THE EFFECT OF IDIOPATHIC PARKINSON’S DISEASE ON SEATED TRUNK REACTIONS

by

KATHERINE ELIZABETH PAUHL

B.Sc., University of Waterloo, 2005

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

MASTER OF SCIENCE

in

THE FACULTY OF GRADUATE STUDIES

(Human Kinetics)

THE UNIVERSITY OF BRITISH COLUMBIA

(Vancouver)

July 2008

© Katherine Elizabeth Pauhl, 2008
ABSTRACT

A common symptom of Idiopathic Parkinson’s disease (IPD) is decreased trunk and balance control. These deficits in patients with IPD are not treatable, and their underlying mechanisms are not well understood. Additionally, it is not known to what extent decreased trunk control contributes to postural instability in patients with IPD. Previous work by Martin (1965) observed that patients with post-encephalitic Parkinson’s disease would fall in the direction of the tilt when perturbed while seated. In order to better understand the underlying causes of these observed trunk deficits and attempt to replicate Martin’s findings, this study investigated postural corrective movement of the trunk while seated in patients with IPD and age-matched healthy controls. Participants’ range of motion (ROM) was tested actively and passively while lying supine, following which, bilateral electromyography (EMG) (rectus abdominis (RA), external oblique (EO), and erector spinae (ES₉, L₃)) and 3-D kinematic measures were recorded while participants were seated on a modified chair and received unexpected perturbations, 7° at 40°/sec, in four different directions (forward, backward, left, and right). EMG responses were normalized to participant’s maximum voluntary contractions. We observed patients with IPD to have decreased active and passive ROM only in the frontal plane relative to controls. Patterning of muscle responses to rotational perturbations did not vary between groups in any direction, except backward, and trends toward significantly greater ES₉ activity were observed during backward and left tilts in patients with IPD. Despite this both patients with IPD and controls were able to make appropriate trunk corrective movements opposite the direction of the tilt. However, two patients, who were most severely affected, did make incorrect trunk movements in the
direction of the tilt during left and right tilting perturbations which, upon visual inspection, appear to be due to improperly modulated and timed muscle responses. Thus, our data counters the findings of Martin, and suggests the trunk is posturally stable in IPD. Therefore, balance instabilities during stance are likely due to improper responses of the lower limbs. However, as disease severity increases, the contributing influence of an improperly responding trunk may add to their postural deficits.
# TABLE OF CONTENTS

Abstract .......................................................................................................................................................... ii

Table of Contents .......................................................................................................................................... iv

List of Tables ................................................................................................................................................. vii

List of Figures ............................................................................................................................................... viii

Acknowledgements ....................................................................................................................................... ix

1. BACKGROUND ...................................................................................................................................... 1
   1.1 Quiet standing ................................................................................................................................... 5

   1.2 Anticipatory postural adjustments (APAs) ....................................................................................... 7

   1.3 Standing reactive postural responses in a healthy age matched control population ....................... 8

   1.4 Standing reactive postural responses in an IPD patient population .................................................. 10

   1.5 Seated postural responses in a healthy age matched control population ......................................... 13

   1.6 Seated postural responses in an IPD patient population .................................................................... 19

   1.7 Influences on postural responses ..................................................................................................... 23

   1.8 Hypotheses ....................................................................................................................................... 24

2. METHODS ............................................................................................................................................. 25

   2.1 Subjects ........................................................................................................................................... 25

   2.2 Active and passive ranges of motion (ROM) .................................................................................... 26

      2.2.1 Setup ......................................................................................................................................... 26

      2.2.2 Procedure .................................................................................................................................... 27

      2.2.3 Measures and data analysis ....................................................................................................... 29

      2.2.4 Statistical analysis ..................................................................................................................... 30
2.3 Tilting perturbations ................................................................. 30
    2.3.1 Setup ............................................................................... 30
    2.3.2 Measures ........................................................................... 32
    2.3.3 Procedure ........................................................................... 33
    2.3.4 Data analysis ...................................................................... 34
    2.3.5 Statistical analysis .............................................................. 36
3. RESULTS .................................................................................. 37
    3.1 ROM kinematics ................................................................. 37
        3.1.1 Sagittal plane trunk flexion and extension ......................... 37
        3.1.2 Frontal plane trunk flexion (left and right) ......................... 39
    3.2 Tilting perturbations ......................................................... 39
        3.2.1 Sagittal plane perturbations .............................................. 39
        3.2.2 Frontal plane perturbations ............................................... 46
    3.3 Case studies .......................................................................... 47
4. DISCUSSION .............................................................................. 51
    4.1 Lateral range of motion (ROM) of the trunk was not influenced by IPD ... 51
    4.2 Timing and amplitude of corrective trunk movements in the sagittal plane are not influence by IPD ................................................................. 53
    4.3 Timing, but not amplitude of corrective trunk movements in the frontal plane are influenced by IPD ................................................................. 54
    4.4 Evidence for lateral trunk instability was observed in most severely affected patients with IPD ................................................................. 56
4.5 Patients with IPD have altered patterns of muscle responses to backward perturbations .................................................................57

4.6 Larger muscle response magnitudes were observed in patients with IPD when responding to backward and leftward tilting perturbations ......................58

4.7 Possible mechanisms for muscle co-contraction and improperly modulated muscle responses in patients with IPD.................................................................59

4.8 The role of the trunk in postural stability.................................................61

4.9 Strengths and limitations.........................................................................64

5. CONCLUSION ..............................................................................................67

5.1 Future research..........................................................................................67

REFERENCES ..................................................................................................69

APPENDIX A ....................................................................................................83

APPENDIX B ....................................................................................................85
LIST OF TABLES

Table 1  Demographic and disease characteristics of controls and patients with IPD

Table 2  Normalized MVC % and raw group 100 ms area averages for rectus abdominis (RA), external oblique (EO), erector spinae (ES$_{T9}$, ES$_{L3}$)
# LIST OF FIGURES

<table>
<thead>
<tr>
<th>Figure</th>
<th>Description</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>Figure 1</td>
<td>Tilting reactions from Martin (1965)</td>
<td>20</td>
</tr>
<tr>
<td>Figure 2</td>
<td>Range of motion (ROM) apparatus</td>
<td>27</td>
</tr>
<tr>
<td>Figure 3</td>
<td>Passive range of motion apparatus</td>
<td>28</td>
</tr>
<tr>
<td>Figure 4</td>
<td>Tilting perturbations apparatus</td>
<td>31</td>
</tr>
<tr>
<td>Figure 5</td>
<td>Group averages of active and passive ranges of motion, and stiffness graphs</td>
<td>38</td>
</tr>
<tr>
<td>Figure 6</td>
<td>Average group traces of trunk corrective movements from the T3 rigid body</td>
<td>40</td>
</tr>
<tr>
<td>Figure 7</td>
<td>Patterns of muscle activation during forward and rightward tilts</td>
<td>42</td>
</tr>
<tr>
<td>Figure 8</td>
<td>Normalized ES&lt;sub&gt;T9&lt;/sub&gt; MVC %, 100 ms area data during forward and backward tilting perturbations</td>
<td>45</td>
</tr>
<tr>
<td>Figure 9</td>
<td>Case studies: Tilting kinematics from T3 rigid body of incorrect corrective trunk movements</td>
<td>49</td>
</tr>
<tr>
<td>Figure 10</td>
<td>Case study: Muscle response characteristics of an incorrect and correct trunk response to a left tilt</td>
<td>50</td>
</tr>
</tbody>
</table>
ACKNOWLEDGEMENTS

I would firstly like to thank my supervisor Dr. Mark Carpenter. Mark, I would like to thank you for your patience and encouragement throughout my Masters. I deeply appreciate all you have done for me, and can not thank you enough for taking me on as your first graduate student. Your unwavering support and guidance throughout the past three years has shaped and influenced not only who I am as a researcher but has directly impacted me personally. I am proud to say I am leaving UBC with not only a Masters degree and the accredited qualities that are associated with it, but also a new found outlook at life and personal expectations. I only hope that I am able to bring as much devotion to my future endeavors as you bring to your research and students.

To my committee members Dr. Timothy Inglis and Dr. Romeo Chua, I would like to express my gratitude for all your support, encouragement and guidance during my time at UBC. Your informative suggestions and knowledge helped increase the depth of my project. While your caring, friendly natures made it easy to approach both of you with questions no matter how big or small and helped to make this a truly enjoyable experience. I would also like to thank Dr. Inglis for welcoming me and allowing me to be apart of his lab during my first year.

To my lab members Adam Campbell, Justin Davis, Chantelle Murnaghan, Brian Horslen and Katie Fukushima, I could have asked for no one better to have shared this experience with and I would like to thank all of you for taking the time to review and edit my thesis, providing helpful suggestions and supporting me. I would also like to thank Melanie
Lam for her friendship and the time she committed to editing and providing feedback on my thesis.

This project would not have been possible without the help and support of the recruiting staff at the Pacific Parkinson’s Research Centre. Thank you for all your hard work to ensure we had participants and for seeing the value and importance of this research.

To my fellow graduate students, whose names are too many to list, thank you for your support, friendships and many laughs.

I would like to thank all my friends and family back in Ontario for their support and kind words, and would specifically like to thank my parents. Their untiring support now and throughout my life has been essential for battling life obstacles and has been the backbone of my success. Thank you for all you have sacrificed and all you have given me.

Lastly I would like to thank my husband Andrew Pauhl who selflessly put on hold his academic pursuits so that I could follow mine. His support, love and encouragement were my crutch in times of weakness and were what saw me through to the end. You are a blessing in my life and inspire me to be a stronger person.
1. BACKGROUND

Idiopathic Parkinson’s disease (IPD) was classically described in 1817 by James Parkinson. An English physician, and paleontologist, James Parkinson described and depicted the classic symptoms of Parkinson’s disease in his book, “An Essay on Shaking Palsy”. However, it was not until the 1830’s when Wilhelm von Humboldt (1767-1835) self documented, in a series of letters, the progression and development of the disease that it became well known and accepted in the medical profession. However, it still took until the end of the 19th century to be accepted as a special disease (Horowski et al., 1995).

Today, the human population is anything but unaware of Parkinson’s disease. With major Hollywood celebrities, such as Michael J Fox, presenting with the disease an increased amount of attention and research has been focused on it; and for good reason. Presently, 10% of the American population is now over the age of 65yrs, and over a million of those individuals are believed to have IPD (Morris, 2000). It is thought that by the year 2020 more than an estimated total of 40 million people world wide will have this disease (Morris, 2000).

Though no set cause has been pinpointed as of yet, current thoughts regarding the cause of the disease are extensive and can include; mitochondrial dysfunction and oxidative metabolism, excitotoxins or neurotrophic factors (Lang & Lozano, 1998).

Idiopathic nature of Parkinson’s disease

Idiopathic Parkinson’s disease (IPD) is known foremost as a progressive movement disorder, resulting from degeneration of dopaminergic cells within the basal ganglia (BG). The BG itself is made up of four main nuclei: (1) striatum (putamen, and caudate); (2) globus pallidus (internal and external segments); (3) subthalamic nucleus and (4) substantia nigra (pars reticulata, and compacta parts). The degeneration of dopaminergic cells, in IPD,
been found to be localized to the substantia nigra pars compacta (SNpc). The major input nuclei of the BG is believed to be the striatum, which receives projections from the frontal cortex and limbic areas. In contrast, the major output nuclei of the BG is the globus pallidus internus (GPi).

There are two different pathways within the BG nuclei, the output of which influences the level of cortical excitation in motor control areas via the thalamus. Both pathways originate in the putamen, the main input nuclei in the BG. The ‘direct’ pathway involves the putamen, and GPi and has a net excitatory effect on the thalamus. The indirect pathway involves the putamen, GPe, sub-thalamic nucleus and GPi, and has a net inhibitory effect on the thalamus. One analogy is to think of these pathways as the ‘accelerator’ and ‘decelerator’ respectively. These pathways work in parallel and are regulated by dopaminergic inputs originating from the SNpc to facilitate voluntary movement.

In healthy individuals dopamine has an excitatory effect on the direct pathway and an inhibitory effect on the indirect pathway. In this way movements are properly facilitated and can be performed with ease. Conversely, diseases involving dopamine deficiencies, such as IPD, result in decreased activity of the direct pathway enabling the indirect pathway to over activate the GPi due to overly active STN projections (Ferrarin et al., 2004).

Degeneration of dopaminergic cells results in motor control deficits. These deficits can range, but are not limited to, shuffling gait, bradykinetic movements, tremors, akinesia, postural instability and increased occurrences of falls (for review see (Jankovic, 2008). In addition to the above stated symptoms of IPD, clinical symptoms present such as reduced flexibility (range of motion – ROM) (Pedersen et al., 1997) and impaired axial control (such as turning in bed) in IPD patients (Stack & Ashburn, 2006; Steiger et al., 1996). In many
cases, patients with IPD present with inconsistent sleep patterns in conjunction with fatigue due to restless night’s sleep (Stack & Ashburn, 2006), and must fully sit up to turn themselves in bed because they lack the ability to rotate their trunk (Stack & Ashburn, 2006).

Supplementary Balance Disorders

In addition to IPD, there are a number of supplementary balance disorders that despite their IPD symptomatic commonalities (such as those listed above) are quite different in terms of their mechanistic causes. Some of the current parkinsonism movement disorders include corticobasal degeneration, diffuse lewy body disease, encephalitis, vascular parkinsonism, progressive supranuclear palsy (PSP) and Wilson's disease (Lang & Lozano, 1998). For instance, PSP involves the deterioration and death of cells in selected areas of the brain and is not isolated to the BG. Therefore, the possibility exists that balance control deficits seen with PSP may not be directly related to specific BG dysfunction because additional areas of the brain may be affected. Advances in imaging techniques and quality now enable researchers to better understand the neuroanatomical differences that occur with PSP pathology. For instance through magnetic resonance imaging (MRI) researchers have found that PSP patients have an enlarged third ventricle, lower midbrain diameter and a smaller/thinning quadrigeminal plate compared to patients with IPD (Barsottini et al., 2007). Similarly, post encephalitic parkinsonism symptoms are caused by swelling of the entire brain and the death of cells as the brain is forced into the side of skull. This again speaks to the point that cell death is not localized to the same brain regions in these parallel disorders and concurrent damage in additional cortical sites may be occurring.
Treatment

Common treatment for IPD can come in two forms; pharmaceutical or through surgical means such as deep brain stimulation (DBS). During early stages of IPD most patients are very receptive to drug therapy, such as levodopa (L-dopa) or other dopamine agonists. These drugs provide relief from the most established symptoms such as bradykinesia, akinesia, tremor and deficits in gait (Ferrarin et al., 2004; Loher et al., 2002; Steiger et al., 1996). **However, both trunk and postural control deficits do not benefit from this treatment** (Bonnet et al., 1987; Koller et al., 1989; Maurer et al., 2003; Vaugoyeau et al., 2007). Although some studies have observed IPD patients to have a short term benefit from L-dopa treatment on clinical axial measures such as turning in bed (Steiger et al., 1996), over time, as the disease progresses, the effect of L-dopa diminishes and improvements are not as noticeable (Ferrarin et al., 2004; Maurer et al., 2003).

As IPD progresses and reaches its more advanced stages and when drug treatment efficacy declines, DBS can be used as a supplement therapy (Hallett & Litvan, 1999) to aid in managing symptoms (Bejjani et al., 2000; Shivitz et al., 2006). DBS involves surgically implanting a stimulating electrode into the GPi or STN. A wire is then passed from the electrode under the skin to a control unit placed subcutaneously below the clavicle. Patients are then capable of triggering the stimulation by placing an additional magnet over the termination unit under the skin. Once triggered DBS results in a decrease in the over active indirect pathway by removing inhibitory output from the GPi to the thalamus, resulting in an improvement in motor performance (Ferrarin et al., 2004; Maurer et al., 2003). However, postural stability does not appear to benefit from DBS and postural responses are slower compared to controls (Shivitz et al., 2006).
The current lack of an effective treatment for postural instability and trunk control deficits in patients with IPD, results in patients suffering from an increased risk of falling (Grimbergen et al., 2004). In addition IPD patients adopt inefficient and improper response strategies to balance perturbations. These responses include stiffening their trunk and pelvis through increased co-contraction (Carpenter et al., 2004), narrowing their base of support (Dimitrova et al., 2004b) and limiting and or altering their protective arm movements (Carpenter et al., 2004). When all these factors are combined, IPD patients become severely prone to falls. The importance of better understanding the postural deficits that exist within the IPD patient population is clearly demonstrated by Bloem et al., (2001). In their six month study, 50% of IPD patients reported a fall. Of these individuals 35% suffered a serious injury from the fall or experienced more than one fall over the six month course.

Based on these findings it is clear that patients with IPD are prone to falling. Although we are aware of these features of IPD, we do not understand what is causing these deficits to develop, or to what degree these deficits affect balance control. One question that arises from this base level knowledge is whether there is a relationship between decreased trunk control and postural stability in patients with IPD? The following section will review current literature on postural instability of IPD, with the aim to identify potential relationships between postural control and trunk control deficits in IPD.

1.1 Quiet standing

Characteristics of standing postural control in a healthy and IPD patient populations

For most healthy individuals standing quietly is not a demanding task. As participants maintain a straight upright posture, changes in body position are constantly
occurring. Humans continuously sway and change their centre of pressure (COP) as they stand as a means to preserve balance.

Such an assumed “simple” task becomes challenging for individuals with balance deficits such as IPD. During the initial stages of the disease patients present with a more backward tilt to their posture. However, as the disease progresses increased muscle rigidity and decreases in ROM begin to set in and patients are observed to have more of a forward lean to their posture (Vaugoyeau et al., 2007). The forward lean is a classical characteristic of IPD known as stooped posture or camptocornia (Umapathi et al., 2002).

However, it has also been postulated that camptocornia may be a strategy used by patients to bring themselves away from the direction in which they most often experience falls, backward (Jacobs et al., 2005). Research has found that if asked to close their eyes while standing, patients with IPD will actually move back toward their more feared position, backward. This finding suggests that IPD patients may be consciously maintaining a more forward lean of their trunk (Kitamura et al., 1993) and that camptocornia is more of a postural strategy than it is a direct symptom of the disease. It has also been postulated that myopathy may be a mechanistic cause of camptocornia. The effects of improperly functioning muscle fibers within the trunk and neck extensors, compounded by fat stores replacing depleted muscle and altered motor unit potentials, none of which are common with aging, could possibly account for changes in IPD posture (Schabitz et al., 2003).

Not only are there changes in IPD patient’s postural angle, but there are also marked changes in their ability to maintain quiet standing. This is evident from increased COP oscillations and decreased postural limit boundaries (Schieppati et al., 1994). Despite these changes, it is thought that patients with IPD are fairly stable while standing and problems
arise when IPD patients attempt to move from a static position to a more demanding motor control scenario. For instance, when asked to perform a “simple” task, such as rising onto their toes, patients have difficulty generating the necessary amount of torque and have delayed centre of mass (COM) responses (Frank, Horak, & Nutt, 2000).

1.2 Anticipatory postural adjustments (APAs)

APA characteristics; control population

In order for the body to generate a discrete movement or move from a static position such as quiet stance to walking, changes within the body’s centre of gravity are required. These movements result in a disequilibrium of balance (Cuisinier et al., 2005; van der Fits et al., 1998). In anticipation of this upcoming movement, a strategy of activating trunk musculature in the opposing direction of the upcoming events occurs to counteract the expected mechanical affects of the perturbation (Cuisinier et al., 2005; Massion, 1992). The activity seen in the postural muscles has been termed an anticipatory postural adjustment (APA). APAs are temporally and spatially adaptable depending on the speed, displacement, load, symmetry and postural support used during the movement (van der Fits et al., 1998). Research examining APA activity when participants perform various upper limb movements while standing, has been documented in a control population (Lee et al., 1995; van der Fits et al., 1998). In most cases, APA activity while standing is seen in both the leg musculature (rectus femoris, biceps femoris, tibialis anterior, soleus) and trunk superficial musculature (rectus abdominis, and erector spinae) and are direction specific to the upcoming movement (Aruin & Shiratori, 2003; P. Hodges et al., 1999).
APA characteristics; IPD population

Patients with IPD demonstrate abnormalities when attempting to generate anticipatory muscle action prior to a self generated movement (Frank, Horak, & Nutt, 2000; Lee et al., 1995). Research examining the activity of APAs in a Parkinson population while standing and performing upper limb movements suggests that APA activity is rarely present, approximately 5% of time, compared to 100% of time in otherwise healthy controls (Bazalgette et al., 1987). Similar changes in APAs with IPD are observed during voluntary leg movements (Lee et al., 1995) gait initiation (Martin et al., 2002) and rising to the toes (Frank, Horak, & Nutt, 2000). However, postural adjustments made by patients with Parkinson’s disease appear to be subject to change and depend on the magnitude of the perturbation used to trigger the activity (Aruin et al., 1996).

1.3 Standing reactive postural responses in a healthy age matched control population

Biomechanics

When we compare self generated movements to standing reactive postural control, a clearer understanding of the balance deficits seen in IPD patients emerges. As would be expected, humans are posturally more stable when self triggering a movement in comparison to having to reactively respond to a perturbation (Nougier et al., 1999). In a laboratory setting, standing reactive perturbations can be generated through either translational or rotational perturbations. Responses to rotational or translational perturbations are typically directionally specific. Current literature regarding the order of muscle activation of these postural responses to a dynamic disturbance is divided. While some believe muscle onsets and balance correcting responses ascend in a distal to proximal fashion (Horak et al., 1996),
others have found a more proximal to distal activation order (Allum et al., 2002; McIlroy & Maki, 1995). During anterior-posterior translations or pitch plane rotations, plantar or dorsiflexor torques within the ankle are required to maintain stability. Typically speaking, regardless of perturbation direction, the required torque is generated within the first 150-200ms following platform movement (Carpenter et al., 2004; Horak et al., 1996). In control participants, the generation of torque is adaptable to both velocity and perturbation amplitude (Horak et al., 1996). Depending on perturbation direction, changes in trunk position are seen. For instance, fast backward perturbations cause a forward inclination of the body (Dietz et al., 1993); whereas a forward, or anterior perturbation, cause the body to become more erect (Dietz et al., 1993). In most cases, trunk responses are seen approximately 100ms following the perturbation (Allum & Honegger, 1992; Carpenter et al., 2004). Quick responses observed in trunk activity stress its importance and contribution to balance correcting activity. As the trunk is a key factor in maintaining postural stability, it can also limit an individual’s ability to respond to a perturbation. This may be due to intersegmental caudo-rostral spinal motion and anatomical restrictions (Preuss & Fung, 2007).

The spinal column is designed to have a greater ROM in the sagittal plane. Ligaments, muscle attachments and spinous processes all contribute to stabilizing the spine and in doing so limit it’s ROM in the posterior direction. This makes it more difficult to respond as efficiently to perturbations such as anterior translations. Such anatomical restraints, as well as those associated with other joints such as the ankle; also make it more challenging to respond to perturbations in the medial-lateral direction. Due to limitations in ROM quick movements, such as quick backward movements, cause the CoM to fall outside the base of support and are difficult to recover from.
Muscle responses

As previously stated, muscle responses to reactive perturbations are typically direction specific and act to oppose the upcoming perturbation. Muscle responses to a backward or posterior perturbation elicit activity in GAS, hamstrings and paraspinals (Horak et al., 1996). Anterior perturbations tend to cause activity in TIB, soleus and biceps femoris (Dietz et al., 1993; Dimitrova et al., 2004b). In comparison to healthy young controls, elderly participants generate atypical responses when the velocity of the posterior perturbation is increased. In instances such as these, elderly participants generate activity in the quadriceps with reciprocal activity occurring between hamstrings and GAS (Horak et al., 1996).

1.4 Standing reactive postural responses in an IPD patient population

Biomechanics

When perturbed, patients with IPD tend not to produce sufficient amounts of torque in response to the perturbation (Carpenter et al., 2004; Horak et al., 1996). Similar to the negligible effect anti-parkinson medication has on balance control, torque generation is not enhanced nor does it greatly improve while participants are ON anti-parkinsonian medication (Horak et al., 1996). However, patients are capable of scaling torque production to various velocities and small changes in amplitude; although, these changes are not as adequately scaled as those observed in controls (Horak et al., 1996). The limited ability to accurately generate torque is a major concern for IPD patients as this deficit has been found to correlate with a patient’s degree of postural instability (Frank, Horak, & Nutt, 2000), which also declines with disease progression.

When IPD patients are required to respond to translational perturbations alternating
between a wide and narrow base of support, they are unable to adapt their postural responses correctly (Dimitrova et al., 2004a; Dimitrova et al., 2004b). During instances such as these, IPD patients are most unstable when standing with a narrow base of support (Dimitrova et al., 2004b). This finding is most perplexing considering the fact that patients with IPD naturally adopt a narrower base of support with disease onset and progression. Such an unstable base of support could be one of the factors that contribute to their postural instability. Of great interest is the fact that patients with IPD also respond to perturbations with fewer corrections and modulations in the upper body or trunk compared to controls and when trunk corrections are present they are delayed in onset (Dietz et al., 1993). Their inability to accurately control and modulate trunk activity may be a key factor in explaining and better understanding their postural deficits. These inabilities may be a causal factor to their increased susceptibility to falls.

Muscle responses

The ability to finely control and modulate postural responses is accomplished by reciprocal asymmetric muscle activation based on multi-sensory afferent information. The inability to effectively incorporate these two aspects of balance control can cause an individual to become prone to postural imbalances and falls. This inability is one of the fundamental deficits that persist in patients with IPD. Although their muscle onsets to reactive perturbations are similar to those of healthy age matched elderly controls (Horak et al., 1996), patients with IPD react to dynamic perturbations with symmetrical co-activation of both agonist and antagonist muscles (Carpenter et al., 2004; Dietz et al., 1993; Dimitrova et al., 2004a; Dimitrova et al., 2004b; Horak et al., 1996). Additionally, they have tendencies
to activate antagonist muscles earlier than agonists especially when perturbed at higher velocities (Dietz et al., 1993; Dimitrova et al., 2004a; Dimitrova et al., 2004b; Horak et al., 1996). These tendencies are not only seen in the lower musculature, but also in the trunk (Dimitrova et al., 2004b; Horak et al., 1996). These unfavorable postural responses create a situation where postural modulation is not feasible as the trunk becomes more rigid. This stiffening strategy is exacerbated as the magnitude of the response is increased beyond typical balance limits (Carpenter et al., 2004; Dimitrova et al., 2004a; Dimitrova et al., 2004b).

Atypical muscle responses are common among all perturbation directions indicating that the greater instability observed in lateral and backward perturbations could in part be due to biomechanical limitations (Dimitrova et al., 2004b).

The importance and impact of sensory information on postural responses, especially for patients with IPD, becomes clear when a given sensory system is removed. When required to respond to perturbations with no vision, IPD patients lose the ability to make a reactive counter movement. The dependency these individuals place on the visual system is evident and could possibly aid in explaining their overactive use of antagonist flexor muscle activity versus agonist extensors. Flexor muscles have been found to be directly affected and modulated by visual information in comparison to extensors (Dietz et al., 1989; Dietz et al., 1992; Dietz et al., 1993).

Although this evidence supports the improper and detrimental balance correcting strategies employed by patients with IPD, it is difficult to isolate where the deficits are originating. It is also unclear whether the body as a whole is improperly responding or whether one segment is the main contributor to postural deficits. Literature to date has
focused on the contributions and reactions made by the lower limbs to dynamic perturbations, while little emphasis has been placed on the relative contributions made by the trunk. Because there is such a dominant bias and focus on dynamic balance control research on the lower limbs, it is not known how the trunk alone, irrespective of the lower limbs, responds to dynamic balance disturbances especially in a patient population such as IPD. Furthermore, it is not known how atypical muscle activity in the trunk could be contributing to an overall decreased ability to maintain balance. The following section reviews the limited research of trunk control during dynamic perturbations in both control and patient populations.

1.5 Seated postural responses in a healthy age matched control population

Seated postural responses in a control population

Completely isolating the trunk is not an easy task. In different seated paradigms participants may sit with their feet resting on either the apparatus surface itself (Forssberg & Hirschfeld, 1994), on low friction surface (Zedka et al., 1998), or secured to a chair (Preuss & Fung, 2007). It is also common for participants to sit self supported with their legs free to hang from the support surface (Martin, 1965). Despite the differences in apparatus set up, each paradigm directly assesses one’s ability to dynamically control their balance with almost complete isolation of the trunk.

Feedforward and feedback reactions

An additional way to gain insight into the relative contributions of the trunk in postural control is to have participants perform dynamic balance tasks such as maintaining
balance on a wobble board. In contrast to reactive perturbations, such as those mentioned above, this paradigm requires participants to use both feedforward and feedback control methods to maintain postural stability and balance. The balance aspect of this task is different in that the participant must actually balance/stabilize the support surface instead of their body position relative to the support surface. During acts of dynamic balance control, an increased difficulty in maintaining balance in the medial-lateral direction during sitting (Cholewicki et al., 2000) could reflect inadequate feedback processing to modulate phasic muscle activation patterns.

Important factors that contribute to an individual’s ability to maintain postural control, especially in this scenario, are trunk length and body weight. Individuals who are longer in the trunk and are greater in weight have an increased difficulty stabilizing themselves during such challenging feedforward feedback balance scenarios (Cholewicki et al., 2000). A raised COM combined with a greater body weight to support requires increased muscular force to overcome the torque generated at the hip joint. Unsuccessful force or muscle pattern generation would otherwise provide ample freedom for the COM to deviate from outside of the base of support and a less stable and unsuccessful trial could occur.

**Biomechanics of seated postural adjustments in a control population**

Whether translational or rotational perturbations are given, one of the cardinal characteristics observed across subjects is rotation of the pelvis following a flexion or extension of the hip joint respectively. Backward or forward tilts of the pelvis, seen during forward translations or legs up rotations and backward translations or leg down rotations respectively, are consistently observed within milliseconds of platform movement (Forssberg
Based on what little research there is, it is hypothesized this pelvic activity triggers the pattern of postural response required to compensate for rotational perturbations (Forssberg & Hirschfeld, 1994; Hirschfeld & Forssberg, 1994). The initial trigger is thought to act in a manner similar to that of a central pattern generator (CPG) as seen with gait. Further shaping of the response occurs based on previous experience (ie internal models) and the integration of sensory information arising from changes in such as head orientation/position (Forssberg & Hirschfeld, 1994; Hirschfeld & Forssberg, 1994).

Changes in trunk position, as seen through changes in thorax positioning, occur following rotations of the pelvis (60-70ms) (Forssberg & Hirschfeld, 1994). Those responses described above for toe up rotations or forward translations are also accompanied with a downward acceleration, then backward rotation of the head. Toe up and backward translations are subject to greater response variability (Forssberg & Hirschfeld, 1994). Though sequences of body segment responses are somewhat similar between rotational and translational perturbations there are a few variations. For instance, changes in hip responses are seen depending on perturbation type. Forward translations of a platform cause an extension of the hip joint, while leg up rotations cause a flexion of the hip joint. Similarly, movements of the head and thorax are in the backward direction during a forward translation, but are in a forward direction during a leg down rotation (Forssberg & Hirschfeld, 1994). Changes in biomechanical characteristics between these two perturbation types should not be unexpected as each possesses different restraints on the responding muscles. That is, translation perturbations cause stretch reflexive responses in the same muscles that are required to counteract the perturbation, whereas rotational perturbations do not stretch those
muscles required to maintain stability and balance. The advantage of using rotational perturbations is that there is a clear onset of muscle activity indicative of the actual balance correcting response. In contrast, muscle response onsets during translation perturbations have onsets that are intermixed with stretch reflexive activity.

Directional activity of seated postural muscles and muscle characteristics

Seated participants have a greater degree of background, or tonic, activity in the dorsal muscles (erector spinae (ES), latisimus dorsi (LD)) compared to ventral muscles such as external and internal oblique and rectus abdominis (Zedka et al., 1998). Similar to what would be expected during standing (Preuss & Fung, 2007), seated postural muscle activity is dependent on the direction of the perturbation. When participants are seated and translated quickly forward or given a legs up rotational perturbation, activity of more ventral muscles (ie. rectus abdominis (RA) and internal and external obliques (IEO) are seen while dorsal muscles (ie ES or paraspinal muscles) relax (Forssberg & Hirschfeld, 1994; Zedka et al., 1998). Following the perturbation a gradual increase in dorsal muscle activity is representative of postural correcting or long latency muscle responses. In some instances, activity of rectus femoris (RF) has been found to precede activity in ventral muscles in response to a quick leg up rotation. The trigger for this activity may originate from fast flexion of the hip created by the perturbation (Forssberg & Hirschfeld, 1994). Early onset of this muscle activity may also be indicative of a stabilizing strategy used to limit rotation of the pelvis.

The similar directional sensitivity seen during fast leg up rotations is additionally seen during legs down. However, the decrease in muscle activity seen in the ventral muscles
during a forward rotation (legs down) is not as great as that seen in dorsal muscles during backward (leg up) perturbations (Zedka et al., 1998). As well, stretch reflexive activity is not as apparent, nor is a consistent muscle pattern maintained (Forssberg & Hirschfeld, 1994).

With respect to fast rotational perturbations in the frontal plane, muscle activity increases in those muscles contralateral to the perturbation while ipsilateral muscles become more relaxed (Zedka et al., 1998). That is, if a leftward perturbation were to be given, left ES (antagonist) activity would decrease while the right ES (agonist) activity would increase. In conjunction, after the initial perturbation, phasic muscle activity occurs between these two agonist and antagonist muscles (Zedka et al., 1998). The directional specificity that is seen with respect to activation patterns of these muscles is not unexpected. A tilt to the left would not be well compensated for with increased left ES activity as this would further destabilize the participant and pull them in the direction of the perturbation instead of counteracting it. Also, modulation of such a quick response in a phasic manner is advantageous as a more accurate and specific response can be generated to countermand the perturbation.

Unlike dorsal muscles, ventral muscle activity (RA) during frontal plane perturbations do not demonstrate phasic muscle activation patterns (Zedka et al., 1998). The uncharacteristic responses seen in the ventral muscles could be related to their role in ventilation (Hodges et al., 2007).

For the most part a similar pattern of direction specific muscle activation is seen during translational seated perturbations (Forssberg & Hirschfeld, 1994). However, there are small differences in muscle responses between rotations and translation perturbations. For instance, rotational perturbations elicit some dorsal muscle activity first (RF) during legs up rotations prior to ventral muscles, translated perturbations generate initial responses in the
ventral muscles as a whole prior to dorsal muscle activity (Forssberg & Hirschfeld, 1994). Variations in neck muscle onsets also occur. Neck flexors have an early onset time during forward translations (Forssberg & Hirschfeld, 1994). In contrast, legs up rotations elicit early onset times in neck extensors. In general neck muscle activity and the changes seen with it, correspond well to opposing the movement generated from the trunk and head (Forssberg & Hirschfeld, 1994).

Influences on seated postural responses

The speed of a perturbation does appear to impact muscle activation patterns. Slow backward perturbations (legs up rotation) induce stretch reflexive muscle activity in the dorsal muscles, while a more gradual increase in ventral or abdominal muscles are seen. This is in contrast to what is seen during the quick large bursts of muscle activity during a fast perturbation. Forward rotations (legs down) at this slower speed cause parallel and gradual increases in both dorsal and ventral muscle activations. A phasic muscle activation response in the frontal plane diminishes while the directionally specific pattern of muscle activity remains constant (Zedka et al., 1998).

In contrast, the impact manipulations to vision and expectancy of a perturbation do not seem to influence muscle activation patterns while seated in a control population (Zedka et al., 1998).

An additional factor that does not appear to influence muscle activation patterns during seated rotational perturbations is participant’s body position relative to the axis of rotation. Changing participants seated position relative to the axis of rotation does not alter muscle activation patterns despite changes in head acceleration that this causes (Forssberg &
Hirschfeld, 1994). Because of this, the relative contribution of the vestibular system in coordinating a postural adjustment in these paradigms remains unclear.

Although we appear to have some understanding regarding seated postural control, when we compare this to what is documented in the field of standing balance, relatively speaking, little is actually known. As this field advances the importance of trunk control in maintaining balance and postural stability is becoming more apparent. Especially when looking at populations with balance deficits such as patients with Parkinson’s disease. With that stated, it is necessary to point out the fact that even less is known regarding trunk control in this clinical population. The following sections outline what information is currently available with respect to seated trunk control in an IPD patient population.

1.6 Seated postural responses in an IPD patient population

*Feedforward and feedback reactions*

Previous research by van der Burg (2006) observed IPD patients changes in COP measures during a postural feedforward and feedback task. From these data a general understanding of how muscle activation may be coordinated to maintain dynamic seated balance can be formulated. When required to balance using the wobble board paradigm, van der Burg et al (2006) observed that patients demonstrated increased COP excursions in combination with increased medial-lateral (ML) root mean square (RMS) values and decreased mean power frequency (MPF) values. These data demonstrate the large variability and lack of trunk control in IPD patients. As previously mentioned delays in balance correcting adjustments and greater COP excursions may be reflective of decreased asymmetrical muscle activation. As much as this study demonstrates IPD patient’s inability
to properly maintain balance and trunk control, it does not reflect their ability to reactively respond to an external perturbation because this task requires both feedforward and feedback information processing while a reactive perturbation task does not.

**Biomechanics of seated postural adjustments in an IPD patient population**

Martin (1965) investigated postural responses in patients with post encephalitic Parkinson’s (PEP) disease, among other patient groups, while patients were seated on a manually tilted bed (Figure 1). Following the perturbation in the seated condition, patients appeared to have no postural response and would fall or tip in the direction of the tilt (Figure 1B). In contrast, a healthy control participant would have a balance correcting response in the opposing direction of the tilt (Figure 1C).

**Figure 1: Tilting reactions from Martin (1965)**

These figures illustrate the trunk responses of subjects with; (A) complete loss of labyrinthine function, (B) a post-encephalitic Parkinsonian patient and (C) control participant as observed by Martin (1965).

**Directional activity of seated postural muscles and muscle characteristics**

Despite the need to gain an increased understanding of muscle activity in the trunk within this patient group, research to date is limited. With this stated, when attempting to better understand the muscle responses during a seated paradigm, one could make sound
hypotheses based on our knowledge of muscle activation patterns during standing reactive perturbations. However, these data are neither specific nor detailed enough to truly understand the deficits that may exist within the trunk and how balance responses vary depending on the postural condition (ie standing or sitting) (Preuss & Fung, 2007). Although there have been studies investigating the relationship between bimanual limb coordination, trunk control (Tunik et al., 2004) and functional reaching tasks (Poizner et al., 2000; Stack et al., 2005) in patients with IPD. This research does not directly isolate reactive trunk responses, but provides insight, more so, into the degree of stability that is required in the trunk in order to have a stable base from which to execute such movements.

Although no direct measures of EMG activity are available, one could draw from the previous work of Martin (1965) to predict the patterns of muscle activations that may occur during a seated reactive perturbation. Based on the responses made during the perturbation, falling in the direction of the tilt, one could hypothesize that patients with IPD may have responded with a similar muscle activation pattern as seen in response to perturbations while standing. These muscle activation patterns may include co-contraction (symmetrical muscle activation) and or insufficient muscle modulation (Carpenter et al., 2004). These factors, coupled with the decreased ROM and increased resting tonic muscle activity observed in patients with IPD, could have caused the trunk to stiffen. Therefore, this stiffening may have facilitated falling in the direction of the tilt. Such an explanation for the observed responses may more accurately account for the documented postural responses than those proposed by the author. Martin (1965) suggests that the lack of postural response witnessed in PEP patients was due more to a complete lack of labyrinthine function than basal gangliar dysfunction, and as such, tilting reactions would be an efficient means by which to test
pallida integrity. Martin’s (1965) interpretations of his results were based on the fact that PEP patient responses simulated those of patients tested with complete loss of labyrinthine function (Figure 1A). In either case, the fact that an anecdotal hypothesis is required to currently explain trunk activity and its contribution to balance control in patients with IPD highlights the need to further expand our understanding in this field of research.

Limitations of current seated perturbation research in both control and patient populations

Not only is research investigating seated postural responses in patients with IPD limited in the amount of information available, but much of the current literature is limited in their design consistency. Perturbing and isolating the trunk specifically is challenging. To do so requires a machine or technique that is able to perturb a participant with a consistent velocity and amplitude. In attempts to substitute or make due with limited equipment, perturbations have been elicited manually by an experimenter. In the study by Martin (1965) patients sat on a bed while a researcher would hold onto the corner and pull the bed upward or downward. Inconsistencies in force generation could have greatly influenced both the degree and type of postural response elicited by all participants within the study. A similar limitation has also been observed in control research (Zedka et al., 1998).

As mentioned previously, when recruiting clinical populations, especially IPD, distinguishing between disorders that present with similar symptoms as IPD and true IPD is required. During Martin’s study post encephalitic Parkinson’s patients were tested instead of IPD patients. As stated in the clinical section, the issue with generalizing the Parkinsonian symptoms, as caused by an ailment, is that it’s effects are not isolated specifically to the basal ganglia and additional neurological deficits may be present.
1.7 Influences on postural responses

Effects of fear

Besides the aforementioned influences on seated postural control, fear of falling can also greatly impact a patient’s ability to maintain balance. The effect of fear of falling and or anxiety on postural control has not only been documented during seated responses in an IPD patient population (van der Burg et al., 2006), but also during standing (Adkin et al., 2003).

During seated balance tasks, patients with IPD demonstrate an increased fear of falling not only through increases in COP excursions, but decreases in MPF when compared to “non-fearful” patients and controls (van der Burg et al., 2006). If seated balance performance as measured by COP and MPF data are compared to what is currently known with respect to standing balance literature in a healthy population an inconsistent result is seen. During times of higher anxiety (or increased fear of falling) while standing, control participants COP excursions decrease, and their MPF increases creating a quickly gyrating inverted pendulum (Adkin et al., 2000; Carpenter et al., 1999; Carpenter et al., 2001). Similarly, a reduction in COM displacement is observed even when responding to tilt during high threat/anxious situations (Carpenter, Frank et al., 2004). The inconsistent findings between patients and controls may be related to their response characteristic of increased symmetric muscle activation. This would limit their ability to make fast corrective movements, and could result in the larger COP traces seen.

Better understanding postural stability and trunk control as it relates to falls is important not only to IPD patients but to healthy elderly populations as well. During 2001 an estimated 15,000 individuals died due to fall related injuries. The cost of falls alone in the USA reached 20 billion dollars in 2001 and is only expected to rise over the years to come.
(Bloem et al., 2003). The importance of better understanding the contributing factors to falls and developing ways to prevent them is not just important economically for our health care system, but is also important for ensuring and maintaining overall quality of life.

The lack of supporting data, specific to trunk muscles, in a reactive paradigm in an IPD patient population and its contributing role in balance control and fall prevalence is clear. In an attempt to further this field of research and replicate or refute previous findings, such as those by Martin (1965), the study proposed here aims to investigate trunk muscle responses in an idiopathic Parkinson’s disease population and healthy age matched controls while seated.

1.8 Hypotheses

On the basis of previous literature we hypothesize that during seated tilting perturbations:

(1) Relative muscle response patterning in patients with IPD will be similar to that of controls.

(2) The magnitude of IPD patients postural muscle activity will be modulated incorrectly, and over amplified.

(3) Patients with IPD will have a greater degree of postural instability in the medial-lateral (left, right), and backward directions.

(4) Lastly, IPD patient’s range of motion will be decreased in both sagittal and frontal planes.
2. METHODS

2.1 Subjects

Seven individuals diagnosed with Idopathic Parkinson’s disease (IPD) according to the UK Brain Bank criteria (Hughes et al, 1992) and 10 otherwise healthy age-matched controls, recruited from a local community program, volunteered to participate in this study (Table 1). Disease severity was assessed using the Unified Parkinson’s Disease Rating Scale (UPDRS) and Hoehn and Yahr scale.

| Table 1: Demographic and disease characteristics of controls and patients with IPD |
|-------------------------------------------------|-----------------|------------------|
| The numbers associated with each medication represent the number of patients that were on that medication at the time of testing. Significant differences between groups are represented by (**). Values in parenthesis represent the standard deviations, with the exception of No. women whose value in parenthesis represents the percentage of women that participated in that group. |
|-------------------------------------------------|-----------------|------------------|
| | Patients (n = 7) | Controls (n = 10) |
| Age (years) | 70.4 (7.59) | 68.4 (6.0) |
| No. of women | 3 (42%) | 5 (55%) |
| Height (m) | 1.7 (0.09) | 1.7 (0.09) |
| Weight (lbs) | 172 (44.5) | 149.6 (33.7) |
| Trunk length (cm) | 50.4 (6.0) | 49.35 (4.1) |
| Duration of disease (years) | 8.0 (3.2) | - |
| No. of falls in the past 6 months | 4.7 (9.6) | 0.1 (0.3) |
| Fear of falling (%) | 22.1 (23.8) | 7.8 (11.7) |
| UPDRS score | 35 (9.0) | - |
| Hoehn and Yahr stage | 2.5 (0.41) | - |
| Medication | | |
| Sinemat | 4 (57%) | - |
| Sinemat CR | 3 (43%) | - |
| Mirapex | 3 (43%) | - |
| Synthroid | 1 (14%) | - |
| Hydrazine | 1 (14%) | - |
| Propranolol | 1 (14%) | - |
| Comtan | 1 (14%) | - |

Inclusion criteria for both control and patient groups were the following: 1) being able to stand and sit independently without external support, and 2) be free from any additional confounding neurological impairments such as vestibular imbalance or orthopedic pathologies of the hip or spine. Furthermore, patients with IPD had to have been classed
between 1.5 – 3.0 on the Hoehn and Yahr scale. IPD participants were asked to take their normal anti-Parkinson medications one hour prior to arriving for their testing session to ensure an optimal “ON medication” state was achieved during the testing session. All procedures were approved by the University of British Columbia ethics review board and participants provided their informed consent prior to participating in the study.

Prior to the experiment, participants’ fear of falling and fall histories were assessed using a series of verbal questions including: “1) Have you ever fallen? 2) If yes, can you recall how many times you have fallen in the past 6 months?; and 3) Rate your fear of falling on an incremental scale from zero to one hundred, where zero is not fearful and one hundred is very fearful.” In addition, we recorded participants’ height, weight, age and trunk length measured from the first thoracic vertebra (T1) to the top of the sacrum (Table 1).

2.2 Active and passive ranges of motion (ROM)

2.2.1 Setup

Participants were positioned atop a customized swivel table with foam support on either their back (to test frontal plane ROM) or on their right side (to test sagittal plane ROM). Their hips were positioned at the most superior edge of the fixed portion of the table while their trunk was positioned onto a trunk support table (21.5” x 16”). As shown in Figure 2, the trunk support board moved freely around a base table (61.5"x 49") covered with nylon balls, 1/2" in diameter, to create a nearly frictionless surface (Parkinson, 2004). The trunk support board was positioned so that its inferior edge was aligned with the eleventh thoracic vertebra (T11), ensuring similar placement across participants. Stabilizing straps were placed across participants’ hips, thighs and chest. A rigid body was mounted onto a
wooden dowel at the upper right hand corner of the trunk support (Figure 2), and an Optotrak Certus motion sensor (Northern Digital Inc, Waterloo) was positioned behind the swivel table. The trunk support could translate freely (for active ROM) or be pulled by a cable attached at one end to the upper right or left hand corner of the trunk support for passive ROM. This cable was attached to a weighted pulley system that could be lowered manually.

2.2.2 Procedure

Once positioned on the swivel table, participants completed all of the required ROM tests in one plane (frontal; left and right lateral trunk flexion) before continuing in the second plane (sagittal; on their right side; for trunk flexion and extension). Measures of active and passive ROM as well as corresponding measures of relative trunk stiffness were performed in each test direction. Movement direction and task (active/passive) were counterbalanced across subjects. Active ROM tests were performed 3 times in each direction and required participants to voluntarily bend their trunk to their perceived end ROM. To help relax
participants prior to the initiation of each trial, they were instructed to inhale and slowly release their breath (Mak et al., 2007). During passive ROM testing, participants were additionally instructed not to aid or resist the movement. To calculate passive ROM and relative trunk stiffness, incremental weights, applied from lightest to heaviest (2.5lbs, 5lbs, 7.5lbs, 10lbs, 17.5lbs) were secured to the pulley system, and slowly lowered manually by the researcher. A baseline pull angle, relative to the trunk support board and cable, of 45° was used while pulling participants in the frontal plane during left and right lateral trunk flexion and sagittal backward extension. During passive sagittal forward trunk flexion tasks, a baseline pull angle of 25° was used (Figure 3).

Figure 3: Passive range of motion apparatus
(A) Depiction of experimental setup during passive left frontal plane ROM.
(B) Depiction of experimental setup during passive backward extension ROM in the sagittal plane. Similar to active ROM testing, support straps were placed over the participants’ chest, hips, and thighs. The white arrow in each image indicates the cable that was attached to pulley system and trunk support and has been outlined in white as well. In both (A) and (B) the cable is attached to the upper left hand corner of the trunk support. For both right frontal plane and forward sagittal plane flexion the cable was attached to the upper right hand corner of the trunk support. The red arrows point to the weights which were attached to the pulley system and lowered by a researcher.
Baseline angles of pull were chosen based on pilot testing and were the most accurate representation of participants’ active path of motion. The first four weights were lowered to a point where they were able to hang freely (if the 5th weight was too heavy, it was slightly supported by the researcher) and the position was held for three seconds. The weight was then lifted slowly upward, removed and the subsequent weight added. If the participant reached their end ROM during implementation of one of the lower weights, as confirmed through their verbal report, subsequent weights were not added. The greatest weight combination was used to determine the participants end ROM. If end ROM was not achieved with the maximum weight (17.5lbs) a manual pull trial was performed.

2.2.3 Measures and data analysis

Peak active and passive ROM (deg) values were determined from the positional data collected from the mounted rigid body placed on the dowel on the trunk support. The rigid body consisted of four infra-red light emitting diodes that were recorded by an Optotrak Certus motion sensor with a sampling frequency of 50Hz. Before each task, a resting 3 sec control trial was collected with the participant lying as straight and relaxed as possible. The maximum active and passive ROM was calculated by subtracting the resting mean from the maximum angular displacement recorded for each trial, and then averaged across trials for each subject. Incremented passive stiffness values were measured as the slope of the force (N) versus displacement (deg) curve (Panjabi, 2003) as determined from the last three maximum weights applied during passive ROM.
2.2.4 Statistical analysis

Non-parametric statistical analysis was used to account for small and uneven sample sizes and non-normally distributed data. Maximum ROM and stiffness values were ranked then analyzed using a 2x2 (group x direction) analysis of variance (ANOVA) for frontal and sagittal planes independently with a p-value of 0.05. Significant trends were considered for 0.05<p<0.10. Post-hoc comparisons were performed using the Mann-Whitney U test for interaction effects. Multiple comparisons were corrected using the Bonferroni method with an adjusted p-value of 0.025 for significant effects and 0.025<p<0.05 for trends toward significant.

2.3 Tilting perturbations

2.3.1 Setup

Participants were seated in a modified chair mounted onto a wooden support (79 cm long and 61 cm wide) that was fixed to a custom made multidirectional tilting platform (University of Basel, Switzerland). The difference in chair height between the front and back edge was not adjusted from its original position (back edge of chair 20mm higher than front), in order to keep a more natural sitting posture (Forssberg & Hirschfeld, 1994). Participants’ legs were positioned against a wooden support (43 cm long), with knees flexed at 50° (Figure 4).
To ensure distal segments such as the pelvis and lower limbs did not influence postural responses in the trunk, two straps were used to stabilize these segments to the seat and leg support respectively. One strap was placed over the upper thigh while another was placed across the anterior superior iliac spine (ASIS). The ASIS strap was secured so that it pulled the pelvis into the posterior support of the chair to limit, if not completely remove, pelvis rotation since it appears data are inconsistent as to the effect of pelvic motion on triggering postural responses (Hirschfeld and Forssberg, 1994). An additional strap was placed across participants ankles to stop any lifting of their feet from the support. A visual target was positioned 2.5 m away from participants at eye level and was used as a visual reference point throughout the experiment. Furthermore, foam padding was placed along the edge of the wooden support, inside the posterior edge of the chair, and under each strap to ensure for participant comfort.
2.3.2 Measures

Electromyographic (EMG) activity was collected from 10 different muscles using the Telemyo 2400R Telemetry system (Noraxon Inc., USA). Two self adhesive disposable Ag/AgCl surface electrodes were placed on each muscle belly 3cm apart from one another. EMG recordings were attained bilaterally from: rectus abdominis (RA) (3cm lateral of the umbilicus), external oblique (EO) (15cm lateral of umbilicus), erector spinae (ES) (at the thoracic level of T9, ES\textsubscript{T9}, 3cm lateral of the spinous process): at the lumbar level of L3 (3cm lateral to the spinous process, ES\textsubscript{L3}). EMG was also recorded unilaterally from the right sternocleidomastoid (SCM), and right medial deltoid (MD). Muscle landmarks were adapted from (Preuss & Fung, 2007). Raw EMG signals were pre-amplified 500X, band pass filtered online between 10-500Hz and sampled at 1500Hz. These data were then A/D converted (Power 1401, CED, UK) and sampled at 1000Hz, before being digitally filtered (band-pass filter between 40-300Hz) and rectified offline (Spike2, Cambridge Electronic Design, UK). Filtering frequency was based on the optimal recommended frequencies for removing heart rate artefact from axial muscle EMG (Drake & Callaghan, 2006; Redfern et al., 1993; Zhou et al., 2007).

Kinematic data were collected from four rigid bodies, each defined by three infra-red markers, placed on the posterior aspect at the thoracic level of T3, at the lower lumbar level just above the L3 EMG electrodes, on a head mount and on the posterior edge of the tilting platform. Three dimensional position data from each rigid body was recorded with Optotрак Certus motion sensors with a sampling rate of 100Hz and was used to calculate angular displacements in the sagittal and frontal planes (Figure 4).
2.3.3 Procedure

Prior to any tilting perturbations, participants were asked to sit in a comfortable posture with arms crossed, looking straight ahead at a target that was positioned at eye level on a white board. During this time their mean trunk position, in the anterior-posterior and medio-lateral directions (X,Y coordinates), relative to the T3 rigid body marker, were recorded and established as participants’ “home position”. All participants were required to start each tilting perturbation from their “home position” (± 5mm) which allowed us to control for perturbation anticipation or any postural drift that may have occurred during testing. Starting from their “home position” with their arms across their chest, elbows away from their body and eyes open, participants were perturbed in one of four directions: forward (legs down): backward (legs up): left: or right. Perturbation order was presented randomly to each participant. Rotation velocity (40°/sec) and amplitude of rotation (7°) were held constant across all trials. These perturbation parameters are similar to those used in previous seated and standing perturbation studies (Carpenter, Frank et al., 2004; Forssberg & Hirschfeld, 1994; Hirschfeld & Forssberg, 1994; Zedka et al., 1998). Perturbation velocity was determined from pilot testing and was set at a velocity that elicited consistent postural responses. Faster speeds did not appear to be as destabilizing, or as consistent at generating balance correcting responses. Following each rotational perturbation, the tilting platform returned to its original starting position (parallel to the floor).

To avoid fatigue, participants were instructed to relax their arms between trials. The direction of platform perturbation was changed between each trial by shifting the axis of rotation relative to the seated participant. To ensure participants remained unaware of perturbation direction, they were asked to close their eyes between trials and covers were
used to hide the position of the axis of rotation relative to the participant. Participants were not provided with any warning cue prior to the actual perturbation and a random period of 10sec to 1min was used between trials. Participants were perturbed an average of seven times in each direction.

2.3.4 Data analysis

The first trial for each participant was removed from the data analysis to account for any possible first trial affects and one control participant was removed from data analysis due to previous exposure to experimental protocol. Also, one IPD patients EMG data was removed from the analysis based on past medical history.

Kinematic dependent measures were determined from the angular displacement of the T3 rigid body and were based on the participant’s corrective trunk movement in response to the platform perturbation. A corrective trunk movement was considered to be trunk motion that was opposite to the direction of the tilting perturbation. Onset times for both trunk and platform displacements were determined as the time when the angular displacement exceeded the mean background position by ±2 standard deviations (SD) and remained above this level for 200 and 100ms respectively. Corrective trunk onset latency was calculated as the difference between the onset time of the platform and onset time of the corrective trunk movement. The absolute average angular difference in position was calculated as the net change from their initial starting position to peak angular corrective movement. Time to peak was calculated as the onset of the corrective trunk movement to peak movement.

Area and muscle onset latencies were calculated for muscles on the left side, but did not included analysis of MD or SCM. Muscle and platform onsets were determined for each
trial using an algorithm, then approved and/or manually corrected after visual inspection for accuracy (Allum et al., 2002; Carpenter, Frank et al., 2004). Any trials that had an undetectable muscle onset time due to remaining artefact were not further analyzed. Muscle and platform onsets had to be maintained above the calculated 300ms background activity ±2 (SD) for 10 and 20ms respectfully. To capture the earliest possible reflexive abdominal muscle onset time (Beith & Harrison, 2004) and the latest possible postural balance correcting activity (Allum et al., 1996; Carpenter et al., 1999), muscle onsets were calculated between 10 and 500ms. A muscle was considered to have a detectable average onset if 40% of the trials within a direction had onsets recorded within the set criteria.

Rectified EMG area for each muscle was calculated from the background muscle activity (BGA) recorded during the 100ms interval prior to the onset of the platform perturbation. Likewise, rectified EMG area was calculated 100ms after each muscle onset and referenced to the level of BGA. To calculate the average area of an EMG response for a given muscle, all trials with a detectable EMG onset, as per the defined criteria, were included. If no detectable onset occurred, BGA was used as the data point for that given trial (Carpenter et al., 2008).

Area data were normalized to each participant’s maximum voluntary contraction (MVC) for each recorded muscle. MVC data were collected upon completion of the testing protocol (Carpenter et al., 2005), while participants remained seated on the tilting platform. Participants were then encouraged to pull or push against manual resistance, applied by an experimenter, and maintain the maximal contraction for 3sec. To maximally activate ES₉₉, ES₃₃, EO and RA participants pushed backwards into extension; pulled their shoulders across their bodies; and pulled forward into a crunch, respectively. Lastly, SCM MVCs were
calculated by having participants put their own hands on their cheeks and push maximally into them.

2.3.5 Statistical analysis

Non-parametric statistical analysis was used to account for small and uneven sample sizes and non-normally distributed data. All kinematic variables were analyzed in a 2x2 (group x direction) ANOVA within each plane (frontal and sagittal) independently with a p-value of 0.05. Muscle onset latencies were ranked and compared in a 2x4 (group x muscle) ANOVA within each perturbation direction with a p-value of 0.05. EMG area calculated from both normalized and non-normalized EMG, was ranked and compared in a 2x2 (group x direction) ANOVA within each plane (frontal and sagittal) independently with a p-value of 0.05. Significant trends were considered when 0.05<p<0.10. Post-hoc comparisons were performed using the Mann-Whitney U test for interaction and group main effects and the Wilcoxon signed-rank test for significant muscle effects. Multiple comparisons were corrected using the Bonferroni method with adjusted p-values of 0.008 and 0.025 respectively. Statistically significant trends were considered as 0.008<p<0.016 and 0.025<p<0.05 respectively.
3. RESULTS

3.1 ROM kinematics

3.1.1 Sagittal plane trunk flexion and extension

No significant differences were observed between patients with IPD and healthy age matched controls in either active or passive ROM in the sagittal plane. However, maximal active ROM was significantly influenced by direction ($F_{(1,15)} = 54.334, \ p < 0.001$), with a larger ROM observed in flexion compared to extension (Figure 5A). Similarly, passive ROM was significantly influenced by direction ($F_{(1,15)} = 75.763, \ p < 0.001$) with a greater maximal ROM occurring during flexion compared to extension. Trunk stiffness calculations were not found to be significantly different between groups, however there was a main effect of direction ($F_{(1,13)} = 25.601, \ p = 0.000$) whereby a greater degree of stiffness was present in extension compared to flexion (Figure 5C).
Figure 5: Group averages of active and passive ranges of motion, and stiffness graphs

Group averages during active (A) and passive (B) sagittal plane forward flexion and extension. Note how both groups are able to reach similar peak ranges of motion. Trunk stiffness during sagittal ROM was greatest during backward extension compared to forward for both participant groups (C). Active (D) and passive (E) frontal plane left and right side flexion was decreased in patients with IPD. Trunk stiffness during frontal plane was similar between groups and directions (F). Significant differences ($p < 0.05$) are indicated by (*).
3.1.2 Frontal plane trunk flexion (left and right)

Patients with IPD displayed a significant reduction in maximal active ROM positions compared to controls (Figure 5D), independent of direction in the frontal plane ($F_{(1,15)} = 12.774, p = 0.003$). Likewise, maximum passive ROM was also significantly decreased for patients with IPD compared to controls (Figure 5E) independent of direction ($F_{(1,15)} = 6.379, p = 0.023$). There was a trend towards a significant effect of direction on stiffness ($F_{(1,15)} = 3.553, p = 0.079$). A greater degree of trunk stiffness tended to be seen when moving to the left compared to right (Figure 5F).

3.2 Tilting perturbations

3.2.1 Sagittal plane perturbations

Kinematics

The onset latencies of balance correcting movements were significantly dependent upon perturbation direction ($F_{(1,14)} = 13.029, p = 0.003$), with earlier corrective movements observed during backward compared to forward perturbations, $0.084 \pm 0.005$ and $0.097 \pm 0.009$ sec, respectively. Similarly, time to peak movements were significantly dependent upon direction ($F_{(1,14)} = 5.266, p = 0.038$) and were earlier during backward compared to forward perturbations, $0.198 \pm 0.009$ and $0.235 \pm 0.016$ sec, respectively. There were no significant differences in onset latencies, time to peak movements or absolute angular displacements between control and patients with IPD when perturbed during forward directions (Figure 6).
Figure 6: Average group traces of corrective trunk movements from the T3 rigid body

Group averages of trunk corrective movement from the T3 rigid body are represented by the solid lines while the dashed lines, in corresponding colour, represent the positive and negative standard error (STDE). Perturbation direction is listed with each figure, such that forward indicates a forward (Legs-down) perturbation and right a rightward perturbation and so on. Notice the corrective trunk movements opposite the direction of the tilt for each direction. As can be seen both groups make similar corrective trunk movements opposite to the direction of the tilting platform. The dashed line represents the onset of the platform.
Patterns of EMG activation

As can be seen in Figure 7, the onset latencies of muscle responses during forward (legs-down) tilts were dependent upon muscle ($F_{(3,24)} = 23.266, p < 0.001$). Post-hoc analyses revealed that earlier onsets occurred in RA compared to ES_L3 ($p = 0.007$) and EO compared to both ES_T9 and ES_L3, $p = 0.001$ and $p = 0.001$, respectively. A significant muscle by group interaction was observed ($F_{(3,18)} = 3.762, p = 0.029$) for backward tilts. Although, post-hoc comparisons revealed no significant differences there was a visual difference between onset latencies in EO and ES_T9 for control participants. However, similar onset times were observed between these muscles in patients with IPD ($0.176 \pm 0.020; 0.178 \pm 0.022$ sec).
Figure 7: Patterns of muscle activation during forward and rightward tilts

Patterning of muscle response activity to forward and rightward tilting perturbations. Each trace represents a single trial for a control (A) and a patient with IPD (B). The dashed line represents the onset of platform movement, while the red line represents the earliest muscle onset for that pattern of muscle activity. Notice the similarities between the two groups for the forward tilts, where EO is earlier than EST9 and RA is earlier than ESL3. Likewise, a similar pattern of response is observed between groups during rightward tilts where EO is earlier than EST9.
Normalized EMG Amplitudes

There was a significant interaction between group and direction for the EST9 muscle \( (F_{(1,13)} = 6.322, p = 0.026) \) for normalized EMG area. Post-hoc tests revealed a trend toward significance for increased EMG area in patients with IPD compared to controls in the backward direction \( (p = 0.045) \) (Figure 8); however, no differences between groups were observed in the forward direction (Table 2). A trend toward a significant main effect of direction for ESL3 \( (F_{(1,13)} = 3.535, p = 0.083) \) was also observed with a greater degree of activity seen during forward compared to backward tilting perturbations. No significant differences were found for either RA or EO muscles.
Table 2: Normalized MVC % and raw group 100 ms area averages for rectus abdominis (RA), external oblique (EO), erector spinae (ES\textsubscript{T9}, ES\textsubscript{L3}). Analysis of both normalized and raw 100 ms area showed similar statistically significant patterns and trends. On average a general trend of increased muscle response magnitudes can be seen for patients with IPD compared to controls. The highlighted boxes and (#) signify the trend toward significantly larger ES\textsubscript{T9} for patients with IPD in comparison to controls.

<table>
<thead>
<tr>
<th></th>
<th>Forward</th>
<th>Backward</th>
<th>Left</th>
<th>Right</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Control</td>
<td>PD</td>
<td>Control</td>
<td>PD</td>
</tr>
<tr>
<td>RA</td>
<td>Normalized MVC %</td>
<td>8.99 ± 2.55</td>
<td>14.60 ± 7.84</td>
<td>12.99 ± 3.94</td>
</tr>
<tr>
<td></td>
<td>Raw µV</td>
<td>0.406 ± 0.127</td>
<td>0.373 ± 0.109</td>
<td>0.518 ± 0.194</td>
</tr>
<tr>
<td>EO</td>
<td>Normalized MVC %</td>
<td>19.37 ± 5.09</td>
<td>33.28 ± 15.05</td>
<td>15.56 ± 3.30</td>
</tr>
<tr>
<td></td>
<td>Raw µV</td>
<td>1.42 ± 0.313</td>
<td>2.09 ± 0.712</td>
<td>1.32 ± 0.373</td>
</tr>
<tr>
<td>ES\textsubscript{T9}</td>
<td>Normalized MVC %</td>
<td>27.22 ± 6.48</td>
<td>39.55 ± 9.47</td>
<td>10.31 ± 2.23</td>
</tr>
<tr>
<td></td>
<td>Raw µV</td>
<td>1.97 ± 0.375</td>
<td>2.34 ± 0.567</td>
<td>0.804 ± 0.146</td>
</tr>
<tr>
<td>ES\textsubscript{L3}</td>
<td>Normalized MVC %</td>
<td>11.42 ± 4.40</td>
<td>22.55 ± 7.68</td>
<td>6.46 ± 1.43</td>
</tr>
<tr>
<td></td>
<td>Raw µV</td>
<td>0.544 ± 0.131</td>
<td>1.20 ± 0.391</td>
<td>0.394 ± 0.0947</td>
</tr>
</tbody>
</table>
**EMG normalized background activity (BGA)**

Significantly greater levels of BGA were found in IPD subjects compared to controls for both ES$_T$9 ($F_{(1,13)} = 5.749, p = 0.032$) and ES$_L$3 ($F_{(1,13)} = 6.296, p = 0.026$) muscles (Table 2). No significant differences were observed for RA or EO.

*Figure 8: Normalized ES$_T$9 MVC %, 100ms area data during forward and backward tilting perturbations*

Group averages for normalized ES$_T$9 100 ms area are represented with their associated standard error. Note the trend toward significantly greater ES$_T$9 activity for patients with IPD when being perturbed backward. Trend toward significant differences ($p < 0.05$) are indicated by (#).
3.2.2 Frontal Plane Perturbations

Kinematics

Onset latencies of balance correcting responses were significantly dependent upon group ($F_{(1,14)} = 6.339$, $p = 0.025$) and direction ($F_{(1,14)} = 8.299$, $p = 0.012$). Trunk displacements in control participants had onset latencies that were significantly earlier than those in patients with IPD ($0.062 \pm 0.016$, $0.111 \pm 0.018$ sec). Furthermore, both control and patient groups had earlier responses when tilted to the right then to the left ($0.083 \pm 0.009$, $0.090 \pm 0.018$ sec). No differences were seen in the time to peak movements or in the absolute angular displacements between groups or directions (Figure 6).

Patterns of EMG activation

The onset latencies of muscle responses to leftward perturbations were dependent upon muscle ($F_{(3,6)} = 4.810$, $p = 0.049$) whereby earlier onset times were observed in EO compared to RA ($p = 0.008$). Similarly, the onset latencies of muscle responses during rightward perturbations were also dependent upon muscle ($F_{(3,21)} = 8.036$, $p = 0.001$). Post-hoc tests revealed that EO muscle activity onsets were significantly earlier compared to both $ES_{T9}$ ($p = 0.002$) and $ES_{L3}$ ($p = 0.003$) (Figure 7).

Normalized EMG Amplitudes

Analysis of the integrated EMG area revealed a group by direction interaction for the $ES_{T9}$ muscle ($F_{(1,13)} = 6.119$, $p = 0.028$). Post-hoc analysis showed a trend towards significant increases in $ES_{T9}$ activity going to the left ($p = 0.059$) for patients with IPD compared to controls. EMG area for $ES_{L3}$ was dependent upon direction ($F_{(1,13)} = 54.655$, $p <$
0.001) with larger areas observed during perturbations to the right compared to the left. A trend for a group main effect was also found for ESL3 (F(1,13) = 3.247, \(p = 0.095\)), with larger ESL3 muscle activity observed in patients with IPD compared to controls. No statistically significant differences were seen for RA or EO.

*EMG normalized background activity*

A trend towards a significant group main effect of EST9 BGA was observed (F(1,13) = 4.415, \(p = 0.056\)) with IPD patients tending to have greater levels of BGA compared to controls. BGA for EST9 was dependent upon direction (F(1,13) = 5.926, \(p = 0.030\)) with greater activity present prior to perturbations to the right compared to the left. ESL3 activity was significantly dependent upon group (F(1,13) = 4.702, \(p = 0.049\)). Patients with IPD presented with a greater amount of ESL3 BGA compared to controls. No significant differences were detected in either RA or EO.

Analysis of raw EMG data for both response perturbations and background activities, were similar in terms of significance and trends to those observed for normalized EMG measurements. A detailed description of the statistical results based on the raw EMG data can be found in Appendix A and Table 2.

### 3.3 Case studies

As shown in Figure 9, two patients with IPD responded with trunk corrections in the direction of the tilt when tilted to the left and right. For each tilting perturbation to the left, Subject 17 did not make a corrective trunk movement consistent with what would be expected to correct for the induced postural instability (Figure 9A). While Subject 12’s
initial five trials were accurate and corrective in nature to the direction of the tilt, subsequent trials consisted of inaccurate movements that occurred in the direction of the tilt. During the initial rightward tilting perturbation trials, Subject 17 appeared to respond with a corrective movement to the tilting perturbation, however then began making incorrect corrective movements with increasing trial number and started to respond in the direction of tilt. Subject 12 seemed to have increased difficulty making corrective movements to rightward versus leftward tilts (Figure 9B). An inconsistent patterning of corrective movements was also seen across trials during rightward tilts for Subject 12, but compared to tilts to the left they had a greater number of incorrect movements to the right. It should be noted that of the seven patients with IPD analyzed in this study, Subject 17 and 12 were the two that were the most affected by IPD and both were initially unilaterally affected on the left hand side of their body. Subject 17’s UPDRS and Hoehn and Yahr scores were 44/105 and 3/5, respectively, while subject 12’s UPDRS and Hoehn and Yahr scores were 45/105 and 2.5/5, respectively. As can be seen in Figure 10, IPD patients’ inability to make corrective trunk movements may be due to increased muscle co-contraction and increased muscle activity from balance correcting muscles.
Figure 9: Case studies: Tilting kinematics from T3 rigid body of incorrect corrective trunk movements

Individual trials for frontal plane perturbations of two patients with IPD who made incorrect trunk movements to the tilting perturbations. Each trial for the left and right perturbation directions is graphed against the averaged control corrective movement, with Subject 17 graphed in (A) and Subject 12 graphed in (B). Subject 17 was most prone to incorrect trunk movements when perturbed to the left, as can be seen in (A) left tilt, their trunk would go in the direction of the tilt during each trial. Subject 12 (B) does not appear to be as prone to incorrect trunk movements in one direction; however they seem to become maladapted at making corrective trunk movements over time more so when perturbed to the right. Notice the correct movements the control participants make (solid purple line) in response to these perturbations. The corresponding movement of the platform is depicted on the bottom for both left and right perturbations.
Figure 10: Case study: Muscle response characteristics of an incorrect and correct trunk response to a left tilt
Normalized single trial muscle responses to peak muscle activity in volts for Subject 17 with IPD (A) and a healthy control (B). The dashed line represents the onset of the platform and a schematic of the platform movement is shown in the bottom trace. The light grey images represent the right side of a muscle i.e. right RA, while the dark lines represent the left side of the muscle i.e. left RA. Notice the increased degree of background activity, co-contraction and larger muscle response magnitudes from the patient with IPD (A). While the response by the control participant follows the correct muscle pattern, EO earlier than RA and is balanced by overall larger muscle responses from right sided muscles compared to the left [rectus abdominis (RA), external oblique (EO), erector spinae at T9 (ES\textsubscript{T9}) and erector spinae at L3 (ES\textsubscript{L3})].
4. DISCUSSION

This study was inspired by the work of JP Martin (1965). Martin observed that patients with post-encephalitatic Parkinson’s disease would fall in the direction of a tilting perturbation when seated, making little to no corrective trunk movement. He reasoned these deteriorated responses arose from a diminished or altered vestibular system as vestibular loss patients responded to the identical tilting perturbations in a similar fashion. In order to better understand the underlying causes of these observed trunk deficits and attempt to replicate the findings by Martin (1965), this study investigated postural corrective movement of the trunk during controlled support-surface rotations while seated in patients with idiopathic Parkinson’s disease and age-matched healthy controls.

4.1 Lateral range of motion (ROM) of the trunk was influenced by IPD

No difference was found between patients with IPD and healthy controls maximum ROM during both active and passive trunk forward flexion and extension. In contrast, we observed patients with IPD to have a significantly decreased ROM compared to controls during active and passive trunk lateral flexion in the frontal plane (Figure 5). Our findings are not consistent with previous research comparing ROM in patients with IPD and healthy controls. Bridgewater and Sharpe (1998) found patients with IPD to have a significantly decreased active ROM compared to controls during trunk forward flexion and extension movements, as well as trunk lateral flexion. Furthermore, all participants in our study had greater active ROM in all directions compared to those reported by Bridgewater and Sharpe (1998). Differences in the observed maximum active ROM achieved by participants between these two studies may be attributed to the experimental apparatus used. In the study by
Bridgewater and Sharpe (1998), ROM was tested while patients were standing and strapped to a dynamometer. Although this standing paradigm is commonly used (Mak et al., 2007; Wright et al., 2007), it is limited in that participants must maintain some degree of postural stability due to the effects of gravity. This will inherently increase the amount of tonic background activity (Wright et al., 2007) which can then limit the degree of ROM achieved by participants. Although we acknowledge that this condition may increase the degree of external validity associated with the standing paradigm as most postural corrections are executed from this position, our paradigm enables the degree of passive stiffness relating to tissues other than muscles to be observed, as they do play a role in the maximum end range of motion that is achieved (Brown & McGill, 2008).

In addition to decreased active ROM in trunk lateral flexion, we also observed that patients with IPD had a significantly decreased ROM during passive trunk lateral flexion compared to controls. To our knowledge this is the only study that has tested passive ROM in patients with IPD in any direction of trunk movement, either in a standing or supine position. Our data suggest, that additional causes not related to muscle tone, such as changes in visco-elastic properties (Lee, 1989), or morphologic changes in trunk muscles (Schabitz et al., 2003) may be influencing the degree of ROM in patients with IPD. This finding in part, explains and supports why patients with IPD have increased instability during frontal plane perturbations while standing, since the relative biomechanical responses require greater flexion from the trunk and hips, which if limited would decrease patients postural stability (Carpenter et al., 2004; Gruneberg et al., 2004; Horak et al., 2005; Vaugoyeau et al., 2007).
Spinal stiffness and stability has been described by Panjabi (2003) as the degree of trunk movement with increasing loads, where limited movement under higher loads represents a more stable spine. These spinal column characteristics can be observed in our data for both our control and IPD patient groups in Figure 5. Despite observed differences in active and passive ROM during lateral trunk flexion between patients with IPD and controls, there was no significant difference in spinal stiffness between the groups in any plane of movement.

4.2 Timing and amplitude of corrective trunk movements in the sagittal plane are not influenced by IPD

In response to titling perturbations, patients with IPD were able to make corrective seated trunk movements with appropriate timing and amplitude regardless of the perturbation direction in the sagittal plane.

Compared to the current findings, Forssberg and Hirschfeld (1994) observed earlier and smaller corrective trunk movements in young healthy controls during both forward and backward perturbations. These discrepancies in response characteristics may be due to differences in participant age or the experimental apparatus used between studies. For instance, a large age range separated participants in the two studies. Our study recruited participants between the ages of 61 to 80, while Forssberg and Hirschfeld (1994) recruited participants between the ages of 18 to 32. Therefore, the observed differences between the two studies may provide support for potential age-related effects on postural control of the trunk, such as; reduced elasticity of articulating tissues, loss of disc cartilage and arthritic changes (Buckwalter et al., 1985), or changes in muscle response characteristics such as
timing and amplitude. Unfortunately EMG comparisons cannot be made with Forssberg and Hirschfeld as normalized EMG data is not available for comparison. Variations in the participant’s seated postural orientation may have also influenced the extent to which they were able to make corrective trunk movements. Forssberg and Hirschfeld’s experimental procedures required participants to sit with their legs fully extended and allowed the pelvis to move freely. In contrast, our participants sat with their knees bent and pelvis restricted. Following pilot testing in a similar seated position as Forssberg and Hirschfeld, we found participants had great difficulty maintaining the seated posture and experienced some discomfort in their hamstrings. Since these muscles cross both the knee and pelvis, sitting with an outstretched leg may increase their tension, which could be further exacerbated in less flexible participants. Movements of the trunk and pelvis in the Forssberg and Hirschfeld study could have increased tension in the hamstrings and restricted movement. These issues provide further support for the body positioning used in our study.

4.3 Timing, but not amplitude of corrective trunk movements in the frontal plane are influenced by IPD

Although no differences in corrective trunk movements were observed during sagittal plane perturbations, delayed onset times of corrective trunk movements were observed for patients with IPD during frontal plane perturbations. Delayed corrective movements may be related to increased rigidity found in patients with IPD in this plane of motion; however, if this were fully the case differences in trunk displacement would also be expected, in addition to delayed onsets, between the two groups. However no differences in the amplitudes of trunk corrective movements were observed in the frontal plane. In all, these results support
that patients with IPD were able to make corrective trunk movements, despite initial delays in trunk corrective movements in the frontal plane.

It is difficult to say with certainty whether the characteristics of trunk movements made by our participants in seated frontal plane perturbations are consistent with what would be expected as literature in this area is limited. However, based on the subjective observations from cinematic data from one study we may be able to draw some conclusions. Martin (1965) observed that healthy older adults were capable of making corrective trunk movements opposite to the direction of a seated tilting perturbation in both the sagittal and frontal planes. In contrast, patients with post-encephalitic Parkinson’s disease were not able to make or maintain an appropriate corrective trunk response and would fall in the direction of the tilting perturbation. Our data do not support this finding as we observed that patients with IPD were capable of making corrective trunk movements in the frontal plane, which although delayed, were generally in the appropriate direction of the perturbation. The difference in results between the current study, and Martin (1965) may be explained by differences in the perturbation magnitudes used. Martin (1965) used a tilting platform with a much larger range of motion (Figure 1) which therefore may have been more posturally-destabilizing and required a greater corrective trunk movement than the tilting perturbations used in the current study. Also, the perturbations used by Martin (1965) were elicited manually by a researcher, putting into question the consistency of perturbation velocity and magnitude. In comparison, our study maintained a constant tilting velocity, with a set perturbation magnitude. These criteria not only ensured consistency across trials, but enabled us to decipher when participants made responses to the perturbation and deceleration components respectively. This distinction could not be made in the study by Martin (1965),
and could be important as recent research has observed that deceleration components of a perturbation can influence postural responses (Carpenter et al., 2005). Also, visual condition may have affected how patients in the study by Martin (1965) responded as perturbations were elicited while participants had their eyes closed, which for patients with IPD, has been found to be a dominant system of reliance for making postural corrections (De Nunzio et al., 2007; Vaugoyeau et al., 2007). In contrast, patients in our study performed the experiment with their eyes open.

Alternatively, observations by Martin (1965) may have been confounded by co-morbidities due to the encephalitis his patients experienced. For instance some of his patients had decreased proprioceptive awareness and could not maintain upper limb positions with their eyes closed. Furthermore, confirmation of properly functioning vestibular systems was only conducted on 20% of his testing group. These altered sensory inputs could have influenced the responses observed by Martin (1965), while our patients did not present with proprioceptive loss as found during UPDRS testing, nor vestibular imbalances as reported verbally or through testing.

### 4.4 Evidence for lateral trunk instability was observed in most severely affected patients with IPD

Although average results indicated that IPD patients were able to make corrective trunk movements to a tilting perturbation, two patients with IPD in the current study made incorrect trunk movements in the direction of the tilting platform when perturbed in the frontal plane. Despite their best efforts, these two IPD patients made either a corrective trunk movement to the tilting perturbation, then switched to respond with an incorrect trunk
movement in subsequent trials; or initially made incorrect trunk movements in every trial. Interestingly, previous literature has found that patients with IPD have difficulty adapting postural responses to changing postural perturbation scenarios (Dimitrova et al., 2004a; Dimitrova et al., 2004b), however, reverting to incorrect following postural responses has not been reported. These altered trunk responses may be linked to disease severity, which has been shown to correlate with decreased postural stability in patients with IPD (Adkin et al., 2003; Jankovic, 2008; Stack & Ashburn, 2006). Both patients who demonstrated incorrect trunk corrections were the most affected by IPD, as evidenced by their UPDRS score (45 and 44 respectively) and Hoehn and Yahr scale (2.5 and 3 respectively).

4.5 Patients with IPD have altered patterns of muscle responses to backward perturbations

Our recorded EMG activity allowed us to examine the underlying response characteristics that enabled patients with IPD to make corrective trunk movements to external perturbations. In both groups, during responses to forward tilting perturbations earlier muscle onsets were observed in muscles that were ipsilateral to the direction of the tilting perturbation, with RA and EO activated earlier than ES T9. Both groups also had similar frontal plane response patterns with earlier onsets in muscles ipsilateral to the perturbation direction. For instance, leftward perturbations had earlier onsets in EO compared to RA. In contrast, during backward perturbations, patients with IPD tended to respond with symmetrical muscle activation compared to healthy older adults with similar onset times of EO and ES T9, however, this did not affect their ability to make appropriate corrective trunk movements. Responding with co-activation creates an increased amount of rigidity in a body
segment such as the trunk and limits an individual’s ability to properly maneuver the centre of mass (CoM) within their base of support. In contrast, response strategies with asymmetrical muscle responses, such as those utilized by our healthy older adults, allow appropriate postural responses to be made away from the ensuing displacement of the external perturbation by moving the CoM and keeping it within the base of support. Earlier muscle onsets in muscles ipsilateral to the tilting perturbation are most likely associated with stretch reflexive activity of the muscle, followed by suppression in the magnitude of their response activity. One may think these earlier muscle onsets would be destabilizing as they would draw the trunk in the direction of the tilt, however, these responses are countered by greater response amplitudes in muscles that are contralateral to the tilting perturbation.

Similar patterning of muscle responses have also been observed in the trunk in previous seated literature. The directional sensitive pattern of trunk muscle onsets in our elderly controls corresponds to what has been observed in young healthy individuals when tilted backward (Forssberg & Hirschfeld, 1994) and when translated in the sagittal and frontal planes (Preuss & Fung, 2007). These data together suggests that directional sensitivity of trunk muscle responses are not influenced by age.

4.6 Larger muscle response magnitudes were observed in patients with IPD when responding to backward and leftward tilting perturbations

Earlier onsets of muscles ipsilateral to the tilting perturbation were countered by directionally sensitive changes in muscle response amplitudes in both patients with IPD and control participants. As hypothesized, patients with IPD had a tendency to respond with larger magnitudes of muscle responses, however this was limited to just backward and
leftward tilting perturbations and specific muscles, namely ES\textsubscript{T9}. Despite this, increased response magnitudes in this muscle did not appear to affect IPD patients’ ability to make corrective trunk movements opposite the direction of the tilt.

Directionally sensitive changes in the magnitude of muscle responses to both sagittal and frontal plane perturbations in our study are consistent with findings from previous seated perturbation studies involving young adults (Forssberg & Hirschfeld, 1994; Preuss & Fung, 2007; Zedka et al., 1998), such that muscles which are opposite or contralateral to the direction of the perturbation respond with greater amplitudes than those ipsilateral to the perturbation. By doing this a balance correcting response drawing the trunk away from direction of instability is achieved.

4.7 Possible mechanisms for muscle co-contraction and improperly modulated muscle responses in patients with IPD

Although the causes of improperly modulated muscle responses and co-contractive responses in patients with IPD are not specifically known, they may be related to altered autogenic or reciprocal inhibition, respectively, or increased muscle background activity. A recent study by Potter et al. (2004) observed changes in spinal autogenic inhibition in the soleus muscle in patients with IPD. These reflex processes feedback Ib afferent information to Ib interneurons, which in turn act back on the homologous muscle. This inappropriately functioning reflex may partially explain the increased amplitude of responses in patients with IPD. If tensile changes within a muscle are not fed back to control its activity, it may be allowed to elicit a greater magnitude of response which normally would be inhibited. Furthermore, Meunier et al. (2000) also observed patients and controls to have similar Ia
reciprocal inhibition in forearm flexors at rest, but upon activation of wrist flexors patients had almost a complete loss of this inhibition on their most affected side. This finding could account for why patients have such a difficulty turning ‘off’ their antagonist muscles during postural responses. However, additional research in each respective area mentioned is needed. In addition, patients with IPD are known to have increased background muscle activity at rest (Carpenter et al., 2004; Horak et al., 1996; Mak et al., 2007; Wright et al., 2007; Zedka et al., 1998). Since a muscle response is dependent on a variety of factors including changes in intention, experience, initial conditions and background motor neuron and muscle activity, (for review on the cortical control of posture see (Jacobs & Horak, 2007), if patients with IPD initially have increased tonic drive to many of their balance correcting muscles, this may cause the motor neuron pools to have greater excitation, lowering threshold levels and enabling greater responses to be generated with less neural command. Our data support this proposed mechanism, as we found increased background activity in ES_T9 and ES_L3, which were the only muscles to show trends toward significantly increased response amplitudes. Despite this, patients were still able to make corrective movements and maintain postural stability.

In essence, due to the interconnections between brainstem nuclei (Chen & Lemon, 2004; Potter et al., 2004) and the basal ganglia, the altered pathways responsible for the changes in muscle inhibition should progressively deteriorate as disease severity increases. If this is the case then it is likely that decreased postural control of the trunk could result from increasing co-contraction and improperly scaled muscle responses, leading to increased symmetry of responding muscles and thus rigidity of the trunk. Our data, though speculative, provide support for this theory, because as mentioned, two of our patients with IPD
responded to tilting perturbations with incorrect movements of their trunk in the direction of the tilt and these patients were most severely affected with IPD. Visual analysis of their ‘worst’ responses showed an increased degree of co-contraction between agonist and antagonist muscles in both timing and amplitude (Figure 10). Since these characteristics were not significantly present for the remainder of our patients, it suggests that these altered response patterns may account for the inappropriate responses of these two patients.

4.8 The role of the trunk in postural stability

Kinematic and EMG data from standing postural responses in patients with IPD allows for interesting comparisons with responses to seated postural perturbations. Patients with IPD are capable of making appropriate standing balance correcting response to forward perturbations in the sagittal plane similar to healthy older adults (Carpenter et al., 2004; Horak et al., 2005). However, patients with IPD have been found to have increased difficulty when responding to standing backward perturbations with slower forward corrective trunk movements (Carpenter et al., 2004) and smaller stability margins (Horak et al., 2005). Similarly, patients with IPD have been found to have decreased postural control when responding to frontal plane perturbations while standing (Dimitrova et al., 2004a) and appear to be particularly unstable with a narrowed stance width due to slower centre of pressure corrections (Horak et al., 2005).

Parallel to these findings, we also found no difference between balance correcting movements between patients with IPD and healthy older adults when perturbed forward. In contrast, we did not observe any prominent deficits in the ability of IPD patients to make appropriate postural trunk corrections to backward or frontal plane perturbations.
Analysis of IPD patient’s postural responses generated while standing has revealed that the underlying muscle mechanics are altered, in comparison to healthy older adults. Patients with IPD are able to generate similar patterns of muscle response which are directionally specific to both translational (Horak et al., 1996) and rotational perturbations (Carpenter et al., 2004). It has also been reported that patients with IPD respond with early muscle responses from antagonist muscles and have overall larger muscle responses (Carpenter et al., 2004; Dietz et al., 1993; Dimitrova et al., 2004b; Horak et al., 1996). These findings have been observed in patients trunk muscles when responding to translational perturbations, irrespective of whether they had a wide or narrow stance (Dimitrova et al., 2004b) and when responding to rotational perturbations (Carpenter et al., 2004). Furthermore, these response characteristics have also been observed in the lower limbs of patients with IPD in response to postural perturbations, resulting in an increased symmetry of muscle activation and decreased production of torque about the ankle, limiting the corrective movements they are able to make (Carpenter et al., 2004; Dimitrova et al., 2004a; Dimitrova et al., 2004b; Horak et al., 1996), and may account for their decreased stability margins (Horak et al., 2005).

Our data support that patients with IPD are able to respond with similar muscle patterning relative to healthy older adults. However, our observed changes in muscle co-contraction and increased muscle response amplitude do not fully support what has been previously shown in standing, as these changes were limited to specific muscles and directions. While typically in standing they are seen across and between multiple muscles and directions.
Since our data shows that patients with IPD are capable of making corrective trunk movements with muscle responses that are directionally specific, and are adaptable to changing perturbation directions, then it is unlikely that the trunk is the sole proprietor to increased instability in patients with IPD, especially during the initial stages of the disease. Initial deficits in torque production and inability to adjust torque gain to changing postural circumstances (Horak et al., 1996) may have a direct impact on the functional outcome of the trunk. If the body responds to postural perturbations as a multi linked system (Gruneberg et al., 2004) then altered torque production in the lower limbs could directly affect the trunk. Preuss and Fung (2007) compared trunk responses in participants while seated and when standing and observed that trunk responses while standing were smaller in amplitude relative to those made while seated. They postulated that these differences are due to the initial dynamic responses of the lower limb to the perturbation, and that these responses cause an initial perturbation to the trunk prior to that of the platform. Therefore, it seems likely that any change from the norm of lower limb response would directly affect the trunk’s ability to assist in balance correction and instead may inflict greater instability. In support of this, it is predicted through modeling that any limited torques in the leg must be countered by rapid torques in the hip and trunk in order to maintain stability (Kuo, 1995; Winter et al., 1990).

As disease severity increases, it becomes apparent that these altered lower limb responses will have a greater affect on the trunk, especially if the response pattern of its musculature adapts a more co-contractive and improperly scaled muscle response schema. If patient’s response pattern from the trunk increases the degree of rigidity through symmetric muscle activation, then this will severely inhibit their ability to adequate generate the counter trunk torques needed to regain stability.
Furthermore, over time the natural level of rigidity associated with increased background muscle activity within the trunk increases with age (Allum et al., 2002; Gruneberg et al., 2004) and disease severity (Bridgewater & Sharpe, 1998; Carpenter et al., 2004; Wright et al., 2007). Biomechanical response criteria for postural responses in the frontal plane require an increased degree of both hip and trunk flexion, and an increased level of active control over the trunk (Henry et al., 1998) to respond to these perturbations. As our data has shown a decreased ROM in the frontal plane in patients with IPD, it is likely that over time the combination of altered lower limb and trunk responses combined with increased rigidity of the trunk, creates an increasingly rigid system incapable of making the appropriately required postural response, especially in the frontal plane.

Since our data did not show any global changes in patients with IPD ability to make appropriate corrective trunk movements, it is postulated that postural deficits may originate from deficiencies in lower limb responses. However, disease severity may directly affect this relationship altering the contributing factors to decreased postural stability in patients with IPD. Therefore, it is not that patients with IPD lack the ability to generate any corrective trunk activity (Martin, 1965), but is due more to the culmination of inappropriate responses from separate segments acting together that ultimately results in increased instability in this patient group.

4.9 Strengths and limitations

This study was strong in experimental design and provides critical information for better understanding postural instability in patients with IPD. Nevertheless there are some limitations that should be taken into account. Firstly, the number of patients included in this
study were limited. However, this was a result of our stringent inclusion criteria which allowed us to ensure that there were no co-morbidities that would confound our findings. Therefore, we are certain that differences observed between patients and healthy controls are due to the effects of IPD and not additional co-morbidities. In addition, sample sizes of similar magnitude have also been used in previous literature (Carpenter et al., 2004; Horak et al., 2005; Jacobs & Horak, 2006) and the fact that we were able to reach significance despite our small sample size provides support for the magnitude of the differences we observed. Although our perturbation criteria was based from previous literature (Forssberg & Hirschfeld, 1994), the magnitude of the postural perturbation may not have been large enough to require a demanding postural response, which could have impacted the response patterning we observed. In addition, participants were asked to hold their arms across their chest, removing their ability to make protective arm movements which could have altered their postural responses. Carpenter et al. (2004) recently observed that patients with IPD make abnormally directed arm movements during standing perturbations. Since these arm movements have been observed to be in the direction of the perturbation, by removing IPD patients ability use corrective arm movements to our tilting perturbations we may have made them more posturally stable. Thirdly, one may also argue that our normalized EMG data may not fully represent patients MVCs. Previous reports have found that patients are not able to produce the same isometric force patterns as controls (Bridgewater & Sharpe, 1998). However, it is doubtful that this is the case in our data, as similar findings were observed for both raw and normalized values. We also limited motion of the pelvis which may have altered automatic triggers for postural responses because previous literature has suggested that postural triggers may originate from the pelvis (Allum et al., 1998; Forssberg &
Hirschfeld, 1994). By limiting the motion of the pelvis we could have altered the automatic triggers for postural corrections. However, the fact that we were able to observe similar trunk corrective movements in both participant groups, which were similar to previous seated literature in patterning and directional sensitivity, suggests this did not influence our data. Interestingly, this could also mean that either the pelvis is not needed to trigger postural corrections or that we did not completely isolate pelvis motion. With respect to the ROM testing, participants may have shifted their body position while on the table which may have affected their maximum ROM achieved. However, a great deal of care was taken to ensure limited movement of the trunk occurred during testing by implementing support straps and through visual inspection. Additionally, because ROM was based on the relative position of the trunk support, calculating relative changes in ROM from participant’s initial stationary position would have accounted for any shifts of the trunk support.
5. CONCLUSION

In conclusion we have found that patients with IPD are capable of making corrective trunk movements to seated perturbations with the appropriate muscle responses, which may become altered over time with increasing disease severity, contributing to decreased postural stability. Response characteristics did not vary between groups substantially, however, limited ROM in the frontal plane may contribute to postural deficits in this plane. Lastly, our novel findings do not support the previous findings of Martin (1965). In contrast, to what he believed was the contributing cause for incorrect trunk responses, specifically vestibular loss, we attribute the occurrence of incorrect trunk responses to deficits in improperly modulated muscle response amplitudes coupled with symmetric muscle activity. This is further confirmed by the fact that vestibular loss patients have been found to respond to standing tilting perturbations with delayed onsets of balance correcting responses (Allum et al., 1998) which we did not observe in our patients with IPD.

Overall, it is clear that the associated postural deficits of the trunk in patients with IPD are not fully understood and requires further research; however our data may provide beneficial information regarding timed changes in trunk muscle responses with increasing disease severity.

5.1 Future Research

Future research addressing the contributing factors of decreased ROM in patients with IPD is required. In order to focus on this issue, the degree of tonic trunk muscle activity should be recorded through use of EMG while participants lay supine. In this way the amount of tonic trunk muscle activity relative to changes in passive tissues could be
compared and additional factors contributing to IPD patients decreased ROM explored. In addition, IPD patients’ spinal stiffness should be determined from a larger number of incremental weights in order to provide a more detail look into the changes of spinal stiffness in patients with IPD and elderly controls when laying supine.

Lastly, research examining the factors that contribute to standing and seated balance instability in patients with IPD is required. In particular, future studies should be designed to examine trunk responses in young, elderly and patient groups while seated, then in standing so both age and posture related changes in trunk muscle responses to external perturbations can be accounted for. The contributing influence of the pelvis should also be accounted for by ensuring similar pelvic positioning in both standing and seated positions (Preuss & Fung, 2007). Additionally, based on our two case studies, it may be beneficial to conduct this research with IPD patients of varying levels of disease severity in order to determine the possible altered changes in postural responses in the trunk. The resulting benefit of this research may lead to the development of a grading system of postural instability and possibly, preventative rehabilitation programs.
REFERENCES


APPENDIX A

RESULTS

Sagittal plane perturbations

Raw 100(ms) area data

A significant interaction between group and direction for $EST_9$ ($F_{(1,13)} = 5.423$, $p = 0.037$) was observed, with a greater degree of activity during forward perturbations compared to backward for patients with IPD. However post-hoc analysis did not reveal any significant comparisons. $EST_9$ response areas were significantly dependent on perturbation direction ($F_{(1,13)} = 19.389$, $p = 0.001$) and were greatest during perturbations forward compared to backward. Trends for group ($F_{(1,13)} = 4.130$, $p = 0.063$) and direction ($F_{(1,13)} = 3.483$, $p = 0.085$) main effects for $ES_{L3}$ were also found. Patients with IPD had a tendency to have an overall greater amount of EMG activity compared to controls, while both groups appeared to have larger responses when perturbed forward compared to backward. RA and EO did not show any significant differences between groups or directions.

Raw 100(ms) background area

A significant group main effect was seen for $ES_{L3}$ ($F_{(1,13)} = 5.704$, $p = 0.033$). Patients with IPD had a greater amount of background activity in comparison to controls. Raw EMG for RA had a trend for a significant main effect of direction ($F_{(1,13)} = 3.225$, $p = 0.096$). No differences were seen in RA or EO.
**Frontal plane perturbations**

Raw 100(ms) area data

Raw EMG area values for the ES\textsubscript{T9} muscle were dependent on direction ($F_{(1,13)} = 46.293, p < 0.001$) with a greater amount of activity observed during perturbations to the right compared to the left. A trend toward a significant group by direction interaction was also found for ES\textsubscript{T9} ($F_{(1,13)} = 4.083, p = 0.064$) with increased ES\textsubscript{T9} activity during leftward perturbations for patients with IPD. However no significant, or trend toward significant, post-hoc comparisons were observed. Area averages for ES\textsubscript{L3} were significantly dependent on group ($F_{(1,13)} = 5.980, p = 0.029$) and direction ($F_{(1,13)} = 67.172, p < 0.001$). Patients with IPD had significantly larger response areas than did controls, while both groups had greater area responses during tilting perturbations to the right than to the left. A trend toward a significant main effect of direction was observed for RA ($F_{(1,13)} = 4.022, p = 0.066$). Both groups responded with greater activity going to the right then to the left.

Raw background activity

A trend toward a significant main effect of direction was found for ES\textsubscript{T9} ($F_{(1,13)} = 3.651, p = 0.078$) for perturbations to the right compared to left. Background activity for ES\textsubscript{L3} was dependent on group ($F_{(1,13)} = 5.426, p = 0.037$) and was greater in patients with IPD compared to controls. No significant differences were seen in either RA or EO.
APPENDIX B

The University of British Columbia
Office of Research Services
Clinical Research Ethics Board – Room 210, 828 West 10th Avenue,
Vancouver, BC V5Z 1L8

ETHICS CERTIFICATE OF EXPEDITED APPROVAL: RENEWAL

PRINCIPAL INVESTIGATOR: DEPARTMENT: UBC CREB NUMBER:
Mark G Carpenter

INSTITUTION(S) WHERE RESEARCH WILL BE CARRIED OUT:

<table>
<thead>
<tr>
<th>Institution</th>
<th>Site</th>
</tr>
</thead>
<tbody>
<tr>
<td>UBC</td>
<td>Vancouver (excludes UBC Hospital)</td>
</tr>
<tr>
<td>Other locations where the research will be conducted:</td>
<td>N/A</td>
</tr>
</tbody>
</table>

CO-INVESTIGATOR(S):
Bastiaan Bloem
Katherine Baker
A. Jon Stoessl
Z. Jane Wang
Martin J. McKeown
Meeko Oishi

SPONSORING AGENCIES:
- National Parkinson Foundation (US) - "A failure of multi-tasking: the combination of postural perturbations and reaching puts PD subjects at risk for falling"
- Parkinson Foundation of Canada - "Trunk Control and Balance Impairment in Parkinson’s Disease"

PROJECT TITLE:
Trunk Control and Balance Impairment in Parkinson’s Disease

EXPIRY DATE OF THIS APPROVAL: July 23, 2009

APPROVAL DATE: July 23, 2008

CERTIFICATION:
In respect of clinical trials:
1. The membership of this Research Ethics Board complies with the membership requirements for Research Ethics Boards defined in Division 5 of the Food and Drug Regulations.
2. The Research Ethics Board carries out its functions in a manner consistent with Good Clinical Practices.
3. This Research Ethics Board has reviewed and approved the clinical trial protocol and informed consent form for the trial which is to be conducted by the qualified investigator named above at the specified clinical trial site. This approval and the views of this Research Ethics Board have been documented in writing.

The Chair of the UBC Clinical Research Ethics Board has reviewed the documentation for the above named project. The research study, as presented in the documentation, was found to be acceptable on ethical grounds for research involving human subjects and was approved for renewal by the UBC Clinical Research Ethics Board.

Approval of the Clinical Research Ethics Board by:

Dr. Gail Bellward, Chair

85