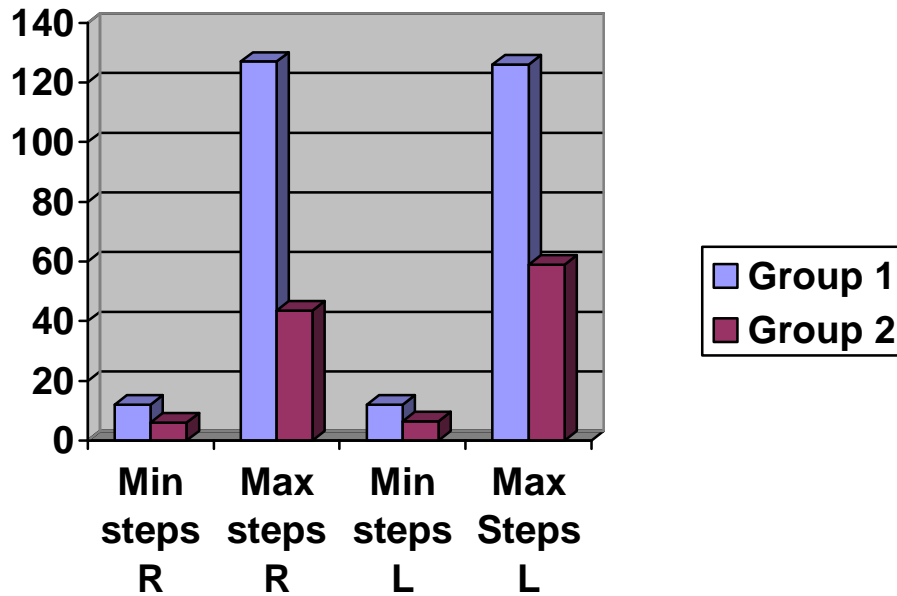
a statistical change in the average step length between baseline and week twenty four (24) ($p=0.7213$). The change in the average step length with FESS and FES&MS at week fourteen (14) was better in Group 1 than in Group 2, but not with statistically indicated significance ($p=0.1463$).

The difference in the average step length from week fourteen (14) to week twenty four (24) was statistically bigger in Group 1 than in Group 2 ($p=0.0258$). This means that the average step length decreased more in Group 1 than in Group 2. The increase in the average step length at week fourteen (14) in Group 1 and in Group 2 was not sustained at week twenty four (24).

4.3.7 Performing a 360-degree turn.

Freezing/akinesia and bradykinesia/hypokinesia of gait occur regularly while the patient with PD is turning. Many falls also occur while turning. In this clinical trial the patients were requested to turn 360 degrees on one spot. With each patient the time taken to turn and the number of steps involved in this movement were recorded.

The parameters observed during turning on one spot of the two groups also show large differences as illustrated in Table 4.5, Table 4.6, Table 4.7 and Table 4.8. In both groups a large difference between the minimum and maximum number of steps involved in completing the 360-degree turn (to the left and to the right) at the baseline measurement. This is better portrayed in Graph 4.6, than in table format.



Graph 4.6: Comparison of minimum and maximum number of steps in turning to the left and to the right at baseline

It can be seen that there is a large difference between the minimum and maximum number of steps to the right in Group 1 (12 and 127) and the same applies in Group 2 (6 and 43.5). The same is evident for number of steps to the left in Group 1 (6 and 126) and for Group 2 (6.5 and 59).

4.3.8 Number of steps to turn 360-degrees to the right

Table 4.5 Turning: Number of steps to the right

Week	Group 1 (N=10)	Group 2 (N=10)	P-Value*		
			Between groups	Within group1	Within group 2
Baseline	39.3 (34.80757)	19.9 (15.82157)	0.0754 (#)		
Week 14	14.3 (9.900056)	13.2 (11.19325)			
Week 24	30.3 (34.87454)	17.8 (15.62619)			
Change from baseline to week 14	25 (26.59783)	6.7 (11.89818)	0.1549	0.0051(*)	0.1820
Change from baseline to week 24	9 (17.63204)	2.1 (10.54567)	0.7554	0.2613	0.4112
Change from week 14 to week 24	-16 (25.14845)	-4.6 (8.617811)	0.1927	0.0093(*)	0.1658

- Values between brackets indicate the standard deviation

- p-values from ANCOVA for ranks between groups and from Wilcoxon's matched pairs signed ranks test for within groups
- (*) Statistical significant difference in the number of steps to turn 360-degrees to the right.
- A negative result indicates that there has been an increase in the number of steps to turn to the right
- (#)P-value from the M-W test compares between groups at baseline

Table 4.5 illustrates that the number of steps taken to turn 360-degrees to the right differs largely between the patients in Group 1 and in Group 2 at baseline. This difference is marginally significant according to the M-W test ($p=0.0754$). (There is also a larger SD in Group 1 (34.80757) than in Group 2 (15.82157).

The number of steps to turn to the right decreased significantly in Group 1 ($p=0.0051$) but not in Group 2 ($p=0.1820$).

After the FESS and the FES&MS were removed at week fourteen (14) (after the measurements had been recorded) the number of steps to turn to the right increased again in both groups. In Group 1 the increase in the number of steps from week fourteen (14) to week twenty four (24) was statistically significant ($p=0.0093$). There was no statistical change from baseline to week twenty four (24) ($p=0.2613$).

In Group 2 the increase in the number of steps to the right between week fourteen (14) and week twenty four (24) was not statistically significant ($p=0.1658$). The change from baseline to week twenty four (24) was also not statistically significant ($p=0.4112$). There was no statistically significant difference in the number of steps to turn to the right between Group 1 and Group 2 at week fourteen (14) or at week twenty four (24) ($p=0.1927$ and $p=0.7554$ respectively). The decrease in the number

of steps to turn to the right with FESS and FES&MS was not sustained in Group 1 or in Group 2 at week twenty four (24), although the number of steps taken to turn to the right was still less than that at the baseline measurement.

4.3.9 Turning: number of steps to turn 360-degrees to the left

Table 4.6 Turning, number of steps to the left

Week	Group 1 (N=10)	Group 2 (N=10)	P-Value*		
			Between groups	Within group1	Within group 2
Baseline	44.45 (37.5355)	19.485 (16.53723)	0.0411 (#)		
Week 14	14.8 (9.437514)	17.7 (20.11108)			
Week 24	33.4 (34.92913)	15.3 (8.615877)			
Change from baseline to week 14	29.65 (31.91312)	1.785 (19.43282)	0.3458	0.0125(*)	0.2613
Change from baseline to week 24	11.05 (21.18497)	4.185 (11.53656)	0.4162	0.2014	0.9593
Change from week 14 to week 24	-18.6 (27.04811)	2.4 (14.97554)	0.4393	0.0058(*)	0.7592

- Values between brackets indicate the standard deviation

- p-values from ANCOVA for ranks between groups and from Wilcoxon's matched pairs signed ranks test for within groups
- (*) Statistical significant difference in the number of steps to turn 360-degrees to the left
- A negative result indicates that there has been an increase in the number of steps to turn to the left.
- (#)P-value from the M-W test compares between groups at baseline

Table 4.6 illustrates that there is a statistically significant difference in the number of steps taken by the two groups to turn to the left at the baseline measurement to perform the walking activity according to the M-W test ($p=0.0411$).

When compared to the baseline measurement, the number of steps that the patients in Group 1 and in Group 2 took after fourteen (14) weeks with FESS and FES&MS decreased significantly in Group 1 ($p= 0.0125$), but not in Group 2($p= 0.2613$).

Although the decrease in the number of steps is higher in Group 1 than in Group 2, this difference between the two groups is not statistically significant ($p= 0.3458$).

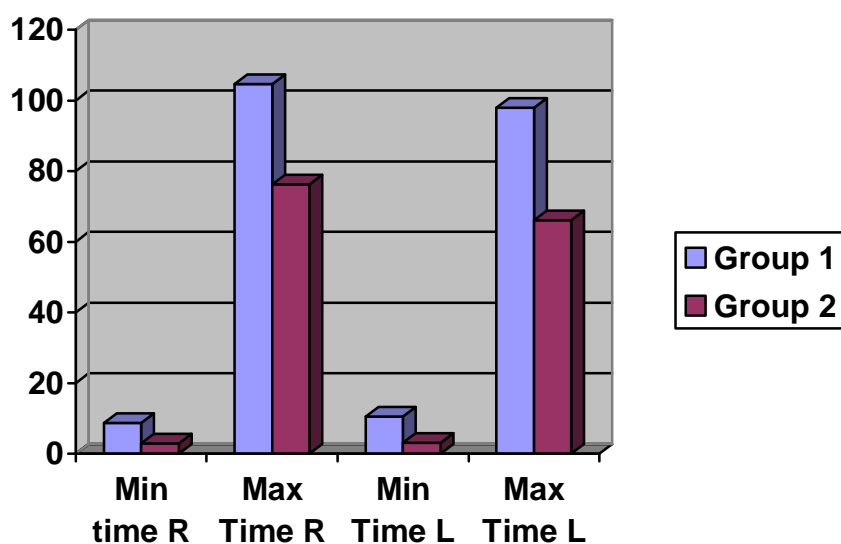
After the stimulation was removed at week fourteen (14) (after the measurements had been recorded), by week twenty four (24) the number of steps once again increased with statistical significance in Group 1 ($p=0.0058$), but not in Group 2 ($p= 0.7592$).

There was also no statistical significant difference between Group 1 and Group 2 in the change (increase) in the number of steps to turn to the left as measured at week

fourteen (14) and at week twenty four (24). It would seem that some carry over effect occurs with the FES&MS on the number of steps to turn to the left.

4.3.10 Time taken to turn to 360-degrees to the left and to the right

Each patient was requested to turn 360-degrees on the spot, to the left and to the right. The time taken to perform each turn was measured to determine the effect of freezing/akinesia and bradykinesia/hypokinesia on the time to turn 360-degrees in the patients with PD in this clinical trial. The time was measured in seconds from the start of the turn until a full 360-degree turn had been completed.



Graph 4.7: Comparison of the min and max times for turning to the left and to the right for both groups at the baseline measurement

From Graph 4.7 it can be seen that, when considering the 360 degree turn to the right, there is a large difference in minimum and the maximum times in Group 1 (8.75s and 104.61s) and in Group 2 (2.95s and 76.27s). When considering the 360

degree turn to the left, this is also true in Group 1 (10.525s and 98s) and Group 2 (3s and 66.07s).

Table 4.7 Time to turn 360-degrees to the right in seconds

Week	Group 1 (N=10) (s)	Group 2 (N=10) (s)	P-Value*		
			Between groups	Within group1	Within group 2
Baseline	41.687 (36.55575)	23.3195 (28.56364)	0.0963 (#)		
Week 14	12.228 (13.43276)	9.67 (12.25308)			
Week 24	35.043 (60.95188)	18.29 (24.67984)			
Change from baseline to week 14	29.459 (30.37893)	13.6495 (22.75641)	0.6042	0.0051(*)	0.0926
Change from baseline to week 24	6.644 (44.85633)	5.0295 (17.68959)	0.6786	0.2026	0.3863
Change from week 14 to week 24	-22.815 (47.98866)	-8.62 (13.80747)	0.6928	0.0069(*)	0.0745

- Values between brackets indicate the standard deviation
- p-values from ANCOVA for ranks between groups and from Wilcoxon's matched pairs signed ranks test for within groups

- (*) Statistical significant difference in the time in seconds to turn 360-degrees to the right.
- A negative result indicates that there has been an increase in the time to turn the 360-degrees since the previous assessment
- (#)P-value from the M-W test compares between groups at baseline

In Table 4.7 the marginal significant difference in the number of steps taken by the patients to turn to the right at the baseline measurement and to perform the walking activity according to the M-W test ($p=0.0963$) is illustrated. The time it took the patients in Group 1 to turn 360-degrees to the right was considerably slower than that of Group 2 (41.687 and 23.3195 seconds respectively).

The decrease in the time that the patients in Group 1 and Group 2 took to turn 360-degrees at week fourteen (14) with FESS and FES&MS was statistically significant; $p= 0.0051$ for Group 1 and $p=0.0926$ for Group 2.

After the FESS and FES&MS were removed at week fourteen (after the measurements had been recorded), the time it took the patients in Groups 1 and 2 to turn 360 degrees at week twenty four (24) increased, again statistically significant in Group 1 ($p= 0.0069$), and a trend towards statistical significance in Group 2 ($p= 0.0745$).

Although the times at week twenty four (24) were still better than that at baseline, it is clear that the effect of the FESS and FES&MS is not sustained at week twenty four (24) in either group.

4.3.11 Turning: Time in seconds to turn 360-degrees to left.

Table 4.8 Time to turn to left in seconds

Week	Group 1 (N=10) (s)	Group 2 (N=10) (s)	P-Value*		
			Between groups	Within group1	Within group 2
Baseline	40.3625 (33.53)	20.8555 (23.29659)	0.0696 (#)		
Week 14	13.625 (12.51362)	14.345 (24.63677)			
Week 24	41.89 (72.10026)	13.645 (14.84102)			
Change from baseline to week 14	26.7375 (30.10105)	6.5105 (22.29833)	0.4494	0.0166(*)	0.2026
Change from baseline to week 24	-1.527499 (54.05223)	7.2105 (18.61843)	0.9257	0.2845	0.4139
Change from week 14 to week 24	-28.265 (62.83066)	0.7000003 (11.16356)	0.0295(*)	0.0367(*)	0.1688

- Values between brackets indicate the standard deviation
- p-values from ANCOVA for ranks between groups and from Wilcoxon's matched pairs signed ranks test for within groups

- (*) Statistical significant difference in the time in seconds to turn 360-degrees to the left.
- A negative result indicates that there has been an increase in the time to turn the 360-degrees since the previous assessment.
- (#)P-value from the M-W test compares between groups at baseline

There is a marginally significant difference between the two groups, in the number of steps to turn to the left at the baseline measurement, to perform the walking activity according to the M-W test ($p=0.0696$). From Table 4.8 it is clear from the baseline measurement that it took Group 1 twice as long as Group 2 to turn the 360-degrees to the left; 40.3625 and 20.8555 seconds respectively.

The time to turn the 360-degrees to the left with FESS and FES&MS is statistically significant in Group 1 ($p= 0.0166$), but not in Group 2 ($p= 0.2026$) at week fourteen¹⁴.

Although the patients in Group 1 responded better on the stimulus by turning faster to the left than Group 2, the difference between the two groups is not statistically significant ($p= 0.4494$).

After the FESS and FES&MS were removed at week fourteen (14) (after the measurements had been recorded), the time to turn the 360-degrees increased again in Group 1, with statistical significance ($p= 0.0367$). In Group 2 there was

virtually no change in the time it took to turn the 360-degrees to the left and this had no statistical significance ($p=0.1688$).

However there is a statistical significant difference between the changes that took place from week fourteen (14) to week twenty four (24) between Group 1 and Group 2. This indicates that the time taken to turn 360 degrees increased more in Group 1 than in Group 2.

It appears that there was no lasting effect of the FESS on turning to the left in Group 1, in contrast to Group 2, where the measurement at week twenty four (24) was better than that at week fourteen (14), when the time was decreased.

Clinical aim 3, the possible reason/s for the observed differences in effects on freezing/akinesia and bradykinesia/hypokinesia using only a FESS or FES&MS, could be explained by looking at the results of the change in the heart rate and the physiological cost index (PCI) of the patients during the walking activity. The changes in these parameters could explain the effect on the other parameters of gait in the patients who participated in the clinical trial.

4.4 The effect of the walking activity on the heart rate and physiological cost index of gait.

The resting heart rate of each patient was first recorded and as was the change in heart rate after the walking activity, as this would form part of the calculation to determine the PCI or the effort of walking. Any change noted in the effort of walking will indicate an effect of the application of FESS and FES&MS on the parameters of

gait in the patients who were part of the trial. If the PCI decreases, i.e. there is less effort used to complete the walking activity with FESS and FES&MS, this will have a positive effect on the quality of life of the patients as they will then be enabled to walk longer distances before tiring.

The resting heart rate of each patient was recorded at the beginning of each assessment session. As the patients completed the walking activity, their heart rate was again recorded, applying the same heart rate monitor used at the beginning of the walk. With this information and the indicated time it took to walk the 20 metres it was possible to calculate the physiological cost index (PCI), or effort of walking for each patient. The PCI gives an indication of how much work has been done and how tiring the walking activity is for the patient. The formula to calculate the PCI is the change in the patient's heart rate minus the resting heart rate, divided by the speed to walk the 20 metres, divided by 60. A high PCI (above 0.5) indicates a large effort required to walk the distance of 20 metres. A lower PCI (below 0.5) indicates less effort for the walking task.

4.4.1 Resting heart rate in beats per minute

The resting heart rate of each patient was measured on the heart rate monitor at the beginning of each assessment. The recorded figure was then used to calculate the change in heart rate and then the PCI for each patient. The FESS and FES&MS is not expected to have any effect on the resting heart rate of the patients and is therefore not going to be discussed in this chapter or portrayed in a table format. Any

change in the resting heart rate would be due to an increased fitness level of the patient who participated in this clinical trial and not directly related to the use of the FESS or FES&MS.

4.4.2 Comparison of the change in heart rate

A change in heart rate during the walking activity was measured to then calculate the PCI for each patient.

Table 4.9: Change in heart rate recorded in beats per minute after completing the walking activity

Week	Group 1 (N=10) (Beats per minute)	Group 2 (N=10) (Beats per minute)	P-Value*		
			Between groups	Within group1	Within group 2
Baseline	8.281 (3.209475)	11.414 (5.404019)	0.1612 (#)		
Week 14	5.067 (3.523846)	11.799 (5.64563)			
Week 24	7.333 (5.076453)	16.767 (12.67201)			
Change from baseline to week 14	3.214 (2.179552)	-0.3849999 (3.485139)	0.0111(*)	0.0093(*)	0.4446
Change from baseline to week 24	0.948 (3.179147)	-5.353 (14.65502)	0.1066	0.2619	0.3077
Change from week 14 to week 24	-2.266 (2.787389)	-4.968 (13.77493)	0.4816	0.0092(*)	0.1683

- Values between brackets indicate the standard deviation

- p-values from ANCOVA for ranks between groups and from Wilcoxon's matched pairs signed ranks test for within groups
- (*) Statistical significant difference in the time in beats per minute.
- A negative result indicates that there has been an increase in the change in heart rate since the previous assessment
- (#)P-value from the M-W test compares between groups at baseline

In Table 4.9 the change in the heart rate for the two groups after walking the distance of 20 metres and performing the 180-degree turn is illustrated. There is a non significant difference in the change of heart rate at the baseline measurement to perform the walking activity according to the M-W test ($p=0.1612$).

At week fourteen (14) the decrease in the change in the heart rate in Group 1 with FESS and the increase in the change of heart rate in Group 2 with FES&MS was statistically significant in Group 1 ($p= 0.0093$) but not in Group 2 ($p=0.4446$). Further, the indicated change in the heart rate was statistically significantly lower in Group 1 than in Group 2 ($p=0.0111$) at week fourteen (14).

After the FESS and FES&MS were removed at week fourteen (14), following the assessment, the assessment at week twenty (24) showed a statistically significant increase in the change in heart rate in Group 1 ($p= 0.0092$), but not in Group 2 ($p=0.1683$). The change in heart rate at week twenty four (24) was higher than the baseline measurement in Group 2, but with no statistical significance ($p= 0.3077$).

4.4.3 Physiological cost index (PCI) to complete the walking activity

Table 4.10 PCI to walk 20 metres with a 180-degree turn at 10 metres

Week	Group 1 (N=10)	Group 2 (N=10)	P-Value*		
			Between groups	Within group1	Within group 2
Baseline	0.846 (0.766905)	0.547 (0.5414189)	0.2568 (#)		
Week 14	0.1566 (0.1631361)	0.575 (0.8441597)			
Week 24	0.463 (0.3323001)	1.725 (3.557915)			
Change from baseline to week 14	0.6894 (0.7948362)	-0.028 (0.5361447)	0.0130 (*)	0.0051 (*)	0.8785
Change from baseline to week 24	0.383 (0.6414012)	-1.178 (3.294527)	0.1720	0.0926	0.3329
Change from week 14 to week 24	-0.3064 (0.2665013)	-1.15 (3.656443)	0.5491	0.0051 (*)	0.5076

- Values between brackets indicate the standard deviation

- p-values from ANCOVA for ranks between groups and from Wilcoxon's matched pairs signed ranks test for within groups
- (*) Statistical significant difference in the PCI
- A negative result indicates that there has been an increase in the change in PCI since the previous assessment
- (#)P-value from the M-W test compares between groups at baseline

In Table 4.10 it is shown that the PCI in Group 1 (0.846) at the baseline measurement was greater than that of Group 2 (0.547). There is a non significant difference in the change PCI at the baseline measurement according to the M-W test ($p=0.2568$). Both groups had a low SD of 0.766905 and 0.5414189 respectively.

At week fourteen (14) the PCI for Group 1 with FESS was statistically significant lower than at the baseline measurement ($p= 0.00031$). The PCI for Group 2, however, increased slightly with no statistical significance ($p=0.8785$). The difference between the PCI for Group 1 and Group 2 was statistically significantly higher in Group 1 ($p=0.0130$).

After the FESS and FES&MS were removed, the PCI at week twenty four (24) in both the groups increased again with statistical significance in Group 1 ($p= 0.0051$), but not in Group 2 ($p= 0.5076$). The increase in the PCI in Group 2 from week fourteen (14) to week (24) was more than the baseline measurement.

These results indicate that the effect of FESS is not sustained once the stimulus has been withdrawn. While FES&MS does not have any effect on the PCI, the PCI in Group 2 at week twenty four (24) was higher than the baseline measurement.

4.5 Comparison of the participation level between the two groups and within the two groups

On participation level of the ICF, all patients were requested to complete outcomes measures relating to PD and the effect the disease has on their quality of life. These outcomes measures (in the format of questionnaires) are internationally recognised scales and the result is expressed in a numerical value, through which the impact of the disease on the quality of life of the patient with PD can be interpreted. The outcomes measures used in this clinical trial are:

- The Freezing of gait scale (FOG) to determine the severity of freezing on the patients' functional gait;
- The motor score from the Unified Parkinson's Disease Rating Scale (UPDRS); and
- The Parkinson's disease Questionnaire (PDQ 39), to determine the impact on the patients' functional ability and QoL.

4.5.1 The effect of freezing of gait on the quality of life of the patients with PD

The higher the score, the more frequent the freezing episodes occur.

Table 4.11 Freezing of Gait Scale

Week	Group 1 (N=10)	Group 2 (N=10)	P-Value*		
			Between groups	Within group1	Within group 2
Baseline	22.8 (1.032796)	14.8 (5.308274)	0.0013 (#)		
Week 14	17 (2.748737)	10.9 (5.877452)			
Week 24	20.8 (1.549193)	14.9 (5.384133)			
Change from baseline to week 14	5.8 (2.820559)	3.9 (4.931757)	0.9824	0.0057 (*)	0.0158 (*)
Change from baseline to week 24	2 (1.885618)	-0.1 (2.514403)	0.7466	0.0074 (*)	0.6803
Change from week 14 to week 24	-3.8 (1.873796)	-4 (5.120764)	0.0244(*)	0.0055 (*)	0.0215 (*)

- Values between brackets indicate the standard deviation
- p-values from ANCOVA for ranks between groups and from Wilcoxon's matched pairs signed ranks test for within groups

- (*) Statistical significant difference in the FOG scale scores
- A negative result indicates that there has been an increase in the FOG score since the previous assessment
- (#)P-value from the M-W test compares between groups at baseline

At week two (2) (baseline measurement) freezing during gait occurred more frequently in the patients in Group 1 than in Group 2. The difference between the two groups was statistically significant. Patients in Group 1 experienced significantly more freezing episodes during the walking activity than patients in Group 2 ($p=0.0013$ according to the M-W test).

Group 1 had a smaller SD (1.032796) than Group 2 (5.308274) indicating a larger variance within Group 2.

The FOG score decreased statistically significantly at 14-week in Group 1 ($p=0.0057$) and in Group 2 ($p=0.0158$) when the relevant stimuli was switched on. There was no significant difference between the change that took place in Group 1 compared to the change that took place in Group 2 ($p=0.9824$). This indicates that there was a decrease in the Freezing of Gait score during the walking activity at week 14. After the stimuli were removed at week 14, the number of freezing episodes during the walking activity increased in both groups, with statistical significance in both Group 1 ($p=0.0055$) and in Group 2 ($p=0.0215$). The number of freezing episodes at week twenty four (24) returned almost to the number of freezing episodes at the baseline measurement in both groups. The increase in freezing episodes was statistically higher in Group 1 than in Group 2 ($p=0.0244$).

From these results it is clear that FESS and FES&MS decreases the FOG score, indicating fewer freezing episodes during gait. A decrease in the freezing episodes should have a positive effect on the quality of life of the patients as they are able to move around with more ease.

4.5.2 Unified Parkinson's Disease Rating Scale (UPDRS)

Only the part of the UPDRS that measures the motor effect of PD on the lives and quality of life of patients with PD was used in this clinical trial, which was used to measure the effect of FESS and FES&MS on the motor performance of patients with PD in this trial. The higher the score on this scale the more the PD is affecting the patient's life. The maximum score for this part of the Outcomes measure is 56.

Table 4.12 Unified Parkinson's Disease Rating Scale

Week	Group 1 (N=10)	Group 2 (N=10)	P-Value*		
			Between groups	Within group1	Within group 2
Baseline	33.9 (8.491172)	20.7 (6.092801)	0.0025 (#)		
Week 14	31.7 (8.340663)	20.7 (9.369573)			
Week 24	32.8 (8.175845)	22.2 (8.337332)			
Change from baseline to week 14	2.2 (2.859681)	0 (4.496913)	0.2200	0.0575	0.4694
Change from baseline to week 24	1.1 (1.100505)	-1.5 (3.17105)	0.0441 (*)	0.0099 (*)	0.2562
Change from week 14 to week 24	-1.1 (2.424413)	-1.5 (3.17105)	0.3874	0.0980	0.0858

- Values between brackets indicate the standard deviation
- p-values from ANCOVA for ranks between groups and from Wilcoxon's matched pairs signed ranks test for within groups

- (*) Statistical significant difference in the UPDRS
- A negative result indicates that there has been an increase in the change in UPDRS since the previous assessment
- (#)P-value from the M-W test compares between groups at baseline

In Table 4.12 the significant difference (M-W $p=0.0025$) in the UPDRS score between Group 1 and Group 2 at the baseline measurement is illustrated. In both Group 1 and Group 2 the SD (of 8.491172 and 6.09280 respectively) is high.

The change in the outcome of the motor score of the patients in Group 1 at week fourteen (14) was moderately statistically significant ($p= 0.0575$) when compared to the baseline measurement. This indicates that the patients in Group 1 showed an improvement in their motor performance at week fourteen (14). After the FESS in this group was removed at week fourteen (14), these patients' motor performance decreased statistically significantly ($p=0.0099$) between week fourteen (14) to week twenty four (24). However the difference in motor performance did not return to the baseline value at week twenty four (24). The difference between the baseline measurement compared to week twenty four (24) in Group 1 is still statistically different ($p=0.0980$). It appears that at week twenty four (24) some functional carry over effect has taken place in this group.

No change in the motor score (Motor performance) of the patients in Group 2 took place between the baseline measurement and week fourteen (14). From this it can be said that the FES&MS on the patients in Group 2 did not have any effect on their

motor score at week fourteen (14). However after the stimulus was removed their motor performance deteriorated compared to baseline measurement with a tendency towards statistical significance ($p=0.0858$). Clinically this implies that FES&MS had no effect on the motor performance of patients in Group 2 and that deterioration in the patients in this group's motor performance took place when the FES&MS was removed. The deterioration showed a tendency toward statistical significance ($p=0.0858$)

The difference in the change in motor performance between the patients in Group 1, when compared to the change in patients in Group 2 at week fourteen (14) was not statistically significant ($p= 0.2200$) . The change in motor performance reported by the patients in Group 1 at week 24 was however statistically significant higher when compared to the change in patients in Group 2 ($p= 0.0441$).

4.5.3 Parkinson's Disease Questionnaire-39 (PDQ-39)

The PDQ-39 questionnaire gives an indication of how severely the person living with PD is afflicted by this condition. ADL's and relationships are covered in this questionnaire. A change of 3 points in this questionnaire indicates statistical significance in this clinical trial. The higher the score, the more severely the quality of life is affected (Jenkinson, et al, 1997).

Table 4.13 Parkinson's Disease Questionnaire-39

Week	Group 1 (N=10)	Group 2 (N=10)	P-Value*		
			Between groups	Within group1	Within group 2
Baseline	68 (19.91091)	65.4 (16.6012)	0.6759 (#)		
Week 14	59.6 (18.96898)	59.8 (15.4474)			
Week 24	63.7 (20.4018)	63.2 (9.2336)			
Change from baseline to 14	8.4 (3.864367)	5.6 (2.065591)	0.0607(*)	0.0049 (*)	0.0047 (*)
Change from baseline to 24	4.3 (2.869379)	2.2 (1.75119)	0.0501 (*)	0.0058 (*)	0.0063 (*)
Change from 14 to 24	-4.1 (2.078995)	-3.4 (2.1187)	0.2717	0.0057 (*)	0.0054 (*)

- Values between brackets indicate the standard deviation

- p-values from ANCOVA for ranks between groups and from Wilcoxon's matched pairs signed ranks test for within groups
- (*) Statistical significant difference in the PDQ-39 scores
- A negative result indicates that there has been an increase in the change in PDQ-39 score since the previous assessment
- (#)P-value from the M-W test compares between groups at baseline

In Table 4.13 a non significant difference in the PDQ-39 scores in Group 1 and Group 2 (M-W $p=0.6759$) is illustrated at the baseline measurement. The SD in Group 1 (19.91091) and in Group 2 (16.6012) was high.

The PDQ-39 score decreased statistically significantly in Group 1 ($p= 0.0049$) and in Group 2 ($p=0.0047$) at week fourteen (14). Clinically this indicates that the patients experienced an improvement in their quality of life in most aspects when using the FESS and the FES&MS.

The improvement on the PDQ-39 of Group 1 when compared to that of Group 2 tends to be statistically significantly higher ($p=0.0607$).

After the FES and the FES&MS were removed, the PDQ-39 score increased statistically significantly in both Group 1 ($p= 0.0057$) and Group 2 ($p= 0.0054$). This decrease in quality of life measured at week twenty four (24) in both groups did not return to the same degree of quality of life than what was measured at baseline.

4.6 Evaluation of the two groups on impairment level according to the ICF

In Chapter 2 (paragraph 2.14) the topic of muscle tone in PD was discussed. The aim with the assessment of these patients' muscle tone in accordance to the Modified Ashworth Scale before and after the intervention (i.e.: at fourteen (14) weeks and twenty four (24) weeks) was to determine if the effect of the FESS and FES&MS on a patient's gait, which also has an effect on the patients' muscle tone, as they walk more easily.

There is no evidence that patients with PD present a condition of sensory impairment. However, based on the fact that PD is a movement disorder and that sensory impairment has an effect on movement, it was decided to include the testing of these patients' discrimination of a light sensory touch and a painful stimulus.

The impairment level assessment of the two groups of patients who received the specific intervention in this study entailed the assessment of:

- Muscle tone in the lower limbs that was graded according to the Modified Ashworth Scale.
- Skin sensation, specifically the discrimination between painful stimulus (pin-prick) and light-touch according to the ASIA scale.

4.6.1 Assessment according to the Modified Ashworth scale

This scale is used to grade increased tone (spasticity level) in patients' limbs and trunk. The scale is a 6 point grading scale that ranges from 0-5, where 0 indicates no increase in muscle tone and 5 indicates the limbs are rigid in flexion or extension.

Table 4.14 Patients muscle tone according to the Modified Ashworth scale

Week	Group 1 (N=10)	Group 2 (N=10)	P-Value*		
			Between groups	Within group1	Within group 2
Baseline	2.4 (1.577621)	1.6 (1.264911)	0.2176 (#)		
Week 14	1.9 (1.286684)	1.3 (0.9486833)			
Week 24	2.3 (1.494434)	1.5 (1.178511)			
Change from baseline to week 14	0.5 (0.7071068)	0.1 (0.3162278)	0.7422	0.0466 (*)	0.0833
Change from baseline to week 24	0.3 (0.4830459)	0.1 (0.3162278)	0.6496	0.3173	0.3173
Change from week 14 to week 24	-0.4 (0.5163978)	-0.2 (0.421637)	0.5903	0.0455 (*)	0.1573

- Values between brackets indicate the standard deviation

- p-values from ANCOVA for ranks between groups and from Wilcoxon's matched pairs signed ranks test for within groups
- (*) Statistical significant difference in the Ashworth scale scores
- A negative result indicates that there has been an increase in the Ashworth scale since the previous assessment
- (#)P-value from the M-W test compares between groups at baseline

As can be seen from Table 4.14, there was not much change from baseline to week fourteen (14) and week twenty four (24). There is a non significant difference in the Ashworth score at the baseline measurement according to the M-W test ($p=0.2176$).

A slight decrease in muscle tone on the Ashworth scale was seen at week fourteen (14), with statistical significance in Group 1 ($p= 0.0466$). There was an improvement in Group 2, but the change was not statistically significant ($p= 0.0833$). This change in the muscle tone could also have contributed to the improvement in the walking speed, step length and PCI seen in both the groups at week fourteen (14). The reverse is also true, namely, that the improved walking performance due to the external cueing with the FESS and FES&MS may have contributed to the decrease in abnormal muscle tone. Both these groups had an increase in their muscle tone after the intervention had been removed at week fourteen (14). Group 1 showed a statistically significant increase in muscle tone change ($p= 0.0455$), but there was no significant increase in muscle tone in patients in Group 2 ($p= 0.1573$) at week twenty four (24). The increase in muscle tone from week fourteen (14) to week twenty four (24) tends to be significantly ($p=0.0593$) more than in Group 2 during the same period.

4.6.2 Painful stimulus in the form of pin-prick and light-touch measured on the ASIA scale

The somato-sensory changes were recorded on the ASIA scale. This was done for a pin-prick and light-touch. The ASIA scale is usually used to determine the sensory level in a spinal cord injury. It was however used in this clinical trial to determine any sensory fall out that the patients with PD may have in the upper and in the lower limbs. The highest score of 112 indicates that there is no sensory fall out. As the score decreases a gradually increased level of sensory impairment is indicated.

Table 4.15 Pin-prick according the ASIA scale

Week	Group 1 (N=10)	Group 2 (N=10)	P-Value*		
			Between groups	Within group1	Within group 2
Baseline	112 (0)	111.6 (1.264911)	0.3173 (#)		
Week 14	112 (0)	111.6 (1.264911)			
Week 24	112 (0)	111.6 (1.264911)			
Change from baseline to week 14	0	0	0	0	0
Change from baseline to week 24	0	0	0	0	0
Change from week 14 to week 24	0	0	0	0	0

- Values between brackets indicate the standard deviation
- p-values from ANCOVA for ranks between groups and from Wilcoxon's matched pairs signed ranks test for within groups

- (#)P-value from the M-W test compares between groups at baseline

There was no difference detected in the pin prick sensory stimulus in Group 1 or Group 2 at week fourteen (14) or at week twenty four (24). All the patients in Group 1 had normal sensation with regards to pin prick. However, one (1) patient in Group 2 had fall out in one (1) dermatome and this situation did not improve with the application or removal of FES&MS.

4.6.3 Light-touch according to the ASIA scale

Table 4.16 Light-touch according to the ASIA scale

Week	Group 1 (N=10)	Group 2 (N=10)	P-Value*		
			Between groups	Within group1	Within group 2
Baseline	111.9 (0.3162278)	111.6 (1.264911)	0.9422 (#)		
Week 14	111.9 (0.3162278)	111.6 (1.264911)			
Week 24	111.9 (0.3162278)	111.6 (1.264911)			
Change from baseline to week 14	0	0	0	0	0
Change from baseline to week 24	0	0	0	0	0
Change from week 14 to week 24	0	0	0	0	0

- Values between brackets indicate the standard deviation

- p-values from ANCOVA for ranks between groups and from Wilcoxon's matched pairs signed ranks test for within groups
- (#)P-value from the M-W test compares between groups at baseline

There was no difference detected in the light touch sensory stimulus in Group 1 or Group 2 at week fourteen (14) or at week twenty four (24). All the patients in Group 1 had normal sensation with regards to light touch. However, one (1) patient in Group 2 had fall out in one (1) dermatome and this situation did not improve with the application or removal of FES&MS.

4.7 Conclusion

In Chapter 4 the tabulated results of the clinical trial were explained. These results were recorded in accordance with predetermined parameters of gait for a baseline measurement (which was the calculated average of the measurements recorded at week zero (0) and week two (2)), and compared with similar readings recorded at week fourteen (14) and at week twenty four (24) of the trial. The biographical data (which entailed the age of the patients, gender distribution in the two groups and the time since PD was diagnosed) were discussed separately for each group. Although the patients had been allocated to either of the two groups on a random basis, it became evident that, on average, older patients who recorded a slightly longer time since the time of diagnosis of PD were placed in Group 1, while this was not the case for the patients in Group 2. The results of the gait assessment included the incidence of falls, the patient's posture, use of walking aid and the effort of walking calculated by the PCI. The participation level was assessed according to the FOG,

UPDRS and the PDQ-39. The impairment level of the patients was evaluated according to the Modified Ashworth scale and the testing of sensory fall-out.

The findings reported in this chapter will be further discussed in Chapter 5 based on the literature study previously explained.

CHAPTER 5

DISCUSSION AND CONCLUSION OF RESULTS

5.1 Introduction

The result of this study (the process of which is described in Chapter 4), in which FESS and FES&MS was administered to patients with episodes of freezing/akinesia and/or bradykinesia/hypokinesia during gait, is interpreted and discussed in this chapter. This discussion will follow the sequence as set out under the heading The Aims and Objectives of this Study, as recorded in Chapter 1, paragraph 1.7. Each discussion is followed by an indication of the acceptance of either the null or alternative hypothesis stated in Chapter 1, paragraph 1.6 and in Chapter 3, paragraph 3.3.3.

This clinical trial was conducted with the aim of contributing to the effectiveness of physiotherapy management of patients with PD. The administering of the FESS and FES&MS by using the ODFS is used as an external cue to overcome episodes freezing/akinesia and/or bradykinesia/hypokinesia and thereby improve the gait of these patients to a more functional level.

5.2 Comparison between the biographical data of the patients in Group 1 and in Group 2.

From the results described in Chapter 4 it can be concluded that the two groups are not comparable with each other when regarding age and time since onset of PD. In the discussion of the results and measurements within Group 1 and within Group 2, the results of the baseline measurements at week fourteen (14) and week twenty

four (24) of Group 1 is also compared to that of Group 2. There are a number of reasons that could account for the differences in the two groups at the baseline measurement. These may include:

The minimum, maximum and mean age of the patients in Group 1 (74.9 years) was slightly higher than in Group 2 (69.5 years); however, the age distribution of the patients in the two groups are similar as indicated in Graph 4.1 and in Graph 4.2. Despite the fact that patients participating in this study were randomly allocated to either Group 1 or Group 2, there were slight differences in the minimum, maximum and mean ages of the patients in the two groups. However, the effect of the age on the participant's mobility was also minimised in that these patients were selected according to specific criteria that are described in Chapter 3, paragraph 3.4.3.2.

The representation of gender between the two groups was unequal (Graph 4.3). This corresponds with Maera and Hobson's (2000) findings that it would appear that more males are diagnosed with PD than females. No reference could be found in the literature that neither the signs and symptoms nor the treatment of PD differs in males and females patients. It is therefore assumed that there will be no difference in the presentation of freezing/akinesia or hypokinesia/bradykinesia during gait in males and females and that male and female patients will not react differently to FESS and FES&MS. All research conducted this far has not reported a difference in onset in the age of PD in females and males. The diagnostic signs and symptoms as

well as common reactions to pharmacological and physical interventions for PD also appear to be similar in both genders.

The dose of the medication that these patients were on (as well as their “on” and “off” periods) did indicate a difference; however, each time a patient’s performance on the different outcomes measures used in this study was assessed careful note was taken that this was done after the same time-span after taking their medication. Patients were requested that, for the duration of this trial, not to change the dose or the time in the day that they take their medication.

The variations in the minimum, maximum and mean time that patients in both groups had been diagnosed with PD (Graph 4.5), would seem to be small, (the difference in mean age of three (3) years); however, the signs and symptoms of PD may have been more pronounced in Group 1 than in Group 2. This may relate to the fact seven (7) out of ten (10) patients in Group 1 used walking aids, compared to three (3) out of ten (10) patients in Group 2. The difference in the number of years since the onset of PD may also have had an effect on the type and level of external intervention, in the form of ongoing therapy, medication and advice that had been offered to these patients. As has already been stated in Chapter 2, paragraph 2.12, the effects of the medication and of coping strategies usually given during therapy have been reported to wear off over time and patients with PD are constantly looking for a new intervention to help them overcome the debilitating symptoms of PD, especially for episodes of freezing/akinesia (Morris, 2001).

The time since the diagnosis can also have an effect on the patients' perception of his/her quality of life. They will either have learnt coping strategies, or have a decreased quality of life due to various issues of mobility or in the environment. The younger patients may also have had different views on the quality of life due to the progressive worsening of the signs and symptoms and the quality of life. Younger patients may also perceive their quality of life as decreasing or poor when they can no longer continue with certain activities that she/he did not have a problem with previously.

5.2.1 Conclusion on the difference in biographical data of patients in the study

The differences between patients in Group1 and Group 2 with regards to gender, medication and dosage of medication was not expected to have any influence on the outcome of the intervention (FESS and FES&MS) on the dependent variables (Parameters of gait) due to the fact that:

- Gender does not have an effect on the signs and symptoms of PD
- The time-span after these patients had taken their medication and the time of the assessment of these patients and their dosage of medication were kept stable throughout this study.

The fact that:

- The average age of the patients in Group 1 was five (5) years older than the patients in Group 2, and
- The patients in Group 1 had been diagnosed with PD for three (3) years longer than the patients in Group 2,

- Seven (7) patients in Group 1 made use of a walking aid compared to three (3) in Group 2,

indicates that the patients in Group 1 might be slightly more affected in terms of the parameters of gait than the patients in Group 2. Whether this difference is statistically significant or not will be discussed in the following paragraphs.

The first aim of this study was to determine the effect of an external cue in the form of FESS and FES&MS in people with PD in overcoming episodes of freezing/akinesia and bradykinesia/hypokinesia.

The objectives related to this aim are to determine whether FESS and FES&MS had an effect on the following parameters of gait:

- time taken to walk ten (10) meters, turn 180 degrees and walk ten (10) meters back to the starting point.
- the speed with which patients walked ten (10) meters, turned 180 degrees and walked ten (10) meters back to the starting point.
- the number of steps (half gait cycles) patients took to walk ten (10) meters, turn 180 degrees and walk ten (10) meters back to the starting point.
- the average step length (distance of a half gait cycle).
- the time and number of steps patients will take to turn 360 degrees, to the right and to the left
- the patient's episodes of freezing/akinesia as measured on the Freezing of Gait Scale (FOG).

5.3 Comparison of the parameters of gait during the walking activity

The parameters of gait that were measured during the clinical trial, namely: time, speed, number of steps and average step length, will be discussed following Table 5.1.

Table 5.1 tabulates the P-values of all the parameters of gait as well as the Mann-Witney value at baseline. All four (4) parameters of gait are tabulated in one (1) table for the sake of convenient comparison, discussion and conclusion of the effects of FESS and FES&MS on the parameters of gait in this chapter, And these were recorded between baseline and week fourteen (14), as this was the period that FESS (Group 1) and FES&MS (Group 2) was applied to the patients. The comparison between baseline and week twenty four (24) are also tabulated as this will indicate any carry over effect of the FESS or FES&MS after the stimulation has been withdrawn.

Table 5.2 illustrates the p-values of the Freezing of Gait (FOG) scale. This table has been included in this paragraph as FOG has a direct effect on the parameters of gait and this may assist in clarifying some of the results obtained during the walking activity.

Table 5.1: P-Values of the comparison of the parameters of gait to complete the walking activity

Parameter of gait	Week	Comparison of groups (M-W) At baseline	Between Groups (p-value)	Within Group 1 (p-value)	Within Group 2 (p-value)
Time	Baseline to week 14	0.0588	0.0257	0.0051	0.0593
	Baseline to week 24		0.5452	0.3863	0.7213
Speed	Baseline to week 14	0.0694	0.0315	0.0093	0.0367
	Baseline to week 24		0.8296	0.3860	0.3860
Number of steps	Baseline to week 14	0.4057	0.1463	0.0051	0.1688
	Baseline to week 24		0.6536	0.7989	0.7989
Average step length	Baseline to week 14	0.7055	0.1463	0.0051	0.3329
	Baseline to week 24		0.3855	0.5076	0.3329

The patients in Group 1 receiving FESS completed the walking activity faster at week fourteen (14) than compared to the baseline measurement with statistical significance ($p=0.0057$). At week fourteen (14) the speed to complete the walking activity also increased with statistical significance ($p=0.0093$) in Group 1. The patients used fewer steps over the distance and the average step length also increased with statistical significance ($p=0.0051$).

At week twenty four (24) the time to complete the walking activity in Group 1 increased again, but was still less than the baseline time measured. The speed to complete the activity decreased at week twenty four (24). The number of steps increased and the average step length shortened or decreased at week twenty four (24) after the FESS was removed. All the parameters of gait returned to near baseline measurements by week twenty four (24). None of these changes were statistically significant, indicating a poor functional carry over of the effect of the FESS on the patient's gait.

In Group 2, the time in which the patients performed the walking activity decreased marginally significantly ($p=0.0593$) at week fourteen (14). The decrease in the time taken to complete the walking activity was statistically significant ($p=0.0367$) with FES&MS. The number of steps decreased and the average step length increased during the walking activity, but the change was not statistically significant ($p=0.1688$ and $p=0.3329$) the standard deviation in group 2 remained relatively high. And at week twenty four (24) all four of the parameters of gait returned to baseline or near baseline measurements, once again indicating no functional carry over effect of the FES&MS.

At baseline, patients in Group 1 walked moderately significantly slower in terms of time and speed than the patients in Group 2 (M-W $p=0.0588$ for time and 0.0694 for speed). However with regard to the number of steps and average step length there was no significant statistical difference between the groups at baseline (M-W $p=0.4057$ and 0.7065 respectively). The standard deviation of all four parameters of

gait in both groups was, initially, high. This decreased at week fourteen (14) and then increased again at week twenty four (24). It would seem that the intervention with FESS and FES&MS decreased the effect of the outliers within each group.

Patients in Group 1 performed better with the FESS than patients in Group 2. This is not only according to the calculated p-values, but also, according to the decrease in the standard deviations, was especially noticeable in Group 1 (Table 4.1, 4.2, 4.3 and 4.4).

The change in time that patients in Group 1 took to perform the walking activity, and the speed with which they walked were significantly statistically better than the patients in Group 2 ($p=0.0257$ and 0.0315 respectively). With regard to the number of steps they took and the average step length, there was no significant statistical difference between the changes that took place from baseline to week fourteen (14).

Table 5.2 Freezing of Gait (see Addendum 4 for Freezing of Gait scale)

Freezing of Gait scale	Week	Comparison of groups (M-W) At baseline	Between Groups (p-value)	Within Group 1 (p-value)	Within Group 2 (p-value)
	Baseline to week 14	0.0013	0.9824	0.0057	0.0158
	Baseline to week 24		0.7466	0.0074	0.6803

The change in the FOG scale between baseline and week fourteen (14) in both Group 1 and in Group 2 is statistically significant ($p=0.0057$ and $p=0.0158$ respectively). The difference between the FOG at baseline measurement and week twenty four (24) in Group 1 is still statistically significant, which indicates that the number of freezing episodes did not increase to the baseline number of freezing episodes after the FESS was removed ($p=0.0074$). This is not in line with the other parameters of gait that are displayed in Table 5.1, which indicate there was no functional carry over effect in the change of parameters of gait at week twenty four (24).

In Group 2 however there was no significant statistical difference between the number of freezing episodes at week twenty four (24) compared to the baseline measurement ($p=0.6803$), indicating there was no carry over effect of FES&MS on freezing/akinesia. This observation is in line with the changes that took place in the parameters of gait displayed in Table 5.1.

The SD in Group 1 was lower than in Group 2 at baseline, week fourteen (14) and week twenty four (24). The SD in Group 2 remained relatively high, but constant at baseline, week fourteen (14) and week twenty four (24) (See Table 4.11).

5.3.1 Discussion of results on the parameters of gait and freezing of gait scale

According to the Mann-Witney test there was a moderately significant difference in the time and minimally significant difference in the speed with which patients in Group 1 completed the walking activity, compared to Group 2. Group 1 took more time and walked with less speed to complete the walking activity.

All four the parameters of gait in Group 1 (FESS), namely, time, speed, number of steps and the average step length to complete the walking activity, as well as the FOG scale showed a significant statistical change from the baseline measurement to the week fourteen (14). All four parameters of gait measured in Group 1 also showed a significant statistical change back to near baseline measurements at week twenty four (24). (At week twenty four (24) there was therefore no statistical difference between the baseline measurements compared to the baseline measurement).

In Group 2 however there was a moderately significant statistical change in the time and a significant change in the speed to complete the walking activity between the baseline measurement and the measurement at week fourteen (14). The change in the number of steps and step length in

Group 2 was not statistically significant. By week twenty four (24) all the parameters of gait in Group 2 had also returned to baseline values.

The return of the speed (and the other parameters of gait, for that matter) to near baseline measurement at week twenty four (24) indicates that the effects of the FESS and/or the FES&MS were not sustained. This means that brain plasticity due to motor learning did not occur due to the intervention of FES. The reason for this may be the nature of PD being a progressive disorder (Krack, et al, 2003) and that the pathology in the basal ganglia is not reversible - at least not within twelve (12) weeks of intervention.

Clinically this indicates that FESS improves the gait parameters of a patient with PD, but has a poor carry over effect once the FESS has been removed.

The speed at which the patients walked may have been statistically significantly affected because FESS and FES&MS probably changed the timing of the muscle contraction between tibialis anterior and the gastrocnemius and soleus muscles. The FESS and FES&MS probably caused a longer contraction of the tibialis anterior muscle during the pre-swing and swing phases which may have contributed to a longer inhibition of the gastrocnemius and soleus muscles which assisted the patient to get toe-clearance during the swing phase of gait and as such to overcome short steps and therefore increase the speed of gait. (Nieuwboer, et al, 2004).

The change in the walking time, speed, number of steps and step length at week fourteen (14), all indicate that a decrease in the number of freezing episodes and/or the bradykinesia that should have taken place. The fact that at week fourteen (14) Group 1's average speed of walking was 0.809 meter/sec and that of Group 2 was 0.74 meter/sec indicates that the walking speed in both groups had increased to such an extent that they would be able to maintain a functional walking speed

within the community (see Table 4.2.). Functional community walking speed is considered to be 0.8m/s (Whittle, 2007). This speed decreased again at week twenty four (24) to near baseline measurements.

A change in the parameters of gait due to the application of FESS and FES&MS might also have an effect on the participant's frequency of falling during gait, the use of a walking aid(s), the freezing of gait, and walking within the community.

The gait of a person with PD is characterised by bradykinesia/akinesia and a decrease in stride length. One of the reasons for the decrease in bradykinesia /akinesia is that foot clearance during the pre-swing and swing phases of gait is increased and this leads to a decreased incidence of tripping, which results in a fall.

From the increase in the scores of the FOG scale and the return of all the parameters of gait to baseline measurements by week twenty four (24), it can also be said that FESS and FES&MS did not have an effect on the pathophysiology of PD, as the effects of the FESS and FES&MS are not maintained once the intervention has been withdrawn.

Patients with PD often use a walking aid to assist them in overcoming the episodes freezing/akinesia and to prevent them from falling. In this clinical trial seven (7) of the patients in Group 1 used a walking aid at the baseline assessments, compared to three (3) of the patients in Group 2. At the week fourteen (14) assessments with the relevant stimulus, only five (5) of the patients in Group 1 and two (2) of the patients

in Group 2 were using a walking aid. At the week twenty four (24) assessment however all seven (7) in Group 1 and all three (3) of the patients in Group 2 who were using the walking aids at the baseline assessment were once again using the walking aid. This was due to them feeling unstable without the relevant stimulus and the fear of falling that had increased again. The spontaneous decrease in the use of a walking aid in both groups at week fourteen (14), confirms the results discussed above that FESS and FES&MS have a positive effect on the parameters of gait. This could be due to the increase in step length (see Table 5.1), decrease in rigidity (paragraph 5.6) and probably also the improvement in the postural reactions as the patients in the clinical trial reported less incidents of falling and generally feeling more stable when using FESS and FES&MS (Bloem, et al, 1999, Oliverira, Gurd, and Nixon, 1997; Shumway-Cook and Woollacott, 2001).

Various reasons for falling in people living with PD have been described in the literature and also observed in the patients in this clinical trial. The loss of dynamic standing balance predisposes patients with PD to falls, as they have difficulty in responding to unexpected postural perturbations (Smithson, Morris and Iansak, 1998). As discussed in Chapter 2, paragraph 2.7.3, it is estimated that 60% of patients with PD fall compared to 29% of falls in the elderly (Shuratova, Morris and Huxam, 2004). Most falls occur indoors, due to decreased postural control and the fact that patients with PD restrict walking in the community (outdoor walks) because of this fear of tripping and falling. The falls also usually occur in the “on” period of medication, because this is when the patient is most mobile and thus more active (Bloem, et al, 2003).

Fear of falling occurs due to an increased incidence of tripping while walking, especially when mobilising in the community. Many patients who participated in this clinical trial who had already experienced falls found that falling occurred most of the times during a freezing episode and while turning.

Fear of falling has also been found to be related to postural instability, but it remains unclear whether falling results from postural instability or whether falling exaggerates postural instability that exists due to the progression of the disease (Adkin, Frank, and Jog, 2003). Reasons for falling in patients with PD include:

- Postural instability due to rigidity in the antagonistic muscles. Stiffness in the muscles results from this rigidity and there is a loss of postural control that affects the dynamic standing balance. Postural instability is also hypothesized to be caused by a decrease in proprioception, (Melnick in Umphred, 2007), but it has not been shown in the clinical trial on FESS and FES&MS on akinesia in gait.
- The decrease in the postural control that results from the rigidity in the antagonistic muscles causes decreased dynamic standing balance (Bloem, et al, 1999).

The somato-sensory sensation levels for both groups appeared not to be affected in the patients who participated in this clinical trial and remained unchanged throughout the clinical trial.

There was a slight improvement in the Ashworth scale (refer to Table 4.14), at week fourteen (14), more so in Group 1 than in Group 2. A change of 0.5 in patient's

muscle tone in Group 1 and 0.3 in Group 2 as well as a decrease in the standard deviation in both groups indicate that some patients must have experienced a clinical decrease in muscle tone. In the case of patients with a Grade 2+, as indicated in Group1, the tone decreased from 'considerable increase in muscle tone with passive movements that might have been difficult' to 'more marked increase in muscle tone through most of the range of motion, but affected part moved easily. With a slight decrease in tone the ability to perform functional activities will be easier and this may have a positive impact on the patient's perception of the quality of life of the patient. The range of active movement of the limbs will also be clinically better with a decrease in tone. The change in muscle tone could be ascribed to the improved ability to walk due to the timed external cue (FESS or FES&MS). Functional improvement decrease abnormally increased muscle tone. This decrease in the muscle tone could also have contributed to the improvement in the walking speed and step length seen in both the groups at week fourteen (14). The reverse was also true, namely, that the improved walking performance due to the external cueing with the FESS and FES&MS may have contributed to the decrease in rigidity. Both these groups had an increase in their muscle tone after the intervention had been removed at week fourteen (14).

- All the patients who participated in this clinical trial were required to keep a falls diary. At the beginning of the trial all of them expressed a fear of falling. At week fourteen (14) all the patients in Group1 and in Group 2 reported that they did not have a fear of falling while they were using the FESS or the FES&MS. This could also be seen by the decreased need for the walking aid at week fourteen (14) in the

patients that used a walking aid at the baseline assessment. At week twenty four (24) all the patients once again reported that the fear of falling had returned and the three (3) patients in Group 1 and two (2) patients in Group 2 who stopped using the walking aids started using it again.

Two (2) of the patients in Group 1 described the decrease in the fear of falling as:

"I feel more stable while I use the FES, and I do not have freezing episodes as before." (Patient 1)

This is where (when experiencing a freezing episode), I used to get unstable and topple over." (Patient 2)

Tripping and falling occurs as a result of the decrease in the amplitude of the movements, in particular the stride length and non-toe-clearance of the ground with the swing phase of gait (Morris, 2005). The decrease in the stride length is also characterised by akinesia and this increases the risk of tripping and falling in patients with PD. The increase in the step length experienced by both Group 1 and Group 2 at week fourteen (14) decreases the chances of tripping and falling, but the return to the near baseline measurement at week twenty four (24) again increased the chances of tripping and falling.

When falling in patients with PD occurs due to non-toe-clearance (decreased timing in the reciprocity between tibialis anterior and gastrocnemius during the terminal stance and pre swing phase of gait), and the application of FESS or FES&MS encourages an improvement in the timing between the TA and the GS muscles during these phases of gait, falling episodes should decrease.

The question remains whether FESS and FES&MS would result in a decrease in falling due to a change in the posture, decreased automatic postural reactions and rigidity.

- Stooped posture. The centre of gravity of the patient with PD is shifted from the midline of the supporting basis forward, and falling may occur (Marsden, 1994, Oliverira, Gurd, and Nixon, 1997; Melnick in Umphred, 2007). Only one patient in Group 2 had a marked stooped posture and a scoliosis to the right in the thoracic region. Nineteen (19) of the patients in the clinical trial had a slightly stooped posture. At week fourteen (14), the patients in both Group 1 and Group 2 did appear to walk more uprightly, as they did not focus on the ground during gait in the effort to avoid tripping. At week twenty four (24) the patients were once again focussing on the ground during gait, in the effort to avoid tripping and this shifted the centre of gravity forward again, thereby causing a more flexed posture. This was, however, a subjective observation.

The results of the time and number of steps to turn 360 degrees to the right and to the left are discussed before the null hypothesis or alternative hypothesis can be accepted or rejected. The reason for this is that patients often experience freezing episodes when they want to turn. Turning on the spot is part of functional walking and it was expected that the result obtained after testing the participants against the parameters of turning on the spot would coincide with results obtained from the testing of the participants against the parameters measured during the walking activity.

5.4 Turning 360 degrees “on the spot”

5.4.1 Time to turn 360 degrees “on the spot” in seconds

Table 5.3 and Table 5.4 illustrate the results obtained after testing the participants against the criteria for the turning activity (turning 360 degrees “on the spot”). Table 5.3 illustrates the time it took the patients in Group 1 and in Group 2 to complete this test. Table 5.4 illustrates the number of steps that it took the patients to turn 360 degrees. Tables 5.3 and 5.4 show the P-values at week fourteen (14) and at week twenty four (24) The M-W value at baseline is also indicated.

	Time to turn to the right in seconds				Time to turn to the left in seconds			
Week	Comparison of groups (M-W) At baseline	Between Groups	Within Group1	Within Group 2	Comparison of groups (M-W) At baseline	Between Groups	Within Group1	Within Group 2
Change from baseline to week 14	0.0963	0.6042	0.0051(*)	0.0926	0.0696	0.4494	0.0166 (*)	0.2026
Change from baseline to week 24		0.6786	0.2026	0.3863		0.9257	0.2845	0.4139

Table 5.3 P-values of time to turn 360-degrees to the right and to the left in seconds

The time it took the patients to turn to the right at week fourteen (14) decreased significantly in Group 1, but not so in Group 2.

In both Groups there was no significant statistical difference between the baseline measurements and the week twenty four (24) measurements - confirming again that there is a poor carry over of the effect gained with the FESS and the FES&MS at week fourteen (14).

There was also a significant statistical change in the time taken to turn to the left for the patients in Group 1 at week fourteen, but not so for Group 2.

The SD in Group 1 (SD=30.37893) at week fourteen was high. This increased to (SD=44.85633) at week twenty four. Group 2's SD was lower at week fourteen (14) (SD=22.75641). This decreased to (SD=17.68959) indicating an overall improvement in this group.

In both Groups there was (as with turning to the right) no significant statistical difference between the baseline measurements and the week twenty four (24) measurements with their turning to the left. It therefore also confirms that there is a poor carry over of the effect gained in turning to the left with the FESS and the FES&MS at week fourteen (14).

There was a large SD for the patients in Group 1 between baseline and week fourteen (14) (SD=30.10105). This increased (SD=54.05223) between baseline and week twenty four (24). These results indicate that there were outliers at week twenty four (24), even though there was improvement in the time taken to perform the task. In Group 2, there was a slight increase in the SD at week fourteen (14)

(SD=18.61843), but the SD decreased at week twenty four (24) (SD=11.16356), indicating that the whole group showed a decrease in the time taken to turn to the left.

5.4.2 Number of steps to turn 360 degrees “on the spot”

Table 5.4 P-values of turning: Number of steps to the right and left

	Number of steps to turn to the right				Number of steps to turn to the left			
Week	Comparison of groups (M-W) at baseline	Between Groups	Within Group 1	Within Group 2	Comparison of groups (M-W) at baseline	Between Groups	Within Group 1	Within Group 2
Change from baseline to week 14	0.0754	0.1549	0.0051 (*)	0.1820	0.0411	0.3458	0.0125 (*)	0.2613
Change from baseline to week 24		0.7554	0.2613	0.4112		0.4162	0.2014	0.9593

There is a tendency towards a significant statistical difference in the baseline measurement of the number of steps that patients in Group 1 gave when turning to the right, compared to Group 2.

When turning to the left, there was a definite significant statistical difference (M-W= 0.0411) when turning to the left between the two groups.

The number of steps taken to turn 360 degrees to the right decreased significantly in Group 1 at week fourteen (14), but not in Group 2, though there was still a decrease in the number of steps taken by that group. The same pattern is seen for the number of steps to turn to the left in both Group1 and in Group 2. The number of steps taken increased again in both groups at week twenty four (24), again confirming that there is no carry over effect of the FESS and the FES&MS.

5.4.3 Discussion of Turning

The statistical significance in the decrease in the time and the number of steps to turn “on the spot” (to both the left and to the right) that took place between baseline and week fourteen (14) in Group 1 corresponds with the outcome of Group 1 in the walking activity; namely, a decrease in the episodes of freezing/akinesia/bradykinesia.

At week twenty four (24) there was neither a significant statistical change in the time nor in the number of steps taken to turn the 360 degrees, when compared to the baseline measurement. It confirms again that there is a poor carry over effect of the FESS in patients with PD at week twenty four (24), after the application of FESS was

removed at week fourteen (14). The results of the turning “on the spot” to the left and to the right confirms the outcome of the walking activity and the effect of FESS and FES&MS on the parameters of gait.

Group 2 showed no significant statistical change in the time nor in the number of steps to turn 360 degrees to the right or to the left. This corresponds with the results found in the parameters of gait during the walking activity.

5.5 Discussion on hypotheses

As stated in Chapter 3, the hypotheses on which this research was based was stated as:

Hypothesis 1 (H1)

FESS and FES&MS decrease freezing/akinesia and bradykinesia/hypokinesia during gait in patients suffering from PD.

Null Hypothesis (H0)

FESS and FES&MS do not decrease freezing/akinesia and bradykinesia/hypokinesia during gait in patients suffering from PD.

From the results of the parameters of gait, the FOG scale and from the turning on the spot as indicated in the preceding paragraphs:

The alternative hypothesis 1 (H1) is accepted for Group 1, but the null hypothesis 1 (H0) is accepted for Group 2.

5.6 The effect of FESS and FES&MS on the quality of life of patients with PD

The quality of life of the patients who participated in the clinical trial was measured using the PCI during the walking activity, the PDQ-39 questionnaire, UPDRS and the FOG scale. The number of falls is discussed in paragraph 5.3.1.

Table 5.5 illustrates the results of the PCI, UPDRS and the PDQ-39. These have been grouped to more easily discuss the results of the clinical trial on the quality of life of the patient with PD. The P-values are tabulated as well as the M-W value at baseline.

Table 5.5 Quality of life measurements

Outcomes measure	Week	Comparison of groups (M-W)	Between Groups (p-value)	Within Group 1 (p-value)	Within Group 2 (p-value)
PCI	Baseline to week 14	0.2568	0.0130 (*)	0.0051 (*)	0.8785
	Baseline to week 24		0.1720	0.0926	0.3329
UPDRS	Baseline to week 14	0.002	0.2200	0.0575	0.4694
	Baseline to week 24		0.0441 (*)	0.0099 (*)	0.2562
PDQ-39	Baseline to week 14	0.6759	0.0607	0.0049 (*)	0.0047 (*)
	Baseline to week 24		0.0501 (*)	0.0058 (*)	0.0063 (*)

5.6.1 Discussion of the PCI, UPDRS and PDQ-39

The changes in the PCI between baseline and week fourteen (14) are statistically significant in Group 1 ($p=0.0051$).

The change in PCI for Group 1 at week fourteen (14) confirms the results of the parameters of gait as patients were able to walk faster, with less number of steps and an increased step length. The PCI decreases at week twenty four (24), once again confirming that there is no carry over effect of the FESS or the FES&MS once the stimulus had been withdrawn.

There were no significant statistical changes in the PCI in Group 2 at week fourteen (14) or at week twenty four (24) ($p=0.8785$ and $p=0.3329$ respectively). It is interesting to note that the PCI increased at week fourteen (14) in Group 2 and increased even further at week twenty four (24). These results at week fourteen (14) do not correspond with the results observed in the parameters of gait.

The motor part of the UPDRS shows a marginally significant change in Group 1 and a non-significant statistical change in Group 2 at week fourteen (14) ($p=0.0575$ and $p=0.4694$ respectively). The change from baseline to week twenty four (24) was statistically significant in Group 1 ($p=0.0099$). In Group 2 there was no change between the baseline measurement and week fourteen (14), but the score increased at week twenty four (24). The results in Group 1 confirm the outcome of the parameters of gait, as the motor part of the UPDRS was used in this trial. It is, however, also confirmed that there was a poor carry over effect of the FESS and the FES&MS ten (10) weeks after it was removed.

It is interesting to note significant statistical changes in the PDQ-39 for both Group 1 and Group 2 took place by week fourteen (14) ($p=0.0049$ and $p=0.0047$ respectively) and at week twenty four (24) ($p=0.0058$ and $p=0.0063$ respectively). The quality of life of all these patients with PD was therefore perceived as been significantly better with the application of the FESS and FES&MS than when it was not applied. Both Groups also showed a significant statistical increase in the score at week twenty four (24) ($p=0.0058$ and $p=0.0063$ respectively) after the FESS and the FES&MS was removed indicating a decrease in the quality of life once the FESS and FES&MS has been

removed. This indicates a poor carry over effect of the FESS and FES&MS once the stimulation has been withdrawn.

The results regarding Group 1, as it is displayed in Table 5.5, indicate that by week fourteen (14) their effort to perform the walking activity had decreased significantly ($p=0.0051$), their ability to move around measured with the motor part of the UPDRS improved most significantly statistically ($p=0.0575$), their freezing episodes during gait (Table 5.2) decreased significantly statistically ($p=0.0057$). And these factors probably contributed to the fact that these patients experienced an increase in their quality of life.

Although the quality of life for the patients in Group 2 improved significantly from the baseline statistics to week fourteen (14), it is not supported by statistically significant improvement in their effort to perform the walking activity (PCI) ($p=0.8785$). Their ability to move, as measured on the motor part of the UPDRS, showed no significant statistical change from baseline to week fourteen (14), as there was no difference in the scores on the questionnaire. There was a decrease in the score at week twenty four (24), indicating a decrease in the ability to move once the FES&MS was removed. Their freezing episodes measured on the FOG scale (Table 5.2) decreased significantly statistically ($p=0.0158$). The supporting evidence for the increase in the quality of life at week fourteen (14), of patients in Group 2, is therefore not as strong as in Group 1.

Although freezing/akinesia during gait in Group 1 (Table 5.2) at week twenty four (24) is still better than the baseline measurement ($p=0.0074$), their effort to perform the

walking activity (PCI) increased mildly ($p=0.0926$). Their mobility as it was measured on the motor section of the UPDRS as well as their perceived quality of life were still statistically significantly better than the baseline measurement ($p= 0.0099$ and $p= 0.0058$ respectively).

Patients in Group 2 experienced a statistically significant improvement in their quality of life on the PDQ-39 at week fourteen (14) and week twenty four (24) compared to what they experienced at the baseline measurements although there is no supporting evidence that their effort to perform the walking activity (PCI), their mobility on the motor section of the UPDRS and the FOG scale was better than the baseline measurements.

The younger patients in Group 2 may have perceived the quality of life to be worse as they now have to cope with the sudden loss of independence and adapting to coping strategies. The patients in Group 1, on average, have been diagnosed with PD for a longer period of time and may have accepted the diagnosis by now, whereas the younger patient may still be in denial.

5.6.2 Discussion of hypothesis on quality of life

As stated in Chapter 3, the hypotheses on which this research was based was stated as:

Alternative hypothesis 2 (H2)

FESS decreases freezing/akinesia and bradykinesia/hypokinesia during gait in patients suffering from PD to the extent that it improves the quality of life of these patients.

Null hypothesis 2 (H02)

FESS and FES&MS do not decrease freezing/akinesia and bradykinesia/hypokinesia during gait in patients suffering from PD to the extent that it improves the quality of life of these patients.

Based on the results of Group 1 on the PCI, UPDRS, FOG and the PDQ-39, it is clear that the effort to perform the walking activity (PCI), the fact that these patients experienced improvement in their motor ability (UPDRS), a decrease in freezing episodes during gait (FOG) and the improvement in their quality of life (PDQ-39).

Although patients in Group 2 showed a statistically significant improvement in their quality of life, it is not supported by the fact that all the parameters of gait nor the PCI and the motor part of the UPDRS changed statistically significantly. The significant statistical difference on the PDQ-39 may therefore have occurred due to the Hawthorne effect.

The alternative hypothesis 2 (H2) is accepted for Group 1.

Neither the alternative hypothesis 2 (H2) nor the null hypothesis 2 (H02) is accepted for patients in Group 2.

5.7 Comparison between the effect of FESS and FES&MS on the gait in patients with PD

Based on the results from the parameters of gait, the FOG, PCI, UPDRS and the PDQ-39, it is clear that the patients in Group 1 receiving FESS did respond better than the patients in Group 2 receiving FES&MS.

As stated in Chapter 3 the hypotheses on which this research was based was stated as:

Alternative hypothesis 3 (H3)

FESS decreases freezing/akinesia and bradykinesia/hypokinesia during gait in patients suffering from PD more than FES&MS.

Null Hypothesis 3 (H03)

FESS does not decrease freezing/akinesia and bradykinesia/hypokinesia during gait in patients suffering from PD more than FES&MS.

The alternative hypothesis 3 (H3), is therefore accepted for Group 1 and for Group 2.

The reason for the acceptance of the alternative hypothesis is not clearly understood and can not be explained from the results in this clinical trial. Some of the reasons for this may be:

- FES&MS could be an over-stimulation for the patient with PD. Some of the patients in Group 2 responded well to FES&MS and others showed little to no response.
- Emotionally and cognitively the FES&MS may have been too much stimulation. Therefore, there is a need for further investigation to identify the criteria that indicate a patient is a candidate for FESS and FES&MS.

5.8 General Discussion

Extensive research on the medical treatment and physiotherapy for patients with PD has been done in the past. This includes pharmacological, surgical and external cueing, to optimise patient's functional ability in spite of the ever worsening signs and symptoms of PD.

A wide variety of treatment modalities that are used to treat patients with PD are described in the relevant literature. And that reflects a variety of approaches to intervention, as described in Table 2.2. The treatment modalities, as well as the approaches to intervention reveal that patients with PD are treated by a multidisciplinary team.

It is important that an extensive assessment of the patient with PD be done, to determine the stage of the disease and to plan and implement the relevant

physiotherapy (Bloem, et al, 2004). Continual assessment and adaptation of the treatment plan will be necessary, due to the fact that PD is a progressive disease. This will also be the case with the use of the FESS, as the long term effects of FESS still need to be determined.

The timing and dose of medication also still needs to be continually monitored when applied together with the use of the FESS, as it was not within the scope of this clinical trial to test the effects of the FESS or FES&MS during “off” periods. The outcome measures used in this clinical trial can be adopted for use during assessments as they are clear and easily reproducible.

5. 8.1 The use of FESS and FES&MS in a physiotherapy programme

The main aim of the physiotherapy programme is to ensure that the patient with PD remains as independent as possible, for as long as possible, through applying exercises and external cues (Morris and Iansek, 1997; Del Olmo and Cudeiro, 2005; Mak and Hui-Chan, 2004). It is of vital importance that the patient with PD remains active within society and the community, to prevent withdrawal and immobility issues that result in secondary complications, such as contractures, bladder infections etcetera. (Viliani, et al, 1999) All the exercises should be integrated into the home or the work environment, to ensure the patient remains active in that environment. It is the responsibility of the multidisciplinary team to assess the condition of the patient on a regular basis and to adapt or adjust the exercises/home programme as necessary, to maintain the level of independence (Morris, et al, 1997).

Rehabilitation is offered to patients with PD as they show difficulty in functioning during ADL. The symptoms of PD result in the impairment of gross and fine motor coordination. These become problematic and result in the patient becoming functionally dependant on others (Gage and Storey, 2004). While pharmacological interventions slow down the progression of the disease, this is known to become ineffective over an extended period of time. Physiotherapy is therefore indicated through all the different stages of the disease as discussed in paragraph 2.8 (Cutson, Laub and Schenkman, 1995).

FESS can be used as a tool in a physiotherapy programme to assist in maintaining and improving gait patterns, decreasing the number of freezing episodes and decreasing the number of falls. All this will improve the quality of life of the patient living with PD.

5.8.2 FESS and FES&MS as an external cue to treat akinesia and hypokinesia in patients with PD

There are five factors that potentially contribute to bradykinesia and akinesia, namely, tremor, muscle weakness, rigidity, movement variability and slower thought processes (Berardelli, et al, 2001). Patients with PD do not loose the ability to move, but rather the ability to activate the movement (Ilansek, et al, 1995; Cunnington, et al, 1995).

In addition to the drugs such as levo-dopa, a number of studies have revealed that external sensory cues, such as tactile cues, e.g. mild sensory shock, rhythmic auditory

cues or auditory cues, together with visual cues serve to assist the patient to initiate with step taking during gait (Burleigh-Jacobs, et al, 1997; Jacono, Casadio, Morasso, and Sanguineti, 2004; Jiang, O' Mara, Chen, Stern, Vlagos, and Hanson, 1999)

External cueing has been used to enhance the patient with PD's motor performance since 1942 (Gage and Storey, 2004). External cues can compensate for the decrease in dopamine in the defective basal ganglia (Jahanshani, Jenkins, Brown, Marsden, Passingham, and Brooks, 1995; Morris, 2000; Morris, 2005; Morris and Lansak, 2002; Perry, et al, 2004.). External cues that are described in the literature (Del Olmo and Cudeiro, 2005; Dibble, et al, 2004; Suteerawattananon, et al, 2004) to initiate a functional movement, such as gait, include visual, auditory or somato-sensory information. In combination, they contribute towards improving the quality of the patients' movement. This is contradictory to the results described in this clinical trial as the sensory only (FESS) cue had better effects on the impairment, participation and functional activity levels of the patients than the combination of a sensory and motor cue (FES&MS).

With appropriate external visual and auditory cues patients with PD reveal a gait pattern of normal velocity, cadence and stride length (Lewis, et al, 2000). Research by Dam, Tounin, and Casson, (1996) showed that external cueing has also found to have positive effects in performing the functional tasks such as dressing or eating. The results from this clinical trial that indicated that FESS decreases akinesia and bradykinesia in patients with PD significantly, is based on the measured parameters of

gait during the walking activity. After turning they had to walk the same route back to the starting point back again. The patients however used the ODFS through which the FESS was administered constantly for twelve (12) weeks when they were walking.

The principles underlying teaching the patients compensatory movement mechanisms entails breaking down of the a functional movement into simple movement components, rearranging of the components into a logical order, applying prior mental rehearsal of the movement before performing it, performing each component of the movement separately, avoiding simultaneous motor or cognitive tasks and the application of the appropriate external cues to initiate and maintain momentum of the movement (Morris, 2000; Morris and Iansek, 1997).

The above concepts are true even with the use of FESS and FES&MS. A rehabilitation programme still needs to be followed while using the external cue of FESS or FES&MS. The rehabilitation programme will still be adapted as individually required by the patient. The FESS and FES&MS can be used to assist in increasing cardiovascular fitness where walking is involved and the programme aims at improving the general mobility.

Internal cues can also be utilised to improve the quality of the movement of the patient with PD. The effectiveness of the internal cues may relate to how much cognitive attention is used to effectively perform the task at hand (Morris, et al, 1996). However Marchese et al (2000) found that by teaching patients to use compensatory movement strategies and sensory cues (a combination of internal and external cueing) effect on

gait was longer lasting and positive, than was found to be the case when compared to those programmes that focus on exercises and functional activities alone.

Cubo, et al (2004) did, however, conclude that rhythmic auditory cueing actually slows the walking pace of the patient with PD and is not beneficial in overcoming their freezing episodes.

The precise mechanism behind the reduced stride length in patients with PD is still poorly understood. As yet there is no treatment method for assisting patients with PD to regulate their stride length throughout the course of this progressive neurological disease (Morris, et al, 1996). The increased cadence is a compensation for the decrease in stride length (Morris, et al, 1994). A common finding in these research articles is that normal range of movement can be achieved with some form of external cue in the form of visual, sensory and/or auditory in patients with PD under the correct conditions. This clinical trial may have bridged this gap in information, as it would appear that, with continued use of FESS for twelve (12) weeks, the average step length may be regulated in patients with PD. However, as stated before, the effect of using FESS in the long term on patients with PD still needs to be determined in a clinical trial.

Dibble, et al (2004) conducted a study to determine the effect of sensory cueing on the maximal gait speed that patients with PD could achieve during gait. The outcome of this trial by Dibble, et al (2004) suggests that when the speed of gait is the primary goal

of the participant, sensory cues may interfere with body movement outcomes of the lower limb, causing a slower swing phase and gait speed over the specific distance, probably due to rigidity in patients with PD. The trunk movements were less and the arms appeared to be more adducted and braced to the side of the body, giving a rigid appearance of the posture during gait. The results of the clinical trial in the trial with the FESS and FES&MS do not correspond with these results as the use of FESS did increase the speed of gait, although an increase in speed was not the primary goal with the application of the FESS.

Although it has been shown by the studies already discussed that visual, auditory or somato-sensory information can improve the quality of movement in patients with PD. From the results discussed in Chapter 4 and Chapter 5 of this study on the effects of FES on akinetic gait, it is indicated that FES in the form of an external sensory stimulation (FESS) (external cue) improves the gait speed, number of steps and average step length. The improvement in all these parameters of gait resulted in patients overcoming episodes of freezing and akinesia which corresponds with the results from the Freezing of Gait scale and the outcomes measures used to determine the quality of life (PDQ-39)

5.8.3 FESS and FES&MS compared to other forms of treatment in optimising impairment, functional activity and participation

The improvement and maintenance of a patients' functional ability entail that the deterioration in the patients physical ability due to secondary muscle weakness, a

decrease in their joint range of movement, and cardiovascular endurance and the development of spinal deformities (Morris, et al, 1997) should be prevented.

The aims of most of the general exercise programmes for patients with PD are to promote function through improvement of functional muscle strength, flexibility, coordination, balance and relaxation (Viliani, et al, 1999). Through a well planned exercise programme, that takes factors such as aging and other medical conditions that the patient may present into consideration, exercise endurance and general functional mobility on participation level is maintained/optimised. This enhances such activities as continued ambulation within the community (walking to the shops, to church or to visit friends, etcetera) and optimises the patients' quality of life.

Physiotherapy needs to focus on maintaining muscle strengthening, range of movement and endurance once the diagnosis of PD has been made, to ensure that the patient will be able to remain as mobile as possible, for as long as possible. This will also ensure that the patient with PD will remain socially active for a longer period of time. The patient should be encouraged to participate in some form of recreational sport, such as bowls, swimming, yoga, walking, etcetera (Fertl, et al, 1993) to optimise cardiovascular endurance. The maintenance of their endurance will also ensure the maintenance of motor function and help to reduce stiffness and other musculo-skeletal impairments. The use of the FESS will ensure that patients with PD remain active for longer as they will be more mobile and will be safer when mobile. Patients are able to remain active and this assists with maintaining muscle strength, endurance general circulation. Secondary effects of immobility will also therefore be avoided such as constipation, stiffness and even swelling of the lower limbs.

Although PD is a progressive disorder, physical activity may slow down the progression of the impairments and functional limitations, as well as reduce the mortality rates of PD. This may also occur in bedridden patients where passive and active-assisted activities and strengthening exercises may slow the progression of the disease and patients may be able to assist slightly with their care. Patients with PD who participated in regular exercises were found to have a normal gait velocity (Canning, Alison, Allen, and Groeller, 1997) which coincides with the literature that states that patients need to be involved in a rehabilitation programme that includes regular exercise and maintenance exercises. Strengthening exercises have also found to be beneficial to patients with PD. An increase in stride length and gait velocity was found after the programme was implemented (Scandalis, Bosak, Berliner, Helman, and Wells, 2001). It was not indicated how long the effect lasted on patients with PD. Pellecchia, et al, (2004), have shown that there is functional improvement as well as sustained improvement in the motor skills of PD patients that follow a long term comprehensive rehabilitation programme that is designed by a physiotherapist. A study on hypokinetic patients with PD revealed that there is consistency in the components of gait, such as speed and stride length, provided that the subjects were measured in the “on” state of medication, i.e. at least thirty (30) minutes post drug administration. The same subjects were assessed during the peak phase of the medication and then again thirty (30) minutes prior to the next administration of medication. Marked differences in the gait pattern of these patients could be observed. The change in the gait performance thirty (30) minutes before the next administration

of the medication strongly suggests that medication has a marked impact on the gait pattern in patients with PD (Urquhart, et al, 1999).

The results of the effect of FESS on akinetic gait are consistent with the findings of the trials mentioned above. From the improvement of the PCI at week fourteen (14) with FESS it can be concluded that the patients experienced less effort to perform the walking activity. The patients were more mobile with the FESS, thereby improving their cardiovascular output. The improvement in the step length and speed that patients experienced during the completion of the walking activity were also subjectively reported by the patients i.e. that they went on walks with their spouses, went shopping, could be left alone at home for longer periods, could get to the phone to answer it when it was ringing and go visit friends again and get involved in the community again. FESS can also be combined with an exercise programme that the patients may participate in to further improve their functional mobility.

All the patients who participated in this clinical trial were assessed during the “on” state of medication and at a fixed time after their medication had been taken. (It can be noted that the effects of FESS and FES&MS in the “off” state of medication needs still to be determined.)

Walking: The main problems patients with PD experience with gait is the deficit in regulation of their stride length, initiating gait as well as walking slower once they have started walking, until they reach a period of rest.

The use of external cues and cognitive strategies to overcome difficulties of gait and freezing/akinetic episodes are the focus of the physiotherapist when trying to improve the functional gait of a patient with PD.

In paragraph 2.9 external cueing to overcome freezing/akinetic episodes is discussed in greater detail. The use of cognitive strategies was investigated by Bond and Morris (2000) who found that if the attention of these patients was diverted away from the action they are in the process of performing, to focus on a second task, their stride length and gait speed decreased immediately. There has been no research in the field of long-term effects of external cues and cognitive strategies on gait in PD.

Teaching patients to concentrate on the heel strike and push off phase of gait (i.e. the normal sequence of movement components during gait) also helped patients in increasing the stride length and decreased the episodes of freezing (Morris, 2000).

At the beginning of the clinical trial in this study I assumed that the FES&MS would assist the patient in getting a good heel strike due to the active muscle contraction of the tibialis anterior and the peronei muscles; and, as such, improve the timing of the contraction of the gastrocnemius and tibialis anterior muscles during the pre-swing and swing phases of gait. This has, however, proven not to assist these patients with improving their functional gait or decreasing the episodes of freezing as effectively as the FESS, which acted as an external cue. The reason for this phenomenon could be that the FES&MS over-stimulated the patient, but this will need to be further explored in future clinical trials.

Turning (as part of functional gait): Patients with PD find turning especially difficult when they experience episodes of freezing. During the act of turning each consecutive step gets smaller until all movement ceases. Consequently, patients with PD are taught to turn in large arcs instead of on one place (Yekutiel et al, 1991). The use of FESS has proven to assist the patient to turn on the spot by decreasing the number of steps taken to turn and decreasing the time to turn 360 degrees.

Preventing tripping and falling: Postural instability results in a stooped posture with increased flexor tone and a decrease in the stride length that results in a shuffling gait (Marsden, 1994). The patient with PD probably compensates with a flexion position of the trunk, hips and knees in the attempt to lower the center of gravity of the body and to contribute in improving his/her stability. When the patient steps forward, a shuffle gait occurs, probably due to the protective steps which are a compensatory postural mechanism to avoid falling forwards. People with basal ganglia impairment are prone to tripping and falling, due to the decrease in stride length and non toe-clearance of the ground during the initial and early swing phase of gait (Morris, 2005). The reduction in stride length, that is characteristic of akinesia/bradykinesia, adds to the increased risk of falling because of the risk of tripping over obstacles (Plotnik, et al, 2005).

Treating the injuries as a result of falling is costly to society and to the health care funders. The combination of the risk of falling and the decrease in speed of ambulation contributes to the limited community ambulation of patients with PD, resulting in an overall decrease in their quality of life (Morris, 2000).

Most falls occur under harmless circumstances, such as turning around (Bloem, et al, 2003). Dual or multi-tasking while walking or standing also commonly contributes to the incidence of falling among patients with PD. This is due to patients not being able to prioritize the movement patterns automatically, so as to maintain balance under complex circumstances.

The FESS and FES&MS (FESS more so than FES&MS) appear to have decreased the chances and the fear of falling in patients with PD. The patients reported feeling more stable with the stimulus and some of them even left their walking aids when walking with the stimulus.

No other research on the prevention of tripping and falling in PD could be found. From results found in this study it became evident that FESS decreased the incidence of falling after the patients who participated in this study started using the FESS and increased again after it was removed at week fourteen (14). This finding was concluded from the entries in the falls diaries, as no falls occurred among these patients during the twelve (12) weeks of intervention. The results of falls in this clinical trial are discussed in paragraph 5.3.1.

Posture: The stooped posture in patients with PD can either be the result of musculo-skeletal impairment or be a compensatory mechanism for basal ganglia impairment such as found in PD.

The improvement in the time and the decrease of the number of steps to complete the turning activity in this clinical trial show that turning is statistically significantly easier for the patients when they use FESS. Subjectively, the patients reported that they feel

safer when using FESS or FES&MS although turning on the spot did not show any statistical significant improvement in Group 2.

5.8.4 FESS and FES&MS compared with other forms of treatment in optimising quality of life

Improvement was also seen in patients with PD functional activity and on the participation levels in patients with PD. Their scores on the quality of life questionnaires improved, indicating an improvement in their participation in their environment. Patients reported that their difficulties with gait, especially that of freezing/akinesia and bradykinesia/hypokinesia, is the main reason that they withdraw from the community, become isolated and, thereby, lead a life of poor quality. With the improvement of the quality of life with, especially when using FESS, a patient with PD can live a more productive life and still contribute to society for a longer period of time. This also improves the quality of life of the family and or the primary care giver. FESS, more so than FESS&MS, improved the quality of life of the patients who participated in the clinical trial. This can be seen from the results of the participation level scores (Table 5.5). Most of the patients reported to have preferred wearing the ODFS for gait than to walk without it. They said to feel safer and that they could walk longer distances in their environment with the stimulation. FESS also decreased their episodes of freezing/akinesia and/or bradykinesia/hypokinesia. Fourteen (14) of these patients have purchased stimulators post trial, as they felt that their walking and quality of life was better with the use of the ODFS. And I have now implemented FESS on all fourteen (14) patients as part of their ongoing treatment.

From this clinical trial it can be concluded that FESS does have a positive effect on the functional outcome of gait in patients with PD. However, it must be noted there is poor carry over of the effect of FESS ten (10) weeks after the stimulus was withdrawn. The poor carry over effect after twelve (12) weeks of using the FESS indicates that no motor learning has taken place. This result indicates that an external cue such as FESS and to some extent FES&MS fulfills the role of the basal ganglia but confirms the fact that PD is a degenerative condition. In the case of PD, this is therefore an intervention that will need to be ongoing. Though it is not yet known whether the effectiveness of FESS will wear off over time.

The outcome of the trial that tested the effect of FESS and FES&MS on freezing/akinesia and bradykinesia/hypokinesia in patients with PD has the potential of playing a major role in maintaining the patient's functional independence and participation in their communities. When this happens the detrimental effect of immobility resulting in secondary complications, such as poor posture, muscle contractures joint stiffness, constipation and other complications, will be postponed or even avoided. If this is the case health care funders (medical aid schemes) will save the cost of treating the consequences of immobility and falling. The cost of an ODFS is minimal, compared to the cost of chronic medication for freezing/akinesia, and health care/rehabilitation to prevent tripping and falling, as well as the secondary complications of immobility

Chapter 5 presented the results of administering FESS and FES&MS on patients who experienced freezing/akinesia and/or bradykinesia/hypokinesia were integrated and discussed. Lastly, the result of this study was discussed in relation to the relevant results of previous research done on freezing/akinesia and bradykinesia/hypokinesia in patients with PD

In Chapter 6 a summary of these results will be presented, the shortcomings of this clinical trial will be discussed and recommendations for further studies in this field are made.

CHAPTER 6

SUMMARY, LIMITATIONS AND SUGESTIONS FOR FURTHER RESEARCH

6.1 Introduction

The focus of this study was to determine whether FESS and FES&MS, as different forms of external cues, have a significant effect on akinesia during gait in patients with PD. The motivation behind this study was that the effect of medication presently used to treat akinesia wears off over time (Gray and Hildebrand, 2000; Morris, 2000; Schrag, et al, 1998) and, in consequence, new methods of intervention need to be explored for to assist the patient with PD in overcoming akinesia and other movement difficulties related to PD. Akinesia, which is used synonymously with freezing, as one of the characteristic signs of PD is defined as 'a lack of initiation of gait that occurs when the activity in the primary motor cortex or in the supplementary motor area is decreased' (Bronchie, et al, 1991; Cunnington, et al, 1995).

Bradykinesia, which is used synonymously with hypokinesia, is defined as 'slowness of movement'. While bradykinesia is the term used to describe the slowness of movement, akinesia refers to the lack of spontaneous or associated movement or the prolonged time to initiate the movement (Evarts, et al, 1981).

Akinesia and bradykinesia results from a disruption in the neurotransmitters that occur in the neural projections from the internal segment of the globus pallidus to the cortical regions of the supplementary motor area and the primary motor cortex (Alexander and

Crutcher, 1990) due to a progressive degenerative disorder that is associated with the malfunctioning of the basal ganglia (Diaz and Bronstein, 2005).

The degeneration of the dopaminergic nigro-striatal neural pathways leads to an imbalance in the output of the striatal neural pathways. The loss of dopamine leads to an increase in activity of the striatal neurons in the indirect pathway and a decrease in the activity of the striatal neurons in the direct pathway. The decreased inhibition through the direct pathways results in less activation of the motor cortex. Bradykinesia and akinesia are thought to be caused by decreased inhibition of the motor cortex (Cote and Crutcher, 1991).

PD is a progressive neurological movement disorder, due to degeneration mainly in the basal ganglia (Whittle, 2007).

6.2 The Research questions that guided the study

The research questions that guided this study were:

1. Does FESS or FES&MS decrease freezing/akinesia or bradykinesia/hypokinesia during gait in patients suffering from PD?
2. If FESS or FES&MS does make a statistical and clinical difference to a patient suffering freezing/akinesia or bradykinesia/hypokinesia's gait, is the difference large enough to improve the quality of life of the patient?
3. Is there a difference between FESS and FES&MS on freezing/akinesia or bradykinesia/hypokinesia?

To answer these questions, a single blinded, randomized clinical trial was conducted. Twenty (20) patients that complied with the inclusion and the exclusion criteria were recruited for the clinical trial. They were randomly placed into two groups with Group 1 receiving FESS and Group 2 receiving FES&MS. Assessments were conducted at week zero (0) and again at week two (2). The average of these measurements was calculated to obtain a baseline measurement with which the result of the assessments at weeks fourteen (14) and twenty four (24) were compared. The assessment at week fourteen (14) was performed with the FESS and FES&MS switched on. After the performance of this test, the FESS and FES&MS were removed and patients in both groups were again assessed at week twenty four (24). In addition, the patients in both groups were monitored at weeks eight (8) and eighteen (18) respectively.

The assessment performed at weeks zero (0), two (2), fourteen (14) and twenty four (24) consisted of measuring the parameters of time, speed, step length and number of steps during a pre-determined distance of twenty (20) meters. At ten (10) meters the patients in both groups had to perform a 180 degree turn and walk the same distance back to the starting point.

The time and the number of steps it took the patients to turn 360 degrees 'on the spot' were measured. The reason for including this activity is that turning is a functional part of gait and patients with PD do find turning difficult and tend to experience episodes of freezing while turning. And it is usually while involved in the activity of turning that postural instability occurs.

The patients were required to complete the freezing of gait (FOG) scale questionnaire. This scale was used to indicate the number of akinetic (freezing) episodes experienced during community walking and activities of daily living. The score was used by the primary investigator to assist in assessing the influence of akinesia on these patients' functional during ADL and, as such, on their quality of life

These patients' perceived quality of life was determined by asking them to complete the PDQ-39 questionnaire, as well as the motor part of the UPDRS questionnaire at each of the assessments. Their PCI was also measured during the walking activity, which gave an indication of the patients' effort to walk and, as such, the PCI (though a physiological measure) is also related to their perceived quality of life.

The following null/alternative hypotheses were accepted for the parameters of gait, the FOG scale and 'turning on the spot' based on the outcome of this clinical trial:

The alternative hypothesis 1 (H1) was accepted for Group 1: "FESS and FES&MS decreases freezing/akinesia and bradykinesia/hypokinesia during gait in patients suffering from PD".

The null hypothesis 1 (H0) is accepted for Group 2: "FESS and FES&MS do not decrease freezing/akinesia and bradykinesia/hypokinesia during gait in patients suffering from PD."

The alternative hypothesis 2 (H2): “FESS decrease freezing/akinesia and bradykinesia/hypokinesia during gait in patients suffering from PD to such an extent that it improves the quality of life of patients“ was accepted for Group 1.

Neither the alternative hypothesis 2 (H2) nor the null hypothesis 2 (H02) is accepted for Group 2.

The alternative hypothesis 3 (H3): “FESS decreases freezing/akinesia and bradykinesia/hypokinesia during gait in patients suffering from PD more than FES&MS”, was accepted for Group 1 and for Group 2.

The somato-sensory sensation levels for both groups appeared to not be affected in the patients who participated in this clinical trial and this remained unchanged throughout the clinical trial.

There was a slight improvement in the Ashworth scale (see Table 4.14) at week fourteen (14), more so in Group 1 than in Group 2. With a slight decrease in tone the ability to perform functional activities would be easier and this change may have a positive impact on the patient’s perception of the quality of life. The range of active movement of the limbs would also be clinically easier with this decrease in tone. This change in muscle tone could be ascribed to the improved ability to walk due to the effect of the external cue (FESS or FES&MS). Both the groups had an increase in their muscle tone after the intervention had been removed at week fourteen (14).

The degree to which the motor learning capability is affected by the cognitive deficits and motor problems in patients with PD are not completely understood (Morris, 2000). The result of this study revealed that no functional carry over of the improvement in the parameters of gait, FOG as well as in the QoL measures of patients who received the FESS or the FES&MS took place. This implies that if motor learning had taken place during the twelve (12) weeks of stimulation, it was either reversed after the stimulation was removed at week twelve (12) of the trial or it may indicate that no motor learning had taken place at all during the twelve (12) weeks of using the stimulation. This therefore confirms Morris's statement that motor learning capabilities in patients with PD is not well understood (Morris, 2000).

From the discussion in Chapter 5, it can also be concluded that FESS appears to have a more significant difference on akinesia and bradykinesia than FES&MS. There were statistically significant changes in the parameters of gait with the use of FESS but, despite the recording of a slight improvement, the same could not be said of the use of FES&MS.

A possible reason for this could be that FES&MS is an over-stimulation for the affected muscles. FESS would also feel slightly more comfortable on the lower limb than FES&MA, because FES&MS requires a higher intensity of the asymmetrical bi-phasic wave form to obtain the desired muscle contraction of the peronei and tibialis anterior muscles. Involuntary muscle contractions caused by the FES&MS may also be distracting during gait.

The reasons that FESS and FES&MS would have improved the functional gait of patients with PD could be that both FESS and FES&MS aided in increasing the postural stability of these patients, by improving postural control and by decreasing rigidity. Further, the decrease in the rigidity allowed these patients to move with more ease.

The fact that the five (5) patients in Group 1 and two (2) patients in Group 2 started walking without their walking aids indicates that their confidence level during walking improved.

FESS and FES&MS is an extraceptive cue that probably redirects the locus of control away from the dysfunctional basal ganglia, leading to an improvement in the quality of the desired movement. This is similar to the findings of Morris 2005.

The focus of most of the general exercise programmes created for patients with PD is to promote function through improvement of functional muscle strength, flexibility, co-ordination, balance and relaxation (Viliani, et al, 1999). FESS promotes functional gait in patients and, as such, muscle strength, flexibility, co-ordination and balance are improved, due to the improved functional movement.

Bond and Morris (2000) found that it is the habitual mechanisms that are most affected in patients with PD. The authors suggest that patients with PD be taught to practice multiple tasking from the early stages of PD. Patients with PD benefit from the practice of breaking the sequence of more complex movements/tasks into smaller components of movement and avoiding dual task performances (Bond and Morris, 2000).

Morris (2000) explains the need for a lasting external cue to assist patients in overcoming mobility issues, including that of akinesia in PD. Evidenced-based therapy is necessary to determine the effects of interventions on the functional outcome of gait and quality of life in patients with PD.

The results of this clinical trial indicate that FESS may be a valid tool to use as an external cue, in overcoming movement impairments, specifically akinesia and bradykinesia in patients with PD.

Conditions under which this clinical trial was conducted are described in Chapter 3, paragraph 3.4. The shortcomings that were identified during this trial are discussed in the following paragraph.

6.3 Limitations of the clinical trial

From the large standard deviations that are reflected in the results (see Chapter 4) it is clear that a larger sample group would have given a more statistically valid indication of the effect of FESS and FES&MS on akinesia and bradykinesia during the gait of patients with PD. The reason for the small sample group used for this clinical trial was that patients who would probably have met the inclusion and exclusion criteria had already been recruited by medical specialists to participate in one of the many drug trials being conducted for PD. Patients participating in one trial were not permitted to simultaneously enter another trial, as this situation might have confounded the results of both the trials.

The sample group of patients who participated in this study was only recruited from within the Western Cape Province and did not include patients from the low socio-

economic income group as no volunteers from this group came forward to participate in the trial. Although this study did not discriminate between people from different cultural groups, only one patient from the African cultural group could be identified and recruited to participate in this study.

The walking activity used in this clinical trial consisted of a series of controlled circumstances:

- Distance of twenty (20) meters; which was not, it should be noted, representative of the total distance these patients could walk per day during ADL or while in the community, i.e., going shopping or visiting friends, going to church, etcetera;
- Followed by the completion of a 360 degree turn (which is a major stimulus that triggers akinesia and bradykinesia) was included in this study. It should be noted this trial took place under controlled circumstances and not under daily changing/varying circumstances similar to that which patients may encounter during their ADL and/or community ambulation.

In spite of the limitations mentioned, the chosen walking activity was an acceptable way to control the circumstances to be enabled to measure the parameters of time, speed, number of steps and step length during the walking activity as well as during turning on the spot. These results could form the basis of further studies in the future.

Although the FOG scale and PDQ-39 questionnaire gave some insight into the problems that patients may have experienced with gait, in particular, and other aspects that affected their quality of life, in general, other measures should be investigated to determine whether FESS and/or FES&MS have an effect on the way in which those

aspects of the patients' activities of daily living, whether in their home/social, recreational or work environments are affected.

The objective observation and measurement of parameters of gait, such as step length, was difficult to do accurately; although the trials were videotaped and a second (independent) therapist was given the task of measuring the result obtained for each patient against the set parameters, to limit the possibility of recording inaccurate measurements. It is recommended that a force plate that is connected to an electronic movement analysis system be used in future studies, to avoid the recording of inaccurate measurements.

6.4 Recommendations for further study

The trial was conducted over a period of twenty four (24) weeks, of which the patients used the FESS and FES&MS for only the first twelve (12) weeks. In future studies it is recommended the effect of FESS and FES&MS on akinesia during gait be determined over a longer period of time. And such a trial should determine the time span after removal of the FESS and/or FES&MS before patients start to experience either a regression in the measured parameters or a decrease in function. Participating patients could be evaluated/monitored on a weekly basis, to see how quickly they respond to the intervention or how long it takes for the intervention to have some effect on their ambulation. They could also be tested on a weekly basis to determine how quickly the effect wears off, as did appear that no or very little carry over effect has taken place ten (10) weeks after the FESS and FES&MS had been removed in this trial.

It should further be determined what the effect is of a longer period of using the FESS and/or FES&MS and whether this will have a sustained effect on the patients' gait and other functional activities.

Patient reaction on intermittent or periodic use of the FESS and/or the FES&MS should also be determined.

It is recommended to follow up on the progress of the patients post trial, to determine whether they have, of their own decision, continued with the intervention of the FESS and FES&MS or not. This will assist physiotherapists to determine whether there is a lasting long-term effect of the FESS or whether ongoing input is required, as is expected after this clinical trial.

In any follow-up study, patients from all population groups and socio-economic levels should be included in the sample group. This would require that a larger sample group of patients be recruited. To achieve this, other centres that are equipped to perform the assessment and intervention need to be incorporated to participate in a multi-centre clinical trial.

A multi-dimensional movement (gait) analysis should be conducted during the clinical trials to more objectively determine the effect of FESS and FES&MS on the patients' gait. Such a system of analysis could indicate whether FESS and/or FES&MS have an effect on the patients' quality of movement and posture of the whole body. Such a multi-dimensional movement system of analysis could reveal valuable information on

possible changes in the patients' postural control and bradykinesia during gait as a result of the FESS and FES&MS.

EMG-studies during the assessment will assist physiotherapists in understanding the effect of FESS and FES&MS on akinesia and bradykinesia and the patients' postural control during functional activities.

According to a study conducted by Wood, Edwards, Clay, Wadley, Roenker and Ball (2005), there is inconsistency in studying the effectiveness of external sensory cues on sensori-motor performances in PD. It has been suggested by the authors enlisted above that cognition and sensori-motor functioning are not independent of each other, as once believed. Those authors make the statement that sensori-motor functioning and cognition are interrelated, in the sense that cognitive loading decreases the sensori-motor functioning. From this, Block, (2005) and Li, Lindenberger, Hommel, Aschersleben, Prinz, and Baltes, (2004). concluded that sensori-motor decline is associated with a decline in cognitive functioning. It is known that patients with PD show cognitive impairment (Auriel, Hausdorff, Herman, Simons, and Giladi, 2006) therefore the interaction between cognitive functioning and specifically the patients' reaction to the FESS should be investigated.

The strengthening and general exercise programmes that emphasize the increase of gait velocity, step length and PCI should be further explored and compared to the effect of FESS and FES&MS on patient's ambulation.

These exercise programmes should include exercises that are targeted at balance deficits and assist in reducing the incidence of falls and improving function. A

community based fitness programme that includes a variety of exercises may prove to be a cost effective way of maintaining community mobility and physical strength from the early and middle stages of PD after FESS and/or FES&MS had been removed. In the results of the study it was indicated that FESS will assist in maintaining patient mobility, both in the home and community environment that is so necessary in maintaining a good quality of life for the patient with PD.

An increase or maintenance of mobility will assist in preventing other complications of immobility, such as increase circulation, the prevention of constipation, maintenance of good muscle strength, muscle length and endurance and prevent the stiffening of joints, especially of the lower limbs.

It has also been shown in this clinical trial that FESS and FES&MS may play a role in preventing falls, as these patients experience greater stability and, consequently, feel safer while on their feet with the FESS and FES&MS. This might assist in correcting patient postural stability.

The prevention of falls will benefit the health care insurance schemes/companies as there might be a decrease in fractures and hospitalization due to injuries related to falling. Future studies should include investigations of all these aspects of possible improvement in the ambulation and general mobility of the patients due to the application of FESS.

6.5 Conclusion of the study

Morris (2005a) describes interventions that may be effective in the treatment of PD.

The use of external, visual and auditory cues turn the locus of the control of the movement away from the basal ganglia to the intact frontal regions of the brain.

Strategies to enhance cognitive attention to the task at hand can also be applied.

Morris has determined that there is insufficient research available to determine the effectiveness of the cognitive or exercise components for patients with PD and suggests that further research in this field is necessary.

This clinical trial revealed statistically significant results by using FESS as an external cue, despite of the small sample group and this, as such, forms a good basis for further, more complex (possible multi centre) trials.

From this clinical trial it can be concluded that FESS decreases akinesia and bradykinesia statistically significantly during ambulation of patients with PD. A poor carry over effect (lasting effect as evidence of motor learning) of the functional improvement that was observed during the trial, would suggest that motor learning did not take place during the initial period of twelve (12) weeks of this trial.

The effect of FES&MS also has an effect on akinesia and bradykinesia on the gait of patients with PD, but is not statistically significant in all the parameters of gait that were used.

The perception of improved quality of life of all the patients who participated in this trial and used the FESS improved. The improved QoL was sustained in the patients with FESS ten (10) weeks after the ODFS had been removed. However the patients, in

Group 2 (using FES&MS) did not experience a sustained increase in the QoL to the same extent during the same period. The QoL was perceived to be sustained in patients with FESS and FES&MS following the results of the PDQ-39.

From the results of this clinical trial, it is suggested that FESS be incorporated into the rehabilitation programme of patients with PD. In addition, FESS can be offered as an option to assist patients during exercise programmes designed to aid in improving the gait and other functional activities during ADL.

The result of this study has initiated a new direction for consideration in therapy, while indicating numerous possibilities for further research that might eventually shed new light on a variety of aspects with regard to ambulatory and other functional problems experienced by patients living with PD.

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ADDENDUMS

Addendum 1a)

Unified Parkinson's Disease Rating Scale (UPDRS)

MOTOR EXAMINATION

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.

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.

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.

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.

Rigidity (Judged on passive movement of major joints with patient relaxed in sitting position. Cog wheeling 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.

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.

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.

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.

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.

Arising from Chair (Patient attempts to rise from a straight-backed 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.

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.

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.

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.

Body Bradykinesia and Hypokinesia (Combining slowness, hesitancy, decreased arm swing, 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.

Addendum 1b)

Unified Parkinson's Disease Rating Scale. Scoring for motor test

Participant Name / Identification

Date

Name of Examiner

TEST	SCORE (0-4)
Speech	
Facial Expression	
Tremor at rest (head, upper and lower extremities)	
Action or postural tremor of hands	
Rigidity	
Finger taps	
Hand movements	
Rapid alternating movements of hands	
Leg agility	
Arising from chair	
Posture	
Gait	
Postural stability	
Body bradykinesia and hypokinesia	

Total Score _____ (Max=56)

Comments

Addendum 2a)

Mini-Mental State Examine (MMSE)

Record response to each question

Orientation _____ / 5

Year, month, day, date, time

Country, town, district, hospital, ward _____ / 5

Registration

Examiner names three objects (e.g. apple, table, penny)

Patient asked to repeat three names – score one for each correct answer _____ / 3

Then patient to learn three names (i.e. repeat until correct)

Attention to Calculation

Subtract 7 from 100, then repeat from result, etc. Stop after 5. 100, 93, 86, 79, 72, 65, _____ / 5

(Alternative: spell 'world' backwards. DLROW)

Recall

Ask for three objects learnt earlier _____ / 3

Language

Name a pencil and watch _____ / 2

Repeat 'No ifs, ands, or buts'

Give a three-stage command. Score one for each stage (e.g. 'Place index finger of right hand on your nose, and then on your left ear.') _____ / 3

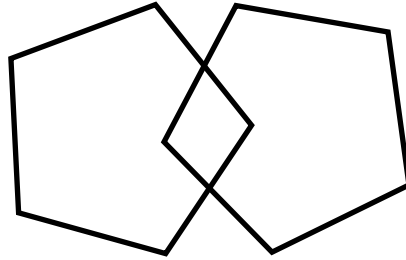
Ask patient to read and obey a written command on a piece of paper stating: 'Close your eyes' _____ / 1

Ask patient to write a sentence. Score if it is sensible and has a subject and a verb _____ / 1

Copying

Ask patient to copy a pair of intersecting pentagons

Addendum 2b)



_____ / 1

Mini-Mental State Examine scoring sheet

Participant Identification

Date

Name of Examiner

Orientation

_____ / 5

What is the (Year), (season), (date), (day), (month)?

Where are we: Country, town, district, hospital, ward?

_____ / 5

Registration

Examiner names three objects (e.g. apple, table, penny). One second to say each. Then ask the patient all three after you have said them. Score one for each correct answer

_____ / 3

Then repeat the trials until he/she either learns all three, or has six trials. Count all trials and record them. *Number of trials* _____

Attention to Calculation

Serial 7's. One point for each correct. Stop after 5 answers.

7,14,21,28,35,42. 100,93,86,79,72,65

(Alternative: spell 'world' backwards. DLROW)

_____ / 5

Recall

Ask for three objects learnt earlier

_____ / 3

Language

Name a pencil and watch

Repeat 'No ifs, ands, or buts'

Give a three-stage command. 'Take a sheet of paper in your right hand,

_____ / 2

fold it in half, and put it on the floor.'

_____ / 3

Ask patient to read and obey a written command on a piece of paper stating: 'Close your eyes'

_____ / 1

Ask patient to write a sentence. Score if it is sensible and has a subject and a verb

_____ / 1

Copy a design

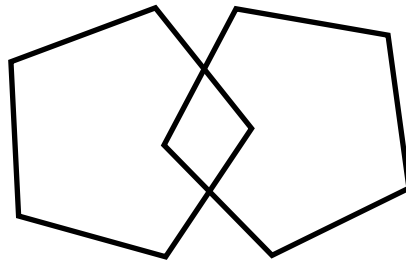
_____ / 1

CLOSE YOUR EYES

Please write a sentence:

Copying

Please copy diagram below



Addendum 3a)

Parkinson's Disease Questionnaire (PDQ 39)

Parkinson's Disease Quality of Life Questionnaire

This questionnaire has 37 questions, which will help us to know you are feeling. Please do not leave out any questions as it is important that they are all answered. Place a tick ✓ in the box that you feel shows how much of a problem each one has been for you in the past 3 months.

How often in the last 3 months have you had trouble with	All the time	Most of the time	Some of the time	A little of the time	Never
1. Stiffness?	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
2. Feeling generally unwell?	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
3. Feeling that you are no longer able to do your hobbies?	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
4. Being tense?	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
5. Feeling insecure of yourself due to your physical limitations?	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
6. Shaking of your hand(s)?	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
7. Feeling worn out or having no energy?	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
8. Difficulties in doing sport or leisure activities?	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
9. Clumsiness?	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
10. Feeling embarrassed about your illness?	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
11. Shuffling when you walk?	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
12. Having to postpone or cancel social activities because of your illness?	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
13. A feeling of extreme exhaustion?	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
14. Difficulties writing?	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
15. Being afraid of possible	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>

progressing of the illness?	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
16. Difficulties writing?	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
17. Being less able to go on holiday than before your illness?	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
18. Feeling insecure of yourself around others?	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
19. Difficulties getting a good night's rest?	Yes <input type="checkbox"/>	No <input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
20. On/off periods?	Friend/neighbour <input type="checkbox"/>	Family member <input type="checkbox"/>	Nurse <input type="checkbox"/>	Other (please specify) <input type="checkbox"/>	<input type="checkbox"/>
21. Difficulty in accepting your illness?					
22. Difficulties talking?					
23. Difficulties signing your name in public?					
24. Difficulties walking?					
25. Drooling?					
26. Feeling depressed or discouraged?					
27. Difficulty with sitting still (for long periods)?					
28. Often needing to urinate and/or wetting yourself?					
29. Difficulties with transport?					
30. Sudden extreme movements?					
31. Difficulties concentrating?					
32. Difficulties getting up (from a chair)?					
33. Constipation?					
34. Difficulties with your memory?					
35. Difficulties turning around in bed?					
36. That your illness inhibits your sex life?					
37. Feeling worried about (the possible consequences of) an operation in connection with your illness?					
Did you need any					

help to complete this questionnaire? If yes, who? partner/spouse <input type="checkbox"/>					
--	--	--	--	--	--

Addendum 3b)

Scoring of the Parkinson's Disease Quality of Life questionnaire:

the questionnaire consists of 37 items, which provide an overall total score, and can be combined to give four sub-scales

Sub-scale numbers	Item
Parkinsonian symptoms (14 items)	1, 4, 6, 11, 16, 20, 22, 25, 27, 30, 32, 35
Systemic symptoms (7 items)	2, 7, 13, 19, 24, 28, 33,
Social functioning items (7 items)	3, 8, 12, 17, 23, 29, 37
Emotional functioning (9 items)	5, 10, 15, 18, 21, 26, 31, 34, 37

The responses to the items are scored for the total score and the sub-scales. Higher scores are indicative of better quality of life

Addendum 4)

Freezing of Gait Questionnaire (FOG)

I
Participant Identification

Date

Name of Examiner

1. *During your worst state—do you walk:*

- 0 Normally
- 1 Almost normally—somewhat slow
- 2 Slow but fully independent
- 3 Need assistance or walking aid
- 4 Unable to walk

Score_____

2. *Are your gait difficulties affecting your daily activities and independence?*

- 0 Not at all
- 1 Mildly
- 2 Moderately
- 3 Severely
- 4 Unable to walk

Score_____

3. *Do you feel that your feet get glued to the floor while walking, making a turn or when trying to initiate walking (freezing)?*

- 0 Never
- 1 Very rarely—about once a month
- 2 Rarely—about once a week
- 3 Often—about once a day
- 4 Always—whenever walking

Score_____

4. *How long is your longest freezing episode?*

- 0 Never happened
- 1 1-2s
- 2 3-10s
- 3 11-30s
- 4 Unable to walk for more than

Score_____

5. How long is your typical start hesitation episode (freezing when initiating the first step)?

0 None

1 Takes longer than 1s to start walking

2 Takes longer than 3s to start walking

3 Takes longer than 10s to start walking

4 Takes longer than 30s to start walking

Score_____

6. How long is your typical turning hesitation? (freezing when turning):

0 None

1 Resume turning in 1-2s

2 Resume turning in 3-10s

3 Resume turning in 11-30s

4 Unable to resume turning for more than 30s

Score_____

Total Score_____

Comments

Addendum 5)

Gait Analysis

WEEK

FES CLINICAL ASSESSMENT									
NAME						Date			
						Assessment		1	2
Side Stimulated R L						Resting Heart Rate			
<u>20m walk with turn</u>		No of Steps	Time	HR	HR increase	Speed m/s	PCI		
No ODFS	1								
	2								
	3								
	Mean								
With ODFS	1								
	2								
	3								
	Mean								
						<u>Speed</u>	<u>PCI</u>		
% change with stimulation									
% change since 1 st assessment (NS)									

<u>3min Endurance Walk</u>	<u>Distance (m)</u>	<u>Speed (m/min)</u>	<u>HR</u>	<u>HR Increase</u>	<u>PCI</u>
No ODFS					
With ODFS					

Comments

VIDEO TAKEN BY.....

Signature of clinician(s)

VIDEO ANALYSIS

<u>20m walk with turn</u>	No. of Clear Steps (22.68m WWT)	Time Taken (22.68m WWT)	No. of Step movements (22.68m WWT)	No. of Steps over 10m	Stride Length over 10m	<u>Cadence Steps/min (22.68m WWT)</u>		Time to Initiate Walking	Episodes of Freezing
						Clear	All		
No ODFS	1								
	2								
	3								
	Mean								
With ODFS	1								
	2								
	3								
	Mean								

Comments / notes

Signature of Clinician.....

Addendum 6)

Determining Range of Movement

LEFT	Range of Motion (ROM)	RIGHT
	Shoulder elevation	
	Depression	
	Protraction	
	Retraction	
	Glenohumeral flexion	
	through elevation	
	Glenohumeral flexion	
	through abduction	
	Glenohumeral	
	Abduction	
	Glenohumeral	
	adduction	
	Glenohumeral	
	Extension	
	Glenohumeral	
	Internal Rotation	
	Glenohumeral	
	external rotation	
	Elbow flexion	
	Elbow extension	
	Forearm pronation	
	Forearm supination	

Wrist extension

Wrist flexion

Wrist ulnar deviation

Wrist radial deviation

Metacarpophalangeal
flexion

Metacarpophalangeal
Extension

Metacarpophalangeal
abduction

Intercarpal joint
movement (accessory
movement)

P I P joints

D I P joints

Thumb abduction
(Web space)

Thumb flexion

Thumb extension

Thumb opposition

Trunk Flexion

Trunk extension

Trunk sideflexion

Trunk Rotation

Thorax rotation

Hip flexion

Hip extension

H Internal rotation

H External rotation

H Abduction

H Adduction

Knee extension

Knee Flexion

Ankle dorsiflexion

Plantar flexion

Eversion

Inversion

Midtarsal joints

(accessory
movement)

Intertarsal gliding
movement (accessory
movement)

Metatarsophalangeal
flexion

Metacarpophalangeal

Extension

Toe flexion

Toe extension

Addendum 7)

Modified Ashworth scale

Modified Ashworth Scale for grading Spasticity

Grade	Description
0	No increase in muscle tone
1	Slight increase in muscle tone, manifested by a catch and release, or by minimal resistance at the end of the range of motion when the affected part(s) is moved in flexion or extension
2	Slight increase in muscle tone, manifested by a catch, followed by minimal resistance throughout the remainder (less than half) of the range of movement (ROM)
3	More marked increase in muscle tone through most of ROM, but affected part(s) easily moved
4	Considerable increase in muscle tone, passive movement difficult
5	Affected part(s) rigid in flexion and extension

Addendum 8)

Data capturing sheet for the Oxford Muscle Strength Test

Muscle Function Chart

LEFT					Myotome	RIGHT				
					Shoulder elevators C3/4					
					Depressors C6/7					
					Protractors C6/7					
					Retractors C5/6					
					Shoulder abductors C5					
					Adductors C5/6					
					Shoulder flexors C5/6					
					Extensors C7/8					
					Sh Int Rot C5/6					
					Sh ext rot C5/6					
					Elbow flexors C5/6					
					Elbow extensors C7					
					Forearm pronators C5					
					Forearm supinators C5/6					
					Wrist ext C6					
					Wrist flexors C7/8					
					Finger flexors C7/8					
					Finger Extensors C8					
					Lumbricals C8/T1					
					Interossei C8/T1					
					Thumb abductor C8/T1					
					Thumb adductor C8/T1					
					Thumb flexor C8/T1					
					Thumb extensor					
					Abdominals					
					Erector spinae					
					Hip flexor L1/2					
					Hip extensor L5/S1					
					Internal rotators S1					
					External rotators S1					
					Abductors S1					
					Adductors L2					
					Knee extensor L3/4					
					Knee Flexor L5/S1					

					Ankle dorsiflexion					
					Plantar flexion S1/2					
					Evertors S1					
					Invertors S1					
					Toe flexors S2					
					Toe extensors S1					

Addendum 9)

Body chart for documentation of sensation

(To Follow)

MOTOR

KEY MUSCLES

	R	L
C2		
C3		
C4		
C5		
C6		
C7		
C8		
T1		
T2		
T3		
T4		
T5		
T6		
T7		
T8		
T9		
T10		
T11		
T12		
L1		
L2		
L3		
L4		
L5		
S1		
S2		
S3		
S4-5		

0 = total paralysis
 1 = palpable or visible contraction
 2 = active movement, gravity eliminated
 3 = active movement, against gravity
 4 = active movement, against some resistance
 5 = active movement, against full resistance
 NT = not testable

Hip flexors
 Knee extensors
 Ankle dorsiflexors
 Long toe extensors
 Ankle plantar flexors

Voluntary anal contraction (Yes/No)

TOTALS + = MOTOR SCORE (100)

SENSORY

KEY SENSORY POINTS

	R	L	R	L
C2				
C3				
C4				
C5				
C6				
C7				
C8				
T1				
T2				
T3				
T4				
T5				
T6				
T7				
T8				
T9				
T10				
T11				
T12				
L1				
L2				
L3				
L4				
L5				
S1				
S2				
S3				
S4-5				

0 = absent
 1 = impaired
 2 = normal
 NT = not testable

Any anal sensation (Yes/No)

PIN PRICK SCORE (max: 112)
 LIGHT TOUCH SCORE (max: 112)

TOTALS + = PIN PRICK SCORE (max: 112)

TOTALS + = LIGHT TOUCH SCORE (max: 112)

NEUROLOGICAL LEVEL

The most caudal segment with normal function

SENSORY MOTOR

R	L
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>

COMPLETE OR INCOMPLETE?

Incomplete = Any sensory or motor function in S4-S5

ASIA IMPAIRMENT SCALE

<input type="checkbox"/>

ZONE OF PARTIAL PRESERVATION

Caudal extent of partially innervated segments

SENSORY MOTOR

R	L
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>

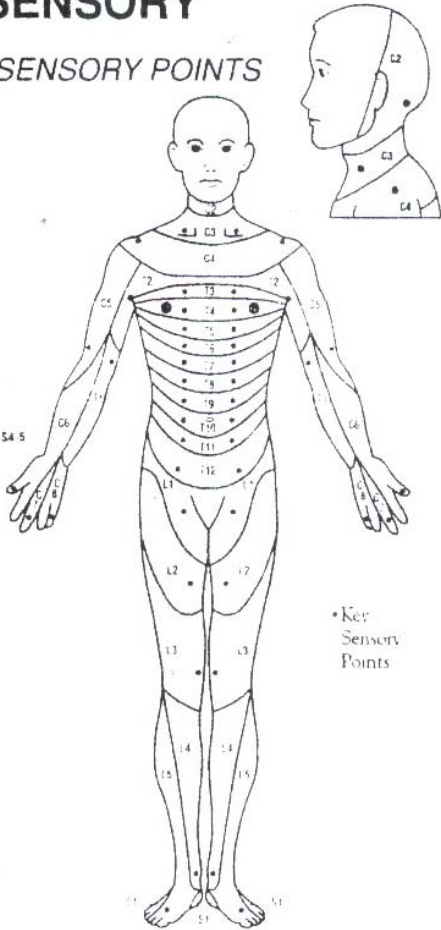
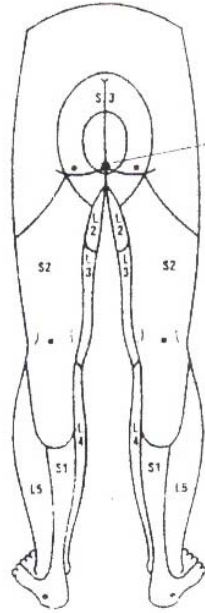
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SENSORY

KEY SENSORY POINTS

0 = absent
 1 = impaired
 2 = normal
 NT = not testable

	LIGHT TOUCH		PIN PRICK	
	R	L	R	L
C2				
C3				
C4				
C5				
C6				
C7				
C8				
T1				
T2				
T3				
T4				
T5				
T6				
T7				
T8				
T9				
T10				
T11				
T12				
L1				
L2				
L3				
L4				
L5				
S1				
S2				
S3				
S4-5				
TOTALS	<input type="checkbox"/> + <input type="checkbox"/>	<input type="checkbox"/> + <input type="checkbox"/>	<input type="checkbox"/> + <input type="checkbox"/>	<input type="checkbox"/> + <input type="checkbox"/>
KIMUM)	(56)	(56)	(56)	(56)



• Key Sensory Points

Any anal sensation (Yes/No)
 + = PIN PRICK SCORE (max: 112)
 + = LIGHT TOUCH SCORE (max: 112)

Addendum 10)

Falls Diary (To Follow)

Time and date of each fall.	Where did you fall – e.g. kitchen or outside?	What were you doing – e.g. turning round or bending over?	Was your FES machine switched on at the time of fall?	Any other comments
Time: Date:				
Time: Date:				
Time: Date:				
Time: Date:				
Time: Date:				
Time: Date:				
Time: Date:				
Time: Date:				

Addendum 11)

Informed consent to participate in the study

The Effect of Functional Electrical Stimulation on Akinesia in patients with Parkinson's Disease.

INTRODUCTION

You are invited to volunteer for a research study. This information leaflet is to help you to decide if you would like to participate. Before you agree to take part in this study you should fully understand what is involved. If you have any questions, which are not fully explained in this leaflet, do not hesitate to ask Nicole. You should not agree to take part unless you are completely happy about all that is involved in the study. In the best interests of your health, it is strongly recommended that you discuss with or inform your personal doctor of your possible participation in this study, wherever possible.

WHAT IS THE PURPOSE OF THIS TRIAL?

You have been diagnosed with Parkinson's disease and Nicole would like you to consider taking part in a research study of functional electrical stimulation (FES). This is using an electrical current to help with your functional activity of walking. We know that FES has a positive effect on the walking pattern of people with Parkinson's. It aids in overcoming freezing episodes and we would like to determine how it helps with this.

You have been shown the inclusion and the exclusion criteria for the trial and all the other participants will also fit these criteria.

During the study you will receive FES to one leg if you are in group A.

WHAT IS THE DURATION OF THIS TRIAL?

If you decide to take part you will be one of approximately 50 participants. The study will last for 16 weeks. You will be asked to visit the investigator five times as during the 16 weeks. You will be asked to fill in forms with questions pertaining to your Parkinson's and how it affects your life. Physical examinations of your walking will also be conducted at each visit. Normal evaluations of your sensation and range of movement of your joints will also be conducted.

Videos will be taken at the assessments and your face will be in the videos. These videos are for use by the investigator only and no third party will have access to the videos. The videos are used to re-evaluate the walking patterns and to aid in counting steps and measuring step lengths. One's posture will also be evaluated from the videos.

Please note that you may be requested to continue with the trial for a further four months to continue to collect data to determine the effects of the FES on walking patterns. It is important that you let the investigator know of any medicines that you are currently taking and to stick with these medications for the duration of the trial. Alterations in medications may lead to your exclusion from the trial.

HAS THE TRIAL RECEIVED ETHICAL APPROVAL?

This clinical trial Protocol was submitted to the Faculty of Health Sciences Research Ethics Committee, University of Pretoria and written approval has been granted by that committee as well as to the Ethics Committee at the University of Cape Town. The study has been structured in accordance with the Declaration of Helsinki (last update: October 2000), which deals with the recommendations guiding doctors in biomedical research involving human/subjects. A copy of the Declaration may be obtained from the investigator should you wish to review it.

WHAT ARE MY RIGHTS AS A PARTICIPANT IN THIS TRIAL?

Your participation in this research trial is entirely voluntary and you can refuse to participate or stop at any time without stating any reason. Your withdrawal will not affect your access to other medical care for Parkinson's. The investigator retains the right to withdraw you from the study if it is considered to be in your best interest. If it is discovered that you did not give an accurate history or did not follow the guidelines of the trial you may be withdrawn from the trial at any time.

IS ALTERNATIVE TREATMENT AVAILABLE?

Alternative treatment in the form of general exercise therapy is often used to aid people with Parkinson's. If you decide not to take part in this study it is possible that your physiotherapist may treat you with other forms of exercise therapy.

MAY ANY OF THESE TRIAL PROCEDURES RESULT IN DISCOMFORT OR INCONVENIENCE?

The FES gives a slight "pins and needles" feeling over the area where it is attached. There are no invasive (breaking or puncturing of the skin) methods, that include needles or injections, that will be used. The machine will be attached to one lower leg by means of easily removable adhesive electrodes (gel pads that are used to transfer the current from the machine to the muscle).

WHAT ARE THE RISKS INVOLVED IN THIS TRIAL?

A rash may develop from the use of the electrodes. This is easily treated by means of washing the area with warm soapy water and removing the electrodes for a short period of time.

ARE THERE ANY WARNINGS OR RESTRICTIONS CONCERNING MY PARTICIPATION IN THIS TRIAL?

One is recommended not to use the FES if one has a cardiac pacemaker. One may not drive when the stimulator is on. It is easily switched off by a press of a button, so one can still drive if involved in the trial.

INSURANCE AND FINANCIAL ARRANGEMENTS

Neither you nor your medical scheme will be expected to pay for any study assessments during the course of the trial.

Assessments and the purchase of the FES after the trial will be at the participants own cost. A stimulator will be provided for the course of the trial. Thereafter stimulators can be purchased from the investigator at a price in the region of R4000. The fitting of the system and further assessments will be at the expense of the participant's medical aid or paid by the participant

SOURCE OF ADDITIONAL INFORMATION

For the duration of the trial, you will be under the care of Nicole Redman, a physiotherapist. If at any time between your visits you feel that any of your symptoms are causing you any problems, or you have any questions during the trial, please do not hesitate to contact her. The telephone number is (021) 442 1867 through which you can reach her or another authorized person. Please stay in contact with your Dr and attend all appointments arranged with the Dr.

CONFIDENTIALITY

All information obtained during the course of this trial is strictly confidential. Information that may be reported in scientific journals will not include any information which identifies you as a participant in this trial. In connection with this trial, it might be important for the ethics committee as well as your doctor to have access to your medical records.

Any information uncovered regarding your trial results will be held in strict confidence. You will be informed of any finding of importance to your health or continued participation in this trial but this information will not be disclosed to any third party in addition to the ones mentioned above without your written permission.

INFORMED CONSENT

I hereby confirm that I have been informed by the investigator, Nicole Redman about the nature, conduct, benefits and risks of clinical trial I have also received, read and understood the above written information (Participant Information Leaflet and Informed Consent) regarding the clinical trial.

I am aware that the results of the trial, including personal details regarding my sex, age, date of birth, initials and diagnosis will be anonymously processed into a trial report.

I may, at any stage, without prejudice, withdraw my consent and participation in the trial.
I have had sufficient opportunity to ask questions and (of my own free will) declare myself prepared to participate in the trial.

Participant's name _____

Participant's signature _____ (Please print) Date _____

I, Nicole Redman herewith confirm that the above participant has been informed fully about the nature, conduct and risks of the above trial.

Investigator's name _____

Investigator's signature _____ (Please print) Date _____

Witness's name* _____ Witness's signature _____