MUSCLE ACTIVATION AND FORCE PRODUCTION IN PARKINSON'S PATIENTS DURING SIT TO STAND TRANSFERS

by

Vincent K Ramsey

(Under the Direction of Michael Horvat)

ABSTRACT

The purpose of this study was to compare the muscle activation, kinematics, and force production, between Parkinson's patients and healthy, age-matched participants, during sit-to-stand transfers. Twenty-four men (71.5 ± 7.1 years) categorized as Parkinson's patients (N=13), and healthy adults (N=11) participated in this study. The trial protocol required participants to rise from a seated to standing position, from a standard seat height of 17 inches, and return to a sitting position after thirty seconds. Two force platforms, positioned one under each foot, measured antero-posterior and vertical force components as well as peak torque moments during each trial. In addition, muscle activation was measured by a six channel, bi-lateral electromyography (EMG) system and recorded muscle frequency levels at 1000 samples per second (Hz). Reflective markers were placed at lower body joint locations and a kinematic assessment was conducted utilizing a high-speed (Peak Performance Technologies, Inc.) motion analysis system. All data collection devices were synchronized and activated upon initial movement of the subject. A 2 X 2 factorial analysis of variance was used to test for significant differences between groups. Based on the data analysis it was concluded that, although mild to moderately afflicted Parkinson's patients are able to adequately perform sit to stand transfers, the higher levels of ground force application and muscle activation patterns required for this task, compared to their healthy peers, indicates the decreased ability to efficiently apply that force in order to perform this simple daily task.

INDEX WORDS: Parkinson's Disease, Electromyography, Ground Reaction Force

MUSCLE ACTIVATION AND FORCE PRODUCTION IN PARKINSON'S PATIENTS DURING SIT TO STAND TRANSFERS

by

VINCENT K. RAMSEY

B.S., Georgia State University, 1996

M.S., Georgia State University, 1998

A Dissertation Submitted to the Graduate Faculty of The University of Georgia in Partial Fulfillment of the Requirements for the Degree

DOCTOR OF PHILOSOPHY

ATHENS, GEORGIA

2002

MUSCLE ACTIVATION AND FORCE PRODUCTION IN PARKINSON'S

PATIENTS DURING SIT TO STAND TRANSFERS

by

VINCENT K. RAMSEY

Approved:

Major Professor:	Michael Horvat
Committee:	Bruce Blasch
	Daniel Hope
	Bryan McCullick
	Mark Wilson

Electronic Version Approved:

Gordhan L. Patel

Dean of the Graduate School
The University of Georgia
May 2002

DEDICATION

I would like to dedicate this dissertation to my wife, Deborah, whose love, support, and encouragement has made the attainment of my education possible.

ACKNOWLEDGEMENTS

I would like to thank my mother, Mary, for her unwavering love and for teaching me persistence. I express my gratitude to the United States Air Force for teaching me how to get things accomplished in a timely fashion and shipping me to all parts of the world where I enjoyed many eye opening experiences. I would also like to express my thanks to those professors and educators that taught me how to think beyond the boundaries of the classroom and appreciate what learning is all about.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

Page

CHAPTER I

INTRODUCTION

Parkinson's disease (Parkinson's disease) is a chronic degenerative neurological condition affecting approximately one out of one hundred persons over the age of 60 (Bradshaw et al., 1998, Gabbard, 2000, Guyton, 1982, National Parkinson's Foundation, 2001). Current demographics estimate 1.5 million people in the United States alone suffer from this non-fatal condition with another 2.5 million patients diagnosed around the world (Canning et al., 1997, The Parkinson's Institute, 2000). This number is expected to increase greatly as the elderly population continues to be the fastest growing societal segment in America.

The first diagnosis of Parkinson's disease was made in 1817 by physician in London, England named James Parkinson for whom the disease is named. His prognosis was based on what he referred to as "shaking palsy", which is one of the more pronounced symptoms of Parkinson's disease (de Goede et al., 2001, Murray et al., 1978, Rogers, 1996). According to Dr. Parkinson "the illness is characterized by involuntary tremulous motion, with lessened muscular power, in parts not in action and even when supported; with a propensity to bend the trunk forward, and to pass from a walking to a running pace, the senses and intellect being uninjured". This description, however, did not relate to the causes of Parkinson's disease. It wasn't until the 1950's that the neuropathology of the disease was determined linking the disease with a cluster of cells in the midbrain whose degeneration cause the symptoms of Parkinson's disease (Cunnington et al., 1999,Georgiou et al., 1998). These symptoms, if left untreated or diagnosed, can cause a number of physical as well as psychological problems. Medical research since the 1950's, has found the cause of Parkinson's disease to be a degeneration of the substantia nigra cells located deep in the midbrain (Hausdorff et al., 1998, Hoehn & Yahr, 1967, Ouchi, et al., 2001). This structure extends the length of the midbrain and is a black color due to its neurons containing melanin and hence its name, nigra (Young & Young, 1997). The substantia nigra is also known to produce the neurotransmitter dopamine, which is essential for the proper transmission of signals throughout the basil ganglia. These neurochemical signals are vital for normal movement of the musculoskeletal system. It is the degeneration of the substantia nigra and subsequent reduction in dopamine production that is thought to elicit the symptoms of Parkinson's disease (National Parkinson's Foundation, 2001).

Contemporary research studies of Parkinson's disease have developed four currently accepted theories of the cause of the disease. The unknown etiology of the degeneration of the substantia nigra is referred to as *Idiopathic* Parkinsonism and may be caused by the mutation of the protein "alpha synuclein" or some other metabolic defect. Another type of Parkinsonism is designated as *Postinfectious* and thought to be caused by viral encephalitis. A third theory of the cause of Parkinson's disease is *Toxic* Parkinsonism and may be the product of exposure to industrial poisons or chemicals including the toxin MTPT. Finally *Arteriosclerotic* Parkinsonism may be due to an infarction or stroke in the basil ganglia causing Parkinson's disease symptoms to appear (Fitzsimmons & Bunting, 1993, The Parkinson's Institute, 2000). Unfortunately, none of these theories have been validated leaving researchers unable to pinpoint the specific cause or causes of Parkinson's disease. The only clear hypothesis validated from the medical research is that as the cells of the substantia nigra die, less dopamine is produced resulting in a multitude of debilitating physical and motor control symptoms.

There are two primary scales that describe and categorize the degree to which Parkinson's disease affects an individual. The "Hoehn and Yahr Staging of Parkinson's Disease" categorizes levels of Parkinson's disease affliction based on a scale from 1-5 with 1 identifying the mildest stage and 5 describing the most severe stage (Appendix A). This scale was developed at Columbia University by Dr. Margret Hoehn and Dr. Melvin Yahr in 1967 and describes only physical determinants of Parkinson's disease such as the degree of tremor and ability to ambulate (Hoehn et al., 1967). A more recent version has been established and is known as the "Modified Hoehn and Yahr Scale of Parkinson's Disease" (Appendix C). The "Unified Parkinson Disease Rating Scale" (UParkinson's diseaseRS) is a recent tool designed to "follow the longitudinal course" of Parkinson's disease (Appendix B) and is completed by interview whereas the Hoehn and Yahr Scale is designed as a clinical assessment without utilizing any patient input. The UParkinson's diseaseRS is comprised of four major sections focusing on the mental state, ability to perform ADL's, motor control and therapeutic response of each subject (National Parkinson's Foundation, 2001). These sections are further delineated into specific components rated on a scale of 0-4. A total of 222 points are possible and represents the most severe disability.

A third useful scale is the "Schwab and England Activities of Daily Living Scale" (Appendix D). This assessment utilizes ADL's as the discerning component of the rating and is based on patient response (The Parkinson's Institute, 2000). The scale is designed as a percentage of independency with 100% describing complete independence down to

0% describing a vegetative state. The scale is factored in increments of 10%. These and other assessment tools enable clinicians and researchers the ability to categorize levels of disability attributed to Parkinson's disease and evaluate components of motor control and daily function.

Another, more quantitative method of assessing individuals' motor function is by measuring the levels of muscular activation and sequence patterns using techniques of electromyography (Rossi et. al., 1996). Typical motor activation is comprised of three distinct phases. Phase one (pre-motor time) is the brief period prior to actual muscle contraction where minimal muscle activity is generated in preparation of the event performance. The second phase (motor time) represents the time from initial muscular contraction until the completion of the movement. This phase is where components of peak amplitude and spectral density of the muscle activation waveform are measured. Phase three (movement time) is the combination of pre-motor and motor time. Comparing these phases of muscle activity allows researchers to determine deficiencies in musculoskeletal mechanics by observing motor phase shifts of diminishment of waveform density. Subtle degradations of muscular motor control, not evident through clinical evaluation, may predispose individuals with neurological disorders to increased falls susceptibility and serious injury (Behrman et. al., 2000).

The work capacity of large muscle groups is directly related to an individual's ability to perform activities of daily living and occupational endeavors (Reuter et al., 1997). In order to measure work capacity, isokinetic strength assessments are used to determine the individual's ability to produce force, and to determine the balance or ratio of opposing muscle groups (e.g. flexors or extensors). Muscle dysfunction or weakness can be a limiting factor in volitional movement or gait in Parkinson's patients and the loss of muscular strength and mass is a common characteristic of the progression of the disease (National Parkinson's Foundation, 2001). Reductions in muscular strength and tone may also lead to deficits in: 1) neuromuscular coordination, balance, and precipitate difficulties in ambulation and susceptibility to falls, 2) predispose the Parkinson's patient to injury because of changes in muscle firmness or tone, and 3) restrict the protective cushioning of the skeletal system. In each context the muscle is weaker and compromises the self-reliance of the individual with as much as 88% of the postural variability in maintaining posture attributed to the lack of strength in the lower extremities (Hirsch, 1998).

Although previous studies have reported decreases in muscle mass and strength, the underlying mechanisms that predispose the individual to lose strength during the clinical progression of Parkinson's disease has proved to be elusive, especially in functional tasks. It is evident from previous work that several issues have not been sufficiently addressed in individuals with Parkinson's disease: 1) the degree of strength loss has not been adequately documented, 2) the affect of muscle coordination, motor recruitment and activation levels have not been correlated with the amount of muscle mass lost, 3) the degradation of physical function in Parkinson's patients has not been assessed via kinematic analysis, 4) the relationship of muscle function to functional tasks has not been established. Examining these and other research questions with regard to Parkinson's disease may provide researchers and clinicians information leading to the development of new and improved interventions to increase the strength, balance, and well being of persons with Parkinson's as well as reducing the rate of degeneration so prevalent with this neurological affliction.

Sit to Stand Transfer

The sit-to-stand transfer is operationally defined as the successful transfer of the body center of mass from a sitting to a stable standing position (Baer & Ashburn, 1995). This ADL is one that many individuals take for granted but perform numerous times in their daily activities. However, many people who are older or have some type of debilitating loss of motor function find the STS to be a significant challenge (Weiner et al., 1993). One of the primary causes of chair rise difficulty is the height of the seat (Rodosky et al., 1989, Vander Linden et al., 1994). Average seat heights range from 16 to 22 inches according to the National Institute for Occupational Safety and Health (1997) but previous studies have indicated that healthy older adults experience greater difficulty in rising as the seat height becomes progressively lower (Arborelius et al., 1992). Consequently individuals with motor control problems, specifically stroke patients with hemiplegia, experienced greater STS difficulty than their healthy age-matched peers (Lee, et. al., 1997, Cheng, P.T., 1998).

While there have been numerous studies examining the biomechanics of different types of chair rise with several different populations, there is a noticeable absence of data regarding participants with degenerative neurological disorders such as Parkinson's disease. Rationale to utilize the STS for this study includes several factors. First, the STS requires activation of the largest muscle groups of the musculoskeletal system, which allows for a greater magnitude of muscular activity (i.e. electrical output) to be recorded and analyzed. Second, the STS movement is perhaps the most common activity that individuals perform numerous times on a daily basis. Finally, the combination of force, EMG and kinematic data collection and analysis for the STS has never been published for a Parkinson's population.

Purpose of the Study

The purpose of this study was to compare the muscle activation levels, kinematics and three-dimensional force vectors between mild to moderate, elderly Parkinson's patients and healthy participants, during sit-to-stand transfers. More specifically, the purpose of the study was to conduct a bilateral assessment of sit-to-stand transfers using eight channel electromyography (EMG), two force platforms and a high-speed motion capture system that will reveal significant differences in mean and median frequency motor recruitment patterns, peak amplitude muscle activity levels, vertical ground reaction forces, and lower limb angular velocity and accelerations between participants with mild to moderate Parkinson's disease and healthy age-matched control participants.

Hypothesis

Although no specific documentation has been uncovered related to muscle activation, kinematics, and force production based on the decrease in functioning in this population, it is hypothesized that there will be significant differences between mild to moderate Parkinson's patients and healthy age-matched control participants, on all parameters, for: 1) lower body kinematics, 2) mean and median frequency motor recruitment, 3) patterns of peak amplitude muscle activity levels, and 4) three-dimensional force vectors assessed during sit to stand transfers. Therefore, the following hypotheses will be tested:

I. There will be significant differences between Parkinson's patients and healthy individuals in lower body kinematics.

II. There will be significant differences between Parkinson's patients and healthy individuals in peak EMG amplitude and median frequency.

III. There will be significant differences between Parkinson's patients and healthy individuals in three-dimensional and bilateral components of force application.

Statement of the Problem

The purpose of this investigation was to compare specific physical and motor characteristics of individuals with Parkinson's disease compared to their healthy peers and more specifically, to document specific differences related to physical functioning. Although clinical assessments have been developed to categorize the state and characteristics of Parkinson's disease, they do not generalize specifically to physical and motor control function.

Limitations of the Study

This study is limited to the sit-to-stand activity and it is an inherent limitation of this study that not every possible activity of daily living can be biomechanically assessed and compared between these populations. The results of this study relate to this sample and may or may not be generalizable to a common population. The participants for this study were randomly selected based on study criteria. However, factors such as 1) type of medication, 2) age, and 3) time of disease onset may account for a portion of the variability with this subject sample.

Delimitations of the Study

This study was delimited to twenty-four male participants, over the age of 60 years, who have been diagnosed with Parkinson's disease and are regarded as being mild to moderately afflicted (I – II on the Hoehn and Yahr scale). Participants were also free of any concomitant disabilities or musculoskeletal conditions that would interfere with their ability to complete the sit to stand task.

Definition of Terms

Terms described in the following section are defined as either functional which, refers to definitions and concepts as they apply to this study, or conceptual, referring to concepts that have been previously defined and accepted by recognized experts and authorities.

Functional Definitions

Electromyography (EMG): Measurement of the electrical activity of the excitable membranes of a muscle or group of muscles. (Kasman, et. al., 1997)

Kinetics: The forces acting on the body during movement and the interactions of sequence of motion with respect to time and forces present. (Thomas, 1997)

Kinematics: Branch of biomechanics concerned with the description of the movements of segments of the body without regard to the forces that caused the movement to occur. (Thomas, 1997)

Motor Recruitment: Refers to the summation of motor unit action potentials within one or more motor units or muscle fibers. (Thomas, 1997)

Torque: Describes the turning or twisting action of a force F when applied to and object with a fixed axis of rotation. (Halliday et al., 1993)

RMS EMG: Method of quantifying a surface EMG signal by summing the area (integrating) under the signal curve.

Mean Frequency EMG: The average frequency of the EMG signal.

Median Frequency EMG: The middle frequency of the EMG signal.

Sit to Stand Transfer (STS): An activity of daily living in which an individual is able to successfully rise from a chair to a stable standing position.

Conceptual Definitions

Activities of Daily Living (ADL): Term used to describe activities related to independent living and include preparing meals, managing money, shopping for groceries or personal items, performing light or heavy housework, and using a telephone.

Biomechanics: Field of study applying the principles of mechanics to the study of animate movement. (Anschel, et. al.,1991)

Akinesia: The complete inability to initiate or execute movement.

Arteriosclerotic: Referring to the hardening and thickening of the arteries causing a loss of elasticity in the blood vessels. (Anschel et al., 1991)

Basal Ganglia: Four masses of gray matter located deep in the cerebral hemispheres that secrete acetylcholine, dopamine, GABA, and serotonin and contribute to some of the subconscious aspects of involuntary movement. (Thomas, 1997)

Bilateral: Pertaining to two sides of the body. (Anschel et al., 1991)

Bradykinesia: Describes difficulty in initiating movement and slowness of movement.

Dopamine: A catecholamine neurotransmitter, or brain messenger, implicated in some forms of psychosis and abnormal movement disorders. (Thomas, 1997)

Dyskinesia: Condition characterized by motor restlessness, abnormal facial movements, and involuntary jerky or writhing movements. (Anschel et al., 1991)

Dysphagia: The inability to swallow or difficulty in swallowing. (Thomas, 1997)

Hemiparetic: Paralysis of only one side of the body. (Thomas, 1997)

Idiopathic: Relates to an unknown cause of disability or morbidity. (Anschel et al., 1991)

Palsy: Paralysis. (Thomas, 1997)

Parkinson's Disease: A chronic nervous system disease characterized by a fine, slowly spreading tremor, muscular weakness and rigidity, and a peculiar gait. (Thomas, 1997)

Power: The amount of work done per unit time. (Anschel et al., 1991)

Rigidity: Refers to difficulty with initiating movement, continuous muscle tension, and uncoordinated reciprocal muscle groups. (Anschel et al., 1991)

Strength: The ability of a muscle or muscle group to exert a force against a resistance. (Anschel et al., 1991)

Substantia Nigra: Largest nuclear mass of the midbrain containing neurons filled with melanin accounting for its black color (Young & Young, 1997)

Tremor: Involuntary, rhythmic, alternating bursts of movement of antagonistic muscle groups. (Anschel et al., 1991)

CHAPTER II

REVIEW OF LITERATURE

Introduction

This chapter presents a review of the current literature regarding the neurophysiology and motor coordination relating to the causes and affects of Parkinson's disease, the types of interventions presently under consideration was effective means to improve the debilitating progression of Parkinson's disease, and elements of the sit to stand transfer, and the biomechanical assessment techniques utilized to evaluate the underlying mechanisms of the motor control necessary to successfully perform this important ADL.

Neurophysiology

Motor coordination and musculo-skeletal movements are controlled by the integrated functions of the nervous system, which is comprised of the central (CNS) and peripheral nervous systems (PNS). The CNS consists of the brain and the spinal cord while the PNS includes neurons outside the CNS and is divided into sensory and motor portions. Sensory neurons that transmit impulses to the CNS and are more commonly referred to afferent nerves fibers. Motor neurons that conduct nerve impulses away form the CNS to the PNS and represent efferent nerve fibers (Goldman et al., 1998, Phillips et al., 1993).

There are essentially three types of relay circuits that transport motor impulses through the nervous system and are categorized according to their function.

These circuits include the pain, visual, and motor paths (Young & Young, 1997). The motor path or motor system is further divided into five groups of neurons including the; 1) lower motor, 2) pryamidal system, 3) basil ganglia, 4) cerebellar, and 5) brainstem. This five-group system is responsible for the activation and responses of the musculoskeletal system during all voluntary movement (Young & Young, 1997). The mechanisms involved in voluntary muscle contraction and movement follow a hierarchical sequence, from the initial thought stimulus to perform a movement, to the actual contraction of the desired muscle structure. Any interruption in the neural transmission pathway could cause a degradation or complete cessation of the desired movement (Johnson et al., 1998).

One of the primary CNS bodies responsible for the initiation of musculoskeletal activation is the basil ganglia (Bradshaw et al., 1998, Gabbard, 2000, Georgiou et al., 1998, Guyton, 1982, Young & Young, 1997). The basil ganglia is a large mass located deep within the midbrain between the cerebral hemispheres and is comprised of the corpus striatum, subthalamic nucleus, and substantia nigra. The basil ganglia has many complex interconnections directly involving other parts of the brain. These include both input and output connections both within and outside the structure (Young & Young, 1997). The basil ganglia is a vital component in the reception and transmission of information received from the cerebral cortex and efferent neurons. The substance that makes motor skills and coordination possible is the neurotransmitter dopamine (Hoehn & Yahr, 1967, National Parkinson's Foundation, Phillips et al., 1993).

Dopamine is produced within the basil ganglia by the substania nigra that gets its name from the black color of its cell bodies caused by the presence of melanin (Phillips et al., 1993, Society for Neuroscience, 1991). This neurotransmitter allows excitatory impulses to reach the subthalamic nucleus and pass to the afferent neurons in the PNS innervating the musculoskeletal system. Without dopamine, signals originating in the cerebral cortex are “stymied” and do not allow for complete and affective muscle responses (Society for Neuroscience, 1991, The Parkinson’s Institute, 2000). The lack of dopamine production by the substania nigra is the primary cause of the movement disorder of Parkinson’s disease (Bradshaw et al., 1998, Young & Young, 1997).

<u>Parkinsonism</u>

References to Parkinson’s disease can be traced back to Hippocrates in 400 BC (National Institute of Neurological Disorders and Stroke, 1998). However, it wasn’t until the early 19th Century that James Parkinson described in detail the nature of the disease that bears his name. At that time the cause of the involuntary palsy was unknown. It wasn’t until the 1950’s that doctors were able to discover that a reduction in dopamine production by the substantia nigra was the cause of Parkinson’s disease (Hoehn & Yahr, 1967, National Parkinson’s Foundation, 2001). It was also found that Parkinson’s disease could afflict even healthy individuals.

Parkinson’s disease affects approximately 1 out every 100 persons over the age of 60 and although there is a slightly greater incidence among men there are no indications that this is a gender related affliction (Fitzsimmons & Bunting, 1993). Currently there over four million persons worldwide that have been diagnosed with Parkinson’s disease and with the over 60 population expected to continue to be the fastest growing segment of society, it is anticipated that this number will rise dramatically over the next decade (The Parkinson’s Institute, 2000).

While there are no definitive conclusions concerning the manifestation of Parkinson's disease, there are four primary theories as to the cause of this movement disorder. Several studies have recently found what are thought to be the "key" to Parkinson's disease based on four currently accepted theories of the cause of the disease. The unknown etiology of the degeneration of the substantia nigra is referred to as *Idiopathic* Parkinsonism and may be caused by the mutation of the protein "alpha synuclein" or some other metabolic defect. Another type of Parkinsonism is designated as *Postinfectious* and thought to be caused by viral encephalitis. A third theory of the cause of Parkinson's disease is *Toxic* Parkinsonism and may be the product of exposure to industrial poisons or chemicals including the toxin MTPT. Finally *Arteriosclerotic* Parkinsonism may be due to an infarction or stroke in the basil ganglia causing Parkinson's symptoms to appear (Fitzsimmons et al., 1993, The Parkinson's Institute, 2000). Unfortunately, none of these theories have been validated leaving researchers unable to pinpoint the specific cause of Parkinson's disease.

Assessment Tools

The first validated assessment tool used to evaluate the physical effects of Parkinson's disease was developed by Drs. Hoehn and Yahr in the late 1960's (Appendix A). This scale was based on a five-stage description of the functional capacity of patients as the disease progressed. Table 2.1 presents the scale in its original form. A modified version of this classification system has been recently developed which further delineates the disease progression in "half stages" (Appendix C).

Table 2.1. Hoehn and Yahr Classification of Disability (Hoehn & Yahr, 1998)

STAGE	CHARACTER OF DISABILITY
I	Minimal or absent; unilateral if present
II	Minimal bilateral or midline involvement. Balance not impaired.
III	Impaired writing reflexes. Unsteadiness when turning or rising from a chair. Some activities are restricted, but patient can live independently and continue some forms of employment.
IV	All symptoms are present and severe. Standing and walking possible only with assistance.
V	Confined to bed or wheelchair.

A more recently developed rating tool is the Unified Parkinson's Disease Rating Scale (UPDRS) and consists of four definitive sections including; 1) mentation, behavior, and mood, 2) ADL, 3) motor, and 4) complications of therapy (Appendix B). The scale is a Likert form of assessment that is administered as a questionnaire and has a point range of 0 (no disability) to 222 (complete disability).

A third useful scale, though not as popular, is the "Schwab and England Activities of Daily Living Scale". This assessment utilizes ADL's as the discerning component of the rating and is based on patient response (Appendix D). The scale is designed as a percentage of independency with 100% describing complete independence down to 0% describing a vegetative state. The scale is factored in increments of 10%. These assessment devices all share a common purpose and that is to describe the clinical and functional manifestations of Parkinson's disease.

Indications

Though different, the above mentioned rating scales are all designed to classify an individual's ability to perform specific physical tasks and describe the degree to which the patient is afflicted with the disease. There are many physical, clinical and psychological symptoms associate with Parkinsonism. Physical manifestations of Parkinson's disease may include tremor or shaking palsy, ataxia, or bradykinesia. Table 2.2 presents an inventory of some of the more common physical impediments caused by Parkinsonism.

Table 2.2. Common physical symptoms of Parkinson's disease (Fitzsimmons & Bunting, 1993).

Resting Tremor	Joint Pain
Slowness of Movement	Fatigue
Weakness	Constipation
Internal Tremor	Lowered Voice Volume
Oily Face or Scalp	Bladder Urgency
Rigidity of Limbs	Restless Legs

Not only do these implications of Parkinson's preclude patients from performing normal daily activities but may also contribute to a reduction in muscle mass, pulmonary function and an overall decline in physiological well being (Cratty, 1989). As the enhanced physical decline of this population, due to age combined with the affects of Parkinson's disease, occurs clinical decrements in function can also be seen.

A combination of functional impairments may include shuffling gait, postural disorders and decreased balance control (Johnson et al., 1998). Table 2.3 provides a synopsis of clinically relevant symptoms associated with Parkinson's disease.

Table 2.3. Clinical impairments and symptoms of Parkinson's disease (Johnson et. al., 1998).

Shuffling Gait	Drooling
Postural Dysfunction	Lack of Arm Swing
Muscle Atrophy	Freezing in Place
Decreased Facial Expression	Decreased Swallowing

These impairments can have a dramatic impact on a patient's ability to maintain independence and quality of life by reducing their capability to perform ADL's at a level commensurate with their needs. As the decline in functional capacity continues the impact on the Parkinson's patient's emotional and psychological well being is also negatively affected (Lou et al., 2001). Feelings of despair and hopelessness can often pervade the individual's thought patterns and outlook on the future. This in turn can lead to an additional decrease in daily function and performance of ADL's due to a lack of motivation brought on by the physical impairment of Parkinson's disease. Table 2.4 provides concomitant psychological factors associated with Parkinson's disease.

Table 2.4. Psychological components of Parkinson's disease (Lou et al., 2001).

Depression	Decreased Motivation
Anxiety	Feeling of Hopelessness
Impaired Memory	Despair
Dementia	Public Embarrassment

It has been suggested that there is a positive linear relationship between the physical degradation associated with Parkinson's disease and the decline in patients' psychological assessment as it relates to emotion and attitude (Lou et al., 2001, Pachetti et al., 2000). However, this theory has not been validated via clinical trials and deserves further investigation.

Motor Rehabilitation/Exercise Therapy

The newest and perhaps least understood forms of therapy involve some form of movement rehabilitation. Recently physical therapists, occupational therapists, exercise physiologists and motor control specialists have formulated interventions, which focus on enhancing the strength and motor recruitment patterns in Parkinson's patients (Chan, 1986, Canning et al., 1997, Eldar & Marincek, 2000). This is being accomplished using resistance training, aerobic conditioning, and stretching and relaxation techniques.

Resistance training programs have shown to be moderately effective in small study samples of individuals (Krebs et al., 1998, Reuter et al., 1999, Scandalis et al., 2001). An increase in cross-section muscle area and density is thought to enhance voluntary motor control and perhaps illicit additional dopamine production by the basil ganglia. Aerobic conditioning protocols have been shown to increase motor coordination (Sunvisson et al., 1997), balance (Miyai et al., 2000), and well-being (Reuter et al., 1999). Additionally, stretching and relaxation programs have provided participants with increased self-confidence and levels of daily function. These methods of physiotherapy are thought to enhance vestibular stimulation thereby enabling the patient to better control their musculoskeletal system during activities of daily living. However, limited clinical trials have been conducted in this arena and further research is needed.

Balance/Vestibular Training

Several studies have examined the effects of balance training on individuals with Parkinson's disease (Behrman et al., 2000, Hirsch et al., 1998). These types of interventions may consist of center of mass perturbation training using Balance Master and Equitest devices, static and dynamic balance training using foam blocks or moveable standing surfaces (Hirsch et al., 1998), and Tai Chi training. It is well known that the degenerated motor control responses, due to Parkinson's disease, increase the risk of falls in this population (Ashburn et al., 2001). It is therefore important to intercede with these types of vestibular interventions to reduce the risk of falls and injury in an already elderly populace.

Cueing

A new type of intervention has recently been introduced that is more a component of psychomotor skills rather than musculoskeletal in nature (Berhman et al., 2000, Cunnington et al., 1999, Lee et al., 1995). Cueing or preparatory postural adjustments (Praamstra et al., 1996) have been shown to diminish the effects of Bradykinesia in Parkinson's disease patients. This training technique has the patient mentally "practice" a movement sequence or provide themselves with a mental "cue" that assists in activating musculoskeletal motor function initiation. Results from this research has shown significant reduction in the effects of Bradykinesia and akinesia in individuals with Parkinson's disease (Praamstra et al., 1996). This type of study provides additional insight to the nature of Parkinson's disease by suggesting that its cause is not entirely due to a decrease in dopamine production but may also be influenced by the strength of the initial motor process originating in the cerebral cortex.

Functional Ability

A final component of rehabilitation for the Parkinson's disease patient is one of assessing and improving the emotional and mental well being of the individual. Previous studies have provided results indicating a definite decrease in self-confidence, self-esteem, and motivation (Fitzsimmons & Bunting, 1993, Lou et al., 2001). This is due not only to the debilitation nature of Parkinson's disease but also in part to the age of most participants afflicted with this disorder. The combination of age and Parkinson's disease can severely limit the activities of this older population thereby inducing feelings of self-pity and depression (O'Sullivan & Schmitz, 2000, Pachetti et al., 2000).

Methods of treating these psychological effects can include drug therapy, family counseling, and support groups (National Parkinson's Foundation, 2001). Many organizations now offer free information, advice and contacts for persons afflicted with Parkinson's disease. The National Parkinson's Foundation (NPF), World Parkinson's Disease Association (WPDA), and The Parkinson's Institute (TPI) are but a few of the non-profit organizations designed to provide support to Parkinson's disease patients.

Summary

The current study utilized biomechanical measurement devices (i.e. videography, force platforms, electromyography) to monitor participants as they performed sit-to-stand transfers in the Veterans Affairs Medical Center, Rehabilitation Research and Development Center's biomechanics laboratory. All of these devices were integrated and synchronized during data collection. This allowed for an accurate assessment of each musculoskeletal component during each phase of the STS. Force and EMG data will be normalized where appropriate.

CHAPTER III

METHODS

This chapter outlines details concerning participant selection and recruitment, sit-to-stand testing protocol (seat height, chair rise format), and equipment set up (high-speed cameras, dual force platforms and 8 channel bi-lateral EMG) and synchronization (Motus system). Additional information includes the study research design (power analysis) and statistical procedures that will be used to analyze the data and present results.

Participants

A total of 24 men, age 60 years and older, were randomly recruited from the Veterans Affairs Medical Center (VAMC) and the Atlanta Parkinson's Disease Association (APDA). Thirteen participants with Parkinson's disease (PARK), who were categorized as a stage I or II Parkinson's patient (Stage II indicates minimal bilateral impairment without affect on balance) according to the Hoehn and Yahr (1967) scale (Appendix A), were selected based on medical data indicating their level of function and confirmed by the individual's neurologist. Eleven healthy age-matched participants were also recruited and grouped as controls (NORM). Inclusion criteria included: 1) the ability to ambulate 50 feet without assistance, and 2) ability to perform a minimum of 5 unassisted sit-to-stand transfers. Individuals with fluctuating responses to medication and functionally disabling dyskinesia or dystonia were excluded from this study. Six additional participants were unable to participate due to the inability to perform an STS without assistance or were unwilling to participate because of concerns regarding EMG skin preparation procedures. Additional exclusion criteria included individuals with pre-existing lung disease, history of cardiac disease, uncontrolled psychiatric illness, dementia, severe depression, and major neurological (stroke), musculoskeletal (rheumatoid arthritis), or metabolic disorders (diabetes).

Effects for medication were controlled, by requiring PARK participants to be tested at 2 hours post medicine ingestion, which is the approximate peak of the medication affect (Ouchi et al., 2001). Subjects read and signed Emory University Human Investigations Committee (HIC) and University of Georgia Internal Review Board (IRB) consent forms prior to beginning any testing.

Upon reading and signing the consent forms, participants were administered a six page demographic questionnaire (See Appendix E). This assessment tool records information regarding, physical condition, educational background, financial status as well as medication intake. Additionally, a 13-point Parkinson's Patient Self-Assessment questionnaire was administered (See Appendix F). This survey provides information regarding difficulties patients may be experiencing due to the onset of Parkinson's disease including speech, hygiene, and gait problems.

Instrumentation

Motion analysis laboratories generally have three common measurement devices used for kinematic data collection. High-speed videography, or motion capture, allows researchers to quantify the linear and angular mechanics of human motion. Referring to the STS movement, the kinematics would be measured by tracking reflective markers at specified joint locations allowing for the calculation of components such as center of mass displacement, angular velocity, and postural response (Stevens et al., 1989, Ikeda et al., 1991, Munro et al., 1998). Furthermore, these types of measures have been shown to be reliable and reproduceable in a laboratory environment (Jeng et al., 1990).

The second most common biomechanical assessment is made using force platforms, which utilize four or more transducers to monitor force, pressure, and torque in three planes of motion. These devices are useful in determining the direction and magnitude of force application during activities such as the chair rise (Riley et al., 1991). These data are normalized, by dividing the recorded value by the subject's bodyweight, which allows for inferential testing.

A third common component of biomechanical testing is that of electromyography (EMG). This measurement system records electrical activation of specific muscle groups during movements such as an STS transfer. The most common method of EMG is to attach surface electrodes over the motor endplate or muscle belly of a particular muscle group. This technique requires a certain amount of skin preparation but is much less invasive while still yielding reliable data. Skin preparation consists of first sterilizing the placement area with alcohol, shaving any excess hair, and then abrading the epidermis to remove the outer layer of dead skin cells. This preparation technique can significantly improve the reception of the EMG signal. Peak amplitude muscle activation, or rms EMG, mean and median frequency, or spectral density, as well as the differentiation of muscle activation sequences and phases, more commonly referred to as fractionated response, during the STS are common components of data recorded using EMG equipment (Kasman et al., 1998).

Kinematics

A kinematic assessment was conducted to examine postural and mechanical responses during the sit to stand movement. Reflective markers were placed at the bilateral lower body joint locations of: 1) distal end of fifth metatarsal, 2) lateral calcaneus, 3) lateral malleolus, 4) lateral tibia condyle, 5) lateral femoral condyle, and 6) greater trochanter. The kinetic and kinematic evaluation was conducted utilizing a Peak Performance Inc. high-speed motion analysis system, which recorded the STS movement at a rate of 120 fields per second. Four digital cameras were positioned at 90° intervals around the subject and recorded each trial in the frontal, sagittal, and transverse planes of movement. A twenty-seven point calibration frame was recorded in the STS "zone" prior to each subject being tested. This frame provides an arbitrary cartesian coordinate system in the recording zone and allows for each marker to be spatially tracked throughout the movement. Spatial data was calculated and analyzed using the Peak Performance Technologies, Inc., MOTUS operating system. Variables that were computed include joint displacement of the right and left knee, measured in degrees of displacement; angular velocity, measured in degrees per second; and angular acceleration, measured in degrees per second squared.

Force Platforms

The application of force to the ground during sit to stand transfers is an important element determining the success or failure in the movement performance. STS forces are applied primarily in the vertical plane, however, components of medio-lateral and antero-posterior forces can help determine participants' stability during the transfer. Two Advanced Medical Technologies, Inc. (AMTI) force platforms, positioned one under each foot, measured antero-posterior, medio-lateral and vertical force components during each trial. Input data was collected at a sampling frequency of 600 samples per second at a gain of 4,000 and was analyzed using a Peak Performance Technologies, Inc. MOTUS analysis software package. The input data from the device is recorded as a voltage level output from the force transducers located at the corners of the force plates. These signals are then filtered and converted to numerical force data (Newtons, Newtons/meter etc.) using standard algorithms. Outcome measures calculated included bilateral and vertical components of ground force application/reaction, and peak torque in the sagittal plane.

Electromyography

Successful completion of the sit to stand movement requires specific coordination of numerous lower body muscle groups. Faulty motor control circuitry caused by diseases such as Parkinson's may adversely affect an individual's ability to properly synchronize the musculature needed to rise from a seated position. Bilateral, bipolar surface EMG was used to determine the electrical activity of the vastus lateralis (LAT), vastus medialis (MED), and medial hamstrings (HAM) muscles during each STS trial. Silver/silver chloride surface electrodes were placed as close as possible to the estimated motor endplates of each muscle according to Kasman et al. (1997). The electrodes were positioned 2.5 cm apart from center to center with a common reference electrode placed over the head of the fibula. The skin was cleaned and abraded to achieve a skin impedance of $< 5K\ \Omega$. The EMG signal was digitized on-line with a sampling frequency of 1,024 Hz using a data acquisition card (DAS-16 Megabyte) processed through a Gateway 2000 Pentium III computer with high and low band-pass filters of 20 and 400 Hz respectively. The gain was set at 1,000 with a common mode rejection of 90 dB. The raw EMG signal was stored and the mean amplitude root mean square (rmsEMG) and median frequency (mfEMG) was calculated over the entire duration of motion of each STS trial. The rmsEMG was to be used as a measure of muscular activity using the formula of Basmajian and Deluca (1985). Normalized rmsEMG for the antagonistic muscle was calculated as a percentage of the rmsEMG activity from the same muscle used in the agonist phase. The mfEMG was processed using a Fast Fourier transform with a Hamming window to determine potential changes in motor unit recruitment occurring between agonist and antagonist contractions. All three data collection devices were calibrated and synchronized to begin recording upon an external trigger supplied by the experimenter.

Testing Protocol

Sit to stand transfer testing protocol required participants to rise from a sitting to standing position, from a seat height of 17 inches (Ikeda et al., 1991), and return to a sitting position after thirty seconds. The chair used was one with a rigid frame, no armrests, and was slightly padded. The chair was affixed to a wooden platform 4ft wide, 4ft long, and 4.5 inches high. This configuration allowed for a level transition between the surface of the force platforms and the chair location. Participants were required to perform three successful transfers. Data was collected on each testing instrument simultaneously, during each STS trial.

Research Design

This quasi-experimental design employed a 2 x 2 (group x leg) factorial analysis of variance (FANOVA) as a means to test for significant main effects between groups as well as within-group interactions (Cohen, 1988). A *post hoc* power analysis of the population sample, using total vertical ground reaction force as the primary outcome variable, revealed that at alpha = .05, n = 30, groups = 2, and a critical effect size = .65, the probability of rejecting a false null hypothesis of was .72 (power). Lower body kinetics were compared for significant differences while a kinematic assessment will be presented to describe differences between subject populations. Raw, three-dimensional, analog force data was divided by subject bodyweight (Kasman et al., 1997), which yields a normalized value of force. Raw electromyographic data was converted to root mean square (rmsEMG) and median frequency values and was compared based on percentage of total time and activation sequence events during the STS movement.

Data Analysis

This study computed means and standard deviations for the quantitative components of lower body kinetics, force production, and EMG mean and median frequency. Means and standard deviations are also calculated for the demographic measures of age, height, weight, and years of military service. A factorial analysis of variance (ANOVA) was employed at the $\alpha = .05$ level to test for significant between-group and within-group differences. Paired samples t-tests at $\alpha = .05$ level were conducted to examine significant within-group interactions. Qualitative comparisons include the use of Person's r zero-order correlation coefficient to determine relationships between groups and variables.

Outcome Measures

Data analysis includes; 1) time to stand, 2) vertical vectors of bilateral force application during rise, 3) rmsEMG peak amplitude, 5) EMG median frequency distribution, 5) knee angular displacements, velocities, and accelerations, 7) demographic data, and 8) Parkinson's patient self-assessment survey data. Data measures were analyzed at the specific phases, of the STS, as described by Riley et al. (1991). These phases include 1) flexion-momentum, 2) momentum transfer, 3) extension, and 4) stabilization. Data from this study was analyzed for the first three phases.

CHAPTER IV

RESULTS

This chapter presents details concerning the statistical analysis of the study data. Results include means and standard deviations, FANOVA and within group t-test results, as well as significant correlations for demographic, kinematic, force, and electromyographical data.

Demographics

Demographic variables assessed for this study include the following; 1) age, 2) height, 3) weight, 4) education level, 5) veteran status, and 6) years of military service. A complete copy of the demographic survey utilized is included in Appendix C. Means and standard deviations were calculated for these variables. A Levene statistic of homogeneity of variance was used to determine group variability differences. Additionally, a one-way analysis of variance was employed to determine significant between-group differences (Cohen, 1988).

The average age of Parkinson's participants was 70.2 ± 7.3 years while the mean age of the control group was 73.4 ± 6.7 years. Similar statistics were also calculated for HGT (PARK = 68.5 in ± 3.2, NORM = 70.4 in ± 2.3) and WGT (PARK = 176.2 lb ± 26.0, NORM = 184.4 lb ± 32.3). A Levene test was conducted to determine homogeneity of variance differences between groups. This statistic is used to compare between and within-group variability to determine if the data set adheres to the requirements of normality. Results indicate no significant differences in variability ($p > .05$) between PARK and NORM participants for the variables of AGE, HGT, and WGT.

Additional demographic data including education level attained, veteran status, and years of military service were also assessed. Approximately 62% of PARK participants completed at least a bachelor's degree program compared to only 22% for NORM participants. Further analysis revealed 92% of PARK participants completed and average of 6.7 years of military service with 89% of NORM participants completing an average of 3.2 years of service. Inferential testing of these variables, via analysis of variance, revealed no significant differences between groups ($p > .05$).

A Parkinson's Patient Self-Assessment questionnaire was administered to all Parkinson's disease participants in this study. This survey provides information regarding difficulties patients may be experiencing due to the onset of Parkinson's disease including speech, hygiene, and gait problems. Approximately 64% of Parkinson's patients reported experiencing some difficulty with speech and/or swallowing. Over 80% of participants reported problems with daily activities such as walking, writing, dressing, or food preparation. In addition, over 80% of Parkinson's participants reported experiencing visible tremors.

A final component of the demographic survey was presented to the Parkinson's participants and queried individual's exercise adherence before and after being diagnosed with Parkinson's disease as well as whether the participants felt exercise had help reduce the effects of Parkinson's disease on their daily activities. Over half (55.6%) of participants responded that they were actively engaged in exercise activities prior to their diagnosis while 78% of the participants admitted to participating in exercise after their diagnosis. When asked whether they felt that exercise had reduced the effects of Parkinson's disease, approximately 83% of participants responded positively.

This finding corroborates previous research examining the effects of exercise on mild to moderately afflicted Parkinson's patients (Rueter et al., 1999).

Kinematics

Included in Table 4.1 are means and standard deviations for the kinematic variables analyzed for this study. To determine if group variability differed significantly, a Levene test was performed (Cohen, 1988).

Table 4.1: Kinematic variables (Mean ± SD) of Parkinson and Control groups.

Variable	PARK (N = 13)	NORM (N = 12)
RISETIME (sec)	4.18 ± 1.63	3.79 ± 0.75
RTKNESO (deg)	93.65 ± 10.37	92.38 ± 7.77
LTKNESO (deg)	98.12 ± 12.19	98.18 ± 5.72
RTKNEVEL (deg/sec)	56.03 ± 23.37	57.58 ±13.68
LTKNEVEL (deg/sec)	58.05 ± 27.66	58.60 ±12.55
RTKNEACC (deg/sec*sec)	74.51 ± 35.51	73.04 ± 25.27
LTKNEACC (deg/sec*sec)	77.89 ± 36.16	89.75 ± 33.51

RISETIME – Total Time for Sit to Stand
RTKNESO – Angle of Right Knee at Seat Off
LTKNESO – Angle of Left Knee at Seat Off
RTKNEVEL – Peak Right Knee Angular Velocity
LTKNEVEL – Peak Left Knee Angular Velocity
RTKNEACC – Peak Right Knee Angular Acceleration
LTKNEACC – Peak Left Knee Angular Acceleration

Based on the results of the Levene statistic, no significant variability differences were apparent between-groups for the kinematic variables ($p > .05$). This finding indicates that the homogeneity of variance between groups is similar and is computed by comparing each cell mean to the absolute mean of the group sample (Cohen, 1988). A factorial ANOVA was then conducted to determine significant between-group mean differences for the kinematic variables. There were no significant between-group differences as determined by factorial ANOVA ($F \leq .451$, $p > .05$). However, when comparing within-group differences, using a normality plot, the PARK group was more variable although not statistically significant. Results from the FANOVA also indicated an interaction effect. A paired samples t-tests revealed significant differences in left and right knee angles at seat off ($t = 2.50$, $p = .028$) in the PARK group, with no right vs. left leg differences found in the NORM group ($p > .05$).

<u>Force</u>

Included in Table 4.2 are the means and standard deviations for the computed force variables. Peak vertical force measures were normalized, by dividing each subject's total bodyweight, in Newtons, into the force value, according to the percentage of total force applied by the right or left foot.

Table 4.2: Force variable comparison (Mean ± SD) of Parkinson and Control groups.

Variable	PARK (N = 13)	NORM (N = 12)
RTZFORNORM	6.21 ± .72	6.12 ± .51
LTZFORNORM	5.54 ± .81	5.79 ± .45
RTMMNTPK (N*m)	32.75 ± 11.63	25.12 ± 5.08
LTMMNTPK (N*m)	28.87 ± 6.76	23.25 ± 5.96
TOTFNORM (N)	11.7 ± 0.97	11.91 ± 0.60

RTZFNORM – Normalized Right Foot Peak Vertical Force
LTZFNORM – Normalized Left Foot Peak Vertical Force
RTMMNTPK – Peak Right Foot Torque Moment
LTMMNTPK – Peak Left Foot Torque Moment
TOTFNORM – Normalized Total Force

The Levene statistic revealed no significant between-group differences of variability ($F \leq 1.03$, $p > .05$), for any of the force variables calculated.

Variability within-groups was similar for the force variables to that of the kinematic results, in that the PARK participants displayed a greater degree of variation for each variable calculated.

A factorial ANOVA revealed no significant ($p > .05$) mean differences between groups at the $\alpha = .05$ level. However, within-group comparisons indicated significant differences ($p < .05$) by leg type (right vs. left). Paired-sample t-tests indicated significant differences between right and left legs in the PARK group for the variables of normalized peak vertical force ($t = 2.22$, $p = .047$) and peak torque moment ($t = 1.98$, $p = .041$). No significant ($p > .05$) within-group differences, by leg type, were found for participants in the NORM group.

Electromyography

Included in Table 4.3 are the means and standard deviations of median frequency by group. Electromyography data was analyzed according to techniques described by Cram et al. (1998).

Table 4.3: Median frequency EMG (Mean ± SD) of Parkinson and Control groups.

Variable	PARK		NORM	
RTLATMED (Hz)	79.06	33.67	73.71	13.21
LTLATMED (Hz)	71.54	26.18	71.30	22.38
RTMEDMED (Hz)	85.86	20.15	79.98	7.88
LTMEDMED (Hz)	80.73	27.31	80.12	10.59
RTHAMMED (Hz)	89.16	30.71	81.49	23.17
LTHAMMED (Hz)	88.30	36.2	73.80	21.81

RTLATMED – Right Vastus Lateralis Median Frequency
RTMEDMED – Right Vastus Medialis Median Frequency
RTHAMMED – Right Hamstring Median Frequency
LTLATMED – Left Vastus Lateralis Median Frequency
LTMEDMED – Left Vastus Medialis Median Frequency
LTHAMMED – Left Hamstring Median Frequency

A Levene statistic was employed to compare the homogeneity of group variance by variable. No significant between-group differences were found for either median frequency (LS ≤ 4.58, $p > .05$) or rmsEMG peak amplitude (LS ≤ 4.03, $p > .05$) for any of the six muscle groups tested. Parkinson's participants consistently exhibited higher median frequencies for both right and left legs as well as presenting higher degrees of variability than their healthy counterparts, indicating the need for this population to activate a greater amount of muscle fibers in order to properly complete the STS movement. A Pearson's r correlation coefficient was utilized to compare within-group relationships between right and left leg knee extensors. Parkinson's participants exhibited significant positive median frequency correlations between right and left vastus lateralis ($r = .839$) and vastus medialis ($r = .807$). In addition, a Pearson's r correlation was used to assess the within-group relationship between the muscular activity of the knee and hip extensors. Figures 4.1 - 4.4 represent the correlations between the vastus lateralis and bicep femoris muscles for the PARK and NORM groups. No significant correlations were found for the NORM group.

Peak amplitude data was calculated for means and standard deviation, factorial ANOVA, and Pearson's r correlation coefficients. Table 4.4 presents rmsEMG peak amplitude mean and standard deviation data.

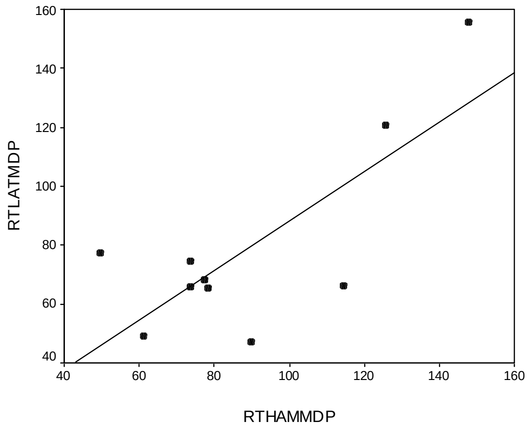
Table 4.4: EMG Peak amplitude (Mean ± SD) of Parkinson and Control groups.

Variable	PARK		NORM	
RTLATPK (v)	4.43	3.81	2.83	2.21
LTLATPK (v)	3.88	1.84	3.29	3.27
RTMEDPK (v)	2.27	1.42	2.23	1.62
LTMEDPK (v)	2.99	2.21	2.25	1.96
RTHAMPK (v)	0.81	0.55	0.71	0.45
LTHAMPK (v)	0.95	0.91	0.98	1.77

RTLATPK – Right Vastus Lateralis Peak Amplitude
RTMEDPK – Right Vastus Medialis Peak Amplitude
RTHAMPK – Right Hamstring Peak Amplitude
LTLATPK – Left Vastus Lateralis Peak Amplitude
LTMEDPK – Left Vastus Medialis Peak Amplitude
LTHAMPK – Left Hamstring Peak Amplitude

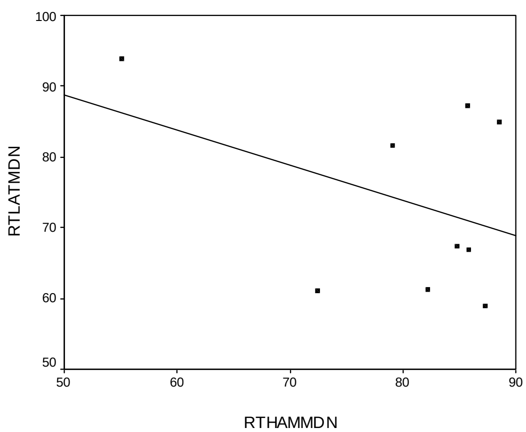
Results from a factorial ANOVA for rmsEMG peak amplitude data revealed no statistically significant differences between the PARK and NORM groups ($F \leq 1.22$, $p > .05$). Again, similar to the median frequency results, means and standard deviations for rmsEMG peak amplitudes were repeatedly higher for the Parkinson's patients, indicating higher levels of muscular activity and variability in the PARK participants.

Figure 4.1: Correlation between right knee and hip extensors for PARK participants (*r = .767).



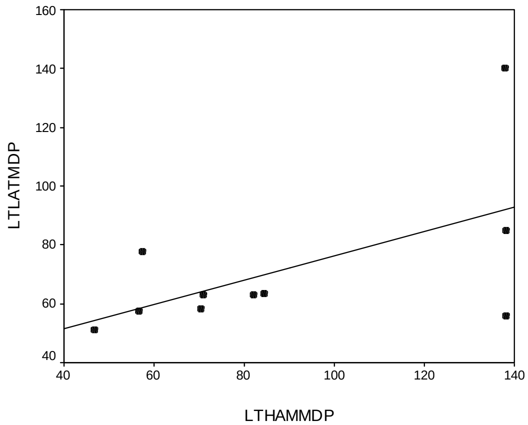
RTLATMDP: Right Lateralis Median Frequency PARK
RTHAMMDP: Right Hamstring Median Frequency PARK
* Represents significance at α = .05

Figure 4.2: Correlation between right knee and hip extensors for NORM participants (r = - .398).



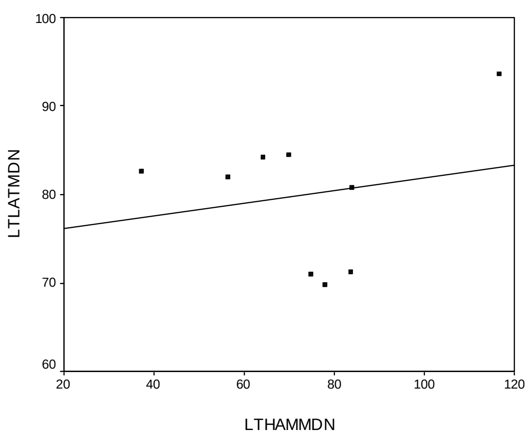
RTLATMDN: Right Lateralis Median Frequency NORM
RTHAMMDN: Right Hamstring Median Frequency NORM

Figure 4.3: Correlation between left knee and hip extensors for PARK participants (r = .570).



LTLATMDP: Left Lateralis Median Frequency PARK
LTHAMMDP: Left Hamstring Median Frequency PARK

Figure 4.4: Correlation between left knee and hip extensors for NORM participants (r = .198).



LTLATMDN: Left Lateralis Median Frequency NORM
LTHAMMDN: Left Hamstring Median Frequency NORM

CHAPTER V

DISCUSSION

The purpose of this study was to compare the muscle activation, kinematics, and force production, between Parkinson's patients and healthy, age-matched controls, during sit-to-stand transfers. The data from this study revealed several important findings. The first item of interest concerns the *lack* of statistically significant between-group (main effect) differences in the kinematics, ground reaction forces and EMG between Parkinson's patients and controls, during a sit to stand transfer. This outcome would lead the researcher to initially reject the *a priori* hypothesis if the statistical results were taken at "face value". However, other components of this study reveal there is indeed significant musculoskeletal and neuromotor variations between individuals with Parkinson's disease and controls.

Results from this research illustrate that individuals with mild to moderate Parkinson's disease are still able to perform the same daily activities as their matched controls, provided they utilize medication to control some of the effects of Parkinsonism (Ouchi et. al., 2001). However, the ability to produce the necessary musculoskeletal forces required to effectively perform ADL's does not necessarily reduce the risk of falls or indicate this population is as physically able as persons without Parkinson's disease. This view is substantiated by the findings that the PARK participants presented significant bilateral differences between right and left legs, during the STS ($p < .05$), for the variables of knee angle at seat off and peak vertical force. This is indicative of the unilateral manner in which Parkinson's disease afflicts persons in its early stages

(Johnson et. al., 1998). The inability to produce constant equilateral force during ADL's could lead to an increased propensity towards falls or other instabilities (Bradshaw et. al., 1998). Further evidence of the unilateral and destabilizing effects of Parkinson's disease can be seen in the higher degree of variability in the kinematics and force production in the PARK group compared to the NORM group. This higher degree of task variability has been presented from previous research (Ebmerier et. al., 1992, Georgiou et. al., 1998) and further reinforces current results that indicate the varying degrees to which Parkinson's disease affects different individuals. All of the kinematic and force variables calculated displayed up to twice the variability in the PARK group. Again, this finding reiterates the varying effects of Parkinson's disease from person to person and reveals the subtle differences between Parkinson's patients and their healthy peers not readily visible via qualitative assessment (Cunnington et. al., 1999).

Perhaps the most important finding from this study is the between and within-group differences in the muscle activation levels of the PARK participants. Parkinson's patients exhibited higher median frequencies as well as substantially higher muscle activation variability for both left and right legs compared to the NORM group. Previous research has indicated that persons with Parkinson's disease have a decreased sensitivity to extensor load mechanisms, which contribute to a diminished gait pattern (Dietz & Colombo, 1998). This diminished load sensitivity is indicated, in the current study, by the higher than normal median frequencies evident in the PARK patients. A reduction in load sensitivity could cause individuals with a neurological impairment to overestimate the amount of muscular contraction necessary to complete a required task such as an STS.

Further, it has been shown that the muscle activation amplitude and motor sequencing in Parkinson's disease patients is markedly disrupted and reduced (Lee et. al., 1995, Palmer et. al., 1991) during postural adjustments as indicated by disrupted center of pressure displacement. Current results revealed co-contraction of the left and right, knee and hip extensors during the STS, indicating that the PARK participants activated both muscle groups simultaneously in contrast to the NORM participants. This co-contraction may be evidence of the difficulties reported in motor sequencing problems exhibited by persons with Parkinson's disease (Rossi et. al., 1996).

Although Parkinson's disease is a progressively degenerative disease and normally treated with medication or through surgical intervention, recent studies have shown physical rehabilitation and exercise to be useful in improving Parkinson's patients' physical function. For example, Scandalis et al. (2001) found that persons with Parkinson's disease were able to increase their strength levels similar to healthy adults with a program that used a resistance training (Scandalis et. al., 2001). In contrast, improvements in strength alone do not necessarily correlate to functional improvements (Powers & Howley, 1997). Therefore it is essential for researchers and clinicians to focus not only on interventions and methods of assessment that will concentrate on improving function and mobility, but also the underlying components of motor control in this population.

Future research should examine the effects of a multitude of physical interventions for Parkinson's patients such as resistance training to improve strength, cardiovascular exercise to enhance endurance and aerobic capacity, and balance and vestibular training to improve spatio-temporal awareness and stability. The lack of literature associated with physical interventions and biomechanical evaluations of persons with Parkinson's disease, especially as it relates to EMG, results in a deficiency of understanding of the neuromuscular control mechanisms so important in effectively performing daily activities. Perhaps the co-contraction seen in this study is more indicative of functioning than the previously discussed scales. The conventions indicated are probably the most noteworthy aspect of this investigation and should be addressed in future studies.

In conclusion, while there were no statistically significant between-group differences found in this study, the distinct differences in ground force application and muscle activation levels and patterns in the PARK group, reveal that persons with Parkinson's disease do exhibit a reduced ability to perform common activities of daily living compared to their healthy peers. These findings also reinforce the notion that biomechanical assessments can reveal subtle physical and mechanical differences between study populations that would ordinarily avoid detection.

BIBLIOGRAPHY

1) Anshel, M., Freedson, P., Hamill, J., Haywood, K., Horvat, M., & Plowman, S.A. (1991). Dictionary of the sport and exercise sciences. Human Kinetics Books. Champaign, IL.

2) Arborelius, U.P., Wretenberg, P., & Linberg, F. (1992). The effects of armrests and high seat heights on lower-limb joint load and muscular activity during sitting and rising. *Ergonomics, 35, (11),* 1377-1391.

3) Ashburn, A., Stack, E., Pickering, R.M., & Ward, C.D. (2001). A community-dwelling sample of people with Parkinson's disease: characteristics of fallers and non-fallers. *Age and Ageing, 30, (1),* 47-52.

4) Baatile, J., Langbein, W.E., Weaver, F., Maloney, C., & Jost, M.B. (2000). Effect of exercise on perceived quality of life of individuals with Parkinson's disease. *Journal of Rehabilitation Research and Development, 37, (5),* 529-534.

5) Baer, G.D., & Ashburn, A.M. (1995). Trunk movements in older participants during sit-to-stand. *Archives of Physical Medicine and Rehabilitation, 76,* 844-849.

6) Basmajian, J.V. & DeLuca, C.J. (1985). Muscles Alive: Their function related by electromyography. *Baltimore: Williams and Wilkens,* 97-98.

7) Behrman, A.L., Cauraugh, J.H., & Light, K.E. (2000). Practice as an intervention to improve speeded motor performance and motor learning in Parkinson's disease. *Journal of the Neurological Sciences, 174, (2),* 127-136

8) Blin, O. Ferrandez, A.M., & Serratrice, G. (1990). Quantitative analysis of gait in Parkinson's patients: increased variability of stride length. *Journal of the Neurological Sciences, 98,* 91-97.

9) Bradshaw, J.L., Georgiou, N., Phillips, J.G., Iansek, R., Chiu, E., Cunnington, R., & Sheppard, D. (1998). Motor sequencing problems in Parkinson's disease, Huntinton's disease, and Tourette's syndrome 1: A review of basil ganglia involvement.

10) Brown, L.E. (2000). Isokinetics in human performance. Human Kinetics, Champaign, IL.

11) Brown, P., Corcos, D.M., & Rothwell, J.C. (1997). Does parkinsonian action tremor contribute to muscle weakness in Parkinson's disease? *Brain, 120,* 401-408.

12) Canning, C.G., Alison, J.A, Allen, N.E. & Groeller, H. (1997). Parkinson's Disease: An investigation of exercise capacity, respiratory function, and gait. *Archives of Physical Medicine and Rehabilitation, 78,* 199-207.

13) Castiello, U., Bennett, K., Bonfiglioli, C., Lim, S., & Peppard, R.F. (1999). The reach-to-grasp movement in Parkinson's disease: response to a simultaneous perturbation of object position and object size. *Experimental Brain Research, 125, (4),* 453-462.

14) Chan, C.W. (1986). Could parkinsonism akinesia be attributable to a disturbance in the motor preparatory process? *Brain Research, 386, (1-2),* 183-196.

15) Cheng, P.T. (1998). The sit-to-stand movement in stroke patients and its correlation with falling. *Archives of Physical Medicine and Rehabilitation, 79,* 1043-1046.

16) Cohen, J. (1988). Statistical power analysis for the behavioral sciences. 2nd ed. Hillsdale, NJ. Lawrence Erlbaum Associates, Publishers.

17) Cratty, B.J. (1989). APE in the Mainstream. 2nd ed. Denver, CO: Love Publishing Company.

18) Cunnington, R., Iansek, R., & Bradshaw, J.L. (1999). Movement –related potentials in Parkinson's disease: external cues and attentional strategies. *Movement Disorders, 14, (1),* 63-68.

19) De Goede, C.J.T., Keus, S. H.J., Kwakkel, G., & Wagenaar, R.C. (2001). The effects of physical therapy in Parkinson's disease: A research synthesis. *Archives of Physical Medicine and Rehabilitation, 82,* 509-515.

20) Dietz, V. & Colombo, G. (1998). Influence of body load on the gait pattern in Parkinson's disease. *Movement Disorders, 13, (2),* 255-261.

21) Doorenbach, C.A., Harlaar, J., Roebroeck, M.E. & Lankhorst, G.J. (1994). Two strategies of transferring from sit-to-stand; The activation of monoarticular and biarticular muscles. *Journal of Biomechanics, 27, (11),* 1299-1307.

22) Ebmerier, K.P., Potter, D.D., Cochrane, R.H., Crawford, J.R., Stewart, L., Calder, S.A., Besson, J.A., & Salzen, E.A. (1992). Event related potentials, reaction time, and cognitive performance in idiopathic Parkinson's disease. *Biological Psychology, 33, (1),* 73-89.

23) Elble, R.J. & Leffler, K. (2000). Pushing and pulling with the upper extremities while standing: the effects of mild Alzheimer dementia and Parkinson's disease. *Movement Disorders, 15, (2),* 255-268.

24) Eldar, R. & Marincek, C. (2000). Physical activity for elderly persons with neurological impairment: a review. *Scandinavian Journal of Rehabilitation Medicine, 32, (3),* 99-103.

25) Fitzsimmons, B. & Bunting, L.K. (1993). Parkinson's disease quality of life issues. *The Nursing Clinics of North America, 28, (4),* 807-818.

26) Gabbard, C.P. (2000). Lifelong motor development. Allyn and Bacon. Boston MA.

27) Georgiou, N., Bradshaw, J.L., Sheppard, D., Chiu, E., Iansek, R., Phillips, J.G., & Cunnington, R. (1998). Motor sequencing problems in Parkinson's disease, Huntington's disease, and Tourette's syndrome 2

28) de Goede, C.J.T., Keus, S.H.J., Kwakkel, G., & Wagenaar, R.C. (2001). The effects of physical therapy in Parkinson's disease: A research synthesis. *Archives of Physical Medicine and Rehabilitation, 82,* 509-515.

29) Goldman, W.P., Baty, J.D., Buckles, V.D., Sahrmann, S., & Morris, J.C. (1998). Cognitive and motor functioning in Parkinson'' disease: participants with and without questionable dementia. *Archives of Neurology, 55, (5),* 674-680.

30) Guyton, A.C. (1982). Human physiology and mechanisms of disease. W.B. Saunders Company, Philadelphia, PA.

31) Halliday, D., Resnick, R., & Walker, J. (1993). Fundamentals of physics. 4th edition. John Wiley & Sons, Inc., New York, NY.

32) Hanke, T.A., Pai, Y.C. & Rogers, M.W. (1995). Reliability of measurements of body center-of-mass momentum during sit-to-stand in healthy adults. *Physical Therapy, 75, (2),* 105-113.

33) Hanks, R.A., Rapport, L.J., Millis, S.R., & Deshpande, S.A. (1999). Measures of executive functioning as predictors of functional ability and social integration in a rehabilitation sample. *Archives of Physical Medicine and Rehabilitation, 80,* 1030-1037.

34) Hausdorff, J.M., Cudkowicz, M.E., Firtion, R., Wei, J.Y., & Goldberger, A.L. (1998). Gait variability and basal ganglia disorders: stride-to-stride variations of gait cycle timing in Parkinson's disease and Huntington's disease. *Movement Disorders, 13, (3),* 428-437.

35) Hayashi, R., Tokuda, T., Tako, K., & Yanagisawa, N. (1997). Impaired modulation of tonic muscle activities and H-reflexes in the soleus muscle during standing in patients with Parkinson's disease. *Journal of the Neurological Sciences, 153, (1),* 61-67.

36) Hirsch, M.A., Rider, R.A., Toole, T. & Hirsch, H.V.B. (1998). Falls prevention in individuals with Parkinson's disease. *Palaestra,* 15-22.

37) Hoehn, M.M. & Yahr, M.D. (1998). Parkinsonism: onset, progression, and mortality. 1967. *Neurology, 50, (2),* 318- 334.

38) Ikeda, E.R., Schenckman, M.L., Riley, P.O. & Hodge, W.A. (1991). Influence of age on dynamics of rising from a chair. *Physical Therapy, 71, (6),* 473-481.

39) Jeng, S.F., Schenkman, M., Riley, P.O., & Lin, S.J. (1990). Reliability of a clinical kinematic assessment of the sit-to-stand movement. *Physical Therapy, 70, 98),* 511-520.

40) Johnson, K.A., Cunnington, R., Bradshaw, J.L., Phillips, J.G., Iansek, R., & Rogers, M.A. (1998). Bimanual co-ordination in Parkinson's disease. *Brain, 121, (4),* 743-753.

41) Kakinuma, S., Nogaki, H., Pramanik, B., & Morimatsu, M. (1998). Muscle weakness in Parkinaon's disease: isokinetic study of the lower limbs. *European Neurology, 39, (4),* 218-222.

42) Kasman, G.S., Cram, J.R. & Wolf, S.L. (1997). Clinical applications in surface electromyography: Chronic musculoskeletal pain. *Aspen Publishing,* Frederick, MD.

43) Kaya, B.K., Krebs, D.E. & Riley, P.O. (1998). Dynamic stability in elders: momentum control in locomotor ADL. *Journal of Gerontology, 53, (2),* M126-134.

44) Knutsson,E. (1972). An analysis of parkinsonian gait. *Brain, 95,* 475-486.

45) Kotake, T., Nobuyuki, D., Kajiwara, T., Sumi, N, Koyama, Y, & Miura, T. (1993). An analysis of sit-to-stand movements. *Archives of Physical Medicine and Rehabilitation, 74,* 1095-1099.

46) Krebs, D.E., Jette, A.M., & Assmann, S.F. (1998). Moderate exercise improves gait stability in disabled elders. *Archives of Physical Medicine and Rehabilitation, 79,* 1489-1495.

47) Lee, M.Y., Wong, M.K., Tang, F.T., Cheng, P.T. & Lin, P.S. (1997). Comparison of balance responses and motor patterns during sit-to-stand task with functional mobility in stroke patients. *American Journal of Physical Medicine and Rehabilitation, 76, (5),* 401-410.

48) Lee, M.Y., Wong, M.K., Tang, F.T., Cheng, P.T., Chiou, W.K. & Lin, P.S. (1998). New quantitative and qualitative measures on functional mobility prediction for stroke patients. *Journal of Medical Engineering and Technology, 22, (1),* 14-24.

49) Lee, R.G., Tonolli, I., Viallet, F., Aurenty, R. & Massion, J. (1995). Preparatory postural adjustments in Parkinsonian patients with postural instability. *Canadian Journal of Neurological Science, 22, (2),* 126-135.

50) Lou, J.S., Kearns, G., Oken, B., Sexton, G., & Nutt, J. (2001). Exacerbated physical and mental fatigue in Parkinson's disease. *Movement Disorders, 16, (2),* 190-196.

51) Lundin, T.M., Grabiner, M.D., & Jahnigen, D.W. (1995). On the assumption of bilateral lower extremity joint moment symmetry during the sit-to-stand task. *Journal of Biomechanics, 28, (1),* 109-112.

52) Millington, P.J., Myklebust, B.M., & Shambes, G.M. (1992). Biomechanical analysis of the sit-to-stand motion in elderly persons. *Archives of Physical Medicine and Rehabilitation, 73, (7),* 609-617.

53) Mitoma, H. (1997). Kinematic and EMG pattern of Parkinsonian gait. *Nippon Rinsho, 55, (1),* 163-167.

54) Miyai, I., Fujimoto, Y., Ueda, Y., Yamamoto, H., Nozaki, S., Saito, T., & Kang, J. (2000). Treadmill training with body weight support: Its effect on Parkinson's disease. *Archives of Physical Medicine and Rehabilitation, 81,* 849-852.

55) Morris, M.E., Iansek, R., Matyas, T.A. & Summers, J.J. (1996). Stride length regulation in Parkinson's disease. Normalization strategies and underlying mechanisms. *Brain, 119,* 551-568.

56) Mourey, F., Grishin, A., d'Ahis, P., Pozzo, T., & Stapley, P. (2000). Standing up from a chair as a dynamic equilibrium task: A comparison between young and elderly participants. *Journal of Gerontology, 55A, (9),* B425-B431.

57) Munro, B.J., Steele, J.R., Bashford, G.M., Ryan, M., & Britten, N. (1998). A kinematic and kinetic analysis of the sit-to-stand transfer using an ejector chair: implications for elderly rheumatoid arthritic patients. *Journal of Biomechanics, 31,* 263-271.

58) Murray, M.P., Sepic, B.S., Gardner, G.M., & Downs, J.D. (1978). Walking patterns of men with Parkinsonism. *American Journal of Physical Medicine, 57, (6),* 278-294.

59) *National Institute of Neurological Disorders and Stroke – Cultured neural stem cells reduce symptoms in model of Parkinson's disease.* [On-line Serial], Available FTP: Hostname: medscape.com/govmt/ninds/1998/07.98/ninds.parkinsons.html.

60) *National Parkinson's Foundation—Parkinson Facts: 2001—What the patient should know* [On-line Serial], Available FTP: Hostname: parkinson.org Directory: pdedu.htm

61) O'Sullivan, S.B. & Schmitz, Thomas, J. (2000). Physical rehabilitation: Assessment and treatment. Philadelphia: F.A. Davis, 4th edition.

62) Ouchi, Y., Kanno, T., Okada, H., Yoshikawa, E., Futatsubashi, M., Nobezawa, S., Torisuka, T., & Tanaka, K. (2001). Changes in dopamine availability in the nigrostriatal and mesocortical dopaminergic systems by gait in Parkinson's disease. *Brain, 124, (4),* 784-792.

63) Pacchetti, C., Mancini, F., Aglieri, R., Fundaro, C., Martignoni, E., & Nappi, G. (2000). Active music therapy in Parkinson's disease: an integrative method for motor and emotional rehabilitation. *Psychosomatic Medicine, 62, (3),* 386-393.

64) Pai, Y.C. & Rogers, M.W. (1990). Control of body mass transfer as a function of speed of ascent in sit-to-stand. *Medicine and Science in Sports and Exercise, 22, (3),* 378-384.

65) Palmer, S.S., Eisenberg, P., & Mortimer, J.A. (1991). Pre-movement silence of EMG activity prior to movement in Parkinson patients. *Electromyographic Clinical Neurophysiology, 31, (7),* 425-431.

66) Phillips, J.G., Bradshaw, J.L., Iansek, R., & Chiu, E. (1993). Motor functions of the basil ganglia. *Psychological Research, 55, (2),* 175-181.

67) Phillips, J.G., Martin, K.E., Bradshaw, J.L., & Iansek, R. (1994). Could bradykinesia in Parkinson' disease simply be compensation? *Journal of Neurology, 241, (7),* 439-447.

68) Polcyn, A.F., Lipsitz, L.A., Kerrigan, D.C., & Collins, J.J. (1998). Age-related changes in the initiation of gait: degradation of central mechanisms for momentum generation. *Archives of Physical Medicine and Rehabilitation, 79,* 1582-1589.

69) Powers, S.K. & Howley, E.T. (1997). Exercise physiology. Theory and application to fitness performance. Brown & Benchmark Publishers, Dubuque, IA.

70) Praamstra, P., Meyer, A.S., Cools, A.R., Horstink, M.W., & Stegeman, D.F. (1996). Movement preparation in Parkinson's disease. Time course and distribution of movement related potentials in a movement pre-cueing task. *Brain, 119, (5),* 1689-1704.

71) Reuter, I., Engelhardt, M., Freiwaldt, J., & Baas, H. (1999). Exercise test in Parkinson's disease. *Clinical Autonomic Research, 9, (3),* 129-134.

72) Reuter, I., Engelhardt, M., Stecker, K., & Baas, H. (1999). Therapeutic value of exercise training in Parkinson's disease. *Medicine and Science in Sports and Exercise, 31, (11),* 1544-1549.

73) Riley, P.O., Schenkman, M.L., Mann, R.W., & Hodge, W.A. (1991). Mechanics of a constrained chair-rise. *Journal of Biomechanics, 24, (1),* 77-85.

74) Riley, P.O., Krebs, D.E., & Popat, R.A. (1997). Biomechanical analysis of failed sit-to-stand. *IEEE Transactions on Rehabilitation Engineering, 5, (4),* 353-359.

75) Rodosky, M.W., Andriacchi, T.P., & Andersson, G.B. (1989). The influence of chair height on lower limb mechanics during rising. *Journal of Orthopaedic Research, 7, (2),* 266-271.

76) Rogers, M.W. (1996). Disorders of posture, balance, and gait in Parkinson's disease. *Clinical Geriatric Medicine, 12, (4),* 825-845.

77) Rossi, B., Siciliano, G., Carboncini, M.C., Manca, M.L., Massetani, R., Viacava, P., & Muratorio, A. (1996). Muscle modifications in Parkinson's disease: Myoelectric manifestations. *Electroencephalographic Clinical Neurophysiology, 101, (3),* 211-218.

78) Scandalis, T.A., Bosak, A., Berliner, J.C., Helman, L.L., & Wells, M.R. (2001). Resistance training and gait function on patients with Parkinson's disease. *American Journal of Physical Medicine & Rehabilitation, 80, (1),* 38-43.

79) Schenkman, M. Hughes, M.A., Samsa, G., & Studenski, S. (1996). The relative importance of strength and balance in chair rise by functionally impaired older individuals. *Journal of the American Geriatrics Society, 44,* 1441-1446.

80) Schenkman, M., Cutson, T.M., Kuchibhatla, M., Chandler, J., Pieper, C.F., Ray, L., & Laub, K.C. (1998). Exercise to improve spinal flexibility and function for people with Parkinson's disease: a randomized, controlled trial. *Journal of the American Geriatrics Society, 46,* 1207-1216.

81) Smithson, F., Morris, M.E., & Iansek, R. (1998). Performance on clinical tests of balance in Parkinson's disease. *Physical Therapy, 78, (6),* 577-592.

82) Society for Neuroscience. (1991, August). *Brain Facts: A primer on the brain and nervous system.* Woodlawn, MD: Joseph Carey, ed.

83) Stack, E. & Ashburn, A. (1999). Fall events described by people with Parkinson's disease: implications for clinical interviewing and the research agenda. *Physiotherapy Research International, 4, (3),* 190-200.

84) Stanley, R.K., Protas, E.J., & Jankovic, J. (1999). Exercise performance in those having Parkinson's disease and healthy normals. *Medicine and Science in Sports and Exercise, 31, (6),* 761-766.

85) Stevens, C., Bojsen-Moller, F., & Soames, R.W. (1989). The influence of initial posture on the sit-to-stand movement. *European journal of Applied Physiology, 58,* 687-692.

86) Sunvisson, H., Lokk, J., Ericson, K., Winblad, B., & Ekman, S.L. (1997). Changes in motor performance in persons with Parkinson's disease after exercise in a mountain area. *Journal of Neuroscience Nursing, 29, (4),* 255-260.

87) *The Parkinson's Institute – Movement Disorders* (2000) [Online Serial] Available FTP: parkinsonsinstitute.org.movement_disorders/parkinsons.html

88) Thomas, C.L. (1997). Taber's cyclopedic medical dictionary. 18th ed., F.A. Davis Company, Philadelphia, Pa.

89) Urquhart, D.M., Morris, M.E., & Iansek, R. (1999). Gait consistency over 7-day interval in people with Parkinson's disease. *Archives of Physical Medicine and Rehabilitation, 80,* 696-701.

90) Vander Linden, D.W., Brunt, D., & McCulloch, M.U. (1994). Variant and invariant characteristics of the sit-to-stand task in healthy adults. *Archives of Physical Medicine and Rehabilitation, 75, (6),* 653-660.

91) Viliani, T., Pasquetti, P., Magnolfi, S., Lunardelli, M.L., Giorgi, C., Serra, P., & Taiti, P.G. (1999). Effects of physical training on straightening up process in patients with Parkinson's disease. *Disability and Rehabilitation, 21, (2),* 68-73.

92) Weiner, D.K., Long, R., Hughes, M.A., Chandler, J., & Studenski, S. (1993). When older adults face the chair-rise challenge. *Journal of the American Geriatrics Society, 41,* 6-10.

93) Wierzbicka, M.M., Staude, G., Wolf, W., & Dengler, R. (1993). Relationship between tremor and the onset of rapid voluntary contraction in Parkinson's disease. *Journal of Neurology and Neurosurgical Psychiatry, 56, (7),* 782-787.

94) Young, P.A. & Young, P.H. (1997). Basic clinical neuroanatomy. Lippincott, Williams & Wilkins. Philadelphia, Pa.

95) Ziv, I., Avraham, M., Michaelov, Y., Djaldetti, R., Dressler, R., Zoldan, J., & Melamed, E. (1998). Enhanced fatigue during motor performance in patients with Parkinson's disease. *Neurology, 51, (6),* 1583-1586.

APPENDIX A

Hoehn and Yahr Staging of Parkinson's Disease

Hoehn and Yahr Staging of Parkinson's Disease

I. Stage One
1. Signs and symptoms on one side only
2. Symptoms mild
3. Symptoms inconvenient but not disabling
4. Usually presents with tremor of one limb
5. Friends have noticed changes in posture, locomotion and facial expression

II. Stage Two
1. Symptoms are bilateral
2. Minimal disability
3. Posture and gait affected

III. Stage Three
1. Significant slowing of body movements
2. Early impairment of equilibrium on walking or standing
3. Generalized dysfunction that is moderately severe

IV. Stage Four
1. Severe symptoms
2. Can still walk to a limited extent
3. Rigidity and Bradykinesia
4. No longer able to live alone
5. Tremor may be less than earlier stages

V. Stage Five
1. Cachectic stage
2. Invalidism complete
3. Cannot stand or walk
4. Requires constant nursing care

APPENDIX B

Unified Parkinson's Disease Rating Scale (UPDRS)

UNIFIED PARKINSON'S DISEASE RATING SCALE

I. MENTATION, BEHAVIOR AND MOOD

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

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

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

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

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

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

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

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

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

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

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

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

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

13. Falling (unrelated to freezing)
0 = None.
1 = Rare falling.
2 = Occasionally falls, less than once per day.
3 = Falls an average of once daily.
4 = Falls more than once daily.

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

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

16. Tremor (Symptomatic complaint of tremor in any part of body.)
0 = Absent.
1 = Slight and infrequently present.
2 = Moderate; bothersome to patient.
3 = Severe; interferes with many activities.
4 = Marked; interferes with most activities.

17. Sensory complaints related to parkinsonism
0 = None.
1 = Occasionally has numbness, tingling, or mild aching.
2 = Frequently has numbness, tingling, or aching; not distressing.
3 = Frequent painful sensations.
4 = Excruciating pain.

III. MOTOR EXAMINATION

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

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

20. Tremor at rest (head, upper and lower extremities)
0 = Absent.
1 = Slight and infrequently present.
2 = Mild in amplitude and persistent. Or moderate in amplitude, but only intermittently present.
3 = Moderate in amplitude and present most of the time.
4 = Marked in amplitude and present most of the time.

21. Action or Postural Tremor of hands
0 = Absent.
1 = Slight; present with action.
2 = Moderate in amplitude, present with action.
3 = Moderate in amplitude with posture holding as well as action.
4 = Marked in amplitude; interferes with feeding.

22. Rigidity (Judged on passive movement of major joints with patient relaxed in sitting position. Cogwheeling to be ignored.)
0 = Absent.
1 = Slight or detectable only when activated by mirror or other movements.
2 = Mild to moderate.
3 = Marked, but full range of motion easily achieved.
4 = Severe, range of motion achieved with difficulty.

23. Finger Taps (Patient taps thumb with index finger in rapid succession.)
0 = Normal.
1 = Mild slowing and/or reduction in amplitude.
2 = Moderately impaired. Definite and early fatiguing. May have occasional arrests in movement.
3 = Severely impaired. Frequent hesitation in initiating movements or arrests in ongoing movement.
4 = Can barely perform the task.

24. Hand Movements (Patient opens and closes hands in rapid succesion.)
0 = Normal.
1 = Mild slowing and/or reduction in amplitude.
2 = Moderately impaired. Definite and early fatiguing. May have occasional arrests in movement.
3 = Severely impaired. Frequent hesitation in initiating movements or arrests in ongoing movement.
4 = Can barely perform the task.

25. Rapid Alternating Movements of Hands (Pronation-supination movements of hands, vertically and horizontally, with as large an amplitude as possible, both hands simultaneously.)
0 = Normal.
1 = Mild slowing and/or reduction in amplitude.
2 = Moderately impaired. Definite and early fatiguing. May have occasional arrests in movement.
3 = Severely impaired. Frequent hesitation in initiating movements or arrests in ongoing movement.
4 = Can barely perform the task.

26. Leg Agility (Patient taps heel on the ground in rapid succession picking up entire leg. Amplitude should be at least 3 inches.)
0 = Normal.
1 = Mild slowing and/or reduction in amplitude.
2 = Moderately impaired. Definite and early fatiguing. May have occasional arrests in movement.
3 = Severely impaired. Frequent hesitation in initiating movements or arrests in ongoing movement.
4 = Can barely perform the task.

27. Arising from Chair (Patient attempts to rise from a 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.

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

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

30. Postural Stability (Response to sudden, strong posterior displacement produced by pull on shoulders while patient erect with eyes open and feet slightly apart. Patient is prepared.)
0 = Normal.
1 = Retropulsion, but recovers unaided.
2 = Absence of postural response; would fall if not caught by examiner.
3 = Very unstable, tends to lose balance spontaneously.
4 = Unable to stand without assistance.

31. Body Bradykinesia and Hypokinesia (Combining slowness, hesitancy, decreased armswing, small amplitude, and poverty of movement in general.)
0 = None.
1 = Minimal slowness, giving movement a deliberate character; could be normal for some persons. Possibly reduced amplitude.
2 = Mild degree of slowness and poverty of movement which is definitely abnormal. Alternatively, some reduced amplitude.
3 = Moderate slowness, poverty or small amplitude of movement.
4 = Marked slowness, poverty or small amplitude of movement.

<u>IV. COMPLICATIONS OF THERAPY (In the past week)</u>

A. DYSKINESIAS

32. Duration: What proportion of the waking day are dyskinesias present? (Historical information.)
0 = None
1 = 1-25% of day.
2 = 26-50% of day.
3 = 51-75% of day.
4 = 76-100% of day.

33. Disability: How disabling are the dyskinesias? (Historical information; may be modified by office examination.)
0 = Not disabling.
1 = Mildly disabling.
2 = Moderately disabling.
3 = Severely disabling.
4 = Completely disabled.

34. Painful Dyskinesias: How painful are the dyskinesias?
0 = No painful dyskinesias.
1 = Slight.
2 = Moderate.
3 = Severe.
4 = Marked.

35. Presence of Early Morning Dystonia (Historical information.)
0 = No
1 = Yes

B. CLINICAL FLUCTUATIONS

36. Are "off" periods predictable?
0 = No
1 = Yes
37. Are "off" periods unpredictable?
0 = No
1 = Yes
38. Do "off" periods come on suddenly, within a few seconds?
0 = No
1 = Yes
39. What proportion of the waking day is the patient "off" on average?
0 = None
1 = 1-25% of day.
2 = 26-50% of day.
3 = 51-75% of day.
4 = 76-100% of day.

C. OTHER COMPLICATIONS

40. Does the patient have anorexia, nausea, or vomiting?
0 = No
1 = Yes
41. Any sleep disturbances, such as insomnia or hypersomnolence?
0 = No
1 = Yes
42. Does the patient have symptomatic orthostasis?
(Record the patient's blood pressure, height and weight on the scoring form)
0 = No
1 = Yes

APPENDIX C

Modified Hoehn and Yahr Scale

STAGE 0 = No signs of disease.

STAGE 1 = Unilateral disease.

STAGE 1.5 = Unilateral plus axial involvement.

STAGE 2 = Bilateral disease, without impairment of balance.

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

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

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

STAGE 5 = Wheelchair bound or bedridden unless aided.

APPENDIX D

Schwab and England Activities of Daily Living Scale

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

90% = Completely independent. Able to do all chores with some degree of slowness, difficulty and impairment. Might take twice as long. Beginning to be aware of difficulty.

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

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

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

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

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

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

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

10% = Totally dependent, helpless. Complete invalid.

0% = Vegetative functions such as swallowing, bladder and bowel functions are not functioning. Bedridden.

APPENDIX E

Demographics

DEMOGRAPHICS

Date of birth ____________________

Gender: Male Female

Military Service Branch: Years:

Height (inches):

Weight (lbs):

Hoehn and Yahr Classification:

What is your ethnic background?

1. African-American
2. Asian
3. Caucasian
4. Hispanic
5. Multi-racial
6. Native American
7. Other

What is your marital status?

1. Single, never married
2. Married
3. Separated or divorced
4. Widowed
5. Other ____________

How many children do you have living at the present time?

1. None
2. One
3. Two
4. Three
5. Four or more

In what type of dwelling do you currently reside?

1. Single-family house
2. Apartment, condo, or townhouse
3. Mobile home/trailer
4. Group home
5. Retirement community (no central dining)
6. Retirement home (central dining)
7. Assisted living facility
8. Nursing home

Who currently resides with you? (Circle all that apply.)

1. No one (lives alone)
2. Spouse
3. Child(ren)
4. Other relative(s)
5. Friend(s)
6. Non-related paid helper
7. Reside in institution

What is the highest level of education you attained?

1. 0-4 years
2. 5-8 years
3. High school incomplete
4. High school completed
5. Post high school, business or trade school
6. Some college, incomplete
7. College completed
8. Post graduate college

What would you estimate your yearly personal income to be before taxes?

1. Under 2,000
2. 2,000-4,999
3. 5,000-9,999
4. 10,000-14,999
5. 15,000-19,999
6. 20,000-24,999
7. 25,000-34,999
8. 35,000-49,999
9. 50,000 or more

MEDICAL HISTORY

Do you have any of the following illnesses or disabilities at the present time? If so, how much does it interfere with your normal activities—not at all, a little, or a great deal? Please circle the appropriate response.

	NO	IF YES		
	(0)	(1) Not at all	(2) A little	(3) A great deal
Heart Circulation				
High blood pressure	0	1	2	3
Circulation trouble in arms and legs	0	1	2	3
Previous heart attack	0	1	2	3
Recurrent chest pain	0	1	2	3
Heart Failure	0	1	2	3
Any other heart trouble	0	1	2	3
	0	1	2	3
Pulmonary/Lung problems				
Ephysema or chronic bronchitis	0	1	2	3
Asthma	0	1	2	3
Tuberculosis	0	1	2	3
Other	0	1	2	3
Orthopedic				
Arthritis or rheumatism	0	1	2	3
Osteoporosis	0	1	2	3
Missing limb or amputation	0	1	2	3
Effects of fracture or broken bone	0	1	2	3
Shoulder pain	0	1	2	3
Metabolic/Endocrine				
Diabetes	0	1	2	3
Thyroid or other glandular disorders	0	1	2	3
Stomach problems				
Ulcers	0	1	2	3
Liver disease or cirrhosis	0	1	2	3
Other	0	1	2	3

	NO	IF YES		
	(0)	**(1) Not at all**	**(2) A little**	**(3) A great deal**
Neurologic/Psychiatric				
Effects of stroke	0	1	2	3
Parkinson's Disease	0	1	2	3
Multiple sclerosis	0	1	2	3
Cerebral palsy	0	1	2	3
Epilepsy or seizure	0	1	2	3
Head injury	0	1	2	3
Spinal cord injury	0	1	2	3
Depression	0	1	2	3
Anxiety	0	1	2	3
Sensory				
Low vision/blindness	0	1	2	3
Glaucoma or cataracts	0	1	2	3
Macular degeneration	0	1	2	3
Diabetic retinopathy	0	1	2	3
Hearing loss	0	1	2	3
Speech impediment or impairment	0	1	2	3
Other				
Cancer or leukemia	0	1	2	3
Anemia	0	1	2	3
Pressure sores, leg ulcers, or burns	0	1	2	3
Serious kidney disease or renal failure	0	1	2	3
Other ______________	0	1	2	3

MEDICATIONS

Are you currently taking, or have you taken in the last month, any of the following medications?

a. Arthritis medication
b. Prescription pain killer (other than arthritis medication)
c. High blood pressure medicine
d. Pills to make you lose water or salt (water pills)
e. Digitalis pills for the heart
f. Nitroglycerin tablets for chest pain
g. Blood thinner medicine (anticoagulants)
h. Drugs to improve circulation
i. Insulin injections for diabetes
j. Pills for diabetes
k. Seizure medication (like Dilantin)
l. Thyroid pills
m. Cortisone pills or injections
n. Antibiotics
o. Medicine for nerves or depression
p. Prescription sleeping pills (once a week or more)
q. Hormones, male or female (including hormone replacement therapy)
r. Other (SPECIFY)

APPENDIX F

Patient Self-Assessment

PATIENT SELF-ASSESSMENT

Patient ______________________________
Date ___________ Time __________

For each of these questions, check the **one statement** that best describes the difficulties you've been having because of your Parkinson's disease. These questions refer to the part of the day when your medicine is providing you with maximum benefit.

1. Have you noticed that your speech has changed? Do you have problems speaking?

- ❑ **No** My speech is normal.
- ❑ **Yes** My speech is mildly affected, but I have no difficulty being understood.
- ❑ **Yes** My speech is moderately affected and I am sometimes asked to repeat myself.
- ❑ **Yes** My speech is severely affected and I am sometimes asked to repeat myself.
- ❑ **Yes** My speech is so severely affected that it is very hard for people to understand me.

2. Have you noticed that you have too much saliva?

- ❑ **No** I do not have too much saliva and I never drool.
- ❑ **Yes** I have a slight excess of saliva. Sometimes I drool onto my pillow at night.
- ❑ **Yes** I have a moderate excess of saliva and I occasionally drool during the daytime.
- ❑ **Yes** I have a marked excess of saliva and I often drool during the daytime.
- ❑ **Yes** I have so much drooling that I often carry a tissue or handkerchief.

3. Do you have problems swallowing or do you choke on your food?

- ❑ **No** I do not have a problem with swallowing and I do not choke.
- ❑ **Yes** I have problems with swallowing, but I rarely choke.
- ❑ **Yes** I have problems with swallowing and I occasionally choke.
- ❑ **Yes** I have problems with swallowing requiring me to eat soft food.
- ❑ **Yes** I am unable to swallow and must use a nasogastric or gastrostomy tube.

4. Have you noticed a change in your handwriting? Do you have difficulty writing?

- ❑ **No** My handwriting is normal.
- ❑ **Yes** My handwriting is slightly slow or small.
- ❑ **Yes** My handwriting is moderately slow or small, but all of the words are readable.
- ❑ **Yes** My handwriting is severely affected. Not all of the words are readable.
- ❑ **Yes** My handwriting is severely affected. Most of the words are **not** readable.

5. Do you have slowness or difficulty using utensils or cutting your food?

- ❑ **No** I do not have slowness or difficulty using utensils or cutting my food.
- ❑ **Yes** I am a little slow or clumsy, but I am able to feed myself without help.
- ❑ **Yes** I am slow and clumsy. I need help cutting some types of food.
- ❑ **Yes** My food must be cut by someone, but I am still able to feed myself.
- ❑ **Yes** I unable to feed myself. Someone else feeds me.

6. Do you have some difficulties with dressing?

- ❑ **No** I do not have slowness or difficulty with dressing.
- ❑ **Yes** I am a little slow, but I don't need help.
- ❑ **Yes** I am slow and I sometimes need help buttoning buttons, tying shoelaces, or getting my arm into a sleeve.
- ❑ **Yes** I need a lot of help getting dressed, but I can still do some things alone.
- ❑ **Yes** I am unable to get dressed without assistance.

7. Have you slowed down or are you experiencing problems with hygiene (bathing, brushing your teeth, combing your hair, going to the bathroom)?

- ❑ **No** I am not slow with these activities.
- ❑ **Yes** I am a little slow with my hygiene, but I do not need help.
- ❑ **Yes** I am slow with my hygiene and I need help to shower and bathe.
- ❑ **Yes** I need help with washing, brushing my teeth, combing my hair, and going to the bathroom.
- ❑ **Yes** I need help with all of my hygiene and I have a Foley catheter.

8. Do you have difficulty turning in bed or adjusting the sheets?

- ❑ **No** I do not have difficulty turning in bed or adjusting the sheets.
- ❑ **Yes** I am a little clumsy and slow with turning in bed and adjusting the sheets, but I do not need help.
- ❑ **Yes** I am only able to turn or adjust the sheets with great difficulty.
- ❑ **Yes** I am able to start turning, but am unable to do it without help.
- ❑ **Yes** I am unable to turn in bed or adjust the sheet without help.

9. Do you have problems with falling?

- ❑ **No** I do not fall.
- ❑ **Yes** I rarely fall.
- ❑ **Yes** I occasionally fall, but less than once per day.
- ❑ **Yes** I fall an average of one time per day.
- ❑ **Yes** I fall an average of more than one time per day.

10. Do you have freezing while you are walking? (“Freezing” occurs when you are unable to walk for a few seconds because your feet seem to be stuck to the ground.)

- ❑ **No** I do not have “freezing.”
- ❑ **Yes** I have “freezing” when I walk, but this happens rarely; OR sometimes, when I first start to walk, I have “freezing.”
- ❑ **Yes** I occasionally have “freezing” when I walk.
- ❑ **Yes** I frequently have “freezing” when I walk. I occasionally fall because of the “freezing.”
- ❑ **Yes** I frequently have “freezing” when I walk. I frequently fall because of the “freezing.”

11. Has your walking changed? Is it difficult to walk?

- ❑ **No** My walking and my arm swing have not changed.
- ❑ **Yes** I don’t swing my arm or I tend to drag my leg.
- ❑ **Yes** I have a moderate amount of difficulty with walking but usually don’t need assistance.
- ❑ **Yes** I have severe problems with walking and usually need assistance.
- ❑ **Yes** I can’t walk at all, even when someone tries to help me.

12. Do you have a visible tremor anywhere in your body?

- ❑ **No** I do not have a visible tremor.
- ❑ **Yes** I have a slight visible tremor that is infrequently present.
- ❑ **Yes** I have a moderate amount of tremor. The tremor bothers me.
- ❑ **Yes** I have a severe tremor and it interferes with many activities.
- ❑ **Yes** I have a severe tremor and it interferes with most activities.

13. Do you have numbness, tingling, discomfort, or aching, which you attribute to your Parkinson’s disease?

- ❑ **No** I do not have numbness, tingling, or aching, which I attribute to my Parkinson’s disease.
- ❑ **Yes** I have occasional numbness, tingling, or aching, which I attribute to my Parkinson’s disease.
- ❑ **Yes** I frequently have numbness, tingling, or aching, which I attribute to my Parkinson’s disease.
- ❑ **Yes** I have frequent painful sensations, which I attribute to my Parkinson’s disease.
- ❑ **Yes** I have excruciating pain, which I attribute to my Parkinson’s disease.

Exercise Component:

14. Did you exercise on a regular basis before you were diagnosed with Parkinson's?

- ❑ **No**
- ❑ **Yes**, one time per week
- ❑ **Yes**, two times per week
- ❑ **Yes**, three times per week
- ❑ **Yes**, for or more times per week

15. Have you exercised regularly since you were diagnosed with Parkinson's?

- ❑ **No**
- ❑ **Yes**, one time per week
- ❑ **Yes,** two times per week
- ❑ **Yes**, three times per week
- ❑ **Yes**, four or more times per week

16. Do you feel like regular exercise has helped you reduce the effects of Parkinson's with regard to your daily activities?

- ❑ **No**
- ❑ **Yes**, a little
- ❑ **Yes**, a moderate amount
- ❑ **Yes**, quite a bit
- ❑ **Yes**, very much