BIOMECHANICAL AND STRENGTH FACTORS UNDERLYING PERFORMANCE OF THE SIT-TO-STAND TASK IN PERSONS WITH PARKINSON'S DISEASE

By

LISA MARY INKSTER

B.Sc.(PT) The University of Alberta, 1989

A THESIS SUBMITTED IN PARTIAL FULFILMENT OF THE REQUIREMENTS FOR THE DEGREE OF

MASTER OF SCIENCE

in

THE FACULTY OF GRADUATE STUDIES

School of Rehabilitation Sciences

We accept this thesis as conforming to the required standard

THE UNIVERSITY OF BRITISH COLUMBIA

April 2001
© Lisa Mary Inkster, 2001
In presenting this thesis in partial fulfilment of the requirements for an advanced degree at the University of British Columbia, I agree that the Library shall make it freely available for reference and study. I further agree that permission for extensive copying of this thesis for scholarly purposes may be granted by the head of my department or by his or her representatives. It is understood that copying or publication of this thesis for financial gain shall not be allowed without my written permission.

Department of Rehabilitation Science

The University of British Columbia
Vancouver, Canada

Date April 17/01
Biomechanical and Strength Factors Underlying Performance of the Sit-to-Stand Task in Persons with Parkinson’s Disease

Purpose: Up to 80% of individuals with Parkinson’s disease (PD) report having difficulty with rising from a chair. The purpose of this study was to 1) quantify how sit-to-stand (STS) performance differs between persons with PD and age-matched controls, and 2) determine if there is a relationship between lower extremity strength and performance of STS.

Methods: Ten male subjects with PD (64.1 ± 10.1 years) and ten male age-matched controls (65.5 ± 12.4 years) participated. Subjects were instructed to rise from a backless, knee-height chair without the use of their arms. Subjects with PD were tested on two separate days in an on and off-medication state. Lower extremity and trunk movements and muscle activity (EMG) were recorded, in addition to forces under the buttocks and feet. A Kin-Com strength dynamometer was used to measure concentric, isokinetic knee and hip extensor torque. Paired t-test assessed differences between groups for 1) biomechanical parameters during the STS and 2) torque from the strength dynamometer. Pearson product correlations were used to assess the relationship between strength (torque) and STS performance (duration).

Results: For the STS, the movement duration, EMG, joint-angles and joint moments (timing and magnitude) were similar between subjects with PD and controls except for a lower and earlier (prior to lift-off of the buttocks) peak knee extensor moment and absence of preparatory abdominal activity in subjects with PD. Hip and knee strength, as assessed by isokinetic torque, was generally lower for subjects with PD. Greater hip strength was related to better STS performance in subjects with PD and greater knee strength was related to better STS performance in controls.

Conclusions: Lower extremity weakness, in addition to altered preparatory activity (early and low peak knee extensor moment and lack of abdominal activity) may contribute to the difficulties that individuals with PD have in rising from a chair. In addition, these findings suggest that performance of the STS task in subjects with PD is more dependent on the hip than on the knee strength.
TABLE OF CONTENTS

Abstract ii
Table of Contents iii
List of Tables vi
List of Figures vii
Acknowledgements viii

GENERAL INTRODUCTION 1

CHAPTER 1: A Biomechanical Analysis of the Sit-to-Stand Task in Persons with Parkinson’s Disease

Abstract 4

1 Introduction 5
1.1 Central Factors 5
1.2 Clinical Signs of Parkinson’s disease 6
1.2.1 Bradykinesia 7
1.2.2 Rigidity 8
1.2.3 Tremor 8
1.2.4 Impaired Postural Reflexes 9
1.3 Peripheral Factors 10
1.4 Evaluation of the Sit-to-Stand Task 10

2 Purpose 11

3 Methods 11
3.1 Setting 11
3.2 Subjects 11
3.3 Protocol 16
3.4 Data Collection 17
3.4.1 Kinetic and Kinematic Data 17
3.4.2 Electromyographic Recordings 18
3.5 Data Analysis 19
3.5.1 Identification of Key Events 19
3.5.2 Symmetry 20
3.5.3 Time-Normalizing 21
3.5.4 Electromyographic Recordings 21
3.5.5 Statistical Analysis 22
4 Results
4.1 Duration of Sit-to-Stand
4.2 Symmetry
4.3 Joint Moments
4.3.1 Joint Moment Profiles
4.3.2 Timing of Peak Joint Moments
4.3.3 Magnitude of Peak Joint Moments
4.4 Joint Angles
4.4.1 Joint Angle Profiles
4.4.2 Timing of Joint Angle Displacement
4.4.3 Magnitude of Joint Angle Displacement
4.5 Electromyographic Recordings
4.5.1 Sequencing of Muscle Activity
4.5.2 Area of EMG

5 Discussion
5.1 Duration
5.2 Preparation Phase
5.3 Lift-off Phase
5.4 Motor Rating Scales
5.5 Clinical Implications

6 Limitations

CHAPTER 2: The Relationship Between Lower Extremity Strength and Performance of the Sit-to-Stand Task in Persons with Parkinson’s Disease

Abstract

7 Introduction
7.1 Strength and the Sit-to-Stand Task
7.2 Performance of Sit-to-Stand in Parkinson’s disease
7.3 Force Generation in Parkinson’s disease
7.4 Relationship Between Strength and Function

8 Purpose

9 Methods
9.1 Setting
9.2 Subjects
9.3 Protocol
9.4 Data Collection
9.4.1 Kinetic and Kinematic Data
9.4.2 Isokinetic Strength Testing
9.4.2 (i) Knee Extension
9.4.2 (ii) Hip Extension
9.5 Data Analysis
9.5.1 Identification of Duration of Sit-to-Stand 52  
9.5.2 Kin-Com Data Analysis 53  
9.5.3 Statistical Analysis 53  

10 Results 54  

11 Discussion 58  
11.1 Isokinetic Average Torque 58  
11.2 Percent Effort 59  
11.3 Relationship Between Strength and Function 61  
11.4 Implications for Physical Therapy 62  

12 Limitations 63  

**GENERAL CONCLUSIONS** 65  

13.1 Strength and Function 66  
13.2 Impaired Preparatory Activity 67  
13.3 Clinical Implications 67  
13.4 Future Ventures 68  

REFERENCES 70  

**APPENDICES**  

<table>
<thead>
<tr>
<th>Appendix</th>
<th>Title</th>
<th>Pages</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Modified Hoehn &amp; Yahr Stages of Parkinson's Disease</td>
<td>78</td>
</tr>
<tr>
<td>B</td>
<td>Hoehn &amp; Yahr Stages of Parkinson's Disease</td>
<td>79</td>
</tr>
<tr>
<td>C</td>
<td>Motor Section of the Unified Parkinson's Disease Rating Scale</td>
<td>80</td>
</tr>
<tr>
<td>D</td>
<td>Electrode Placement Guidelines</td>
<td>83</td>
</tr>
</tbody>
</table>
LIST OF TABLES

CHAPTER 1

Table 1: Characteristics of Subjects with Parkinson’s disease 13
Table 2: Motor Scoring of Subjects with Parkinson’s disease 14
Table 3: Characteristics of Control Subjects 15
Table 4: Mean Duration (N=10) to Perform One Sit-to-Stand Maneuver (Duration in seconds) 24
Table 5: Mean (N=10) Symmetry Index (Ratio of vertical force at lift-off: limb with higher force divided by limb with lower force) 24
Table 6: Mean Timing (N=10) of Peak Joint Moments (% of STS cycle) 25
Table 7: Mean Magnitude (N=10) of Peak Joint Moments (normalized by body mass in Nm/Kg) 27
Table 8: Mean Timing (N=10) of Joint Angle Displacement (% of STS cycle) 30
Table 9: Mean Magnitude (N=10) of Joint Angle Displacement (Degrees) 31

CHAPTER 2

Table 10: Mean (n=10) Average Torque Values for Hip and Knee Extensors for Subjects with PD and for Controls (Nm/Kg) 55
Table 11: Mean (n=10) Peak Torque for Hip and Knee Extensors (Nm/Kg), Mean Peak Hip and Knee Extensor Moments (N/Kg), and Percent Effort (%) 56
Table 12: STS Duration for Self-paced trials, in seconds 56
Table 13: Correlation of STS Duration & Average Torque for Hip and Knee Extension 58
# LIST OF FIGURES

## CHAPTER 1

<table>
<thead>
<tr>
<th>Figure</th>
<th>Description</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Set-up for Data Collection</td>
<td>17</td>
</tr>
<tr>
<td>2</td>
<td>Identification of Key Events During Sit-to-Stand</td>
<td>20</td>
</tr>
<tr>
<td>3</td>
<td>Typical Joint Moment Profiles (mean of 5 self-paced trials) for One Subject with PD (on medication) and One Age-matched Control, for the Ankle, Knee, and Hip.</td>
<td>26</td>
</tr>
<tr>
<td>4</td>
<td>Typical Joint Angle Profiles (mean of 5 self-paced trials) for One Subject with PD (on medication) and One Age-matched Control for the Ankle, Knee, and Hip</td>
<td>29</td>
</tr>
<tr>
<td>5</td>
<td>Typical EMG activity (average of 5 trials) for one subject with PD, on-medication state</td>
<td>32</td>
</tr>
<tr>
<td>6</td>
<td>Typical EMG activity (average of 5 trials) for one control, self-paced</td>
<td>32</td>
</tr>
<tr>
<td>7</td>
<td>EMG onset (% STS) for PD-on</td>
<td>33</td>
</tr>
<tr>
<td>8</td>
<td>EMG onset (% STS) for PD-off</td>
<td>33</td>
</tr>
<tr>
<td>9</td>
<td>EMG onset (% STS) for controls, self-paced</td>
<td>34</td>
</tr>
<tr>
<td>10</td>
<td>EMG onset (% STS) for controls, slow-paced</td>
<td>34</td>
</tr>
</tbody>
</table>
Acknowledgements

I would like to thank my supervisor, Dr. Janice Eng, for imparting a wealth of knowledge and for her support and guidance throughout my graduate work. I would also like to thank my committee members, Dr. Donna MacIntyre, Dr. Jon Stoessl, and Dr. Mark Rogers for their invaluable input, time, and effort. Thanks are also extended to Sharon Yardley, clinical coordinator for the UBC Neurodegenerative Disorders Centre for her valuable clinical information and assistance with recruiting the wonderful individuals who offered to be subjects for this study. I'd like to express my gratitude to Maria Kim for her assistance with data collections and the Kin-Com and to Laurent Mingo for his technical support. Finally, I am grateful for financial assistance from the Vancouver unit of the Canadian Physiotherapy Neuroscience Division, The Royal Canadian Legion and The Jane Hudson award.
GENERAL INTRODUCTION

Many individuals with Parkinson's disease (PD) report that rising from a chair is difficult (Brod et al., 1998). However, it is not known why individuals with PD have difficulty performing this important daily task. The lack of information in this area is surprising since rising from a chair is a task that is commonly used for the purpose of assessing motor function (Koller et al., 1989; Nevitt et al., 1989). Performance of the chair rise task is included in the motor section of the Unified Parkinson's Disease Rating Scale (UPDRS) (Fahn et al., 1987) and the inability of persons with PD to rise from a chair has been positively correlated to a history of falling (Nevitt et al., 1989). Therefore, there is a need to identify why individuals with PD have difficulty with rising from a chair so that appropriate treatment interventions may be subsequently explored. The first objective of this research was to investigate how subjects with PD perform a sit-to-stand task and how the movement strategies employed by these subjects differ from those of age-matched controls. No studies have performed a biomechanical analysis of the STS task in persons with PD. Therefore, in addition to strength measures, this research integrated muscular activity with kinetic and kinematic measures to assess STS performance.

A biomechanical analysis of the sit-to-stand (STS) task has been done with healthy young (Schenkman et al., 1990) and older adults (Ikeda et al., 1991). These studies report that an important event during STS is when the buttocks lift from the chair. At this point, weight is transferred to the lower extremities and maximum joint rotational forces (joint moments) are reached into hip and knee extension (Ikeda et al., 1991; Schenkman, 1990). One study found that older adults used 87% of available knee strength to rise from a chair (Hughes et al., 1996). Therefore, the second objective of this research was to assess the
The aim of this study was to document differences between subjects with PD (tested in both an on-medication and off-medication state) and age-matched controls in the performance of the STS task and in strength of the hip and knee extensors. The following hypotheses were used to guide this study:

1. Kinematics of movement will differ significantly, among the three subject groups (subjects with PD on medications, subjects with PD off medications, and age-matched controls) during STS, as measured by the timing and magnitude of joint angle displacements at the hip, knee, and ankle.

2. Kinetic measurements will differ significantly, among the three subject groups, during STS, as measured by the timing and magnitude of joint moments at the hip, knee, and ankle.

3. The timing and magnitude of muscle activity taken from seven muscles in the lower extremities and trunk will differ significantly among the three subject groups, during the STS task, as measured by the onset latency and magnitude of EMG.

4. A significant correlation will exist between strength (knee or hip extensor force measured by a dynamometer) and STS performance (time to rise from a chair).

This is the first research study to quantify how the performance of the sit-to-stand task in persons with PD (on and off antiparkinson medication) differs from healthy age-
matched controls by using kinematic, kinetic, neuromuscular (EMG) and strength (dynamometer) measures.
CHAPTER 1: ABSTRACT
A Biomechanical Analysis of the Sit-to-Stand Task in Persons with Parkinson’s disease

Purpose: Up to 80% of individuals with Parkinson’s disease (PD) report having difficulty with rising from a chair. The purpose of this study was to quantify how the biomechanical strategies used during the sit-to-stand (STS) task in persons with PD (on and off medications) differ from healthy age-matched controls.

Methods: Ten male subjects with PD (64.1 ± 10.1 years) and ten male age-matched controls (65.5 ± 12.4 years) participated. Subjects were instructed to rise from a backless, knee-height chair without the use of their arms at their comfortable pace. Controls were asked to repeat the task at a slower pace. Subjects with PD were tested on two separate days in an on and off-medication state. Lower extremity and trunk movements and muscle activity (EMG) were recorded, in addition to forces under the buttocks and feet. Comparisons were made between 1) PD-on and self-paced age-matched controls and 2) PD-off and slow-paced age-matched controls. Paired t-tests were used to analyze differences between groups for 1) time to complete one STS maneuver (duration), 2) magnitude and timing of joint angle displacements and joint moments at the hip, knee and ankle, and 3) EMG magnitude. An alpha level of .05 identified significance.

Results: The time to complete one STS maneuver (duration) was not significantly different between PD-on and self-paced controls. Timing of the peak hip extensor moment occurred immediately prior to lift-off (buttocks leaving the chair) in all subject groups. At the knee, a peak extensor moment was reached prior to lift-off in the subjects with PD (on and off medications) and post lift-off in controls. The largest joint moments were generated by the knee extensors, followed by the hip extensors, in all subjects. Subjects with PD, on and off medications, generated knee extensor moments of lesser magnitude than controls. However, only the difference between PD-on and self-paced controls reached statistical significance. Finally, early onset abdominal activity (pre lift-off) was seen in four of the controls, but was absent in subjects with PD.

Conclusions: The early knee extensor moment and absence of early onset abdominal activity, in subjects with PD, could indicate altered preparatory activity. These factors may contribute to the difficulty of persons with PD to rise from a chair.
CHAPTER 1: A Biomechanical Analysis of the Sit-To-Stand Task in Persons with Parkinson’s Disease

Rising from a chair is a daily function required for independent living. This task is particularly difficult for elderly individuals with a musculoskeletal or neurological disorder. In a survey of 379 elderly persons with a diagnosis of arthritis, Parkinson’s disease, multiple sclerosis, and other neurological disorders, 42% reported having difficulty with rising from a chair in the home (Chamberlain and Munton, 1984). In a survey of 101 individuals with Parkinson’s disease, 81% of respondents reported having difficulty with rising from a chair (Brod et al., 1998).

It is not clear why persons with Parkinson’s disease (PD) experience difficulty with rising from a chair. Contributing factors could be both central and peripheral in nature.

1.1 Central Factors:

Current models of the basal ganglia suggest that hypokinetic movement disorders observed in individuals with PD are due to a depletion of dopamine release to the striatum (primary input centre of the basal ganglia), secondary to degeneration of dopaminergic neurons in the substantia nigra pars compacta of the basal ganglia (Wichmann and DeLong, 1996; Wichmann and DeLong, 1993). Depletion of dopamine results in increased inhibition of thalamo-cortical activity by primary output centres of the basal ganglia (the internal segment of the globus pallidus and the substantia nigra pars reticulata) (Wichmann and DeLong, 1996; Wichmann and DeLong, 1993).

How central impairment (i.e. impaired basal ganglia-thalamo-cortical activity) in PD affects the performance of functional tasks is largely unknown. The nuclei of the thalamus
that receive input from the basal ganglia project to the primary motor cortex and to the premotor cortex, including the supplementary motor area (Middleton and Strick, 2000). The supplementary motor area (SMA) plays a role in (a) the generation of internally guided movements (Jahanshahi et al., 1995; Marsden and Obeso, 1994), (b) the regulation of movement amplitude (Beckley et al., 1993), and (c) the preparation of movement (Cunnington et al., 1997; Cunnington et al., 1996). Impaired basal ganglia function in PD has been suggested to cause impaired anticipatory postural control (Rogers et al., 1987; Traub et al., 1980), and ineffective automatic postural responses (Bloem, 1992; Horak et al., 1996; Horak et al., 1992).

Central motor disturbances related to the loss of dopamine in the nigrostriatal system have been investigated by comparing the motor functions of subjects with PD in a medicated and non-medicated state. Levodopa (L-dopa) has been found to alter muscle activity, however its effects may be dependent on the task. Burleigh et al. (1995) reported that L-dopa reduced the background EMG amplitude of the trunk and lower extremity muscles during quiet standing. In contrast, Cioni et al. (1997) found that the administration of L-dopa increased the EMG amplitude of distal muscles during walking.

1.2 Clinical Signs of Parkinson's disease:

When exploring possible reasons for functional impairment, it is important to consider classic motor signs of PD and how these signs affect movement. Clinical signs of PD include bradykinesia, tremor, rigidity, and impaired postural reflexes (Calne et al., 1992).
1.2.1 Bradykinesia:

One of the most disabling symptoms of PD is bradykinesia (Glendinning and Enoka, 1994). While the cause of bradykinesia is unknown, one hypothesis is that there is insufficient excitation of the motor cortex by excitatory circuits from the basal ganglia (Glendinning and Enoka, 1994). Abnormal descending commands from the motor cortex to motor neurons could explain altered motor unit behaviour, and thus bradykinesia, in persons with PD (Glendinning and Enoka, 1994). Another suggestion is that subjects with PD send an incorrect command to the motor cortex which underestimates the magnitude of the agonist burst required to move over a desired range of motion (Berardelli et al., 1986). Normally, the magnitude of the agonist EMG burst is increased for longer movements (Hallet and Khoshbin, 1980). However, it has been suggested that persons with PD use additional bursts to compensate for an inadequate initial EMG burst, which subsequently results in an increase in the duration of the movement (Hallet and Khoshbin, 1980).

If bradykinesia results in a slower rise from a chair, this could result in different motor strategies to complete the functional task. For example, in a study with healthy young adults, it was reported that for increasing speeds of ascent, there was an increase in peak joint torque for hip flexion and knee extension but not for hip extension or ankle plantar flexion (Pai and Rogers, 1991). The authors suggested that the disproportionate increase in torque among joints was an indication that healthy subjects actually changed their movement strategy for progressively faster speeds, rather than simply increase joint torque.

Another study (Doorenbosch et al., 1994) noted that slower speeds of ascent were accompanied by increased trunk flexion and reduced knee extension torque. The authors
suggested that these two factors could be used as compensatory strategies in cases of reduced lower extremity strength.

There is a growing body of evidence to suggest that persons with PD may have reduced force generation even when a slow rate of force development is accounted for (Stelmach et al., 1989). Subjects with PD have been shown to require almost twice as much time to reach a target force as did age-matched controls for an elbow flexion task (Stelmach et al., 1989).

1.2.2 Rigidity:

Rigidity is described as resistance to passive movement, independent of the velocity of the movement (Gregoric et al., 1987). Rigidity has also been defined as the inability to completely relax muscle (Wichmann and DeLong, 1993). Suggested causes of rigidity include changes in passive mechanical properties of muscle fibres (Watts et al., 1986), and increased gain of long-latency stretch reflexes causing increased excitability of alpha motoneurons (Wichmann and DeLong, 1993). Disinhibition of alpha motoneurons, via decreased autogenic inhibition by Ib interneurons, has also been suggested as a cause of rigidity (Wichmann and DeLong, 1993).

1.2.3 Tremor:

The central oscillator hypothesis has been proposed to explain the 3-5 Hz resting tremor observed in persons with PD (Wichmann and DeLong, 1993). According to this hypothesis, oscillatory or phasic neuronal activity is a) produced by the basal ganglia cells, or b) produced by the thalamus secondary to increased tonic basal ganglia output, both of which
could result in phasic activity in the thalamocortical circuit and oscillatory discharges of
corticospinal projection neurons (Wichmann and DeLong, 1993).

In addition to resting tremor, an action tremor of approximately 10 Hz has been
observed in persons with PD (Brown et al., 1998). The presence of this action tremor has
been reported to inhibit fused muscle contraction and thus contribute to weakness when in an
off-medication state (Brown et al., 1997).

Of interest is that studies investigating strength measures in persons with PD have
reported no correlation between the degree of clinical signs, such as tremor or rigidity, and
force (Koller and Kase, 1986; Yanagawa et al., 1990). Thus, the effect of clinical signs on
force generation is not clear.

1.2.4 Impaired Postural Reflexes:

Impairment of postural reflexes can be detected as impaired anticipatory or
preprogrammed postural muscle activity associated with voluntary movement or external
perturbation (Latash and Anson, 1996). Ineffective sequencing of muscle activity in
response to perturbations while standing and an inability to adapt and modify automatic
postural responses to changing support conditions contribute to the postural instability found
in individuals with PD (Beckley et al., 1993; Bloem et al., 1992; Chong et al., 2000; Horak et

Dietz et al., (1988) suggested that increased intrinsic muscle stiffness may cause a
delay in normal postural responses (Dietz et al., 1988). Likewise, rigidity in combination
with bradykinesia may cause this delay and impair voluntary responses (Bloem, 1992).
1.3 **Peripheral factors:**

Since PD is a neurological condition which primarily affects persons over the age of 65, loss of function seen in individuals with this disease could be partly attributed to peripheral changes, such as loss of strength reported in elderly subjects (Brooks and Faulkner, 1994; Grimby and Saltin, 1983; Porter et al., 1995; Stalberg et al., 1989).

In persons with PD, muscle biopsies taken from the biceps brachii (Edstrom, 1970) and tibialis anterior (Rossi et al., 1996) have shown increased type I fibres and decreased type II fibres. However, it is not known if these muscle changes are attributed directly to the disease process or if these changes are secondary to reduced mobility. Furthermore, reported dysfunction in the metabolism of skeletal muscle mitochondria, in persons with PD, (Penn et al., 1995) could affect muscle function in this subject group.

1.4 **Evaluation of the Sit-to-Stand Function:**

Given the many possible impairments in the central and peripheral system, it is not surprising that such a large number of individuals with PD report difficulty with rising from a chair. However, there are no studies to date that evaluate this task in persons with PD.

The motor section of the Unified Parkinson’s Disease Rating Scale (UPDRS; Fahn et al., 1987) is routinely used by clinicians to assess motor function in PD. Rising from a chair is an example of a task in the motor section of the UPDRS and is rated from 0 (normal) to 4 (unable to arise without help). However, the sensitivity of this ordinal scale is questionable. Large changes in motor performance can go undetected if assessment is only done with rating scales (Obeso et al., 1996). Also, an ordinal scale does not provide any information as to the underlying causes of the motor dysfunction.
2 Purpose

The purpose of this study was to investigate muscle activity and movement strategies underlying the ability of persons with PD (1) in an “on-medication state” and (2) in an “off-medication state”, to perform the STS task. Kinematic, kinetic, and electromyography (EMG) profiles, during STS, were compared between (a) subjects with PD in an on-medication state (PD-on) and age-matched controls moving at a self-regulated pace (self-paced controls) and (b) subjects with PD in an off-medication state (PD-off) and age-matched controls moving at a matched pace (slow-paced controls).

3 Methods

3.1 Setting:

Data was collected at the Rehabilitation Research Laboratory located at G.F. Strong Rehab Centre in Vancouver, BC.

3.2 Subjects:

Ten male subjects with PD (64.1 ± 10.1 years; mean ± 1 standard deviation) and ten male age-matched controls (65.5 ± 12.4 years) were recruited from the Vancouver area. Inclusion criteria for subjects with PD included 1) clinical diagnosis of PD for a minimum of one year, 2) ability to rise from a chair, without the use of armrests, in the off-state, and 3) no other neurological, orthopaedic, or cardiovascular condition(s) which could affect their ability to perform the STS task. Control subjects were age-matched and were also screened based on the absence of a neurological, cardiovascular, or orthopaedic condition which could
affect their ability to perform the STS task. All subjects were informed of the research procedures before they gave written consent. The experimental protocol was approved by the University of British Columbia ethics committee.

Subject characteristics are summarized in Table 1 (PD) and Table 3 (controls). The motor characteristics of subjects with PD are summarized in Table 2. All subjects with PD reported unilateral disease with a most and least affected side. This study used data from the most affected side as reported by the subject.

Due to the inclusion criteria, subjects with PD were mildly affected by the disease, as indicated by the modified Hoehn & Yahr scale (Fahn et al., 1987) (Appendix A). For comparison, the original Hoehn & Yahr scale (Hoehn & Yahr, 1967) is described in Appendix B. To avoid confusion, this document will refer to the Hoehn & Yahr stage as opposed to stating “modified” Hoehn & Yahr stage. Hoehn & Yahr stages ranged from 1 (unilateral disease) to 3.0 (mild to moderate bilateral disease; some postural instability; physically independent) (mean 2.1 ± 0.6). There was no difference in the Hoehn & Yahr stage between an on-medication state and an off-medication state. Subjects were also rated with the motor section of the Unified Parkinson’s Disease Rating Scale (UPDRS), (Fahn et al., 1987) (Appendix C). Motor scores (out of a maximum of 108), ranged from 6 to 20, (mean 11.0 ± 4.8), in an on-medication state and 12 to 26, (mean 17.4 ± 4.8), in an off-medication state.

The motor section of the UPDRS includes items 18-31 of the total 42 items. The maximum score for items 18-31 is 108. Table 2 includes a break down of the motor section to illustrate scoring on items that assess primarily bradykinesia (items 23,24,25,26, and 31), action and resting tremor (items 20 and 21), and rigidity (items 22).
Table 1: Characteristics of Subjects with Parkinson's disease

<table>
<thead>
<tr>
<th>Subject</th>
<th>Age</th>
<th>Side</th>
<th>Height (cm)</th>
<th>Mass (Kg)</th>
<th>Years PD</th>
<th>PD Meds (mg)</th>
<th>Activity Level</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>63</td>
<td>L</td>
<td>174</td>
<td>85</td>
<td>3</td>
<td>L-C 50/12.5</td>
<td>H+</td>
</tr>
<tr>
<td>II</td>
<td>81</td>
<td>R</td>
<td>182</td>
<td>76</td>
<td>5</td>
<td>L-C 500/125</td>
<td>H+</td>
</tr>
<tr>
<td>III</td>
<td>67</td>
<td>L</td>
<td>166</td>
<td>84</td>
<td>4</td>
<td>L-C 1500/150</td>
<td>M</td>
</tr>
<tr>
<td>IV</td>
<td>73</td>
<td>R</td>
<td>166</td>
<td>64</td>
<td>5</td>
<td>L-C 400/100</td>
<td>H</td>
</tr>
<tr>
<td>V</td>
<td>59</td>
<td>R</td>
<td>176</td>
<td>88</td>
<td>2</td>
<td>L-C 250/75, Ropinirole 3.25</td>
<td>M+</td>
</tr>
<tr>
<td>VI</td>
<td>61</td>
<td>R</td>
<td>169</td>
<td>106</td>
<td>3</td>
<td>L-C 300/75, Bromocriptine 3.75</td>
<td>L</td>
</tr>
<tr>
<td>VII</td>
<td>68</td>
<td>R</td>
<td>175</td>
<td>69</td>
<td>7</td>
<td>L-C 900/225, Ropinirole 10, Tolcapone 200</td>
<td>Z</td>
</tr>
<tr>
<td>VIII</td>
<td>69</td>
<td>L</td>
<td>172</td>
<td>85</td>
<td>6</td>
<td>L-C 400/100, Ropinirole 3</td>
<td>Z</td>
</tr>
<tr>
<td>IX</td>
<td>56</td>
<td>R</td>
<td>182</td>
<td>109</td>
<td>5</td>
<td>L-C 800/200 and 50/12.5</td>
<td>Z</td>
</tr>
<tr>
<td>X</td>
<td>44</td>
<td>R</td>
<td>177</td>
<td>74</td>
<td>4</td>
<td>L-C 300/75, Tolcapone 300, Ropinirole 3</td>
<td>M</td>
</tr>
<tr>
<td>MEAN</td>
<td>64.1</td>
<td></td>
<td>173.9</td>
<td>84.0</td>
<td>4.4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>STD</td>
<td>10.1</td>
<td></td>
<td>5.7</td>
<td>4.5</td>
<td>1.5</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

1 Most affected side
2 Number of years with diagnosis of Parkinson's disease
3 L-C: levodopa/carbidopa
4 Z (zero): no regular exercise program
   L (low): exercise 1-2 hours per week
   M (moderate): exercise 2-4 hours per week
   H (high): exercise 5 or more hours per week
   + (plus sign): program includes regular strength training in the form of weight training, or in the case of subject I, aquafit
Table 2: Motor Scoring of Subjects with Parkinson's disease

<table>
<thead>
<tr>
<th>Subject</th>
<th>Hoehn &amp; Yahr</th>
<th>Total UPDRS</th>
<th>STS</th>
<th>Bradykinesia</th>
<th>Tremor</th>
<th>Rigidity</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>2</td>
<td>9 on(^2)</td>
<td>0 on</td>
<td>3 on</td>
<td>1 on</td>
<td>2 on</td>
</tr>
<tr>
<td></td>
<td></td>
<td>15 off(^2)</td>
<td>0 off</td>
<td>7 off</td>
<td>1 off</td>
<td>3 off</td>
</tr>
<tr>
<td>II</td>
<td>3</td>
<td>20 on</td>
<td>1 on</td>
<td>9 on</td>
<td>0 on</td>
<td>2 on</td>
</tr>
<tr>
<td></td>
<td></td>
<td>26 off</td>
<td>1 off</td>
<td>10 off</td>
<td>0 off</td>
<td>5 off</td>
</tr>
<tr>
<td>III</td>
<td>2</td>
<td>12 on</td>
<td>0 on</td>
<td>5 on</td>
<td>6 on</td>
<td>1 on</td>
</tr>
<tr>
<td></td>
<td></td>
<td>13 off</td>
<td>0 off</td>
<td>5 off</td>
<td>5 off</td>
<td>3 off</td>
</tr>
<tr>
<td>IV</td>
<td>3</td>
<td>12 on</td>
<td>0 on</td>
<td>2 on</td>
<td>4 on</td>
<td>3 on</td>
</tr>
<tr>
<td></td>
<td></td>
<td>23 off</td>
<td>0 off</td>
<td>11 off</td>
<td>3 off</td>
<td>4 off</td>
</tr>
<tr>
<td>V</td>
<td>2.5</td>
<td>14 on</td>
<td>0 on</td>
<td>5 on</td>
<td>1 on</td>
<td>4 on</td>
</tr>
<tr>
<td></td>
<td></td>
<td>19 off</td>
<td>0 off</td>
<td>5 off</td>
<td>2 off</td>
<td>8 off</td>
</tr>
<tr>
<td>VI</td>
<td>2.5</td>
<td>17 on</td>
<td>0 on</td>
<td>10 on</td>
<td>0 on</td>
<td>2 on</td>
</tr>
<tr>
<td></td>
<td></td>
<td>22 off</td>
<td>0 off</td>
<td>10 off</td>
<td>0 off</td>
<td>6 off</td>
</tr>
<tr>
<td>VII</td>
<td>1.5</td>
<td>7 on</td>
<td>0 on</td>
<td>3 on</td>
<td>0 on</td>
<td>4 on</td>
</tr>
<tr>
<td></td>
<td></td>
<td>15 off</td>
<td>0 off</td>
<td>4 off</td>
<td>6 off</td>
<td>1 off</td>
</tr>
<tr>
<td>VIII</td>
<td>1</td>
<td>6 on</td>
<td>0 on</td>
<td>5 on</td>
<td>1 on</td>
<td>0 on</td>
</tr>
<tr>
<td></td>
<td></td>
<td>12 off</td>
<td>0 off</td>
<td>7 off</td>
<td>4 off</td>
<td>1 off</td>
</tr>
<tr>
<td>IX</td>
<td>2</td>
<td>6 on</td>
<td>0 on</td>
<td>1 on</td>
<td>1 on</td>
<td>4 on</td>
</tr>
<tr>
<td></td>
<td></td>
<td>15 off</td>
<td>0 off</td>
<td>5 off</td>
<td>2 off</td>
<td>6 off</td>
</tr>
<tr>
<td>X</td>
<td>1.5</td>
<td>7 on</td>
<td>0 on</td>
<td>4 on</td>
<td>1 on</td>
<td>0 on</td>
</tr>
<tr>
<td></td>
<td></td>
<td>14 off</td>
<td>0 off</td>
<td>8 off</td>
<td>3 off</td>
<td>0 off</td>
</tr>
<tr>
<td>MEAN</td>
<td></td>
<td>2.1</td>
<td>11.0</td>
<td>0.1</td>
<td>4.7</td>
<td>1.5</td>
</tr>
<tr>
<td>STD</td>
<td></td>
<td>.7</td>
<td>4.9 on</td>
<td>.3 on</td>
<td>2.9 on</td>
<td>2.0 on</td>
</tr>
<tr>
<td>MEAN</td>
<td></td>
<td>17.4</td>
<td>4.8 on</td>
<td>0.1</td>
<td>7.2</td>
<td>2.6</td>
</tr>
<tr>
<td>STD</td>
<td></td>
<td>.7</td>
<td>2.5</td>
<td>.3 off</td>
<td>2.5 off</td>
<td>2.0 off</td>
</tr>
</tbody>
</table>

1 Hoehn & Yahr scores the same for on and off medication states, for all subjects (Appendix A)
2 On: on regular antiparkinson's medication regime; Off: withdrawal of antiparkinson's medications
3 Motor score out of 108 (Appendix C)
4 Score on time #27 of UPDRS "arise from chair" (minimum score= 0, normal; maximum score = 4, unable to arise without help)
5 Items from the motor section of the UPDRS reflecting bradykinesia, maximum of 36 points (items #23,24,25,26, and 31)
6 Items from the motor section of the UPDRS reflecting resting + action tremor, maximum of 28 points (items #20 and 21)
7 Items from the motor section of the UPDRS reflecting rigidity, maximum of 20 points (item #20)
Table 3: Characteristics of Control Subjects

<table>
<thead>
<tr>
<th>Subject</th>
<th>Age</th>
<th>Height (cm)</th>
<th>Mass (Kg)</th>
<th>Activity Level1</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>68</td>
<td>180</td>
<td>79</td>
<td>L</td>
</tr>
<tr>
<td>II</td>
<td>74</td>
<td>173</td>
<td>77</td>
<td>H+</td>
</tr>
<tr>
<td>III</td>
<td>79</td>
<td>186</td>
<td>99</td>
<td>L</td>
</tr>
<tr>
<td>IV</td>
<td>63</td>
<td>164</td>
<td>69</td>
<td>L</td>
</tr>
<tr>
<td>V</td>
<td>77</td>
<td>177</td>
<td>80</td>
<td>L</td>
</tr>
<tr>
<td>VI</td>
<td>66</td>
<td>176</td>
<td>93</td>
<td>M</td>
</tr>
<tr>
<td>VII</td>
<td>44</td>
<td>192</td>
<td>79</td>
<td>M</td>
</tr>
<tr>
<td>VIII</td>
<td>51</td>
<td>170</td>
<td>69</td>
<td>Z</td>
</tr>
<tr>
<td>IX</td>
<td>54</td>
<td>166</td>
<td>73</td>
<td>L</td>
</tr>
<tr>
<td>X</td>
<td>79</td>
<td>181</td>
<td>83</td>
<td>H+</td>
</tr>
<tr>
<td>Mean</td>
<td>65.5</td>
<td>176.5</td>
<td>80.1</td>
<td></td>
</tr>
<tr>
<td>Std</td>
<td>12.4</td>
<td>8.7</td>
<td>9.7</td>
<td></td>
</tr>
</tbody>
</table>

1 Z (zero): no regular exercise program  
L (low): 1-2 hours per week  
M (moderate): 2-4 hours per week  
H (high): 5 plus hours per week  
+ (plus sign): program includes regular weight training
Subjects with PD and age-matched controls were similar with respect to physical activity, or weekly exercise. Activity level has been rated from 0 (no regular physical exercise), to 3 (high level of physical exercise). A plus sign has been added to the rating for subjects who include regular strength training as part of their exercise regime.

3.3 Protocol:

Subjects with PD were tested on two different days and were randomly assigned to commence the first testing in either an on-medication or off-medication state. Pilot tests with on and off state testing done on the same day identified that fatigue was likely a factor contributing to lower strength scores for the second (on-state) strength test. Therefore, two test days were scheduled an average of two to three days apart. Testing commenced between 8:00 a.m. and 10:00 a.m. and required an overnight withdrawal of medications for the off-state testing (CAPIT guidelines) (Langston et al., 1992). Control subjects attended a single morning test session.

Subjects sat on an armless, backless, height adjustable chair instrumented with a force plate under the buttocks. The start position for each subject was with the feet 20 cm apart and with thigh support so that the distance between the anterior edge of the chair and the most anterior point of the patella was 20 cm. The chair height was adjusted for each subject to allow for a 90° angle at the knee, in sitting. Subjects performed approximately 10 to 15 sit-to-stand trials at their own pace (self-paced trials) without the use of their arms. Control subjects also performed an additional 5-10 trials at a slow pace (approximately 50% slower than natural pace).
At the time of instructing the subject to stand up, the investigator pressed a trigger switch to initiate a 6 second collection of simultaneous EMG, force plate, and kinematic data for each STS trial.

3.4 Data Collection:

3.4.1 Kinetic and Kinematic Data

Three 6-component Bertec force plates, 45cm x 50cm, were used to record forces under each foot and under the buttocks. One force plate (flush to the floor) was placed under each foot and the third force plate was mounted on the chair to determine the time of lift-off (Figure 1). Force plate data were collected at 600 Hz.

Figure 1: Set-up for Data Collection

<table>
<thead>
<tr>
<th>Markers</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Fifth metatarsal</td>
</tr>
<tr>
<td>2. Lateral malleolus</td>
</tr>
<tr>
<td>3. Lateral knee joint line</td>
</tr>
<tr>
<td>4. Greater trochanter</td>
</tr>
<tr>
<td>5. Iliac crest</td>
</tr>
<tr>
<td>6. Clavicle</td>
</tr>
<tr>
<td>7. Ear</td>
</tr>
</tbody>
</table>

Force Plate 1 under buttocks

Force Plates 2 and 3 under each foot
An Optotrak (Northern Digital) imaging system was used to track infrared emitting diodes (IREDS which will be referred to as markers) attached to the subjects with double-sided tape. Fourteen markers were used for this study to track segment movements, in the sagittal plane, of the foot, shank, thigh, pelvis, trunk, and head. Markers were placed, bilaterally, at the head of the fifth metatarsal, lateral malleolus, lateral knee joint line, greater trochanter, iliac crest, clavicle and ear (Figure 1). Joint angles were calculated from these markers. Optotrak data were collected at a sampling rate of 60 Hz. Synchronized segment kinematics and force plate data were used to calculate joint moments at the ankle, knee, and hip, using an inverse dynamic model (Winter, 1990).

Ideally, the calculation of hip joint moments prior to lift-off of the buttocks from the chair, requires two force plates (one under each buttock) to provide knowledge of the ground reaction forces of each buttock. Since only one force plate was used under the buttocks, it was necessary to resolve the ground reaction force into a left and right component (reflecting the left and right buttock/thigh). The contribution of the ground reaction force to each side was estimated frame-by-frame by location of the medial-lateral centre of pressure measure relative to the hip markers.

3.4.2 Electromyographic Recordings:

A Bortec 16 channel EMG system was used to collect data from seven muscle groups at a sampling rate of 600 Hz, which was synchronized to the force plate and Optotrak collection. Recordings were taken bilaterally from the following muscle groups: tibialis anterior (TA), medial gastrocnemius (MG), biceps femoris (BF), rectus femoris (RF), vastus lateralis (VL), erector spinae (ES), and rectus abdominus (ABD). Guidelines for the site of
Electrode placement for the seven muscle groups have been described by Winter (1991, pp. 59-68). Electrode placement is described in Appendix D. Raw EMG data were full wave rectified, and low-pass filtered at 6 Hz.

A maximal isometric contraction of each muscle was elicited by each subject in response to manual resistance applied by the investigator. This manually resisted contraction was recorded by EMG to serve as a guide as to the relative amount of muscle activity used during the STS task (Knutson et al., 1994).

3.5 Data Analysis

Data were analyzed for five self-paced trials for PD-on, PD-off, and for controls. Five trials at a slow pace were also analyzed for the control group. The first five trials that did not have any obstructed markers were used for the analysis. Since there were no differences for timing and magnitude of the joint angles and moments between the dominant and non-dominant side for the controls, the dominant side was used to compare to the most affected side of the subjects with PD.

3.5.1 Identification of Key Events:

Figure 2 illustrates the key events identified during STS. Movement onset was identified from the force plate under the buttocks (force plate 1) as the initial horizontal force beyond a baseline level. A drop in vertical force to zero, measured on force plate 1, identified the point in time when lift-off of the buttocks from the chair was achieved. Movement termination was identified as the point in time when the vertical movement of the right ear marker reached a plateau. The number of frames to achieve STS was calculated
from movement onset to movement termination. The number of seconds to complete one STS maneuver will be referred to as duration of the task.

Figure 2: Identification of key events during sit-to-stand

3.5.2 Symmetry:

A ratio of the vertical forces at lift-off was obtained by dividing the limb with the lowest force by the limb with the higher force to provide an index of symmetry (Engardt and Olson, 1992).
3.5.3 **Time-Normalizing:**

Sit-to-stand was viewed as a two phase process. The first phase (preparation phase) was identified as movement onset to lift-off and the second phase (lift-off phase) from lift-off to movement termination. Each phase comprised approximately 40-60% of the STS cycle. Therefore, the joint angle, moment, and EMG data was time-normalized and ensemble averaged across five trials (ie mean and standard deviation at each percent of the STS cycle) so that 0% represented movement onset, 50% represented lift-off, and 100% represented movement termination. An additional 10% was assessed before movement onset and after movement termination to identify key events occurring in these time frames.

The mean of five trials for each subject created a ‘mean’ time-normalized profile to be used to identify the timing (percentage of the movement cycle) and magnitude of 1) peak joint moments, 2) joint displacement, and 3) EMG burst activity. Joint moments were normalized by body mass in kilograms.

3.5.4 **Electromyographic Recordings:**

Onset of EMG activity was recorded in order to compare muscle sequencing among the subject groups. For muscles with two or more bursts, the first burst was used to identify onset of EMG activity. The magnitude of the EMG was calculated as the integration under the EMG curve (area) for the complete STS task and normalized by dividing by the area of a 120 frame section (2 seconds) of the maximal voluntary contraction recording.
3.5.5 Statistical Analysis:

Comparisons were made between 1) PD-on and self-paced age-matched controls and 2) PD-off and slow-paced age-matched controls.

Paired t-tests were used to analyze differences between groups for 1) duration of STS, 2) magnitude and timing of peak hip extensor, knee extensor, and ankle dorsiflexor moments, 3) magnitude and timing of joint angle displacement of the hip, knee, and ankle, and 4) area of EMG. An alpha level of .05 was used to identify significance.
4 Results

4.1 Duration of Sit-to-Stand:

In the on-state, subjects with PD performed STS with the same duration as that for self-paced controls (Table 4). Although the intent of the slow paced controls was to serve as a comparison to PD-off, duration of the slow paced controls was actually slower than the duration of PD-off.

Since the duration for subjects with PD-on and self-paced controls did not differ significantly, motor differences observed between subjects with PD and controls cannot be attributed to the speed of the movement.

Also, there was a significant difference in duration between PD-on and PD-off. As it has been shown that joint angles (Doorenbosch et al., 1994) and joint moments (Doorenbosch et al., 1994; Pai and Rogers, 1991) are affected by how quickly the STS task is performed, further comparisons between the PD-on and PD-off were not performed. Therefore, analysis of kinetic, kinematic, and EMG parameters included comparisons only between 1) PD-on and self-paced controls, and 2) PD-off and slow-paced controls.

It is important to note that there was no association between STS duration and age for each of the three subject groups. In other words, there was not an age effect on the movement duration.
Table 4: Mean Duration (N=10) to Perform One Sit-to-Stand Maneuver (seconds)

<table>
<thead>
<tr>
<th>Group</th>
<th>Pace</th>
<th>Range</th>
<th>Duration</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>PD-on¹</td>
<td>Self-paced</td>
<td>1.27-2.51</td>
<td>1.86 ± (0.37)</td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>Self-paced</td>
<td>1.38-2.65</td>
<td>1.89 ± (0.37)</td>
<td>( p &lt; .05)</td>
</tr>
<tr>
<td>PD-off²</td>
<td>Self-paced</td>
<td>1.52-2.40</td>
<td>1.97 ± (0.27)</td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>Slow-paced</td>
<td>1.87-3.92</td>
<td>2.76 ± (0.71)</td>
<td></td>
</tr>
</tbody>
</table>

¹ Subjects with Parkinson’s disease, on-medication state
² Subjects with Parkinson’s disease, off-medication state

Table 5: Mean (N=10) Symmetry Index (Ratio of vertical force at lift-off: limb with lower force divided by limb with higher force)

<table>
<thead>
<tr>
<th>Group</th>
<th>Pace</th>
<th>Symmetry Index</th>
</tr>
</thead>
<tbody>
<tr>
<td>PD-on¹</td>
<td>Self-paced</td>
<td>0.86 ± (0.121)</td>
</tr>
<tr>
<td>Control</td>
<td>Self-paced</td>
<td>0.92 ± (0.006)</td>
</tr>
<tr>
<td>PD-off²</td>
<td>Self-paced</td>
<td>0.84 ± (0.008)</td>
</tr>
<tr>
<td>Control</td>
<td>Slow-paced</td>
<td>0.89 ± (0.004)</td>
</tr>
</tbody>
</table>

¹ Subjects with Parkinson’s disease, on-medication state
² Subjects with Parkinson’s disease, off-medication state

4.2 Symmetry:

The symmetry index for PD-on and PD-off were lower than values for self and slow-paced controls (Table 5). However, none of the PD values were significantly different from controls.

1 Subjects with Parkinson’s disease, on-medication state
2 Subjects with Parkinson’s disease, off-medication state
± one standard deviation
4.3 Joint Moments:

4.3.1 Joint Moment Profiles:

Joint moment profiles for a typical age-matched pair (one subject with PD on medication and one self-paced control), for the ankle, knee, and hip, are illustrated in Figure 3. The profiles illustrate the importance of lift-off (50%) with respect to both timing and magnitude of changes in joint moments.

All subject groups reached a peak ankle dorsiflexor moment, followed by a peak hip extensor moment, prior to lift-off. However, a peak knee extensor moment was reached immediately prior to lift-off for subjects with PD and immediately post lift-off for controls.

4.3.2 Timing of Peak Joint Moments:

<table>
<thead>
<tr>
<th>Group</th>
<th>Pace</th>
<th>Hip Extension</th>
<th>Knee Extension</th>
<th>Ankle Dorsiflexion</th>
</tr>
</thead>
<tbody>
<tr>
<td>PD-on³</td>
<td>Self-paced</td>
<td>48.0 (± 2.1)</td>
<td>49.7 (± 2.2)</td>
<td>43.4 (± 9.8)</td>
</tr>
<tr>
<td>Control</td>
<td>Self-paced</td>
<td>49.2 (± 1.2)</td>
<td>52.3 (± 2.6)</td>
<td>43.6 (± 4.2)</td>
</tr>
<tr>
<td>PD-off²</td>
<td>Self-paced</td>
<td>48.5 (± 1.9)</td>
<td>49.4 (± 1.9)</td>
<td>42.7 (± 8.4)</td>
</tr>
<tr>
<td>Control</td>
<td>Slow-paced</td>
<td>49.2 (± 2.8)</td>
<td>51.3 (± 2.0)</td>
<td>46.1 (± 4.5)</td>
</tr>
</tbody>
</table>

3 Subjects with Parkinson's disease, on-medication state
4 Subjects with Parkinson's disease, off-medication state
| parenthesis comparison p < .05
± one standard deviation
The only difference in timing of the peak moments between the subjects with PD and controls occurred at the knee joint (Table 6). For PD-on and PD-off, attainment of a peak knee extensor moment occurred prior to lift-off (49.7% of movement and 49.4% of movement, respectively). In contrast, peak knee extensor moment for self and slow-paced controls was reached post lift-off (52.3% of movement and 51.3% of movement, respectively).

Figure 3: Typical Joint Moment Profiles (mean of 5 self-paced trials) for One Subject with PD (on medication) and One Age-Matched Control, for the Ankle, Knee, and Hip. 0% = start of movement; 50% = lift-off; 100% = end of STS. +ve joint moment = ankle dorsiflexor, knee extensor, and hip flexor moment.
Of interest is the overall similarity of timing of peak joint moments among groups for hip extension and ankle dorsiflexion. Both joints reached peak levels prior to lift-off with the peak ankle moment preceding that of the hip. Thus, neither the disease process nor the effect of duration appeared to alter the timing of moments at the ankle and hip.

4.3.3 Magnitude of Peak Joint Moments:

There was no significant difference in height between subjects with PD and age-matched controls. Therefore, joint moments were normalized by body mass (in Kg) only.

Table 7: Mean (N=10) Magnitude of Peak Joint Moments (normalized by body mass in Nm/Kg)

<table>
<thead>
<tr>
<th>Group</th>
<th>Pace</th>
<th>Hip Extension</th>
<th>Knee Extension</th>
<th>Ankle Dorsiflexion</th>
</tr>
</thead>
<tbody>
<tr>
<td>PD-on</td>
<td>Self-paced</td>
<td>0.42 (± 0.23)</td>
<td>1.02 (± 0.22)</td>
<td>0.13 (± 0.009)</td>
</tr>
<tr>
<td>Control</td>
<td>Self-paced</td>
<td>0.42 (± 0.22)</td>
<td>1.13 (± 0.20)</td>
<td>0.11 (± 0.006)</td>
</tr>
<tr>
<td>PD-off</td>
<td>Self-paced</td>
<td>0.40 (± 0.17)</td>
<td>0.94 (± 0.12)</td>
<td>0.11 (± 0.005)</td>
</tr>
<tr>
<td>Control</td>
<td>Slow-paced</td>
<td>0.41 (± 0.25)</td>
<td>1.02 (± 0.20)</td>
<td>0.12 (± 0.007)</td>
</tr>
</tbody>
</table>

1 Subjects with Parkinson’s disease, on-medication state
2 Subjects with Parkinson’s disease, off-medication state

(parenthesis comparison p < .05
± one standard deviation

The controls used a greater magnitude of knee extensor moment when rising from a chair at their self pace. Although knee extensor magnitude for the self-paced and slow-paced controls was greater than that for PD-on and PD-off, respectively, only the comparison
between self-paced controls and PD-on reached statistical significance. The comparison between slow-paced controls and PD-off demonstrated a trend \((p = .067)\).

There were no significant differences between groups for magnitude of joint moments at the hip and ankle. One subject with PD in an on and off medication state, three slow-paced controls, and one self-paced control did not demonstrate an observable ankle dorsiflexor moment prior to lift-off. Therefore, the data in Table 8 excludes these individuals.

4.4 Joint Angles:

4.4.1 Joint Angle Profiles:

Joint angle profiles for a typical age-matched pair (one subject with PD on medication and one self-paced control), for the ankle, knee, and hip, are illustrated in Figure 4. The profiles illustrate the importance of lift-off (50%) with respect to both timing and magnitude of joint angle displacement.

The hip joint first moved into flexion at 12 to 16% of the movement cycle and then switched to extension just prior to lift-off. The ankle joint started with movement into dorsiflexion prior to lift-off, continued into dorsiflexion during lift-off and changed to a plantar flexion movement approximately 10% after lift-off. In contrast, the knee maintained a fairly constant angle until immediately prior to lift-off, at which point the knee moved into extension until movement termination.
Figure 4: Typical Joint Angle Profiles (mean of 5 self-paced trials) for One Subject with PD (on medication) and One Age-Matched Control, for the Ankle, Knee, and Hip. 0% = start of STS; 50% = lift-off; 100% = end of STS. +ve joint angles = ankle dorsiflexion, knee flexion, and hip flexion.
4.4.2 Timing of Joint Angle Displacement:

Table 8: Mean (N=10) Timing of Joint Angle Displacement (% of STS cycle)

<table>
<thead>
<tr>
<th>Group</th>
<th>Pace</th>
<th>Hip Fl*</th>
<th>Hip Ext*</th>
<th>Knee Ext*</th>
<th>Ankle DF*</th>
<th>Ankle PF*</th>
</tr>
</thead>
<tbody>
<tr>
<td>PD-on ¹</td>
<td>Self-paced</td>
<td>14.9(±10.0)</td>
<td>48.5(±6.7)</td>
<td>35.8(±2.4)</td>
<td>23.5(±6.7)</td>
<td>57.0(±2.9)</td>
</tr>
<tr>
<td>Control</td>
<td>Self-paced</td>
<td>12.0(±5.4)</td>
<td>42.9(±10.0)</td>
<td>33.7(±5.2)</td>
<td>24.5(±6.0)</td>
<td>58.7(±3.9)</td>
</tr>
<tr>
<td>PD-off ²</td>
<td>Self-paced</td>
<td>16.0(±8.9)</td>
<td>47.3(±3.1)</td>
<td>37.8(±2.6)</td>
<td>21.8(±8.3)</td>
<td>56.7(±3.9)</td>
</tr>
<tr>
<td>Control</td>
<td>Slow-paced</td>
<td>13.8(±7.5)</td>
<td>52.2(±8.7)</td>
<td>33.5(±4.9)</td>
<td>14.9(±5.8)</td>
<td>61.6(±5.7)</td>
</tr>
</tbody>
</table>

¹ Subjects with Parkinson’s disease, on-medication state
² Subjects with Parkinson’s disease, off-medication state
* Fl=flexion; Ext=extension; DF= dorsiflexion; PF= plantar flexion

Significant differences were only found between PD-off and slow-paced controls for the timing of the onset of movement into knee extension, ankle dorsiflexion, and ankle plantar flexion. For knee extension, the onset of movement for PD-off commenced later than that for slow-paced controls. For the ankle, onset of dorsiflexion was later in PD-off but then switched to ankle plantar flexion earlier in the STS cycle, compared to slow-paced controls. No significant differences were found for the timing of hip angle displacement.
4.4.3 Magnitude of Joint Angle Displacement:

Table 9: Mean (N=10) Magnitude of Joint Angle Displacement (Degrees)

<table>
<thead>
<tr>
<th>Group</th>
<th>Pace</th>
<th>Hip Flexion</th>
<th>Knee Extension</th>
<th>Ankle Dorsiflexion</th>
</tr>
</thead>
<tbody>
<tr>
<td>PD-on 1</td>
<td>Self-paced</td>
<td>21.5(± 14.0)</td>
<td>76.7(± 6.5)</td>
<td>6.9(± 1.2)</td>
</tr>
<tr>
<td>Control</td>
<td>Self-paced</td>
<td>17.0(± 8.6)</td>
<td>75.3(± 24.3)</td>
<td>7.5(± 3.7)</td>
</tr>
<tr>
<td>PD-off 2</td>
<td>Self-paced</td>
<td>23.6(± 10.0)</td>
<td>76.8(± 7.1)</td>
<td>7.9(± 2.4)</td>
</tr>
<tr>
<td>Control</td>
<td>Slow-paced</td>
<td>18.9(± 14.4)</td>
<td>82.6(± 5.3)</td>
<td>8.6(± 4.4)</td>
</tr>
</tbody>
</table>

1 Subjects with Parkinson's disease, on-medication state
2 Subjects with Parkinson's disease, off-medication state
± one standard deviation

Although the joint angle displacements for PD-on and PD-off were greater than controls for hip flexion and less than controls for ankle dorsiflexion, none of these comparisons reached statistical significance. The lack of significance may be partly due to the large variability found between subjects for these variables.

4.5 Electromyographic Recordings:

Typical EMG activity for one subject with Parkinson's disease, on-state, and one self-paced control is illustrated in Figures 5 and 6, respectively.
Figure 5: Typical EMG activity (average of 5 trials) for one subject with PD, on-medication state

Figure 6: Typical EMG activity (average of 5 trials) for one control, self-paced
4.5.1 **Sequencing of Muscle Activity:**

Onset of EMG activity, for the seven muscle groups, is illustrated in Figures 7, 8, 9, and 10 for PD-on, PD-off, self-paced controls, and slow-paced controls, respectively.

**Figure 7:** EMG onset (% STS) for PD-on

- erector spinae, 22.6%
- abdominals, 41.3%
- rectus femoris, 27.0%
- vastus lateralis, 30.6%
- biceps femoris, 30.7%
- medial gastrocnemius, 32.4%
- tibialis anterior, 23.8%

**Figure 8:** EMG onset (% STS) for PD-off

- erector spinae, 22.1%
- abdominals, 35.1%
- rectus femoris, 27.7%
- vastus lateralis, 29.0%
- biceps femoris, 29.7%
- medial gastrocnemius, 45.7%
- tibialis anterior, 27.5%
Figure 9: EMG onset (% STS) for controls, self-paced

- erector spinae, 20.9%
- abdominals, 16.7%
- rectus femoris, 27.5%
- vastus lateralis, 29.4%
- biceps femoris, 30.5%
- medial gastrocnemius, 30.4%
- tibialis anterior, 19.7%

Figure 10: EMG onset (% STS) for controls, slow-paced

- erector spinae, 23.6%
- abdominals, 22.7%
- rectus femoris, 27.6%
- vastus lateralis, 25.8%
- biceps femoris, 30.1%
- medial gastrocnemius, 31.3%
- tibialis anterior, 23.7%
Subjects with PD and controls demonstrated similar onset patterns of EMG activity. Overall, the majority of subjects recruited TA at 20-29% of movement, and VL, RF, and BF, at 20-35% of movement. It was common for these four muscle groups to be recruited within 5% of each other. Erector spinae tended to be recruited either at 20-29% or earlier than 18% of movement (4 subjects with PD and 6 controls).

The only differences observed between subjects with PD and controls were with ABD and MG activity. Early abdominal activity (earlier than 12% of movement) was detected in four control subjects but was not detected in any subject with PD, on or off medication. Late MG activity (later than 50% of movement, or post lift-off) was detected in four subjects with PD. This late MG activity was only demonstrated by one control subject in both self and slow-paced conditions.

4.5.2 Area of EMG:

There were no significant differences between subjects with PD and controls, for area of EMG for the seven muscle groups. Therefore, the amount of EMG activity relative to a maximal voluntary contraction was similar between subjects with PD and age-matched controls.
5 Discussion

5.1 Duration:

This study found that the time to complete one STS maneuver (duration) was not significantly different between PD-on and self-paced controls. Therefore, differences identified between these two groups cannot be attributed to the speed of rising. This is important to note since speed of rising has been reported to be an important factor that influences joint angles and moments used during the STS task (Doorenbosch et al., 1994; Pai and Rogers, 1991).

The finding that subjects with PD did not move slower than controls may be due to the mild degree of PD in these subjects. The mean Hoehn & Yahr score was only 2.1 out of a maximum of 5 for PD-on and PD-off. Secondly, the total UPDRS motor score, out of a maximum of 108 points, averaged only 11.0 for PD-on and 17.4 for PD-off. Thirdly, a 36 point subsection of the UPDRS motor score reflecting the degree of bradykinesia resulted in an average score of only 4.7 for PD-on and 7.2 for PD-off.

5.2 Preparation Phase:

The timing of the peak hip extensor moment occurred immediately prior to lift-off in all subject groups. This is in agreement with previous studies that have reported attainment of a peak hip extensor moment immediately prior to lift-off in young adults (Schenkman et al., 1990) and in older adults (Ikeda et al., 1991).

The timing of the peak knee extensor moment for subjects with PD was not in agreement with the control group or with other published studies using healthy individuals (Rodosky et al., 1989; Schenkmen et al., 1990). Previous studies have reported that healthy
young adults achieve a peak knee extensor moment at the time of lift-off (Rodosky et al., 1989) or immediately post lift-off (Schenkmen et al., 1990) when the body weight has been transferred to both lower extremities. The controls in this study did reach a peak knee extensor moment immediately post lift-off for both self and slow-paced conditions. In contrast, the timing of the peak knee extensor moment in subjects with PD occurred earlier and prior to lift-off. It is also worth noting that further assessment of the knee joint moments revealed that the onset of the knee extensor moment occurred at 31.8% of the STS cycle in subjects with PD compared to 34.0% of the STS cycle in self-paced controls. Therefore, both the onset and peak knee extensor moment occurred earlier in subjects with PD.

The earlier knee extensor moment observed in subjects with PD may reflect impaired timing and sequencing of events to rise from the chair efficiently. For example, the early knee moment could indicate that subjects with PD do not adequately move their center of mass forward, resulting in a STS strategy characterized by an upward, more vertical boost generated by the knee extensor moment prior to lift-off and the transfer of weight to the lower extremities. This finding is in agreement with the speculations by Morris (2000) that persons with PD may fail to bring the center of mass adequately over the base of support.

A recent biomechanical analysis of STS in healthy adults reported that the task involves a distinct preparatory postural component to move the center of mass over the base of support, followed by a centrally programmed sequence of events to rise the body from the chair (Goulart and Valls-Sole, 1999). The lack of early abdominal activity in subjects with PD may suggest altered preparatory postural activity as the abdominals are a key muscle group in moving the trunk over the base of support (Goulart and Valls-Sole, 1999). Alternatively, altered abdominal activity could reflect early changes to the trunk which
precede the characteristic kyphosis of late PD (Bridgewater and Sharpe, 1998). Furthermore, a strong representation of proximal muscles in the somatotopic organization of the basal ganglia (DeLong et al., 1986) suggests that degeneration of neurons in the basal ganglia could lead to preferentially proximal muscle impairment (Weinrich et al., 1988).

It is possible that the lack of early abdominal activity may be compensated by deactivation of tonically active erector spinae to allow the trunk to move forward. However, inhibition of erector spinae at the onset of trunk flexion was not observed.

Overall, the results of our study suggest that subjects with PD may have altered preparatory postural control as indicated by the findings that these subjects 1) reached a peak knee extensor moment pre lift-off and thus prior to transferring weight to the lower extremities and 2) had no pre lift-off abdominal activity. While no studies have explored altered preparatory control of persons with PD during the STS task, altered sequencing of muscle activity during the preparation of movement for a standing arm raise task (Latash et al., 1994; Rogers et al., 1987) and impaired scaling of anticipatory postural responses during a standing arm pull task (Traub et al., 1980) have previously been observed. Also, prolonged activity in the SMA following movement onset in persons with PD has been suggested to interfere with the motor planning process by disrupting the temporal organization of submovements within the total movement task (Cunnington et al., 1996).

Assessment of the magnitude of joint moments during STS indicated that for subjects with PD and controls, the knee extensor joint moments were over two times the magnitude of the hip joint moments. This is in agreement with previous studies using healthy young adults (Schenkman et al., 1990) and older adults (Ikeda et al., 1991). For both the on and off medication states, subjects with PD generated knee extensor moments of lesser magnitude
than controls, to perform the STS task. However, only the difference between PD-on and self-paced controls reached statistical significance.

The presence of lower extremity weakness is one possible explanation for the lesser magnitude of knee extensor moments generated during STS by subjects with PD. Previous studies have reported weakness in subjects with PD, compared to age-matched controls, for isotonic flexion and extension of the wrist, upper extremity, and knee, (Koller and Kase, 1986), isotonic dorsiflexion (Yanagawa et al., 1990), and isokinetic dorsiflexion (Pedersen et al., 1997).

If subjects with PD do not move their center of mass sufficiently forward due to the reduced abdominal activity, then the early knee extensor force may not be adequate to propel the body forward due to the more vertical force projection. Therefore, subjects with PD may have to depend more on the hip joint to accomplish the lift-off. In fact, a post hoc analysis revealed that subjects with PD had a peak hip moment to peak knee moment ratio of .41 compared to a ratio of .37 in controls. This suggests that subjects with PD may have a greater reliance on the hip when performing the STS task.

5.3 Lift-off Phase:

The only difference between subjects with PD and controls during the lift-off phase (lift-off to movement termination) was the late onset of EMG activity in MG seen in 4 subjects with PD. This late MG activity could indicate that subjects with PD are not accurate at producing propulsion forces during lift-off and thus require late MG activity to decelerate the centre of mass. Alternatively, late MG activity might suggest that individuals with PD
demonstrate ineffective sequencing of muscle activity that does not always contribute to the effectiveness of task performance (Horak et al. 1996; Horak, et al., 1992).

Overall, during the lift-off phase, movement profiles for subjects with PD were similar to those of controls. This is in agreement with Marsden (1982) who applied the motor program theory to suggest that while persons with PD are able to call up the appropriate motor programs, there is an impairment in the ability to sequence and execute these programs to achieve the desired motor plan (Marsden, 1982).

5.4 Motor Rating Scales:

Item 27 in the motor section of the UPDRS requires subjects to rise from a straight-back chair with their arms folded across their chest. This item is scored from 0 (normal) to 4 (unable to arise without help). Nine of the subjects with PD scored 0 in an on and off medication state. A score of 1 is defined as ‘slow, or may need more than one attempt’. Only one subject with PD scored 1 as he needed more than one attempt to rise from the chair in both an on and off medication state.

Item 27 is an example of the lack of sensitivity of an ordinal scale. Despite quantitative differences between subjects with PD and controls in the performance of STS, scoring on item 27 was normal for nine of the subjects with PD and therefore did not indicate signs of motor impairment. However, our biomechanical assessment found that there were differences in the on and off duration to complete the task which was not detectable by visual observation. In addition, although the UPDRS was ‘normal’, altered muscle activity, joint motion, and joint moments were found. Thus, although these subjects had only mild PD
signs and the UPDRS rise from a chair scored normal, a biomechanical analysis detected changes in the performance of this task.

5.5 Clinical Implications:

It is important to understand why persons with PD have difficulty with rising from a chair in order to develop appropriate treatment interventions. From this study, a number of factors can be identified to explain why STS is difficult.

Firstly, the subjects with PD in this study generated a peak knee extensor moment prior to lift-off and the transfer of weight to the lower extremities. This finding suggests that subjects with PD used a vertical boost prior to lift-off, to rise from the chair. While STS strategies have not been previously evaluated in persons with PD, one study (Scarborough et al., 1999) reported that some healthy elderly use a vertical rise strategy characterized by lessening of the anterior upper body momentum and dominance of knee extension and vertical momentum at the time of lift-off.

Secondly, subjects with PD generated a peak knee extensor moment of less magnitude than the controls. This finding could partly explain the difficulty that subjects with PD report in rising from a chair. Riley et al. (1997) assessed failed STS trials in elderly subjects with a variety of neuromuscular, musculoskeletal, and vestibular impairments. The authors reported that failed STS trails (identified by either a sit-back or a step-back maneuver) were associated with lower knee torque values, but not hip torque values, during lift-off.

Overall, the subjects with PD in our study 1) demonstrated an absence of preparatory abdominal activity, 2) generated less knee extensor moment than controls, and 3) generated a
peak knee extensor moment prior to lift-off which may have caused the body to move in a more upward, rather than forward, direction.

The findings of this study point to the importance of both lower extremity strength training (specifically of the hip and knee extensors) and the implementation of motor strategies to improve the performance of STS in persons with PD. The type of treatment intervention that would specifically address altered preparatory control of the STS task, is not known. Possibly, the use of external sensory cues to facilitate center of mass transfer and biofeedback to train preparatory abdominal activity could be effective treatment tools to improve STS performance in persons with PD.

6 Limitations

This study used a standardized start position and chair height to assess STS performance. Using a standardized start position and chair height may limit movement strategies utilized by subjects. For example, some studies have identified different movement strategies employed with different initial foot position (Schultz et al., 1992; Vander Linden et al., 1994).

The slow-paced condition for the controls involved an unnatural movement for these subjects and may not be the optimal control for PD-off, particularly since the slow-paced controls moved slower than PD-off. However, there were some similar trends between 1) PD-on and self-paced controls, and 2) PD-off and slow-paced controls. For example, self-paced controls generated a peak knee extensor moment that was significantly greater than that for PD-on. Compared to PD-off, the slow-paced controls still generated a greater peak knee extensor moment despite their slower pace.
The repeated paired t-tests also pose a limitation. While it is understood that there is an increased chance of a type I error when a number of t-tests are run at an alpha level of .05, the aim of this study was to explore trends in movement strategies used by the three subject groups (PD-on, PD-off, and age-matched controls). Furthermore, the small sample size (ten subjects with PD and ten controls) may have increased the chance of a type II error due to large intersubject variability.

Finally, subjects with PD were classified as mild PD. Therefore, motor impairments that could affect the performance of STS in persons with more severe PD cannot be generalized from this study. However, the fact that changes were identified even in these mildly affected subjects suggest that motor function is altered early in the disease process.
CHAPTER 2: ABSTRACT
The Relationship Between Lower Extremity Strength and Performance of the Sit-to-Stand Task in Persons with Parkinson’s Disease

**Purpose:** Rising from a chair is a physically demanding task of daily living that has been reported to be difficult for persons with Parkinson’s disease (PD). The relationship of lower extremity muscle weakness to sit-to-stand (STS) performance has never been evaluated. The purpose of this study was to investigate the relationships between strength (hip and knee extensor torque assessed by a dynamometer) and STS performance (time to rise from a chair, ie duration).

**Methods:** Ten male subjects with PD (64.1 ± 10.1 years) and ten male age-matched controls (65.5 ± 12.4 years) participated. Subjects were instructed to rise from a backless, knee-height chair without the use of their arms at their comfortable pace. Subjects with PD were tested on two separate days in an on and off-medication state. Lower extremity and trunk movements were recorded, in addition to forces under the buttocks and feet. A Kin-Com dynamometer was used to measure concentric, isokinetic knee and hip extensor torque. Paired t-tests were used to compare torque and STS duration between the two subject groups. Pearson product correlations were used to assess the relationship between lower extremity strength (isokinetic torque) and functional performance (STS duration).

**Results:** Average hip extensor torque in subjects with PD was only 70% of that for controls. Average knee extensor torque in subjects with PD was 90% of that for controls. Greater hip strength was related to better STS performance in subjects with PD and greater knee strength was related to better STS performance in controls.

**Conclusions:** This study demonstrated that subjects with PD have less ability to generate lower extremity force compared to age-matched controls and that this weakness may contribute to the difficulty of persons with PD to rise from a chair. In addition, these findings suggest that performance of the STS task in subjects with PD is more dependent on strength at the hip joint than on the knee joint.
CHAPTER 2: The Relationship Between Lower Extremity Strength and Performance of the Sit-to-Stand Task in Persons with Parkinson’s Disease

Rising from a chair, bed, or toilet is a physically demanding activity of daily living. In the United States, over two million non-institutionalized elderly persons report having difficulty with rising from a chair (Dawson et al., 1987). This number is disturbing given the advancing age of the North American population. In Canada, it is predicted that by the year 2021, one person in five will be over the age of 65 (Statistics Canada, 1999).

7.1 Strength and the sit-to-stand task:

The sit-to-stand task requires greater lower extremity strength and range of motion than walking or stair climbing (Berger et al., 1988). The STS task has been defined as four consecutive phases (Schenkman et al., 1990). Phase two, the momentum-transfer phase, starts with the buttocks lifting from the chair (Schenkman et al., 1990) and is the phase most likely to be problematic for individuals with lower extremity weakness. It is during phase two that the largest torque values are reached into hip and knee extension (Kotake et al., 1993; Schenkman et al., 1990).

7.2 Performance of sit-to-stand in Parkinson’s disease:

Parkinson’s disease (PD) is a progressive neurodegenerative disease affecting approximately 1.5% of individuals over 65 years of age and 2.6% of individuals over 85 years of age (Tanner, 1992; Tison et al., 1994). In a survey of 101 individuals with Parkinson’s disease, 81% of respondents reported having difficulty with rising from a chair.
(Brod et al., 1998). However, it is not clear why persons with PD experience difficulty with this task.

7.3 Force Generation in Parkinson’s disease:

There are a number of potential central and peripheral sources which could contribute to a reduced ability to generate force in subjects with PD. Abnormal descending commands from the motor cortex to motor neurons could explain altered motor unit behaviour (Glendinning and Enoka, 1994) and possibly result in reduced force generation in persons with PD. Abnormal motor unit behaviour has been reported for subjects with PD, including firing irregularities of single motor units (Dengler et al., 1990; Glendinning and Enoka, 1994), abnormal coactivation of antagonist muscles (Glendinning and Enoka, 1994), and paired discharges (two discharges from a motor unit with an interval less than 75% of the mean interval between a series of discharges) (Dengler et al., 1990). Also, compared to age-matched controls, persons with PD have been reported to have a greater number of motor units that fire at a lower threshold (Glendinning and Enoka, 1994).

Furthermore, since PD is a neurological condition which primarily affects persons over the age of 65, weakness seen in individuals with this disease could be partly attributed to peripheral changes similar to those reported in elderly subjects. Loss of muscle strength between the ages of 30 and 80 years has been found to vary from 20-40% (Brooks and Faulkner, 1994; Grimby and Saltin, 1983; Porter et al., 1995; Stalberg et al., 1989). Factors to explain loss of strength with age include decreased area of type II muscle fibers, (Brooks and Falulkner, 1994), decreased number and size of muscle fibers, and decreased number of motor units (Booth et al., 1994; Porter et al., 1995).
Assessment of muscle in persons with PD have demonstrated altered muscle properties but it is not known if these muscle changes are attributed directly to the disease process or if these changes are secondary to reduced mobility. Muscle biopsies taken from the biceps brachii (Edstrom, 1970) and tibialis anterior (Rossi et al., 1996) have shown increased type I fibres and decreased type II fibres.

Despite the documented changes in muscle properties, evidence to support a reduced ability to generate force in persons with PD is not definitive. The mixed results may be due to the specific muscles tested, the severity of the disease, the type of muscle action, or the specific strength variable measured.

Isometric muscle testing of elbow flexors has indicated that persons with PD develop muscle torque with a reduced rate (Stelmach et al., 1989; Stelmach and Worringham, 1988). Time to achieve peak isometric force can be up to 3-4 seconds in persons with PD compared to less than 1 second in age-matched controls (Corcos et al., 1996). However, there is a growing body of evidence to suggest that persons with PD may have reduced force generation even when a slow rate of force development is accounted for (Stelmach et al., 1989).

If weakness is found only on one side in persons with PD then the weakness could be attributed to the disease process (Kakimuma et al., 1998; Nogaki et al., 1999). Kakimuma et al. (1998) reported that persons with PD demonstrated significant isokinetic knee flexor and extensor weakness on the affected side but not on the unaffected side. Furthermore, if persons with PD are found to be weaker when tested in an off-medication state, the weakness could be attributed to the disease process (Corcos et al., 1996). Strength decreases of 34%
for isometric elbow extension (Corcos et al., 1996) and 15-30% for isokinetic ankle
dorsiflexion (Pedersen and Oberg, 1993) has been reported after withdrawal of medications.

It should be noted that studies have reported that weakness in PD does not correlate
with the degree of rigidity and tremor (Koller and Kase, 1986; Yanagawa et al., 1990) or
Hoehn and Yahr stage (Yanagawa et al., 1990). However, one study (Jordan et al., 1992)
reported a significant correlation between rate of isometric force generation using a hand­
held dynamometer and clinical measures of rigidity, but not tremor.

7.4 Relationship Between Strength and Function:

Few studies have examined the relationship between muscle strength and functional
performance in persons with PD. One study (Pedersen et al., 1997) examined the
relationship between concentric and eccentric isokinetic strength of ankle dorsiflexors and
gait in persons with PD. For the male subjects, there was a positive but insignificant
correlation between isokinetic strength and measures of stride length and stride frequency.
Another study (Jordan, et al., 1992) reported a significant correlation between the rate of
isometric force generation measured with a hand grip dynamometer and motor disability in
PD assessed with The Kings College Rating Scale (an ordinal scale including measures of
performance of activities of daily livings). A more recent study (Toole et al., 2000) reported
moderate increases in knee flexor and extensor strength and improved static standing
balance, in persons with stage I to III PD, following a strength and balance training program.
No studies have evaluated the role of lower extremity strength and performance of the STS
task in persons with PD. If persons with PD have reduced ability to generate force, one
might speculate that this could be a contributing factor to impaired function.
8 Purpose

The purpose of this study was to compare the ability to generate force of the hip and knee extensors, as assessed by the average torque measured by an isokinetic dynamometer, among three groups: 1) persons with PD in an off-medication state (PD-off), 2) persons with PD in an on-medication state (PD-on), and 3) age-matched controls. Secondly, the relationships (correlations) between the performance of STS (duration of one STS maneuver) and lower extremity strength (average hip and knee extensor torque assessed by an isokinetic dynamometer) were assessed. Thirdly, the percent effort, a measure of the force required for the STS task relative to the available strength (ie. ratio of the peak moment generated during the STS task over the peak torque assessed on the dynamometer) was compared among the three groups.

9 Methods

9.1 Setting

Data was collected at the Rehabilitation Research Laboratory located at G.F. Strong Rehab Centre in Vancouver, BC.

9.2 Subjects:

Ten male subjects with PD (64.1 ± 10.1 years; mean ± 1 standard deviation) and ten male age-matched controls (65.5 ± 12.4 years) were recruited from the Vancouver area.
Inclusion criteria for subjects are described in section 3.2 on page 11 of Chapter 1.

Characteristics of subjects with PD and controls are outlined in Table 1 (p.13 of Chapter 1) and Table 3 (p.15 of Chapter 1), respectively. Motor characteristics of subjects with PD are outlined in Table 2 on page 14 of Chapter 1.

9.3 Protocol:

Subjects with PD were tested in an on-medication state and in an off-medication state according to the protocol previously described in section 3.3 on page 16 in Chapter 1. The STS protocol is also outlined in section 3.3 in Chapter 1.

9.4 Data Collection:

9.4.1 Kinetic and Kinematic Data:

The kinetic and kinematic data collection protocol has been previously described in section 3.4.1 on page 17 in Chapter 1. This information was used to measure the duration of the STS task (see section 9.5.1).

9.4.2 Isokinetic strength testing:

A Kin Com Isokinetic Dynamometer (Chattanooga Group Inc., TN) was used to test bilateral, concentric, isokinetic hip and knee extensor strength. These measurements were chosen based on the findings of previous studies that have reported that the largest joint moments generated during the STS task occur into knee extension, followed by hip extension (Ikeda et al., 1991; Schenkman et al., 1990). This instrument has been shown to be accurate.
and reliable for position, velocity and force (Mayhew et al., 1994; Farrell et al., 1986). The calibration of the instrument was tested prior to the study with known weights and was accurate to within +/- 1 N. Three submaximal cycles and one maximal cycle was completed as practice on the Kin-Com as per the protocol described by Kramer (Kramer, 1990).

Preloading is the magnitude of the activation force and is defined as the force that must be applied to the load cell in order to initiate the movement and has been shown to affect the torque-angle curve generated from isokinetic contractions (Jensen et al., 1991; Kramer et al., 1991). The preload was individualized (Strauss, 2000) for each subject, joint and direction of motion and was set at a minimum of 50% of the peak torque values observed during the warm-up trials and kept constant during the test sessions.

Each test consisted of four maximal repetitions. The first maximal contraction was used for practice and thus was not included with the latter three for data analysis (see p. 53). Moderate and continuous verbal encouragement was provided to encourage subjects to continuously and maximally push against the resistance pad. Positioning and stabilization are documented below.

9.4.2 (i) **knee extension**

Subjects were seated in 90 degrees of hip flexion and with their backs against a rigid support. The Kin-Com lap belt and shoulder strap were used to stabilize the pelvis and trunk. The lateral femoral epicondyle was aligned with the rotational axis of the dynamometer and the cuff of the force transducer was placed three fingerbreadths proximal to the lateral malleoli.
Isokinetic strength was measured between 90 degrees of knee flexion and 10 degrees of knee flexion. This range allowed for strength testing at the angle estimated to match that of knee range at lift-off during the STS task. Previous data collected at the Rehabilitation Research Laboratory of elderly subjects indicated that the average knee range of motion at lift-off is 74 degrees of flexion.

9.4.2 (ii) hip extension

Subjects were positioned in supine and the Kin-Com stabilization straps were used across the pelvis and proximal aspect of the contralateral thigh (Markhede and Grimby, 1980). The greater trochanter of the test leg was aligned with the rotational axis of the dynamometer and the cuff of the force transducer was placed three fingerbreadths proximal to the popliteal fossa.

Isokinetic hip extensor strength was tested between 90° of hip flexion and 10-20° of hip flexion. Isokinetic hip and knee extensor strength was tested at 45°/sec. This angular velocity is similar to the hip and knee extension velocity previously recorded for the STS task in healthy elderly subjects at the Rehabilitation Research Laboratory.

9.5 Data Analysis:

9.5.1 Identification of Duration of Sit-to-Stand:

The number of frames to achieve STS was calculated from movement onset to movement termination. Identification of movement onset, lift-off, and movement termination
has been previously described in section 3.5.1 on page 19 in Chapter 1. The number of seconds to complete one STS maneuver will be referred to as duration of the task.

The amount of muscle force used during the STS task was quantified relative to the strength measured from the isokinetic dynamometer. This variable was referred to as the percent effort and was a ratio of the peak moment generated during the STS task over the peak torque assessed by the dynamometer.

9.5.2 Kin-Com Data Analysis:

Torque values were corrected for the effect of gravity on the lower extremity segment and the effect of gravity on the cuff of the dynamometer. This gravity-correction procedure has been shown to be accurate (Finucane et al., 1994). The three torque-angle curves of each set of contractions were ensembled to obtain a mean curve (i.e., ensemble-averaged with a mean torque value calculated at each angle). Peak torque and average torque over the range were extracted from this single curve as these are the measures most frequently used to assess human muscle performance. Since these measures are derived from three trials, they are more correctly denoted as “mean peak torque” and “mean average torque”, however, the simpler terms of “peak torque” and “average torque” are used in any following discussion to minimize confusion. Peak and average torque values were normalized by dividing by the subject’s body mass (Kg).

9.5.3 Statistical Analysis:

Paired t-tests were used to compare differences between 1) PD-off and age-matched controls, 2) PD-on and age-matched controls, and 3) PD-off and PD-on. The variables
assessed were 1) average torque values, 2) percent effort, and 3) STS duration. An alpha level of .05 was used to identify statistical significance. Since there were no significant differences between the average torque values of the most and least affected sides in subjects with PD and dominant and non-dominant sides in controls, torque analysis only refers to comparisons made between the most affected side in subjects with PD and in the dominant side for controls.

Pearson product correlations were used to assess relationships between STS performance (duration) and lower extremity strength (average torque of the hip and knee).

10 Results

Across all pairwise comparisons, the torque values for the hip and knee of the control group were greater than that for PD-on and PD-off (Table 10). However, only the differences between PD-on and controls for average torque of hip extensors and between PD-off and controls for average torque of knee extensors reached statistical significance. There was a trend, however noted between PD-on and controls for average torque of knee extensors (p = .067).

There was no significant difference in height between subjects with PD and age-matched controls. Therefore, torque values were normalized by body mass (in Kg) only.
Table 10: Mean (N=10) Average Torque Values for Hip and Knee Extensors for Subjects with PD and for Controls (Nm/Kg)

<table>
<thead>
<tr>
<th>Group</th>
<th>Muscle Test</th>
<th>Torque Range</th>
<th>Mean Torque</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>PD-off</td>
<td>Hip Extension</td>
<td>.40-1.35</td>
<td>.76 (±.30)</td>
<td></td>
</tr>
<tr>
<td>PD-off</td>
<td>Knee Extension</td>
<td>.58-1.35</td>
<td>1.00 (±.22)</td>
<td></td>
</tr>
<tr>
<td>PD-on</td>
<td>Hip Extension</td>
<td>.46-1.03</td>
<td>.68 (±.19)</td>
<td>(p&lt; .05)</td>
</tr>
<tr>
<td>PD-on</td>
<td>Knee Extension</td>
<td>.75-1.27</td>
<td>1.07 (±.16)</td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>Hip Extension</td>
<td>.53-1.64 (p = .064)*</td>
<td>.96 (±.32)</td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>Knee Extension</td>
<td>.88-1.64</td>
<td>1.18 (±.24)</td>
<td></td>
</tr>
</tbody>
</table>

* Trend only  
( Parenthesis comparison p < .05  
± one standard deviation

Also of interest is that average torque for the hip extensors in PD-on was only 70% of the value for controls. However, average torque for the knee extensors in PD-on was 90% of the value for controls.

Percent effort and the values which comprise this ratio (peak torque values for hip and knee extensors and peak hip and knee joint moments), are reported in Table 11 for the three subject groups. There were no significant differences between subjects with PD and controls, for percent effort at the hip or at the knee. Although the values for peak torque and peak moments in subjects with PD were lower than the control group, all groups used 36-40% of available hip torque and 70-71% of available knee extensor torque, to perform the STS task.
Table 11: Mean (N=10) Peak Torque for Hip and Knee Extensors (Nm/Kg), Mean Peak Hip and Knee Extensor Moments (N/Kg), and Percent Effort (%)

<table>
<thead>
<tr>
<th>Group</th>
<th>Joint</th>
<th>Peak Torque*</th>
<th>Peak Moment**</th>
<th>Percent Effort</th>
</tr>
</thead>
<tbody>
<tr>
<td>PD-on</td>
<td>Hip</td>
<td>1.13</td>
<td>.42</td>
<td>37.0 (±18.67)</td>
</tr>
<tr>
<td></td>
<td>Knee</td>
<td>1.48</td>
<td>1.02</td>
<td>70.3 (±14.73)</td>
</tr>
<tr>
<td>PD-off</td>
<td>Hip</td>
<td>1.12</td>
<td>.40</td>
<td>36.0 (±11.43)</td>
</tr>
<tr>
<td></td>
<td>Knee</td>
<td>1.38</td>
<td>.94</td>
<td>71.2 (±15.47)</td>
</tr>
<tr>
<td>Control</td>
<td>Hip</td>
<td>1.31</td>
<td>.42</td>
<td>39.2 (±18.36)</td>
</tr>
<tr>
<td></td>
<td>Knee</td>
<td>1.56</td>
<td>1.13</td>
<td>70.8 (±8.43)</td>
</tr>
</tbody>
</table>

* Peak torque measured by an isokinetic dynamometer  
**Peak joint moment generated during the STS task  
± one standard deviation

Mean self-paced duration values for each subject group are documented in Table 12. Although PD-off was slower than PD-on and the controls, only the PD-off and PD-on comparison reached statistical significance. Of interest was that the duration for PD-on and the controls was the same (1.86 and 1.89 seconds, respectively). Therefore, differences identified between these two groups are not due to duration, or time to perform one STS maneuver.

Table 12: STS Duration for Self-paced trials, in seconds

<table>
<thead>
<tr>
<th>Group</th>
<th>Range</th>
<th>Mean Duration</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>PD-off</td>
<td>1.52-2.40</td>
<td>1.97 (±.27)</td>
<td>(p &lt; .05)</td>
</tr>
<tr>
<td>PD-on</td>
<td>1.27-2.51</td>
<td>1.86 (±.37)</td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>1.38-2.65</td>
<td>1.89 (±.37)</td>
<td></td>
</tr>
</tbody>
</table>

Parenthesis comparison p < .05  
± one standard deviation
There was low variability within the trials of each subject for the STS duration measures as indicated by the coefficient of variation (standard deviation/mean of five trials x 100). For self-paced duration, the coefficient of variation for PD-on, PD-off, and controls was 8% ± 4%, 8% ± 2%, and 8% ± 4%, respectively. Within subject variability was also low for the strength measures as indicated by the coefficient of variation (standard deviation/mean of three trials x 100). For average hip torque, the coefficient of variation for PD-on, PD-off, and controls was 8% ± 4%, 7% ± 3%, and 11% ± 7%, respectively. For average knee torque, the coefficient of variation for PD-on, PD-off, and controls was 6% ±1%, 6% ± 2%, and 8% ± 2%, respectively. Thus the low coefficient of variation suggest that the strength and STS performance measures were reliable within the test session.

Pearson Product correlations between duration and average torque values for hip and knee extensors are reported in Table 13. Overall, subjects with PD demonstrated a significant negative correlation between hip torque and duration while controls demonstrated a significant negative correlation between knee torque and duration. Therefore, for subjects with PD, the greater the hip torque, the shorter the duration of STS and for controls, the greater the knee torque, the shorter the duration of STS.
Table 13: Correlation of STS Duration & Average Torque for Hip and Knee Extension

<table>
<thead>
<tr>
<th>Group</th>
<th>Joint</th>
<th>Correlation (duration versus torque)</th>
</tr>
</thead>
<tbody>
<tr>
<td>PD-on</td>
<td>Hip</td>
<td>-.800*</td>
</tr>
<tr>
<td>PD-on</td>
<td>Knee</td>
<td>-.424</td>
</tr>
<tr>
<td>PD-off</td>
<td>Hip</td>
<td>-.707*</td>
</tr>
<tr>
<td>PD-off</td>
<td>Knee</td>
<td>-.584</td>
</tr>
<tr>
<td>Control</td>
<td>Hip</td>
<td>-.337</td>
</tr>
<tr>
<td>Control</td>
<td>Knee</td>
<td>-.667*</td>
</tr>
</tbody>
</table>

* Significant Correlation (p < .05)

11 Discussion

11.1 Isokinetic Average Torque:

At both the hip and the knee the controls produced greater average torque values compared to subjects with PD. However, significant levels were only reached for the comparisons between 1) PD-on and controls at the hip and 2) PD-off and controls at the knee. There was also a trend (p = .067) between PD-on and controls for average torque of knee extensors.

The finding that average torque values were generally lower in subjects with PD is in agreement with previous studies comparing muscle strength between subjects with PD and age-matched controls (Koller and Kase, 1986). One study (Koller and Kase, 1986) measured isometric grip strength as well as isotonic flexion and extension of the wrist, arm, and knee, of subjects with PD and in age-matched controls. The authors reported no significant difference between groups for isometric strength. However, compared to
controls, subjects with PD demonstrated a significant decrease in isotonic strength for all test movements. Our study is the first study to detect reduced hip strength in persons with PD, compared to controls.

There was no significant difference in the ability to generate force (assessed by an isokinetic dynamometer) between PD-on and PD-off. Also, there was no significant difference in torque between the most and least affected sides. Some authors have suggested that strength differences between the most and least affected sides (Kakimuma et al., 1998; Nogaki et al., 1999), or strength differences between an on and off-medication state (Corcos et al., 1996), indicate that decreased strength is due to the effects of the disease process. However, since the subjects with PD who participated in this study were affected by the disease to a mild degree, the lack of differences between on and off-state strength testing does not exclude that the weakness found in subjects with PD was due to central mechanisms. In fact, most of the subjects did not report having a distinct on and off-state. Therefore, we can only report that generation of force was affected similarly between the on and off-states.

11.2 Percent Effort:

All three subject groups used 36-40% effort at the hip and 70-71% effort at the knee to perform the STS task. Thus, the knee force necessary to complete the STS task was very high relative to the available strength. These values are in contrast to the findings of Kotake et al. (1993) who reported that healthy young adults used 27% of available hip strength and only 30% of available knee strength to perform the STS task. Therefore, it appears that age has a large effect on the force used at the knee during a functional task relative to the
available strength. Older adults have been reported to use 87% (Hughes et al., 1996) and 35-87% (Alexander et al., 1997) of available knee strength to rise from a knee height chair and up to 97% of available strength to rise from the lowest chair within the subject's ability (Hughes et al., 1996). Furthermore, Hughes et al. (1996) found that the large percent effort at the knee was due to decreased available strength (denominator) as opposed to a large joint moment generated during the STS task (numerator).

Given that subjects used approximately 70% effort at the knee joint to rise from a chair, it is not surprising that the STS task can be difficult for the elderly, particularly those with knee extensor weakness. One study (Riley et al., 1997) assessed failed STS trials in elderly subjects with a variety of neuromuscular, musculoskeletal, and vestibular impairments. The authors reported that failed STS trials (identified by either a sit-back or a step back maneuver) were associated with lower knee moment values, but not hip moment values, during lift-off.

The percent effort ratios were similar for both groups despite the finding that peak torque (denominator) and peak moment (numerator) values were all greater for the controls. Therefore, both groups likely used a similar motor strategy that required a fixed amount of effort at the hip and at the knee relative to the available strength.

It is possible that the magnitude of joint moments used during the STS task are fixed values of the available strength to result in optimal control of the task for each individual. For example, a subject who uses all of their available strength to rise from a chair may not have any reserve to recover from an unexpected perturbation or change to the task. Alternatively, there could exist a biomechanical relationship between the peak isokinetic
force produced in the selected posture and set angular velocity of the strength test, and the peak isotonic force produced during the STS task.

11.3 Relationship Between Strength and Function:

Overall, greater hip strength (measured concentric, isokinetic average torque) was related to better functional performance (shorter duration of STS) in subjects with PD. In contrast, greater knee strength was related to better functional performance in controls. Since there was no significant difference for STS duration between subjects with PD and controls, differences in the strength-STS performance relationship was not due to how quickly the task was performed. These findings suggest that performance of the STS task in subjects with PD is more dependent on the hip than on the knee. As mentioned in Chapter 1 (page 39), a post hoc analysis indicated that the hip to knee ratio (of peak joint moments) for subjects with PD did reveal a greater dependence on the hip, during STS, compared to controls.

The finding that STS performance in subjects with PD is more dependent on the hip than on the knee may be due to the finding that, compared to controls, the hip showed greater relative weakness. Specifically, average torque for the hip and knee extensors in PD-on was 70% and 90% of the value for controls, respectively. Perhaps the hip strength is the limiting factor in the performance of STS in the subjects with PD. The difference in relative weakness between the hip and the knee may be explained by previous studies that have identified differences between axial and distal motor impairment (Bridgewater and Sharpe, 1998; Weinrich et al, 1988). Axial motor impairment has been identified in persons with early stages of PD (Bridgewater and Sharpe, 1998). Significant differences were found between subjects with mild PD (stages I to II) and age-matched controls for all measures (flexion,
extension, right and left rotation) of trunk range of motion, isometric, and isoinertial strength (Bridgewater and Sharpe, 1998). Furthermore, the authors identified significant differences between subjects with stage I PD and subjects with stage II PD, for measures of trunk range of motion, average isometric torque, and maximal isometric torque. Their findings support the results of our study that indicate that subjects with PD seem to be more affected at the hip (axial) than at the knee (distal).

It is possible that the relationship between strength and STS performance could have been stronger if analyzed with a non-linear statistical method, given that Buchner et al., (1996) reported a non-linear relationship between leg strength and gait speed in older adults. In fact, one might expect a curvi-linear relationship between STS performance and hip or knee extensor strength where STS performance plateaus beyond a certain level of strength. However, observation of scatter plots of STS performance and strength did not provide evidence of a curvi-linear relationship.

11.4 Implications for Physical Therapy:

An important clinical question is: Do persons with PD have difficulty with STS due to the inability to generate extensor force at the hip and knee? The results from this study showed that subjects with PD are not able to generate as much force into hip and knee extension, compared to controls. Also, in subjects with PD, the ability to generate force at the hip is related to their functional performance (duration) of the STS task. However, since correlation studies do not infer causation, further research is required to evaluate whether improving the strength of hip and knee extensors would lead to improved STS performance.
While we were able to detect a reduced ability to generate force in subjects with PD, the relative contribution of the central and peripheral system could not be assessed. However, in support of a strengthening program, studies with healthy elderly subjects have shown that a strengthening program can prevent weakness secondary to disuse atrophy (Glendinning and Enoka, 1994; Judge et al., 1994). It has also been documented that strengthening in healthy elderly has been associated with increased motor unit recruitment (Frontera et al, 1988) and neural adaptations such as increased consistency of EMG activity (Enoka, 1988). Therefore, motor unit abnormalities seen in persons with PD may be amenable to change with a strengthening program (Glendinning and Enoka, 1994).

12 Limitations

One limitation of this study was that strength testing was done at only one speed, 45°/sec. Therefore, the relationship between force generation and speed of testing could not be explored.

Since the STS protocol was developed for maximal reliability of the data, the speeds and postures used during the task may not be exactly simulated by the strength testing protocol. Therefore, the correlations between STS performance and strength may be stronger than what was assessed in this study.

Reliability testing across days was not performed. However, although it is possible that there might be small changes to the absolute strength and STS performance values if tested on another day, it is likely that the relationship between strength and STS performance
would not change. In addition, the very low coefficient of variation for the strength STS duration suggested that the measures are reliable within a single test session.

Repeated paired t-tests were used with an alpha level of significance of .05. Therefore, there is an increased chance of a type I error. While it is understood that there is an increased chance of a type I error when a number of t-tests are run at an alpha level of .05, the aim of this study was to explore trends in STS performance of the three subject groups (PD-on, PD-off, and age-matched controls).

Finally, a small sample size (N=10 PD and N=10 controls) was used. This may increase the chance of a type II error due to large intersubject variability.
GENERAL CONCLUSIONS

Overall, there were a number of similarities in performance of the STS task between subjects with PD and age-matched controls. Firstly, the time to perform one STS maneuver (duration) did not differ significantly between the two groups at a self-pace. Secondly, the maximum joint moments generated by both groups was into knee extension followed by hip extension. Thirdly, there were no differences in EMG patterns for the lower extremity muscle groups between subjects with PD and controls, with the exception of abdominals and medial gastrocnemius. These similarities may be due to the mild degree to which subjects with PD were affected by the disease, as measured by the Hoehn and Yahr stage and the UPDRS motor score. However, even the subjects with mild PD used in this study showed some important differences, compared to age-matched controls, in the performance of the STS task.

During the STS task, subjects with PD, compared to age-matched controls, 1) generated a peak knee extensor moment of lesser magnitude, 2) generated a peak knee extensor moment earlier, and 3) showed no preparatory abdominal activity. Also, isokinetic strength measures of hip and knee extension indicated that persons with PD produced less hip extensor torque and less knee extensor torque than did age-matched controls.

The results of this study suggest that lower extremity strength is related to functional performance. However, strength training has not traditionally been a treatment intervention for persons with PD. This may be partly due to the fact that the widely used clinical assessment tool, manual muscle testing, is often not sensitive enough to detect weakness (Bridgewater and Sharpe 1998; Koller and Kase, 1986). In fact, to address motor function impairment in persons with PD, emphasis has been on increasing joint range of motion
(Schenkman et al., 1998; Schenkman et al., 1989; Viliani et al., 1999), repetitive practice of functional tasks (Corcos, 1991; Schenkman et al., 1989), increasing speed of movement (Homberg, 1993), and use of external cues to stimulate onset of movement (Cunnington, et al., 1999; Homberg, 1993). Only recent literature supports the use of a strength training program for persons with PD (Glendinning, 1997; Toole et al., 2000). However, no studies have investigated the effect of a lower extremity strengthening program on the performance of functional tasks such as STS.

13.1 Strength and Function:

The relationship between lower extremity strength and STS performance has been reported for older adults (Alexander et al., 1997; Bassey et al., 1992; Brown et al., 1995; Schenkman et al., 1996). For example, Alexander et al. (1997) reported that older women with knee extensor weakness had difficulty rising from a chair. Interestingly, weakness of the quadricep muscles and slowness in performing five repetitive chair rises have been identified as risk factors for falling in a sample of older women (Davis et al., 1999). The results from the controls in our study concur with these findings as the STS performance of controls correlated with knee extensor strength and not hip extensor strength. In contrast, the relationship between lower extremity strength and STS performance in PD is not the same as in the elderly. Thus, it is critical to avoid basing interventions for PD simply on performance of healthy elderly and to address impairments specific to individuals with PD.
13.2 **Impaired Preparatory Activity:**

Most of the literature on STS is focussed on the importance of lower extremity muscle force generated at the time of lift-off (Ikeda et al., 1991; Kotake et al., 1993; Schenkman et al., 1990; Schultz et al., 1992). In contrast, a recent study described STS as a two-part task and focused on the importance of postural activity and executional activity (Goulart and Valls-Sole, 1999). The authors defined the postural activity as being postural adjustments aimed at avoiding unwanted displacements during movement execution. Applying this two-part description, we found that persons with PD demonstrated impaired postural activity during the STS task. This was demonstrated by both the absence of preparatory abdominal activity and by the pre lift-off peak of the knee extensor moment. No previous studies have discussed altered postural or preparatory activity with respect to gross motor tasks such as STS. However, altered anticipatory postural control has been described in persons with PD for a standing arm raise task (Latash et al., 1995; Rogers et al., 1987) as well as altered function of the SMA resulting in impaired preparatory activity (Cunnington et al., 1996).

13.3 **Clinical Implications:**

It is important to understand the mechanisms underlying the difficulty to perform STS in persons with PD in order to develop appropriate treatment interventions and to gain insight into reasons why difficulty rising from a chair has been reported to be a risk factor for having multiple falls in persons with PD (Nevitt et al., 1989).
The findings of this study point to the importance of both lower extremity strength training (specifically of the hip and knee extensors) and the implementation of motor strategies to improve the performance of STS in persons with PD.

Support for the use of a strength training program for persons with PD comes from a recent study (Toole et al., 2000) that reported modest gains in the strength of knee flexors and extensors following a thrice weekly, ten week strengthening program in persons with PD (Hoehn and Yahr stages I-III). However, the authors did not look at how gains in strength affect performance of functional tasks such as STS.

Many interventions used to alter the motor impairment of persons with PD are based on the assumption that normal movement can be achieved by teaching movement strategies to compensate for altered basal ganglia activity (Morris, 2000). For example, since persons with PD rely on cortical control to initiate movement and frontal-cortical mechanisms to sustain movement, external sensory cues and other attention strategies (ie mental rehearsal) are recommended treatment tools (Morris, 2000).

The type of treatment intervention that would specifically address altered preparatory control of the STS task, is not known. Possibly, the use of verbal cues to facilitate movement and biofeedback to train preparatory abdominal activity could be effective treatment tools to improve STS performance in persons with PD.

13.4 Future Ventures:

This study provided support for the addition of a strength training program to the treatment of persons with PD for the purpose of improving STS performance. The next
recommended step is to test the effect of a hip and knee strength training program on the performance of the STS task.
REFERENCES


74


Schenkman M, Berger RA, Riley PO, Mann RW, & Hodge WA (1990) Whole-body movements during rising to standing from sitting. Phys Ther 70: 638-651


APPENDIX A

MODIFIED HOEHN & YAHR STAGES OF PARKINSON'S DISEASE


<table>
<thead>
<tr>
<th>Stage</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No signs of disease</td>
</tr>
<tr>
<td>1</td>
<td>Unilateral disease</td>
</tr>
<tr>
<td>1.5</td>
<td>Unilateral disease plus axial involvement</td>
</tr>
<tr>
<td>2</td>
<td>Bilateral disease, without impairment of balance</td>
</tr>
<tr>
<td>2.5</td>
<td>Mild bilateral disease, with recovery on pull test</td>
</tr>
<tr>
<td>3</td>
<td>Mild to moderate bilateral disease; some postural instability; physically independent</td>
</tr>
<tr>
<td>4</td>
<td>Severe disability; still able to walk or stand unassisted</td>
</tr>
<tr>
<td>5</td>
<td>Wheelchair bound or bedridden unless aided</td>
</tr>
</tbody>
</table>
APPENDIX B

HOEHN & YAHR STAGES OF PARKINSON'S DISEASE

Neurol 17: 427-442

<table>
<thead>
<tr>
<th>Stage</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Unilateral involvement only, usually with minimal or no functional impairment.</td>
</tr>
<tr>
<td>II</td>
<td>Bilateral or midline involvement, without impairment of balance.</td>
</tr>
<tr>
<td>III</td>
<td>First sign of impaired righting reflexes. This is evident by unsteadiness as the patient turns or is demonstrated when he is pushed from standing equilibrium with the feet together and eyes closed. Functionally the patient is somewhat restricted in his activities but may have some work potential depending upon the type of employment. Patients are physically capable of leading independent lives, and their disability is mild to moderate.</td>
</tr>
<tr>
<td>IV</td>
<td>Fully developed, severely disabling disease; the patient is still able to walk and stand unassisted but is markedly incapacitated.</td>
</tr>
<tr>
<td>V</td>
<td>Confinement to bed or wheelchair unless unaided.</td>
</tr>
</tbody>
</table>
APPENDIX C
MOTOR SECTION OF THE UNIFIED PARKINSON’S DISEASE RATING SCALE


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 ¼ inch or more

20. Tremor at rest
   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 Rigidit y (judged on passive movement of major joints with patient relaxed in sitting position; ignore cogwheeling)
   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 with widest amplitude possible, each hand separately)
   0=Normal
   1=Mild slowing and/or reduction in amplitude
   2=Moderately impaired. Definite and early fatiguing. May have occasional arrests in movement
   3=Severely impaired. Frequent hesitation in initiating movements or arrests in ongoing movement
   4=Can barely perform the task

24 **Hand Movements** (patient opens and closes hands in rapid succession with widest amplitude possible, each hand separately)
   0=Normal
   1=Mild slowing and/or reduction in amplitude
   2=Moderately impaired. Definite and early fatiguing. May have occasional arrests in movements
   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 or horizontally, with as large an amplitude as possible, each hand separately)
   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 ground in rapid succession, picking up entire leg. Amplitude should be about 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 arise from a straight-back wood or metal 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, and can have had some practice runs)
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
## APPENDIX D

### ELECTRODE PLACEMENT GUIDELINES

<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Tibialis Anterior</td>
<td>Over the greatest muscle bulk just lateral to tibial crest; most proximal half of the leg</td>
</tr>
<tr>
<td>Medial Gastrocnemius</td>
<td>Over the greatest muscle bulk on the medial side of calf</td>
</tr>
<tr>
<td>Biceps Femoris</td>
<td>On lateral thigh, midway between a line from the ischial tuberosity to the head of the fibula</td>
</tr>
<tr>
<td>Rectus Femoris</td>
<td>Distal (and medial) 1/3 between a line from the ASIS to the superior border of the patella</td>
</tr>
<tr>
<td>Vastus Lateralis</td>
<td>Midway between a line from the greater trochanter to the superior border of the patella – on the lateral aspect of the thigh</td>
</tr>
<tr>
<td>Erector Spinae - L1 (Right)</td>
<td>2 cm lateral of the L1 spinous process; count up 2 spinous processes from level of iliac crest</td>
</tr>
<tr>
<td>Rectus Abdominus (Right)</td>
<td>3 cm lateral to the umbilicus</td>
</tr>
</tbody>
</table>