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CLINICAL ASSESSMENT VERSUS FORCE PLATFORM ASSESSMENT

OF POSTURAL INSTABILITY

IN PARKINSON'S DISEASE.
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A THESIS

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A COMPARISON OF CLINICAL ASSESSMENT VERSUS FORCE PLATFORM ASSESSMENT OF POSTURAL INSTABILITY IN PARKINSON'S DISEASE

ROSEMARIE E. SEARS-DUKU, B.H.SC (Physiotherapy) McMaster

Dr. J. L. Starkes

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ABSTRACT

The purposes of this study were: (1) to determine whether there was a correlation between a quantitative assessment of postural sway, and a clinical assessment of postural stability, in patients diagnosed with idiopathic Parkinson's Disease (PD); (2) to plot individual diurnal changes in postural sway characteristics of PD patients over an eight-hour time period; (3) to plot day to day changes in individual postural sway characteristics of PD patients; (4) to determine whether there was a difference in the postural sway characteristics of parkinsonians, with and without vision; (5) to determine whether there was a difference in the postural sway characteristics of the same individuals when using either Sinemet or Deprenyl.

Three male PD patients were recruited into this study. Each subject stood on a stable force platform (AMTI OR6-5-1). Measurements included the standard deviations of the coordinates of the centre of pressure (COP) in the anterior-posterior (a-p) and lateral (lat) directions, the mean velocity of sway, and area of sway. These dependent measures were evaluated in a "quiet standing" condition, once with the eyes open (EO) and once with the eyes closed (EC). These procedures were carried out ten times over the course of an eight hour day. Each subject was tested two days while taking
eight hour day. Each subject was tested two days while taking Sinemet, and two days while on the Deprenyl regimen. Secondly, at two periods of each test day, each patient was evaluated using the postural assessment section of the Sears Parkinson's Assessment Form (SPAF).

The results were: (1) group analyses and individual analyses established the evidence of significant correlations between both the quantitative measures of postural sway (force platform) and the qualitative assessment tool (SPAF); (2) significant variability was evident in the analysis of individual data plots; (3) no statistically significant differences were observed for any subject when measured from day to day; (4) generally, vision was a stabilizing factor in postural control, however, this was quite variable for each subject; (5) significant improvements in postural stability were observed with the introduction of Deprenyl for one out of three subjects.

These findings are discussed in terms of their clinical and behavioral importance, with specific reference to Physiotherapy.
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To Winnie, Quisbert, Djanet, Terese, Celia, Sharon, Milton, Aunt Una, Kirt, Qwyn, Kyla, Donald, Donny, Sheree, Danielle, Mark and Akbar.

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Finally to the subjects who participated in this research, for their commitment throughout the study.

Free at last!
Free at last!
Thank God Almighty.
I am free at last!

MARTIN LUTHER KING JR.
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INTRODUCTION

Parkinson's disease (PD), is idiopathic in nature and corresponds to paralysis agitans, or the shaking palsy, originally described by James Parkinson in 1817.

The onset of this insidious disease, is often imperceptible, yet it is known to progress at variable rates. The major diagnostic symptoms of PD are tremor, rigidity, bradykinesia and disturbances of gait. Even in the early states of PD, some disruption of postural equilibrium is evident, but with disease progression, severe postural instability and falling predominate (Factor & Weiner, 1988).

Another important and ultimately challenging aspect of this disease, is the great variability in clinical presentation of the parkinsonian individual. Functional impairments may seem pronounced at one moment, and then diminished at the next. During the initial stages of PD, these fluctuations may be quite consistent and reflect the medication regimen that the individual has been prescribed. During the advanced stages of the disease however, this fluctuation in performance becomes increasingly inconsistent and may or may not reflect the medication cycle (Rajput & Duvoisin, 1990).

The clinical picture of PD has changed during the last two decades. The clinician today, sees patients with less devastating disability than previously presented. Clinicians
are now more concerned with the patient's complaints of side-effects of the drugs, even though these medication may have partly alleviated disabilities. This change in the clinical spectrum is not due to any alteration in the structural or chemical pathology of PD, but solely due to more effective treatments. Levodopa (L-dopa) remains the backbone of modern treatment, but complications, toxicity, and decreased effectiveness tend to appear with long-term use of all antiparkinsonian drugs and/or with progression of the disease (Forno, 1988; Hefti & Weiner, 1988). Selegiline (Deprenyl), a monoamine oxidase inhibitor, type B (MOA-B) has been shown to potentiate those benefits obtained from L-dopa, as well as to retard the progression of PD (Birkmayer, 1987; Fischer & Bass, 1987; Rinne, 1987; Tetrud & Langston, 1989; The Parkinson Study Group, 1989; Yahr, 1987). This belief has not been completely accepted by the medical community, as some literature denies the effectiveness of Deprenyl with both de novo and long term parkinsonian individuals (Elizan, Yahr, Moros, Mendoza, Pang, & Bodian, 1989a, 1989b, 1990; Friedhoff, 1990; Sudarsky, 1990).

Parkinson's disease results primarily from the degeneration of dopaminergic neurons in the substantia nigra. These neurons are involved in the production of dopamine that is stored within the vesicles of the nerve endings. Under normal conditions, dopamine is released into the synaptic...
space where it acts on dopamine receptors at the post-synaptic nerve ending. The loss of neurons in the substantia nigra results in a depletion of striatal dopamine content. In most patients, symptoms of the disease appear only after this dopamine loss reaches 80 percent. Studies have shown that substantia nigra cell loss and dopamine deficiency proceed in a parallel fashion. The extent of dopamine deficiency correlates well with the severity of akinesia and rigidity, and to a certain extent, postural stability (Rajput & Duvoisin, 1990).

The nature and extent of postural instability is unique and identifiable only by careful subjective and objective examination. One method of evaluating postural stability, is to observe changes in an individual's ground reaction forces. These forces have been termed, centre of pressure (COP). In the analysis of COP, one employs a force platform. This instrument measures the postural sway characteristics of an individual, which in turn provides an indirect evaluation of postural control. Force platform measurements have become one of the more popular methods of analyzing postural control. Measures of postural sway may include standard deviations of COP in the anterior-posterior (a-p) and lateral (lat) directions, velocity of sway, area of sway, amplitude of sway in the a-p and lat directions, as well as the frequency composition, or power spectral analysis of
the sway data (Fernie & Holliday, 1978; Fernie, Gryte, Holliday, & Llewellyn, 1982; Goldie, Bach, & Evans, 1989; Gregoric & Lavric, 1977; Hayes, Spencer, Riach, Lucy, & Kirshen, 1985; Hattori, Starkes, & Takahashi, in press; Kilbreath, 1986; Lucy & Hayes, 1985). A number of studies have identified significant differences in postural sway characteristics between parkinsonian and non-parkinsonian subjects (Gregoric & Lavric, 1977; Hawken, Waterson, Jantti, Tanyerio, & Kennard, 1990; Kilbreath, 1986; Njiokiktjien & De Rijke, 1972; Tokita, Miyata, Matsuoka, Taguchi, & Shimada, 1976; Watanabe, Okubo, & Ishida, 1980). Significant differences in postural sway have also been demonstrated within the parkinsonian population (Cernacek, Brezny, & Jagr, 1973; Folkerts & Njiokiktjien, 1972; Kilbreath, 1986; Kiawans, 1986; Starkes, Riach, & Clarke, 1992). Correlational research has established limited but significant relationships between objective measures of postural sway and clinical features of the disease (Kilbreath, 1986). A few studies have also identified consistent relationships between clinical manifestations of the disease (Kilbreath, 1986; Sears-Duru, 1991).
LITERATURE REVIEW

As this discussion touches on a number of different issues, the literature review shall focus on the following relevant areas: neurophysiology of parkinsonism; parkinsonian medications; assessment tools for PD; postural stability; the role of somatosensory, vestibular and visual systems in postural stability; postural sway; and postural sway and PD.

Neurophysiology of Parkinsonism

The basal ganglia form a conglomerate of nuclei in the telencephalon, diencephalon and midbrain (Forno, 1988). The corpus striatum (caudate nucleus and putamen) and globus pallidus are the most notable components of the telencephalon that are associated with PD. The pallidus is divided into two parts known as the external/lateral and internal/medial segments. The subthalamus, derived from the diencephalon, and the substantia nigra, from the midbrain, complete those structures commonly considered to form the basal ganglia (Forno, 1988).

Of these, the corpus striatum can be divided roughly into a dorsal division, neostriatum, consisting of the caudate nucleus and putamen, and the ventral striatum, consisting of the nucleus accumbens, olfactory tubercle and island of Calleja (Rolls, 1990). The neostriatum receives major inputs
from almost all areas of the neocortex and has major efferent connections with the globus pallidus and substantia nigra (pars reticulata). These in turn are connected to the ventral group of thalamic nuclei and thus to the supplementary motor, premotor, and prefrontal cortex. This pattern of connections suggests that the striatum provides one important route through which the cortex can influence motor structures (Rolls, 1990).

The ventral striatum receives inputs from limbic structures such as the amygdala and hippocampus and projects to the ventral pallidum. The ventral pallidum may influence output regions by the subthalamic nucleus/globus pallidus/ventral thalamus/supplementary motor route, or via the mediodorsal nucleus of the thalamus/prefrontal cortex route. Thus the ventral striatum may be for limbic structures what the neostriatum is for neocortical structures, i.e. a route for limbic structures to influence output regions (Rolls, 1990). The dopamine pathways are at a critical position in these systems, for the nigrostriatal pathway projects to the neostriatum and the mesolimbic dopamine pathway projects to the ventral striatum (Rolls, 1990).

Damage to the striatum produces effects which suggest that it is involved in orientation to stimuli, as well as initiation and control of movement. Depletion of dopamine in the striatum has often been associated with tremor and
akinesia. The effects of damage to different regions of the striatum also suggest that there is functional specialization within the striatum. For example, in monkeys researchers have observed the following functions: (a) neurons in the putamen, which receive inputs from the sensorimotor cortex, have activity related to movements; (b) neurons in the caudate nucleus which receive information from the association cortex have activity related to environmental stimuli. These in turn signal preparation for the initiation of behavioral responses. (c) Neurons in the tail of the caudate nucleus, which receive input from the inferior temporal visual cortex, respond when a patterned visual stimulus changes; (d) neurons in the ventral striatum, respond to emotion-provoking or novel stimuli; (e) the globus pallidus, substantia nigra pars reticulata and subthalamic nucleus have neurons with activity which is clearly related to leg, arm and orofacial movements. Moreover, there is a somatotrophic representation of these body parts within each of these areas (Rolls, 1990).

These findings indicate that there is some segregation of function within the basal ganglia. They suggest that different symptoms might be present depending on the regions of the basal ganglia within which dopamine is depleted. They also suggest that impairment of function of the basal ganglia, as in PD, might produce a variety of changes which are not just motor but might include cognitive changes (Rolls, 1990).
The dopamine-acetylcholine imbalance theory was developed to explain the effects of dopamine deficits in PD. The theory suggested that when dopamine stores were depleted, an excess of acetylcholine resulted. Conversely, an excess of dopamine in the system, resulted in the depletion of acetylcholine. The consequence of each of these scenarios was PD (Marsden, 1984). However, previous research suggests that a decrease in norepinephrine, serotonin and gamma-aminobutyric acid (GABA) also contribute to the symptoms seen in PD (DeLong & Alexander, 1986). Dopamine deficiency contributes to rigidity and hypokinesia, bradykinesia, and akinesia. Norepinephrine deficiency contributes to akinesia; the functional increase in acetylcholine contributes to the parkinsonian tremor; serotonin and GABA deficiencies tend to lessen symptoms (DeLong & Alexander, 1986). What then contributes to postural dysfunction?

Allen and Tsukuhara (1974) have developed a theory that illustrates the relationship between motor control and the basal ganglia. Figure 1 provides a diagrammatic representation of pathways concerned with the execution and control of voluntary movement.

As Figure 1 illustrates, there is a division of motor activity
into three stages: plan, programming and execution. The decision to move is cortical. It may be based upon the arrival of sensory information, or it may originate in the cortex. It is proposed that the intention to move initially achieves expression in patterns of excitation in neurons of the supplementary motor area (SMA). This information is then relayed to the association areas of the cortex from which signals are split into two parallel subcortical streams for processing. One stream enters the basal ganglia, while the other proceeds via the corticopontine tract to the lateral cerebellar hemispheres. Information is then processed in parallel to be returned to the thalamus for recombination. From there it is relayed back to the cortex to complete the programming function. Execution then proceeds via the corticospinal tract. The pars intermedia of the cerebellum updates the movement based on sensory description of limb position and velocity of movement in the planned direction.

Although PD is predominantly characterized by a loss in the dopaminergic striatal projection, Rossor (1981) has suggested that this disease also shows a loss of the noradrenergic projection from the locus coeruleus. The cells of the locus coeruleus, substantia nigra and substantia innominata all share a non-specialised isodendritic pattern which extend from the spinal cord to the basal forebrain. If
PD is seen as a disorder of the isodendritic core, the primary condition would be a loss or dysfunction of the isodendritic cells. The loss of other cell groups outside the isodendritic core may therefore be secondary phenomena resulting from trans-synaptic degeneration. There is evidence that cells are lost within the terminal fields of the projection systems affected in PD. Loss of striatal cells in PD may be an important reason why treatment fails (Mossor, 1981).

Several studies (Cernacek et al., 1973; Folkerts & Njiokiktjien, 1972; Klawans, 1986) have indicated that L-dopa therapy has not been effective in improving postural control. These conclusions would suggest that dysfunction to the dopaminergic system alone does not account for all of the clinical symptomatology evidenced in PD.

Parkinsonian Medication

Drug treatment for Parkinson's disease is divided into two categories: 1) anticholinergic agents and 2) dopaminergic agents (Marsden, 1990). The first category of medications are used to reduce the functional excess of acetylcholine in the system. Anticholinergic agents are primarily useful in treating the parkinsonian tremor; however, they also may relieve some rigidity and bradykinesia. Two classes of drugs with anticholinergic properties are used for this purpose: (a) belladonna alkaloids and synthetic atropine-like agents;
and (b) antihistamines. The choice of agent is based not only on efficacy but also side effects (Lang, 1990).

No single anticholinergic agent has demonstrated superiority to any other anticholinergic agent. Each renders beneficial effects on tremor, primarily due to a sedative effect. It is estimated that no more than 20 percent improvement in parkinsonian symptoms may be expected, although there have been no definitive studies to address this issue. These agents may be efficacious for a period of time in a particular patient, but the disease is progressive and even though the tremor may become less evident, other classes of agents may be needed (Lang, 1990).

The second medication category can be subdivided into (a) agents that increase synthesis of brain dopamine, (b) agents that directly stimulate dopamine receptors, (c) agents that reduce the presynaptic reuptake of dopamine, (d) agents that stimulate endogenous dopamine release, and (e) agents that reduce the catabolism of dopamine (Lang, 1990).

The dopaminergic approach to therapy is aimed at enhancing dopaminergic transmission within the basal ganglia thus helping to restore normal feedback mechanisms. The drugs used for this purpose are: (a) L-dopa, (b) L-dopa plus carbidopa (reduces peripheral metabolism of L-dopa to dopamine), (c) amantidine (stimulates presynaptic dopamine release and reduces the reuptake of dopamine by presynaptic
sites), and (d) aporphines and ergot alkaloids (directly stimulate dopamine receptors) (Marsden, 1984).

Levodopa is an amino acid that crosses the blood-brain barrier in significant amounts only when given in large oral doses. Dopa-decarboxylase, an enzyme, metabolizes L-dopa to dopamine; this reaction occurs peripherally and centrally since the enzyme is located in the liver and gastrointestinal tract, and dopaminergic axons, respectively. Since dopamine will not cross the blood-brain barrier, it is necessary to provide its precursor, L-dopa, to increase the dopamine concentration within the basal ganglia. L-dopa will not reverse or retard the degenerative changes of the substantia nigra, rather, it is an exogenous replacement of dopamine to the corpus striatum and diminishes parkinsonian signs by this mechanism (Marsden, 1984).

Although L-dopa is presently the most effective treatment for parkinsonism, prolonged use (three to five years) is associated with a decrease in efficacy as well as an increase in side effects (Klawans, 1986; Marsden, 1984). Fluctuation in symptomatic responses to L-dopa therapy are a complication in many parkinsonians, particularly in the later stages of the disease (Fahn, 1974). This is often termed the "on-off" syndrome. Barbeau (1972) uses the term "akinesia paradoxica" to describe this phenomenon (Fahn, 1974). Clinicians refer to an "off" period, as one where the
beneficial effects of levodopa have worn off or are not apparent (Fahn, 1974; Rodnitzky & Lang, 1990). 'On' is the responsive phase of beneficial symptomatic effects (Rodnitzky & Lang, 1990).

Berg, Ebert, Willis, Host, Finchan, & Schottelius (1987) describe several types of on-off syndromes:

(1) Early morning akinesia is associated with rigidity and tremulousness upon awakening. It would appear to be due to either a progression of the disease or a depletion of dopamine during sleeping hours.

(2) Freezing episodes are characterized by hesitation upon initiating an act. Freezing shows no correlation with timing of dosage. It is inherent to the disease and may be a sign of disease progression.

(3) End-of-dose deterioration describes a shortened interval in which a given dose is effective. Chorea is seen approximately one hour after a dose, followed by control of parkinsonian symptom, and finally reoccurrence of the symptomatology several hours before the next dose.

(4) Peak-dose dyskinesia and akinesia seem to be a result of drug overdose. These impairments of movement occur when a given dose is at its peak activity and disappear with a reduction in dosage (Berg et al., 1987).

It is not known exactly why some individuals exhibit this pattern of loss of drug effect after several years of
therapy. Rodnitzky & Lang, (1990) suggest that on-off syndromes may occur due to the fact that individuals who have been on long-term replacement therapy have simply lost more dopaminergic cells and have a decreased buffering capacity. There are also fewer remaining cells to take up and store dopamine (Rodnitzky & Lang, 1990).

In the early stages of PD, there is a prompt elevation of plasma L-dopa levels, after the patients take a dose of L-dopa-carbidopa, or similar L-dopa compound. Due to the relatively short half-life of L-dopa, the plasma level drops considerably within four hours, but rises quickly with the next dose. A regular sawtooth pattern of the plasma level may be recorded as doses are taken throughout the day (Rodnitzky & Lang, 1990).

By contrast, the early patient's disability score shows little fluctuation, if any, and many enjoy a long duration of response to a single dose. In fact, some patients mistakenly conclude that L-dopa is ineffective because there is no deterioration in their symptoms, despite missing one dose (Frankel, Pirtosek, Kempster, Bovingdon, Webster, Lees & Stern, 1990; Rodnitzky & Lang, 1990).

Physiologically, in these early patients, the remaining surviving dopaminergic cells are able to take up exogenously-administered L-dopa, convert it to dopamine and release it under neural control when needed. After three or
four years of L-dopa therapy and with further disease progression, the severity of the patient's symptoms may rise and fall with the plasma (L-dopa) levels. In fact, situations occur where random akineti c, dyskineti c and hyperkineti c phases occur in individuals on long term replacement therapy (Frankel et al., 1990; Olanow, 1990).

The role of diet in individuals with PD relates primarily to the ability of certain foods to moderate the effects of drug therapy. The amount of protein in the diet of the parkinsonian patient taking L-dopa is also important, because it can interfere with the absorption of L-dopa at two sites: the intestines and the blood-brain barrier. With the varying types and amounts of amino acids present in the protein we eat, competition for absorption into these areas predominates (Rozovski & Lurie, 1990). In essence, the peripheral and central metabolism of L-dopa becomes inhibited and the effectiveness of the medications becomes questionable. Eriksson, Granerus, Linde & Carlsson (1988) observed that administration of a low protein diet to parkinsonian patients with "on-off" syndromes, consistently increased the total daily time of "on" states when compared with a high protein diet. The authors suggested that the clinical effect of the low protein diet may be due to a marked decrease in the plasma concentration of large neutral amino acids that compete with L-dopa for carrier-mediated transport to the brain.
Deprenyl (Selegiline/Eldepryl), a monoamine oxidase type B (MAO-B) inhibitor extends the effectiveness of L-dopa, by reducing the catabolism of dopamine at the level of the nerve terminals in the basal ganglia. Various clinical trials using Deprenyl with L-dopa-peripheral decarboxylase inhibitor preparations have shown that it prolongs L-dopa's effectiveness, and even retards the natural progression of PD (Birkmayer, 1987; Fischer & Bass, 1987; Rinne, 1987; Tetrud & Langston, 1989; The Parkinson Study Group, 1989; Yahr, 1987). However, some literature denies the effectiveness of Deprenyl with both de novo and long term parkinsonians (Elizan et al., 1989a, 1989b, 1990; Friedhoff, 1990; Sudarsky, 1990).

Assessment Tools for Parkinson’s Disease

Despite the extensive and expanding literature concerning PD, implementation of new parkinsonian evaluative tools, discussion or critical review of the fundamental issues of methodology, reliability, reproducibility, quality and applicability of clinical assessment tools is scarce. With the advent of new and effective medications for PD, evaluation of pharmacologically associated functional changes and the establishment of clinically reliable instruments is crucial.

Variability in individual clinical presentation was acknowledged in early clinical trials evaluating various parkinsonian medications. However, inadequate experimental
protocols failed to consider the wide fluctuations and alteration characteristics of the disease (intrinsic, emotional, as well as drug-related). As a consequence, researchers have had to re-interpret the results of these initial clinical tests. Future research designs must account for individual variability in order to reach reliable and valid conclusions.

Measurements of the clinical symptoms of PD have been performed by many investigators. These assessments were thought to provide a "true" assessment of the parkinsonian state, because they measured fundamental aspects of the disorder. However, few of these assessments were ever evaluated for either reliability or validity.

England and Schwab (1956) conducted a study to evaluate the effect of thalamotomies in patients with PD. The authors developed an instrument to categorize patients into one of five grades related to prognosis and progression of disease. Grade five was the most rapid progression and worst prognosis, whereas Grade one was the slowest progression and the best prognosis. This 10 point scoring system was devised to quantitate activities of daily living as assessed by the patient (subjective score), and aspects of motor function, as assessed by the physician (objective score) (Fahn, Elton & the UPDRS Committee, 1987). The subjective and objective scores represented a percentage of normal function, with 100% of
normal, constituting a score of 10 (England & Schwab, 1956). Guidelines were not available for the examiner to administer a score for the responses attained during the assessment. Nevertheless, the authors concluded variability in symptoms from day to day and hour to hour accounted for less than a five per cent variation in the scores between clinicians.

Canter, de la Torre, and Mier (1961) developed the Northwestern University Disability Scales (NUDS) to measure the extent to which patients with PD lost their pre-morbid proficiency in activities of daily living. This particular scale awarded ten points to individual scores of walking, dressing, hygienic care, and speech. Five points were also awarded each for eating and feeding. The inter-rater reliability for the total scale was .95, with the sub-scales ranging from .84 (speech) to .93 (walking) (Canter, de la Torre, & Mier, 1961). Each task in the NUDS, with its associated scoring system was outlined, however, these were not made available to the examiners. It is imperative that guidelines are made accessible, if only to standardize evaluative procedures and limit individual clinician interpretation.

Hoehn and Yahr (1967) assessed the natural history of 802 patients, who were seen at the Vanderbilt Clinic of the Columbia-Presbyterian Medical Centre from 1949 - 1964. The investigators attempted to classify all patients with a
specific type of parkinsonism and identify the symptomatology associated with the various types of the disease. The extent of disability, the onset, progression and subsequent mortality associated with this disease were also considered. Consequently an arbitrary five point clinical disability rating scale was developed, which presumably reflected the characteristics of the disease. The authors suggested that the lower the score on the scale, the less disabling and the more unilateral the disease. As the disease progressed, the associated score on the scale was higher, and reflected greater disability and bilateral involvement.

This research article provided clinicians with a much needed description of the various types of parkinsonism. One major limitation of the Hoehn and Yahr Scale was that the definition of each stage was quite general and it failed to focus on the significant differences associated with each stage. For example, individuals with unilateral symptoms who were extremely incapacitated would be assigned to Stage I regardless of the extent to which they were functionally impaired. Yet, individuals with bilateral symptoms and minimal impairment would be classified as Stage II, despite the fact that they were less functionally impaired than the other individual classified to Stage I.

Based on the initial uniqueness of this paper, the Hoehn and Yahr Scale became the most popular means of
describing functional deficits of individuals with PD. It has been used traditionally as a means of assessing the effectiveness of parkinsonian medication, and also to indicate disease severity of a patient or population of patients (Lakke, 1990).

Webster (1968) published a clinical rating scale which assessed the physical features of PD, namely bradykinesia, rigidity, posture, gait, tremor, facies, speech and activities of daily living, seborrhea and upper extremity arm swing. Webster assigned value ratings of zero to three for each item, zero indicating no involvement, and scores of one, two and three were assigned to early, moderate and severe disabilities respectively. This scale was additive, with a total score out of 30. The author suggested that the higher the total score obtained on the scale, the greater the extent of disability observed in the subject. All items were specific, well defined and precise. Moreover, this scale was significantly more reflective of the severity of PD than the Hoehn and Yahr scale.

In 1970, the Columbia University Group in their initial trials of L-dopa, developed a five point scale that measured the physical signs of parkinsonism (Duvoisin, 1970). This instrument evaluated the significant signs and symptoms of tremor and rigidity, and a cluster of essentially non-dimensional features, such as monotone speech, simian posture,
bradykinesia, festination, shuffling, propulsion and other characteristic defects of the gait, seborrhea, sialorrhea and diaphoresis.

It took almost fifteen years before an abbreviated version of this scale was evaluated for reliability. Montgomery, Reynolds, and Warren (1985) examined the interobserver agreement of selected scores from the instrument endorsed by Duvoisin. In evaluating differences between observers, the authors suggested that the data were samples from the same behavioral population. Consequently, the Wilcoxon matched-pairs signed ranks test was applied, with the conclusion that there was no significant difference between observers. However, as it was the scoring of the task that was being measured, not the task (behavior) itself, the more appropriate analysis would have been the Mann Whitney U test.

In measuring the linear association between the ratings of the two observers across cases, Spearman Rho scores of .67 for bradykinesia; .89 for gait; .71 for posture; .95 for resting tremor; .72 for postural tremor; and .74 for the Hoehn and Yahr scale, were obtained. These statistical results are rather favourable, but caution must be exercised especially when attempting to generalize these conclusions to such a heterogeneous population.

McDowell, Lee, and Swift (1970) attempted to systematically evaluate the efficacy of L-dopa in 100 patients
with varying symptoms of PD. An instrument was developed to evaluate both functional disability and associated symptomatology. Each item was assigned a "weight", which reflected its importance to function as determined by the investigators. These weighted values were then multiplied by a degree of severity of the symptom at each examination, to achieve a total score. This accounted for a maximum possible score of 88 points for the symptomatology scale and 132 points for the functional disability scale. The higher the score(s) obtained on assessment, the greater the severity of the disease. The authors evaluated changes in function and symptomatology associated with the addition of L-dopa in the individual medication regimen. They concluded that L-dopa taken independently or in association with anticholinergic medications, was effective in the treatment of PD.

Lieberman (1974) also described an evaluative tool that was used to assess change in persons with PD. This was subsequently revised and became known as the New York University Parkinson's Disease evaluation (NYUD). It rated items such as rigidity, resting tremor and bradykinesia for all extremities and parts thereof, as well as gait, postural stability and voluntary movements (functional disability) (Lieberman, Dzitloowski, & Gopinathan, 1980). Over an eight year period, the authors evaluated 520 outpatients with PD utilizing this evaluative tool. Statistical analyses were
performed in only 100 of these patients to determine the relationship between the total score obtained on the NYUD, subsections of the NYUD, and the staging of disease, as described by the Hoehn and Yahr scale.

Lieberman et al. (1980) suggested that reproducible values within five percent for each major sign, total score, involuntary movements and functional disability, were obtained by different observers independently examining the same patient at the same time. Utilizing the Pearson product moment statistic, a significant correlation was found between the total score and the Hoehn and Yahr scale ($r = .66$), as well as for bradykinesia and gait ($r = .63$). Correlations among total score, and functional disability approached but did not reach significance, while correlations between the other signs did not approach significance. The scoring system of this scale was ordinal by design, and therefore, the use of the Pearson product moment is questionable. The Spearman Rho would have been the most suitable statistic. Furthermore, the Fisher's Exact Test was also incorporated to describe these correlations. However, the assumption of this test is that the samples follow a binomial distribution. It has been well documented that PD does not follow a smooth progression. In fact daily and diurnal fluctuations are usually observed; hence the use of the Fisher's Exact Test is also inappropriate. The authors do suggest using the Wilcoxon
signed rank test to evaluate correlations, but these results were not documented.

One of the first documented physiotherapeutic instruments that evaluated PD, was designed by Franklyn (1986). This instrument was developed in order to evaluate functional impairments, changes in gait, posture and balance associated with the disease. Although this evaluative tool addressed some of the needs of the physiotherapist who assesses and treats clients with PD, it was never evaluated for either clinical or statistical significance.

Finally in 1984, a workshop was organized to develop a new rating scale for parkinsonism, that would encompass the best of the existing rating scales. After much deliberation and many revisions, the Unified Parkinson's Disease Rating Scale (UPDRS), version 3.0 was finalized in 1987 (Fahn, Elton, and Members of the UPDRS Development Committee, 1987). The UPDRS was divided into five sections. The first section was a quantitative five point scale which measured the severity of clinical manifestations associated with PD. This section was subsequently divided into mental and historical motor, which included motor functions of activities of daily living, and objective motor features observed at the moment of the examination. Each of these subsections could be summed independently, and all could be combined to achieve a total numerical score (Fahn et al., 1987). The second section was
a qualitative and quantitative assessment of many of the complications of dopaminergic therapy for PD. The third and fourth sections consisted of modified Hoehn and Yahr, and Schwab and England ADL scales, respectively. The fifth section recorded weight, sitting and standing blood pressure, and pulse. The reliability of the UPDRS was examined using the Pearson product moment statistic. The following correlations were observed: Hoehn & Yahr Staging, $r = .78$; Schwab & England Activities, $r = .97$; M entation, Behaviour & Mood (1 - 4 pooled), $r = .55$; Activities of Daily Living (5 - 17 pooled), $r = .82$; Motor Examination (18 - 31 pooled), $r = .90$; Complications of Therapy (32-42 pooled), $r = .85$. All correlations reached significance levels between $p<.01$ and $p<.001$.

The methodology as described by these authors was rather incomplete. There was no description as to the type of subjects used in this study or if pre-test training sessions were provided for the clinicians. In addition, one of the cardinal symptoms of PD, rigidity, which demands physical examination to determine the extent of severity, was measured only as part of the videotape analysis, never as part of a hands on assessment by the clinicians. With a disease in which a significant proportion of symptoms must be physically evaluated to determine severity, this component of physical examination should be mandatory in all clinical trials.
Although the use of videotape has become a routine procedure for most neurologists who specialize in the treatment of patients with movement disorders, there is as yet no uniform method of videotaping parkinsonian patients that has been accepted by investigators in the field. Currently, videotape protocols vary according to the specific needs or purposes of the taping. This method can in and of itself, lead to raise conclusions about "apparently true observations". Nevertheless, videotape analysis may still prove to be an invaluable adjunct to hands-on clinical evaluation (Lang, 1985).

Lakke (1990) stated that "the UPDRS in attempting to encompass the best of the existing scale, appears to have reached its goal by adding more items rather than improving definitions. Derived essentially from the Columbia scale, it is at times confusing and clearly illustrates the difficulty in attempting to strike a reasonable balance between laborious comprehensiveness and practical, succinct clinical needs" (p. 474).

Despite these limitations and objections, one should acknowledge that the UPDRS is a valuable evaluative tool, which provides a wholistic measurement of function for individuals diagnosed with PD.

In Physiotherapy, the implementation of specific treatment techniques is determined by responses during
individual assessments. Physiotherapeutic evaluative tools designed specifically for persons diagnosed with PD are rare. In fact, to date, only one such instrument has been documented, (Franklyn, 1986). While addressing many of the features identified as cardinal symptoms of PD, this tool failed to provide an overall picture of the functional status of the individual.

Consequently, Sears-Duru (1991) developed and subsequently determined the inter-rater and intra-rater reliability of a physiotherapeutic instrument for PD. The focus of the Sears Parkinson's Assessment Form (SPAF) was the description and evaluation of function associated with PD.

The hypotheses associated with the SPAF were as follows:

1. The SPAF would provide an overall assessment of function in the parkinsonian individual.

2. Significant correlations between individual sections of the SPAF, most particularly in the areas of akinesia/bradykinesia, rigidity, tremor, posture, postural stability, and gait would be observed.

3. The greater the overall score observed on assessment, the greater the functional impairment of the individual.

4. High inter-rater and intra-rater reliability characteristics would be observed during the evaluation of
The SPAF was developed out of a need to incorporate a variety of novel instruments, as well existing tools, into one evaluative package for Physiotherapists. The goals of the SPAF were four-fold:

1. Identification of specific problem areas.
2. Easy administration and evaluation.
3. Utilization in primary, secondary and tertiary care centres.
4. Detection of change (improvement or deterioration).

This Instrument was divided into eleven sections, namely, Demographic data, Cognition, Stage of Disease, Akinesia/Hypokinesia/Bradykinesia, Rigidity, Tremor, Gross Motor Performance, Proprioception, Posture, Postural Stability, and Gait. A cumulative score could be obtained for each section, and totalled to produce a tally out of 736. The underlying assumption was that the greater the total score, the greater the severity of the disease.

A comprehensive set of guidelines outlining both patient and examiner position(s) and response(s) was also developed. These guidelines were provided to each examiner prior to each interaction and utilized to assist in the documentation of each individual assessment.

Section I was designed to give general information about the patient's medical history and social situation. The
subsection entitled "Present Parkinsonian Symptoms" was based on the cursory physiotherapeutic assessment described by Franklyn (1986). This component was altered to reflect the subject's perception of the severity of the manifestations of the disease, not the Physiotherapist's perception of the significance of their symptoms, as originally designed by Franklyn. Scoring on this section ranged from zero to three, with zero indicating no impairment, one, two, and three indicating mild, moderate and severe impairments, respectively.

Section II assessed cognitive status utilizing the Folstein Mini Mental State Examination. Originally, James Parkinson assumed that PD was purely a physical ailment. This perspective slowly changed to one that suggested that if cognitive impairments did occur, they were only evident at the end stages of the disease (Brown & Marsden, 1984). Current thought though, is that impairments in cognitive functioning are evident throughout all stages of the disease process (Benson, 1984; Selby, 1990).

Unlike all other sections in the SPAF, where a high score indicated greater functional impairment, in this section, the opposite was true. A lower score reflected "normal" cognitive functioning, whereas a higher score suggested impaired cognitive functioning.

Section III incorporated the Hoehn and Yahr stage of
disease scale. This scale ranged from Stage I to Stage V, as was originally suggested by Hoehn and Yahr (Hoehn & Yahr, 1967).

Section IV assessed akinesia, hypokinesia, and bradykinesia. Tests that are fundamental to the physiotherapeutic evaluation of coordination were incorporated into this section, namely: finger tapping, toe tapping and the finger to nose test/dysdiadokinesia.

Section V evaluated rigidity. This was performed by a measurement of the passive range of motion of the neck and extremities. If cogwheeling was evident upon assessment, an additional score of one point was added to the total tally for each area.

Section VI was an evaluation of tremor. This assessment was based on the observation of the head and extremities while the client was resting. Scores of zero, one and two were indicative of no observable tremor, small amplitude tremor, and large amplitude tremor, respectively.

Section VII evaluated gross motor performance. This section incorporated tests of functional ability that are commonly evaluated by Physiotherapists. Tests included rolling, moving to and from sitting and lying, long sitting, transferring to and from the bed, the chair and the floor.

Section VIII assessed proprioception, by determining the integrity of specific joint proprioceptors.
Section IX evaluated posture. This was measured by observing resting positions of the neck and trunk while either sitting or standing. Scores ranged from zero to five, which reflected increasing impairments of cervical and truncal alignment from the "norm". Additional scores of one and two were allotted if either side flexion or rotation or both of the neck and/or trunk were also observed.

Section X assessed postural stability (Appendix I). This section observed postural reflexes and functional responses to stresses in balance. The areas that were observed included: optical and labyrinthine righting, static and dynamic, dynamic righting in standing, protective reaction of the arm and leg, the fundamental position of kneeling and it’s derived position, half kneel standing and one legged stance. The ability of the individual to attain, maintain and return to each of the starting positions was scored accordingly.

Section XI measured gait. Quantitative aspects, such as distance and stride length, as well as qualitative changes in gait observed at the head, trunk, pelvis, hip, knee and ankle, were measured during this component of the assessment. Gait abnormalities that are specific to PD, such as festination, freezing and impaired arm swing, were also evaluated during this section of the instrument.

Two pilot studies were conducted to determine both the
inter-rater and intra-rater reliability of the SPAF. The methodology has been outlined in a previous article, (Sears-Duru, 1991). The results were very favourable, with both the intra-rater and inter-rater reliability reported at $r = .99$, $p<.05$. Moreover, significant correlations were observed between some of the cardinal symptoms, namely akinesia/bradykinesia, tremor, rigidity, gait impairments, and postural stability. There were minor problems associated with the scoring of this tool and items involved in evaluating cognitive status have since been removed.

Parkinson's disease is a multifaceted manifestation of basal ganglia disease, which at times appears to defy measurement and evaluation. It is important to recognize that throughout the literature, researchers, clinicians, authors and examiners, use different methods to diagnose PD, to evaluate the effects of medication on the course of this disease and to measure functional change (improvement or deterioration) throughout the course of the disease. Each scale is differentially sensitive in registering changes. The magnitude of numeric change as a function of clinical change differs, and the emphasis on particular aspects of disability differs. If studies of PD use different scales, most of which have not been assessed for either reliability or validity, conclusions are questionable and comparisons as essentially impossible (Diamond & Markham, 1983).
Postural Stability

Neurophysiological models of sensorimotor control of posture and movement are evolving rapidly, but few have been applied to the problem of disequilibrium in the elderly. Any model that describes the effect of age on postural control must account both for the increased instability commonly found in the majority of elderly subjects, and the increased variability in postural control in the elderly as a group. One model suggests that the effect of age-related changes in neural function on postural stability. Age-related deterioration in the important sensory modalities, such as vision, vestibular function and somatosensory proprioception; have been well documented (Maki, 1987). This widely accepted model suggests that postural instability is so common, it can be considered an inevitable "aging" effect resulting from widespread degeneration of the musculoskeletal, neuromuscular, and sensory systems. The increase in heterogeneity in postural stability in elderly subjects may be due to an increase in variability about the mean, age-related decrease in neural function, or perhaps due to differences in lifestyle and/or genetic traits (Horak, Shupert, & Mirka, 1989).

An alternative model suggests that the effect of age, per se, on postural control is quite small. However, superimposed upon a small decrease in postural stability due
to age alone, is the increased probability in the elderly of developing specific pathologies which lead to accelerated degeneration in neural and/or musculoskeletal systems. In this model, the probability with which a given pathology will develop is unique to each individual, and therefore the pattern of postural instability will be unique to each individual. In this view, measurable declines in postural control in an individual actually reflect preclinical evidence of specific pathologies, such as PD (Horak et al., 1989). For example, Pyykko, Jantti, and Aalto (1988) performed a study to assess postural control in healthy elderly individuals aged 80+. Postural perturbations were induced by stimulating calf muscles of each leg with vibration. The test was conducted in four conditions: (1) on a rigid surface with visual control; (2) on a rigid surface without visual control; (3) on a foam rubber covered surface with visual control, and (4) on a foam rubber covered surface without visual control. There was a relative shortage of postural information caused by a diminishment or deterioration of proprioceptive and exteroceptive inputs. There was also an absence of tendon reflexes and defective vibration sensation. The authors suggested that these results were indicative of peripheral polyneuropathy. In spite of age, or pathology, perhaps the most obvious of the tasks performed by the central nervous system's postural control mechanisms, is that of attempting to
maintain the upright bipedal stance. This involves generating a series of muscular contractions that produce moments of force about the joints of the musculo-skeletal system to counteract the effects of gravity (Hayes, 1981).

Nashner (1981) indicated that an understanding of the kinematic relations between motions of the body, muscular, gravitational and perturbational forces is important. This is due to the fact that performance of stance is ultimately expressed as the combined orientations and motions of many body parts. Certain mechanical principles of stability have been identified and it would seem that the particular postural control solution employed for a given situation must take these into account. The principles underlying stability, and ultimately determining whether the body is in stable or unstable static equilibrium, may be stated:

1. The degree of physical stability during quiet standing or "static equilibrium" is proportional to the area of the base of support (Nashner, 1981).

2. Stability is directly related to the height of the centre of gravity above the base of support (Hayes, 1981).

3. Stability in a given direction is directly related to the distance of the line of gravity from the edge of the base of support. In addition, it should be remembered that a condition necessary for static equilibrium is that the line of gravity must fall within the area of the base of support.
Stability is maximized in any direction when the centre of gravity (COG) is furthest from the edge of the base of support. Essentially, the bigger the base of support and the closer one is to the line of the COP, the more stable one is (Koozenkani, Stockwell, McGhee, & Firoozmand, 1980). Koozenkani et al. (1980) devised an index of stability to illustrate this concept:

\[ s(t) = \min s_i(t) \quad (i=1,2) \]

The stability margin \( s(t) \) is the shortest distance from the centre of pressure (COP) to either the front \( s_1 \) or back \( s_2 \) of the supporting foot. A person loses balance when the stability margin goes to zero. Individuals who have large stability margins will be better able to withstand a wide range of destabilizing perturbations and therefore, may be less likely to fall (Koozenkani et al., 1980; Mak1, 1987).

A small amount of postural sway is manifest as the central and peripheral nervous systems attempt to keep the body’s centre of gravity (COG) within the area delineated by the supporting base (Murray, Seirig, & Sepic, 1975, Kilbreath, 1986). The reflexes which intervene to maintain this position, may be considered to operate through a system of feedback (Brooks, 1983; Kilbreath, 1986). The three sensory systems that provide feedback input are the proprioceptive, vestibular and visual systems, respectively (Brooks, 1983;
The Role of Somatosensory, Vestibular and Visual Systems in Postural Stability

Postural control, to a large extent, is a reflection of sensory feedback (Oblak et al., 1975). Changes in the orientation of a standing subject are sensed by (1) proprioceptive and cutaneous (support surface) inputs responsive to the contact forces and motions of the feet upon the support surface; (2) visual inputs derived from linear and angular motions of the visual field; and (3) vestibular inputs derived from sway-related linear and angular accelerations of the head (Nashner, 1981).

Three sensory components impart information about the orientation and motions of the standing subject. These are proprioception, exproprioception and exteroception (Nashner, 1981). Proprioception is the sense of position and movement of one part of the body relative to another. Exproprioception imparts information about the position and movement of a part of the body relative to the external environment. Exteroception locates objects in the external environment relative to one another (Nashner, 1981). It was suggested that the somatosensory system utilized the proprioceptive and exproprioceptive points of reference, the vestibular system utilized the exproprioceptive point of reference, and the
visual system used all three points of reference (Nashner, 1981).

Changes in the orientation of a standing subject are sensed by support surface inputs (proprioceptive and cutaneous inputs responsive to the contact forces and motions of the feet upon the support surface), visual inputs (derived from linear and angular motions of the visual field), as well as vestibular inputs (derived from sway-related linear and angular acceleration of the head) (Nashner, Black, & Wall, 1982). However, the orientation information provided by support surface and visual inputs is potentially disrupted by the movements of the external surfaces to which these two senses are referenced. Therefore, support surface and visual inputs can be used to maintain vertical equilibrium only when their reference surfaces are fixed or their motions can be predicted in advance (Nashner et al., 1982). In contrast, the inertial-gravitational reference provided by the vestibular system is unaffected by changes in external surface conditions (Nashner et al., 1982).

**Somatosensory System**

Somatosensory inputs are provided by a number of different types of mechanoreceptors. These sensors are said to be "somatosensory" because they are located in the somatic tissues of the body (i.e. skin, muscles, ligaments, joints and
fascia). They are "mechanoreceptors" in that they respond to mechanical deformation of the receptor or adjacent cells (Maki, 1987).

Cutaneous touch receptors and subcutaneous pressure receptors located on the plantar aspects of the feet provide information about the contact forces between the feet and the supporting surface. Deep pressure receptors located in the tissues of the feet and legs respond to pressure changes resulting primarily from muscle contraction. Relatively little is known about the role that the touch and pressure receptors play in postural control (Maki, 1987).

Proprioception is provided by sensory nerve endings within the joints, throughout the muscles and on the skin (both superficial and deep). Several different types of sensory endings (both encapsulated and unencapsulated) are located in the joint capsule and in the surrounding ligaments. These endings are actually tension receptors, but provide information about the angular displacement of the joint and the rate of displacement (Maki, 1987).

Further proprioceptive information is furnished by spindle receptors which are interspersed throughout the muscles. The muscle spindles respond to changes in muscle length and to rate of lengthening. Each spindle is composed of several small intrafusal muscle fibres which lie in parallel with the extrafusal fibres of the muscle. The
central regions of the intrafusal fibres are innervated by sensory nerves which are excited when the muscle is stretched. The stretch (monosynaptic) reflex is a direct effect of stimulating the muscle spindle. Efferent gamma motor neurons, which innervate the non-receptor end regions of the intrafusal fibres, act to control the threshold and sensitivity of the spindle (Maki, 1987). Active muscle tension is sensed by the Golgi tendon organs. Each organ is located in series with a small number of extrafusal muscle fibres. Although they do respond to passive muscle stretch, the tendon organs are far more sensitive to active muscle contraction (Maki, 1987).

A long latency reflex is also mediated by somatosensors, but is thought to involve higher level neural processing (Brooks, 1983). This reflex is functionally much stronger than the monosynaptic reflexes, and occur in the ankle flexors and extensors. Nashner et al. (1982) suggest that these longer latency reflexes are the dominant stabilizing influence. The rotation of the ankles is the most probable stimulus of the so-called functional stretch reflex (long loop reflex, long latency reflex), that occurs in many persons and seems to be the first useful phase of activity in the leg muscles after a change in erect posture (Nashner, 1981). Whether this is due to afferent inputs from mechanoreceptors in the ankle joints and soles of the feet, or from the spindles of the leg muscles, or from some other
origin is unclear (Era & Heikkinen, 1985).

Several researchers have attempted to measure the importance the proprioceptive system by inducing ischemia (Diener, Dichgans, Guschlbauer, & Mau, 1984), by measuring sway of amputees (Holliday, Dornan, & Fernie, 1978), or by altering the support surface structure upon which the subject stood (Era & Heikkinen, 1985; Nashner et al., 1982; Pyykko et al., 1988). The studies suggested that one of the major sensory factors in deficiencies of postural control may be due to the lack of adequate response to information from joint and possibly muscle receptors. How this affects postural stability is currently being researched.

In the parkinsonian population, the long-latency reflex is impaired (Tatton & Lee, 1975). If the long-latency reflex is the most dominant influence on posture, as Nashner et al (1982) suggested, it is not surprising that deficits in postural stability are predominant in individuals with PD.

**Vestibular System**

The vestibular system is a purely exproprioceptive sense that measures the orientation and the motions of the head with respect to the inertial and the gravitational fields. Because the vestibular system is not subject to external perturbations, it is most useful in recognizing other sensory errors, as when motion of the supporting surface
perturbs the somatosensory inputs. The labyrinthine organs of the vestibular system are located in each inner ear. Each organ comprises a system of membranous sacs and tubes that lie within the temporal bone. The sacs and tubes are filled with fluid (endolymph), as is the space between the membranes and the bone (perilymph) (Maki, 1987).

Each labyrinth has two sacs (the saccculus and the utriculus). Each sac contains a patch of sensory hair cells (the macula) which are embedded in the otolith, a membrane containing numerous calcium carbonate crystals (the otoconia). Displacements of the otoconia, as a result of gravitational or inertial forces, bend and excite the underlying hair cells. Thus, the otoliths function as accelerometers, sensitive to both gravitational force and linear acceleration (Maki, 1987).

Each labyrinth has three semicircular canals. At one end of each canal, there is a patch of sensory hair cells (the crista ampullaris) that projects into a membrane (the cupula) that closes off the end of the canal. Angular accelerations of the head produce movements of the endolymph fluid within the canal, and the resulting deflection of the cupula stimulates the hair cells (Maki, 1987).

The vestibular system also helps to keep images stable on the retina by driving eye movements, called nystagmus, by alternating rapid and slow eye movements (Diener, Dichgans, Guschlbauer & Bacher, 1986).
Martin (1967) suggested that input from the support surface provides the bulk of stability when stance is supported by a fixed, level surface. However he concluded that vestibular inputs are essential for balance whenever support and/or visual surfaces are irregular or in motion.

Diener et al. (1986) performed an experiment to assess postural stabilization in altered vestibular and visual conditions. While standing on a supporting surface, two movement conditions were administered: fast transient and sinusoidal disturbances. Static vestibular input was modified by moving the head with the eyes closed. Visual inputs were also varied by applying stroboscopic illumination, or by moving stripe patterns up or down, as well as by eye-closure. The authors concluded that there were at least two different modes of postural stabilization. One mode subserved only fast corrections and acted through reflex-like responses that were not immediately modified by and possibly not even accessible to inputs from the visual or the vestibular system. These were organized in advance according to prior experience. Within this system there was a certain amount of flexibility in both the time and amplitude domains. Postural stabilization was performed normally as long as at least two of the three afferent systems contained congruent information. (Diener, Bootz, Dichgans, & Bruzek, 1983).

Another mode, a continuous mode was highly dependent
on vestibular, visual and proprioceptive feedback. It subserved the compensation of low frequency disturbances on the one side and of continuous displacement on the other side. This mode was more susceptible to adaptive changes than the previous one (Diener et al., 1986).

Nashner et al. (1981) observed that patients with vestibular impairments were unable to suppress the influence of visual and proprioceptive inputs appropriately whenever motions of the external surface disturbed the orientation information provided by these inputs.

Nashner et al. (1982) performed a study to compare the equilibrium control strategies of normal subjects and subjects with vestibular deficits. All subjects were evaluated over a variety of altered support surfaces and visual environments. The authors observed that patients with mild vestibular impairments performed well in the absence of useful support surface and visual inputs, yet these same patients responded inappropriately and lost balance when exposed to conflicting support surface and visual stimuli. Normal subjects performed equally well when deprived of support surface and visual stimuli or exposed to conflicting stimuli, suggesting that conflicting orientation inputs (proprioceptive and visual systems) are suppressed in favour of those congruent with the internal reference (vestibular system). Diener et al (1986) suggested that the semicircular channels sense best the rate
of postural sway above 0.1 Hz, and the otoliths sense sway below this frequency. Kilbreath (1986) observed parkinsonian subjects to manifest increased sway below 0.5 Hz. If this is the case, one should question the ability of the PD individual to process vestibular input appropriately.

Hawken et al. (1990) studied the effect of manipulating the visual and proprioceptive inputs to the postural control system, in individuals with PD. Nine patients had a Hoehn and Yahr rating of 2 (HY2) and 11 a rating of 3 (HY3). The authors observed that loss of visual and proprioceptive inputs resulted in a loss of balance for 85% of the HY3 patients, but for only 15% of the controls. In this instance subjects relied mainly on vestibular information, so the markedly worse performance of the HY3 group could therefore reflect either peripheral vestibular deficits or difficulties in the central integration of vestibular information.

Visual System

Vision is the most complex of the three modalities because it includes proprioceptive, ex proprioceptive and exteroceptive information (Nashner, 1981). Although it is possible to maintain an upright position with eyes closed, under natural and experimental laboratory conditions, lack of vision may have a large destabilizing effect on posture. This
is observed particularly when visually perceived motion does not adequately correspond to the actual body shift sensed by the vestibular or proprioceptive systems. In fact the visual contribution to postural regulation becomes dominant in patients with defects of the vestibular or somatosensory systems particularly when performing more demanding balancing tasks. Gantchev (1980) indicated that generalized information from visual feedback about body oscillation, as well as information from the different parameters of the oscillations, also played a stabilizing role in attaining and maintaining a vertical posture of the body.

One of the most common measures of postural control, both clinically and behaviourally is the Romberg test of quiet standing (Njokiktjien & Van Parys, 1976; Starkes et al., 1992). This subjective measure was and is used clinically in neurological assessments with comparisons between eyes open and eyes closed conditions (Starkes et al., 1992). The Romberg Quotient (RQ) (ratio of mean extent of sway, eyes closed, to the mean extent of sway, eyes open) is the quantitative estimate of performance on Romberg's test. The formula for RQ is:

$$ RQ = \frac{\text{mean sway with eyes closed}}{\text{mean sway with eyes open}} \times 100\% $$

RQ values for adults are generally greater than 100% indicating that vision improves postural stability (Riach &
Hayes, 1987; Starkes & Riach, 1990). There is however, some concern regarding the accuracy of this measure as a 'true' reflection of postural sway, as prior knowledge of the elimination of vision might trigger other postural strategies (Hamann, Vidal, Sterkers, & Berthoz, 1979).

Pyykko et al. (1980) suggested that elderly individuals (80+), controlled their posture almost entirely by visual influx. When compared to the eyes open condition, closure of the eyes increased the sway velocity by a factor of two. In this sense, vision was a more stabilizing factor for normal elderly individuals.

White, Post, and Leibowitz (1980) observed that postural sway became enlarged with the increase in the frequency of saccades from 3.5 Hz to 4.5 Hz. Body sway also depended on whether movement of the retinal image was voluntarily or externally produced. During externally produced conditions, there was an increase in postural sway. In contrast, there was a decrease in postural sway during voluntarily produced movements.

Body sway in normal human subjects in upright standing has been shown to decrease with periodic saccades (Iwase, Uchida, Hashimoto, Suzuki, Takegami, & Yamamoto, 1979). This decrease was also observed during voluntary rapid eye movements in complete darkness and during eye closure. This indicated that visual information was not primarily concerned
with decreasing postural sway (Uchida et al., 1979).

Based on the above information, "normal" individuals can decrease their postural sway by means of their intact visual system. In contrast, studies have revealed the existence of visual impairments in individuals with PD. Bodis-Wollner and Onofri (1986) have suggested that visual alterations in PD could be caused either by the abnormal functioning of the basal ganglia in sensory motor integration or by the malfunction of dopaminergic systems at different levels of the visual pathways.

Starkes et al. (1992) observed that parkinsonian subjects routinely showed degraded performance when they attempted to stand or move with their eyes closed. Accommodation convergence is impaired from the early stages of PD in the majority of individuals (Selby, 1990). This causes defects in near vision, which may be aggravated by treatment with anticholinergic drugs.

The ability to execute smooth slow pursuit movements is also impaired in individuals with PD (Shibasaki, Tsuji, & Kuroiva, 1979). Tervainen and Calne (1980) observed that fast voluntary saccades are executed in a series of steps of small amplitude (multiple step saccades) which bring the eyes to the desired position more slowly than normal individuals. If, as Uchida et al. (1979) suggest, that it is the execution of saccades itself that is the origin of sway stabilization, then
it would not be unlikely to observe increases in postural sway in parkinsonian individuals.

Postural Sway

"Sway" is the constant small corrective deviation from the vertical when standing upright (Sheldon 1963) and it is often clinically assessed by "Romberg's test" (Odenrick & Sandstedt, 1984). In the past analyses of changes in centre of gravity (COG) were studied to gauge postural stability (Murray, Seireg & Scholz, 1967; Murray et al., 1975). More recently, studies have monitored the moment to moment fluctuations in COP to provide an estimate of postural stability (Goldie et al., 1989; Hattori et al., in press; Kilbreath, 1986; Riach & Starkes, 1989; Starkes et al., 1992).

Gravity is the most consistent force encountered by the human body and behaves in a predictable and describable manner. It is a vector quantity and can therefore be fully described by point of application, action line/direction and magnitude. While gravity acts at all points on an object or segment of an object, its point of application is given as the centre of gravity (COG) of that object or segment. The COG is a hypothetical point at which all mass would appear to be concentrated and is the point at which the force of gravity would appear to act (Murray et al., 1967; Murray et al., 1975).
The COP, on the other hand, is the centre of the distribution of the total force applied to the supporting surface. The total vertical force applied to the platform fluctuates slightly above and below body weight because it includes both body weight and the inertial effects of the slightest movement of the body which occur even when one attempts to stand motionless. There is a reciprocal relationship between COP and COG. The movement of the COP therefore, varies according to the movement of the COG and distribution of muscle forces required to control or produce the movement. (Murray et al., 1975)

To be functional, any postural control mechanism must dampen body sway to ensure proper orientation of the centre of gravity within its base of support. Several investigators have shown that sensory input can serve to reinforce the postural response or modify it (Diener et al., 1986; Iwase et al., 1979; Lucy & Hayes, 1985; Nashner et al., 1982; Njiookikjien & De Rijke, 1972; Uchida et al., 1979). The notion of enhancing sensory flow to the central nervous system forms the basis of many therapeutic techniques. How sensory information controls balance behaviour is not completely clear.

Quantitative measurement of standing balance has never been a routine clinical procedure. The clinical problem has been to develop a convenient method of measuring sway
which is valid, reliable, and readily accessible.

One approach to the assessment of postural sway is to measure the spontaneous postural fluctuations of subjects as they stand quietly on a rigid surface. The commonly measured variable using this format, is the displacement of the COP on the feet (Maki, 1987). Spontaneous a-p and lat fluctuations of COP measured during these tests can be quantified by means of amplitude-based and frequency-based measures (Maki, Holliday, & Fernie, 1990). The amplitude-based measures include: (a) root-mean-square COP displacement relative to the mean COP location (RMS); (b) peak-to-peak range of COP displacement (range); and (c) average speed of COP displacement (velocity) (Maki et al., 1990).

Frequency-based measures consist primarily of the power spectral analysis, with selective Fast Fourier Transformations. These procedures are incorporated to determine the frequency at which the sway energy is concentrated, as well as the degree to which the sway energy "spreads out" to include other frequencies (Maki et al., 1990). Although the measurement of postural control with force platform systems appears to have gained wide acceptance, issues such as validity, retest reliability and sensitivity of different measures have not been addressed (Goldie et al., 1989).

There are several advantages to using the force
platform. First it is a quick, easy and relatively painless test to perform. Secondly, it is an objective measure and hence reproducible. Finally, it is very sensitive to small changes in postural sway, and hence postural stability (Kilbreath, 1986).

Two of the limitations of force platform analysis are the lack of standardized equipment, and variable methodology that exists between researchers. Standardization of variables such as foot, arm positions, head position, and sampling time, limit direct comparisons with other researchers (Goldie et al., 1989; Hattori et al., in press; Kilbreath, 1986; Okubo, Watanabe, Takeya, & Baron, 1979; LeClair & Hlach, 1990). This is slowly being rectified, and attempts to collect normative data have been initiated (Fernie & Holliday, 1978; Hattori et al., in press; Hayes et al., 1985; Lucy & Hayes, 1985; Starkes et al., 1992). Nevertheless methodological differences still limit gross comparisons between groups.

Goldie et al. (1989) conducted a study to evaluate the reliability and validity of force platform measures. They measured three orthogonal force signals (Fx, Fy, Fz) and two horizontal centre of pressure signals. The correlation between these five indices derived from the force platform, showed that the relationship was generally weak. Force measures were more sensitive than COP measures in
discriminating changes in postural stability, which resulted from alteration in base of support. LeClair and Riach (1990) performed a study comparing six postural stability outcome measures. These parameters were: COP in both the a-p (Cpy) and lat (Cpx) directions, sway velocity, and variability of ground reaction forces in the a-p (Fy), lat (Fx) and vertical (Fz) planes. All parameters were measured under two visual conditions, for two stance positions, and for five differing test durations (LeClair & Riach, 1990). The authors indicated that each parameter demonstrated differences for each stance position and test duration, but neither to the same extent, nor in the same direction. Based on these results, the authors suggested that the conclusion of Goldie et al. (1989) as to the superiority of the force parameters could not be supported.

Postural Sway and Parkinson's Disease

Purdon Martin (1967) identified a number of disorders of posture and locomotion in postencephalitic parkinsonism, which were also typical of idiopathic PD. These included disorders of postural fixation, disorders of equilibrium, disorders of righting mechanisms, and disorders of gait. Martin (1967), concluded that postural reflexes which sustained the parts of the body in relation to each other and coordinated them in such a way as to maintain equilibrium,
were dependent upon the basal ganglia. Moreover, associated with every voluntary movement which significantly changed the shape of the body was a postural adjustment which had the effect of protecting and maintaining equilibrium.

The individual with advanced PD tends to stand in a stooped posture. The neck and trunk are flexed, the arms are bent at the elbow and the legs at the knees. The hands tend to flex at the wrist and metacarpophalangeal joints with the fingers extended, while the feet are slightly inverted and plantar flexed. This position clearly places these individuals in a posturally disadvantaged position.

The force platform has been utilized to estimate postural stability for many medical conditions (Fernie & Holliday, 1978; Fernie et al., 1982; Hattori et al., in press; Lucy & Hayes, 1982; Starkes et al., 1992; Watanabe et al., 1980). It has identified differences within parkinsonians, as well as between persons with PD and "normals", and other neurological pathologies (Cernacek et al., 1973; Folkerts & Nijoki, 1971; Gregoric & Lavric, 1977; Hawken et al., 1990; Kilbreath, 1986; Nijoki & de Rijke, 1972; Starkes et al., 1992; Tokita et al., 1976; Watanabe et al., 1980). Although these differences have been observed, high inter and intra subject variability was evident (Gregoric & Lavric, 1977; Hawken et al., 1990; Tokita et al., 1976; Starkes et al., 1992; Watanabe et al., 1980).
Folkerts and Njiokiktjien (1972) performed a study to determine the effectiveness of L-dopa on postural control. Stabilographic measures of the frequency, average amplitude and position of the line of gravity in both directions (a-p and lat), were calculated. It was observed that L-dopa increased postural sway in those with "normal stability", and stabilized those people who were "abnormally unstable" before treatment. The authors also observed that the COP in PD patients was located more lateral (to the left) and posterior than "normals".

Njiokiktjien and De Rijke (1972) compared healthy young (<40 years) and health older individuals (<85 years), to a number of neurological subjects, including PD. Two visual conditions were examined: eyes open (EO) and eyes closed (EC). The authors observed that in the EO condition, healthy young subjects swayed .81 cm, healthy older subjects swayed 1.5 cm and parkinsonian subjects swayed 1.92 cm. In the EC condition, the median values increased to 1.25 cm for the healthy young subjects, 1.87 cm for the healthy older subjects, and 2.55 cm for the parkinsonian subjects.

Cernacek et al. (1973) performed stabilographic recordings of the body axis oscillations in ten parkinsonian patients receiving L-dopa treatment. Five subjects were hypokinetic, while the remaining five subjects exhibited marked tremor. Three types of superimposed oscillations were
observed. Their main frequencies were 0.03 Hz, 0.12 Hz and 0.5 Hz, respectively. The authors observed that the administration of L-dopa increased the amplitude of postural oscillations in hypokinetiC parkinsonians, and decreased the amplitude of postural oscillations in subjects with tremor.

Tokita et al (1976) examined postural sway of both normal subjects and clinical cases with equilibrium disturbances. The sway in cases with PD was characterized by a centripetal sway overlapped with notched waves having two periodic components of about 0.25 Hz and 0.6 Hz. This is similar to the range observed by Cernacek et al. (1973).

Gregoric and Lavric (1977) studied 17 healthy subjects and 34 parkinsonian subjects to determine the postural reaction to the vibratory stimulation of the tendon's mechanoreceptors. By means of the computer, the average displacement of the projections of the COG in all directions was recorded. The authors discovered that in PD patients, the position of the centre of gravity during the Romberg test was on average situated more backwards and more lateral (than a-p) from the centre of the area of support, than in healthy subjects. The amplitude of sway in both directions was on average higher in parkinsonian patients than in healthy age-matched controls. The average area of displacement was also larger in the parkinsonian subjects than the controls, but there was extensive individual variation among the
parkinsonian subjects. Watanabe et al. (1980) compared sway velocity of seven individuals with PD to 16 age-matched controls. Unlike other investigators, the authors observed area of sway to be smaller in the PD subjects than the controls. The authors attributed this difference to the influence of rigidity on postural sway.

Kilbreath (1986) conducted a study to determine the extent and nature of postural instability evident in patients with PD, as well as to determine the relationship between specified characteristics of instability and clinical features of the disease. Thirty seven parkinsonian subjects and age-matched controls were recruited into the study. The author observed that postural sway was clearly exaggerated in subjects with PD. Exaggerated sway was apparent in both eyes open and eyes closed condition. The extent of sway and mean lateral position of the COP increased with the stages of functional disability. The effect of eye closure was greater on a-p rather than lat sway. The severity of hypokinesia, rigidity, and tremor were not correlated with the extent of sway.

Recall the study conducted by Hawken et al. (1990). The authors examined the effect of manipulating visual and proprioceptive inputs to the postural control system. Twenty male subjects with idiopathic PD and 20 age-matched normal control subjects were recruited into the study. Nine subjects
had a Hoehn and Yahr rating of 2 (HY2), and 11 a rating of 3 (HY3). Three visual conditions were tested: eyes open, eyes closed and sway feedback. The three visual conditions were tested once in a quiet standing condition, and once with the ankle stabilized and a hydraulic servo rotating the platform to follow the subject's sway. For each condition, the amplitude of sway was calculated. Comparison of the group means showed that the mean sway for the HY3 group was higher than that for the HY2 group and controls, p<.05. Loss of visual and proprioceptive inputs resulted in a loss of balance for 85% of the HY3 group, but only 15% of the controls. Many HY3 subjects showed little effect of eye closure in the quiet standing condition, but no conclusions about the effect of vision could be drawn because the responses showed considerable variation.

Starkes et al. (1992) conducted a case study to examine how sway velocity changed over time. Sway velocity was recorded for the subject during eight sessions from 9:30 a.m. to 4:30 p.m., for two visual conditions: eye open (EO) and eyes closed (EC). The subject was asked to lean in four directions. The authors observed that the subject routinely showed degraded performance when he attempted to stand or move with his eyes closed. In fact it was also observed on isolated occasions, that the subject would lean to one direction and not be able to recover. The authors concluded
that time of day, time of last dose of medication, drug interaction with meals, exercise, stress and confidence in abilities, all influenced postural performance. There was also extremely high variability within this subject's performance.

PURPOSE

This study was undertaken to address the following issues:

1. Whether there are individual and/or group correlations between the force platform and the clinical assessment of postural stability in PD patients.
2. To plot the individual diurnal changes in postural sway characteristics, over an eight-hour time period of PD patients.
3. To plot day to day changes in individual postural sway characteristics of PD patients.
4. Whether there is a difference in the postural sway characteristics of individual parkinsonians, with and without vision.
5. Whether there is a difference in the postural sway characteristics of the same individuals when using:
   (i) Sinemet
   (ii) Deprenyl.
HYPOTHESES

The following hypotheses were postulated:

1. Significant correlations will exist between the clinical assessment of postural stability (SPAH) and the force platform.
2. Postural sway characteristics of individual parkinsonians will be significantly different with and without vision.
3. Significant differences in postural sway characteristics will be observed between the Sinemet and Deprenyl medication regimen.
4. No significant difference in postural sway characteristics will be observed between the test days.

METHODOLOGY

Three male subjects, each diagnosed with idiopathic PD, were recruited into this study. Table 1 provides a summary of the demographics of the subject population.

Insert Table 1 about here

Subjects were selected using several inclusion and exclusion criteria:
Inclusion Criteria

1. A diagnosis of Parkinson's disease, as determined by a Neurologist.

2. Currently taking:
   (a) Sinemet and Deprenyl; or
   (b) Sinemet and Deprenyl in combination with other anti-parkinsonian medication.

Exclusion Criteria

1. Subjects with evidence of memory loss, confusion or dementia, vestibular impairment;

2. Patients with other Neurological disorders:

3. Patients with severe gastrointestinal disorders;

4. Non-ambulatory patients who were unable to stand, and maintain standing independently for at least thirty seconds.

Force Platform Assessment

Each subject stood on a stable force platform (AMTI OR6-5-1). The force platform measured three linear forces: $F_x$ (lateral), $F_y$ (anterior-posterior), and $F_z$ (vertical). The forces were measured along with three moment components about the $x$, $y$, and $z$ axes, $(M_x, M_y, \text{ and } M_z)$. The forces and moments were measured by foil strain gauges attached to proprietary load cells at the four corners of the platform. The force and moment of force signals were conditioned and amplified prior to A/D conversion at a sampling rate of 10 HZ. The digital
signal was processed by a North Star Horizon Computer using the Computer Automated Stability Analysis program. The subjects' magnitude of sway in the anterior-posterior (a-p) plane, as well as the lateral plane (lat), were assessed. Dependent measures included the standard deviations of the coordinates of the centre of pressure about the mean position—root mean square (RMS)—in the a-p and lat directions, the mean velocity of sway, and the area of sway.

These dependent measures were evaluated in a "Quiet Standing" condition, once with the eyes closed, followed by a trial with eyes open. Each subject stood facing forward with their hands down by their sides, and feet as close together as possible. This position was maintained for thirty seconds during each trial. Two people stood on either side of the subject during each trial in order to protect the individual from losing his balance during the trials. If a patient fell off the platform, that particular trial was discontinued, and a new trial under the same condition was repeated. If a subject was unable to perform the quantitative assessment, the scores were obtained by performing a Yates correction based on the remaining data. This particular procedure provided a "legal" underestimation of performance for the missing tasks. These evaluative procedures were carried out ten times over the course of an eight hour day. This protocol was executed for all subjects on each of four days. Each subject was
tested two days while they were taking Sinemet, and two days
while on the Deprenyl regimen. It should also be noted that
testing times and medication times were recorded for each
day’s testing session.

Clinical Assessment

At two periods of each test day, each patient was
evaluated utilizing the postural stability component of the
Sears Parkinson’s Assessment Form (SPAF), outlined in Appendix
I. The following tasks were evaluated: optical righting,
labyrinthine righting, dynamic sitting, dynamic sitting with
displacement (protective arm), kneel standing, half knee
standing, unsupported stance, dynamic standing, dynamic
standing with displacement (protective leg), and one legged
stance. Each of these tasks were performed in eyes open and
eyes closed conditions. Subjects attained a score of '0', if
they were able to perform the task, '1', if they were able to
perform the task with minimal assistance, or '2'. If they were
unable to complete the task, for a total tally out of 56.
Each subject was tested a total of four times during each of
the two medication regimens.
Data Analysis

Pearson product moment correlational matrices for both individual and group data were calculated, in order to examine the relationship between the various dependent variables. The scores obtained on the SPAF were also included in the correlational analysis. The correlations between the force platform dependent measures and SPAF were used to assess the relationship between a quantitative and qualitative assessment technique.

For each dependent measure, individual data plots were obtained for all subjects, for each condition of days, medication and vision. Each plot yielded information on the performance of ten individual sessions, obtained over an eight-hour period using a time series design. With each subject as his own control, data were plotted for the ten sessions that were conducted on each test day. Data were plotted for all dependent variables, for each visual condition, and testing session. Ninety-five per cent confidence bandwidths were then applied to each of the above graphs to illustrate behaviour, under altered visual conditions. For each graph, the time and type of each medication was indicated to illustrate performance throughout the medication cycle. Individual graphic representations, provided a means of identifying specific testing parameters. They also illustrated the extent of individual variability of
performance that may be observed over a relatively long period throughout one or even a number of days. Individual plots are included in Appendix II.

The analysis determined that the data were quite heterogeneous. Theoretically non-parametric statistics should have been employed. However parametric statistics were applied to the present data, as non-parametric tests have not yet been developed to handle the complex interactions between variables.

For each individual case analysis, a three way analysis of variance with repeated measures (vision X Medication X Day) was applied to determine the influence of the independent variables (visual condition, medication and day), on the dependent variables. Significant effects were further analyzed using Tukey A post-hoc tests.

The quantitative parameters utilized in this study were: RMS in the a-p and lat directions, area of sway and mean velocity of sway. These measures were chosen to answer different questions about postural stability. Fluctuations in COP in the parkinsonian group are highly variable (Gregoric & Lavric, 1975; Hawken et al., 1990; Kilbreath, 1986; Starkes et al., 1991; Tokita et al., 1976; Watanabe et al., 1980). Hence, root mean square measures of the coordinates of COP were incorporated, as they are least affected by variance. The area of sway was measured by summat1ng the triangulated
areas sampled throughout the testing sessions. The mean velocity of sway has been found to be a more reliable measure of postural sway than the range (amplitude) of movement in either the a-p or lateral directions (Fernie et al., 1978). Consequently, this dependent measure was also incorporated into the present study.

RESULTS

Issue #1: Correlational Analysis of the Clinical Assessment and the Force Platform Assessment

Recall that the qualitative evaluation was performed twice in each day. A correlation analysis was used to compare qualitative (SPAF) data with quantitative (force platform) data. Scores were obtained from the force platform for two consecutive assessment sessions. The two sessions selected occurred at times adjacent to the qualitative testing. As the first qualitative analysis was conducted at 1.25 p.m., the quantitative measures for the 1.20 p.m. and 1.40 p.m. sessions were included in the correlational analysis. Similarly, for the 3.05 p.m. qualitative analysis, the postural sway profiles for the 3.00 p.m. and 4.00 p.m. assessments, were included in the correlational analysis. These two independent clinical assessment sessions were designated "Time 1" for the 1.25 p.m. evaluation session, and "Time 2" for the 3.05 p.m. testing session. Pearson product moment correlations were calculated.
for each of the force platform measures, as well as for the SPAF. The two qualitative analyses were correlated with each of the ten testing sessions for that day.

Table 2 indicates that two dependent measures, area of sway and velocity of sway, were correlated with SPAF results during both the Sinemet and Deprenyl trials.

The correlations for area of sway and SPAF were: (a) $r = .45$, $p < .05$, while taking Sinemet; and (b) $r = .60$, $p < .05$, while taking Deprenyl. The correlations for velocity of sway and SPAF were: (a) $r = .51$, $p < .05$, while taking Sinemet, and (b) $r = .52$, $p < .05$, while taking Deprenyl.

As indicated in Table 3, significant correlations were also observed during the individual correlational analysis.

During the first testing session of the Sinemet medication condition, the SPAF was negatively correlated with RMS lat, $r = - .55$, $p < .05$. During the second testing session of the Deprenyl medication condition, the SPAF was correlated with RMS lat, $r = .62$, $p < .05$. 

Subject FR
Subject JA

During the first testing session of the Sinemet condition, the SPAF was negatively correlated with RMS lat, \( r = -0.68, p < 0.05 \). During the second testing session of the Deprenyl medication regimen, the SPAF was correlated with RMS lat \( r = 0.56, p < 0.05 \), area of sway, \( r = 0.68, p < 0.05 \) and velocity of sway, \( r = 0.54, p < 0.05 \).

Subject SK

During the first testing session of the Sinemet medication regimen, the SPAF was negatively correlated with RMS lat, \( r = -0.87, p < 0.05 \), and area of sway, \( r = -0.53, p < 0.05 \). During the second testing session of the Sinemet condition, the SPAF was negatively correlated with RMS a-p, \( r = -0.65, p < 0.05 \), and area of sway, \( r = -0.52 \). During the first testing session of the Deprenyl medication regimen, the SPAF was correlated with RMS lat, \( r = 0.69, p < 0.05 \). During the second testing session of the Deprenyl condition, the SPAF was correlated with RMS a-p, \( r = 0.55, p < 0.05 \).

Issue # 2: Individual Diurnal Changes in Postural Sway

Over an Eight Hour Day

A novel method of evaluating changes in each patient over the course of a day, is to simply plot the person's performance over that day. Then with the patient as his/her
own control, confidence intervals are plotted around the patient's own performance. Confidence intervals demonstrate with ninety-five percent probability, which times of the day are significantly better or worse for the patient. Once the confidence intervals are drawn on the subject's graph, one simply examines which times fall outside the bandwidths. Confidence interval data for each subject are included in Appendix II.

Since postural sway has been shown to be highly unstable in the parkinsonian population (Gregoric & Lavric, 1977; Hawken et al., 1990; Kilbreath, 1986; Starkes, 1991; Tokita et al., 1976; Watanabe et al., 1980), the data were further analyzed with respect to variability. The coefficient of variation (COV) was established for each subject. The coefficient of variation is the standard deviation divided by the mean (performance). High variability is characterized by high coefficient values. A summary table for the coefficient of variation for subject FR is provided in Table 4. Summary tables for the coefficient of variation for subjects JA and SR have been provided in Appendix III.

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Insert Table 4 about here

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For the purposes of this discussion, a case analysis of subject FR will be featured, to illustrate the concepts of
For the first and second test days of the Deprenyl medication regimen, the subject was unable to complete some of the quantitative assessments. On the first test day, the subject was unable to complete the first three testing sessions of the EC condition, as well as the first two testing sessions of the EO condition. On the second test day, the subject was unable to complete the ninth evaluative session in both the EO and EC conditions. In order to deal with these missing entries a Yates correction was applied to the remaining data. This statistic provided an underestimation of the missing values. These estimates were not placed on individual data plots, in order to highlight the clinical importance of the subject’s inability to perform the tasks. Had the subject been able to complete the tasks, the “true” scores would have been extremely high for all of the dependent measures. This clinical significance could not be reflected with the scores obtained by the Yates correction statistic.

Lateral Sway

Sinemet Day One.

The confidence bandwidths for the EO condition were narrower than the EC condition. This indicates that performance was less variable during the EO visual condition.
Coefficient of variation values also support these results. As Table 4 illustrates: (a) COV = .21 for the EC condition, and (b) COV = .30 for the EC condition respectively. Overlapping confidence intervals were also apparent. This suggests that performance may not be unduly altered by changing the visual condition. This premise was not supported. As Table 5 illustrates, RQ was greater than 100%, signifying that vision improved postural stability.

Insert Table 5 about here

At 9:15 a.m. at 12:45 p.m. and 2:10 p.m. respectively, postural sway characteristics were also improved during both visual conditions.

**Sinemet Day Two.**

Extreme variations in performance were observed, particularly during the EO condition. This is documented in Table 4, which indicates: (a) COV = .28 for the EO condition, and (b) COV = .12 for the EC condition respectively. This was also supported by a low RQ (RQ = <100 %), which suggests that vision was destabilizing for this subject.

During the EO condition, at 10:45 a.m., a significant increase in postural sway was observed. Subsequent to this at 1:40 p.m., a significant decrease in postural sway was observed. During both visual conditions, between
approximately 12:45 p.m. and 1:20 p.m. performance appeared to improve.

Deprenyl Day One.

The confidence interval for the EC condition was smaller than the EO condition. This indicates that less variability was evident in the EC condition, or that vision was a destabilizing factor in postural stability. Coefficient of variance values support this belief: (a) COV = .37 for the EO condition, and (b) COV = .19 for the EC condition. Romberg quotient was slightly greater than 100% (RQ = 101%). This indicates that vision was irrelevant in the EO condition.

Overlapping confidence bandwidths were again evident. This suggests that performance may not have been inordinately impaired by altering visual conditions. The RQ value (RQ = 101%) supports this hypothesis.

At 12:45 p.m and 1:20 p.m. during the EC condition, postural sway improved. No significant improvements in postural sway were observed in the EO condition.

Deprenyl Day Two.

Variability was determined to be greater in the EO condition, than the EC condition: (a) COV = .29 for the EO condition, and (b) COV = .20 for the EC condition. Romberg quotient was a little larger than 100% (RQ = 113%). Although there was greater variability in the EO condition, vision improved postural stability.
At approximately 11:30 a.m., during both visual conditions, performance was significantly better than at any other time in the day.

**Anterior-Posterior Sway**

**Sinemet Day One.**

Confidence intervals for the EO condition were larger than the EC condition. Variability was also considerably larger in the EO condition than the EC condition: (a) COV = .31, for the EO condition and (b) COV = .09 for the EC condition.

Overlapping confidence intervals were also evident on this day. Romberg quotient was only 103% demonstrating that vision may not have been completely stabilizing in the EO condition.

At 9:15 a.m. FR displayed improved postural sway characteristics during both visual conditions. Improved performance was also observed at 11:30 a.m and 3:05 p.m. during the EO condition.

**Sinemet Day Two.**

Confidence intervals for the EC condition were a little bigger than the EO condition. Greater fluctuations in performance during the EC condition was also noted by COV values: (a) COV = .22, for the EO condition, and (b) COV = .27 for the EC condition. Romberg quotient was 107% indicating that vision improved postural stability.
At 9:15 a.m. during the EO condition, and at 2:10 p.m. during both visual conditions, episodes of improved postural sway characteristics were observed.

Deprenyl Day One.

Confidence bandwidths for the EC condition were considerably larger than the EO condition. A high RQ value was observed for this dependent measure. In this instance, vision was an extremely stabilizing factor for postural stability. Variability was also greater in the EC condition than the EO condition: COV = .38, for the EO condition and (b) COV = .44 for the EC condition.

At 11:30 a.m. during the EC condition, and at 3:05 p.m. during the EO condition, improved performance was noted.

Deprenyl Day Two.

Confidence intervals overlapped for this test day. This suggests that similar performance might be evidenced in either visual conditions. However the RQ value was greater than 100% indicating that on average vision improved postural stability. Coefficient of variance values indicated that variability was quite similar for both visual conditions: (a) COV = .22 for the EO condition, and (b) COV = .26 for the EC condition.

Little consistency in performance during the EO condition was exhibited. One occasion of improved postural sway was observed at 11:30 a.m for the EO condition however.
At 11:30 a.m., and 12:45 p.m., episodes of improved performance were demonstrated for the EC condition.

**Area of Sway**

**Sinemet Day One.**

The EO confidence bandwidths were smaller than the EC bandwidth. A high RQ value was demonstrated for this dependent measure (RQ = 167%) suggesting that vision improved postural stability. Yet greater variability was apparent in the EO condition more so than the EC condition: (a) COV = .44 for the EO condition, and (b) COV = .38 for the EC condition. Therefore, although vision improved average performance, postural sway characteristics were more variable in the EO condition than the EC condition.

This subject demonstrated relatively consistent postural sway characteristics throughout the day, until approximately 2:10 p.m. Subsequent to this, considerable deterioration of performance was observed for both visual conditions.

**Sinemet Day Two.**

Considerable variability was apparent in the EO condition: (a) COV = .35 for the EO condition, and (b) COV = .25 for the EC condition. Similar to the first day of testing a high RQ value was noted for this dependent measure. Therefore, vision improved performance, but postural sway characteristics remained variable throughout the testing day.
For each visual condition, improved performance was noted between 11:30 a.m and 2:10 p.m. An increase in postural sway was observed 3:05 p.m. followed by a decrease in postural sway at 4:00 p.m.

**Deprenyl Day One.**

Confidence bandwidths were much larger in the EC condition than the EO condition. An extremely high RQ value (RQ = 200%) indicates that vision improved postural stability considerably. Greater variability was evident in the EO condition however, as demonstrated by the COV: (a) COV = .45 for the EO condition, and (b) COV = .30 for the EC condition. Although vision improved overall postural stability, fluctuations in performance were more evident in the EO condition than the EC condition.

Little consistency in performance was observed throughout. During the EO condition, at 1:40 p.m. and 3:05 p.m. significant improvements in postural sway was observed. During the EC condition, two periods of significantly improved performance were observed at 11:30 a.m and 1:20 p.m. respectively.

**Deprenyl Day Two.**

Confidence intervals for the EO condition were noted to be much smaller than the EC condition. Romberg quotient was greater than 100% indicating that vision improved postural stability. Coefficient of variance values were quite similar
however: (a) COV = .33 for the EO condition, and (b) COV = .34 for the EC condition. This difference indicates that although vision improved performance, variability was quite similar in each visual condition.

Between 10:40 a.m and 12:45 p.m., postural sway was observed to decrease during both visual conditions. Postural sway subsequently increased after 12:45 p.m.

**Velocity of Sway**

Sinemet Day One.

This particular pattern of fluctuating performance was quite similar to area of sway. The first day of testing for area of sway approximated the first day of testing for velocity of sway. Deterioration in postural sway, as well as improvements in performance were comparable. An extended period of improved performance was observed between 10:40 a.m. and approximately 2:10 p.m. Increased postural sway was again noted at 3:05 p.m. for both the EO and EC conditions, with a subsequent decrease in postural sway around 4:00 p.m. on both testing days. Romberg quotient was quite high (RQ = 197%) indicating that vision improved postural stability.

Coefficient of variance values were higher in the EO condition than the EC condition: (a) COV = .30 for the EO condition, and (b) COV = .19 for the EC condition. This demonstrates that performance was a little more consistent during the EC condition, despite the fact that vision improved
performance.

Sinemet Day Two.

Once again, performance was similar to area of sway. Confidence intervals were smaller for the EO condition than the EC condition. Romberg quotient was greater than 100% indicating that vision improved performance. Coefficient of variance values were identical for each visual condition (COV = .24).

For both visual conditions, periods from 11:30 a.m. to 1:40 p.m. and 4:00 p.m. exhibited improved postural sway characteristics.

Deprenyl Day One.

Confidence bandwidths for the EC condition were considerably larger than the EO condition. Romberg quotient was quite high (RQ = 170%) indicating that vision improved postural sway. Little consistency in performance was noted, however the COV values were quite similar: (a) COV = .21 for the EO condition, and (b) COV = .17 for the EC condition. Although variability in performance was greater in the EO condition than the EC condition, vision improved postural sway.

Only one period of improved performance was observed in the EC condition: 11:30 a.m. During the EO condition, improved performance was observed at 11:30 a.m., 12:45 p.m. and 1:40 p.m. respectively.
Deprenyl Day Two.

Eyes open confidence intervals were smaller than the EC condition. A high RQ value indicated that vision was a stabilizing factor in postural stability. Variability was a little larger in the EC condition than the EO condition: (a) $\text{COV} = .22$ for the EO condition; and (b) $\text{COV} = .26$ for the EC condition.

A prolonged period of improved performance was observed from 10:20 a.m. until 1:40 p.m. during both visual conditions, at which time performance deteriorated.

Issue #3: Day to Day Changes in Individual Performance

Data for Day 1 and Day 2 were plotted for each dependent variable. Since there was only an eight day separation between test dates, under each specific medication, it was hypothesized that no significant difference would be observed between sessions. Tables 6, 7, and 8 illustrate the means and the significance levels for the independent variable days for subjects FR, JA and SR respectively.

Insert Table 6 about here

Insert Table 7 about here
It was quite evident that extreme variability existed within each subject's daily performance. Fluctuations in performance were observed during both the first and second testing sessions, yet none of the subjects experienced statistically significant differences in performance across days.

**Issue #4 The Influence of Vision on Postural Sway**

A 2 X 2 X 2, three way repeated measures analysis of variance was performed for days (1, 2), medication (Sinemet, Deprenyl) and visual condition (eyes open, eyes closed). This same analysis was performed for each dependent measure for each subject.

**Subject FR**

A three way interaction for visual condition x medication x days was observed for the dependent measure KMS lat, $F(1,9)=7.28$, (p<.05). The means for this effect are illustrated in Table 9.

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*Insert Table 9 about here*
From the Tukey A post hoc analysis, the mean for day 1 Sinemet eyes open were significantly different than the means for day 1 Sinemet eyes closed and day 2 Deprenyl eyes closed, respectively.

A two way interaction for visual condition X medication was observed for the dependent measure RMS a-p, F (1,9)=6.04, (p<.05). The means for this effect are illustrated in Table 10.

Insert Table 10 about here

From the Tukey A post hoc analysis, the mean for Deprenyl eyes open was significantly different than the means for Deprenyl eyes closed and Sinemet eyes closed respectively. Furthermore, a main effect of vision was observed for the dependent measures RMS a-p, area and velocity of sway; it was not observed for RMS lat (Table 11).

Insert Table 11 about here

Subject JA

A two way interaction for visual condition X days was observed for the dependent measures of RMS lat, F (1,9)=8.56, (p<.05). The means for this effect are illustrated in Table
From the Tukey A post hoc analysis, the mean for day 1 EO was significantly different than the means for day 1 EC and day 2 EO. Furthermore, the mean for day 2 EC was significantly different than the mean for day 1 EC.

A two way interaction for visual condition x medication was observed for the dependent measure velocity of sway, $F(1,9)=5.24$, ($p<.05$). The means for this effect are illustrated in Table 13.

From the Tukey A post hoc analysis, the mean for Deprenyl EO was significantly different than the means for Deprenyl EC, and Sinemet EC respectively. Furthermore, the mean for Sinemet EO was significantly different from the means for Sinemet EC and Deprenyl EC. Finally, the mean for Sinemet EC was significantly different than the mean for Deprenyl EC.

A two way interaction for visual condition x medication was observed for the dependent measure area of sway, $F(1,9)=8.56$ ($p<.05$). The means for this effect are illustrated in Table 14.
Although the F-test was significant, the Tukey A post hoc analysis failed to declare any pairwise comparison of the means significant.

Furthermore, main effects of vision were observed for several dependent measures: RMS lat, area of sway, and velocity of sway. The mean values and significance levels are contained in Table 15.

Subject SR

A three way interaction for visual condition x medication x days was observed for the dependent measure velocity of sway, $F(1,9)=6.58$, ($p<.05$). The means for this effect are illustrated in Table 16.

From the Tukey A post hoc analysis, the means for day 1 Sinemet EO and EC, day 1 Deprenyl EO and EC, day 2 Sinemet EO, and day 2 Deprenyl EO respectively, were all significantly
different than day 2 Sinemet EC and day 2 Deprenyl EC, respectively.

Furthermore, main effects for vision were observed for each dependent measures. The mean values and significance levels are contained in Table 17.

Insert Table 17 about here

Romberg Quotients

Tables 5, 18, and 19, provide a summary of the RQ values for subjects FR, JA, and SR respectively.

Insert Table 18 about here

Insert Table 19 about here

RQ values were quite variable for each subject. With very few exceptions, subjects had RQ values of over 100%, indicating that PD patients were highly dependent on vision to control sway. In the instances where RQ values were less than 100% (indicating that vision was destabilizing), one must immediately consider the high variability observed within parkinsonian individuals. These conflicting results would
indicate that sometimes vision helped the PD patients and sometimes vision made performance worse.

As was outlined in Issue #2, the analysis of vision is further strengthened by the individual data plots. In all sessions with subject FR, the width of the confidence bands were different for each dependent measure during the EO and EC conditions. In fact, there were many instances where the confidence bands for the eyes closed data, were thirty to fifty percent larger than the confidence bands for the eyes open data. This effect is readily seen for the following individual data plots: RMS lat-Sinemet days 1 and 2; RMS a-p-Deprenyl day 1 and Sinemet day 1; area of sway-Sinemet day 1 and Deprenyl day 2; velocity of sway-Deprenyl day 1 and Sinemet day 2, respectively.

Issue #5: The Effect of Medication on Postural Sway

The three way repeated measures analysis of variance of day X medication X vision indicated that there was no difference between the effects of Sinemet or Deprenyl on any postural sway characteristic for all but one subject.

Subject FR

The three way repeated measures analysis of variance indicated that there were no significant differences between the effects of Sinemet or the deprenyl medication on any of
the postural sway characteristics measured.

Subject JA

The three way repeated measures analysis of variance indicated that there was a significant difference between the effects of Sinemet and Deprenyl for several dependent measures: RMS lat, $F(1,9)=9.87$, ($p<.05$); area of sway, $F(1,9)=7.19$, ($p<.05$); and velocity of sway, $F(1,9)=11.10$, ($p<.05$) respectively. The means and significance levels are outlined in Table 20.

Insert Table 20 about here

Subject SR

The three way analysis of variance indicated that there were no significant differences between the effects of Sinemet or Deprenyl on any postural sway characteristics.

LIMITATIONS

The major limitation of the present study was the lack of control over the subjects' medications. Had the same medication regimen been applied to all three subjects, more conclusive results about the effects of medication might have been determined. The effect of this limitation is that it a)
may have either reduced or exaggerated the differences between the three subjects, or b) introduced some confounding in the correlations among postural stability and other clinical signs. This area is suspect particularly as studies have indicated that L-dopa selectively influences postural stability or just particular symptoms of PD (Cernacek et al., 1973; Folkerts & Njioiktjien, 1972; Klawans, 1986).

A small sample size of three was utilized for this study. When analyzing a sampling distribution, it is a statistical fact that as the sample size increases, variability decreases. This is reflected when comparing the standard error of the mean to the standard deviation. The standard error of the mean is the standard deviation divided by the square root of the sample size. For example, if the standard deviation is "1" and the sample size is "1", the standard error of the mean is also "1". However, if the standard deviation remains at "1" and the sample size increases by ten, the standard error of the mean would also decrease accordingly.

From a clinical perspective however, increasing the sample size may in fact highlight the differences between subjects, particularly when dealing with diseases of a highly variable nature such as PD. As these three subjects are not necessarily reflective of the parkinsonian population at large, generalization of these results across such a
heterogeneous clinical group is inappropriate.

In the clinical research environment, not only is it extremely difficult to recruit subjects from a random sample, it is also difficult to recruit subjects who can be matched for age, sex, medication and other variables. Although there were neither age-matched male control subjects, data were presented for normal elderly subjects as comparison. The statistical method of analyzing confidence bandwidths, allowed each subject to be his own control and therefore forego the necessity of either age-matched, or sex-matched control subjects. Nevertheless one can still not extrapolate to a more diverse population. Each subject was diagnosed as Stage III of the Hoehn and Yahr scale, by a Neurologist. This select sample is not indicative of the much larger and diverse parkinsonian population, thus once again generalizability is limited.

The diet of the subjects was neither monitored, nor controlled. The influence of protein on the absorption of L-dopa is currently being debated, but some researchers have indicated that a meal high in protein content, may alter the effect of L-dopa, by preventing the absorption of excessive amino acids both in the diet and medication (Rozovski & Lurie, 1990).

The emotional status of the subject while not measured, may have been a confounding variable in this study.
There were times when subjects were initially unable to perform all of the tasks, yet with encouragement and reinforcement by the researchers, tasks were completed. During the initial stages of the study during which all tasks were new, stress and frustration were noted by the clinician. How this may have affected performance is unknown but should be considered in future studies.

DISCUSSION

Issue #1: Correlational Analysis of the Clinical Assessment and the Force Platform Assessment

The main purpose of this present study was to determine the relationship between a qualitative evaluation of postural stability and a quantitative assessment in subjects with PD. The original hypothesis suggested that significant correlations would be observed between the clinical assessment of postural stability and the force platform. One of the most important findings of this study (as hypothesized), was that the SPAF was correlated with several dependent measures for both the group and individual subjects (Tables 2, 3). The SPAF correlated highly with area of sway and velocity of sway for both medication regimens, \( r > .45, p < .05 \), when using group data. A second critical finding was that individual
subject analysis yielded different results from the group analyses. Unlike the overall group data and the individual Deprenyl medication data, all subjects exhibited negative correlations during the Sinemet medication conditions. The impact of the direction of these correlations will be discussed as a component of Issue #5.

Previously, Kilbreath (1986) had not observed many significant relationships between the clinical features of PD and the dependent measures of postural sway. Kilbreath (1986) observed gait to be significantly correlated to hypokinesia, $r = .45$, $p < .01$ and stage of disease, $r = .51$, $p < .01$ respectively. Posture was also correlated with stage of the disease, $r = .53$, $p < .01$. The author attributed the lack of significant relationships between symptomatology and the postural sway measures, to the lack of control of the subject's medication. This particular area may have introduced confounds in the correlations among postural stability and other clinical signs (Kilbreath, 1986).

Few studies have been attempted to correlate qualitative/clinical data with quantitative measures, for PD. Sears-Duru (1991) performed an study to determine the inter-rater and intra-rater reliability of the SPAF. The author performed a correlational analysis of the clinical features of PD with the SPAF. Sears-Duru (1991) observed several correlations between cardinal symptoms of PD that were common
to both reliability studies. The following are a list some of those correlations: stage of disease and posture $r > .89$, $p < .05$; stage of disease and rigidity, $r > .89$, $p < .05$; rigidity and tremor, $r = .84$, $p < .05$; tremor and postural stability, $r > .80$, $p < .05$; and hypokinesia and gait, $r = -.72$, $p < .05$, for the inter-rater reliability study and $r = .72$, $p < .05$, for the intra-rater reliability study.

The inter-rater and intra-rater reliability of this Instrument has been previously reported (Sears-Urru, 1991). Some aspects of the validity of this tool have been demonstrated by virtue of the statistically significant correlations. The SPAF may be one of the best physiotherapeutic evaluations of postural stability in the parkinsonian individual. Many therapists do not have access to objective measurements of postural sway. Utilization of instruments such as the SPAF may still provide clinicians with insight into the status of postural stability for each patient.

There are other symptomalogical factors which may have indirectly influenced these outcomes. For example, Watanabe et al. (1980) suggested that rigidity may actually decrease area of sway. The authors observed area of sway in the parkinsonian subjects to be often smaller than the healthy age-matched control subjects. They attributed these conflicting results to the influence of rigidity on postural
Starkes et al. (1991) observed similar reductions in sway area in a parkinsonian subject as Watanabe et al. (1980). The authors indicated that rigidity may actually decrease area of sway in a quiet standing EO position that is not destabilizing. However if this individual moves or is moved, a period of imbalance and/or falling invariably results. This might produce a scenario in which an individual who is quite rigid may produce a small area of sway, despite exhibiting significant impairments of balance.

Although clinical features such as akinesia, bradykinesia, hypokinesia, rigidity and impairments of gait were not assessed in this present study, their influence on postural sway, whether direct or indirect, must be considered.

Few studies have attempted to correlate qualitative/clinical data with quantitative measures for PD. Although limited, this study did observe significant relationships between specific measures of postural sway and postural stability. Further research into this particular area of quantitative and qualitative relationships is warranted.
In this study, dramatic examples of diurnal variability were observed for each subject. The variable nature of this disease was truly reflected both qualitatively and quantitatively. Each subject fluctuated from the extremes of instability and stability within a few hours. This was particularly apparent when analyzing the data provided by subject FR. Although the inclusion and exclusion criteria for the study were very specific, significant variability was evident throughout FR's daily performance.

Overlapping confidence intervals for the EU and EV conditions were quite apparent for many of the dependent measures. This suggests that performance may not have been unduly altered by changing the visual condition. This premise was not supported by the results obtained for the dependent measure, lateral sway. On the first testing session of the Sinemet medication regimen RQ was greater than 100%, signifying that vision improved postural sway, in spite of overlapping confidence intervals. This inconsistency occurs as a nature of each particular statistic. Calculation of confidence intervals is a complex process which takes into account the nature of each individual response. Romberg quotient on the other hand, is the relative difference of the
average extent of sway with EC, to the average extent of sway with EO. Hence, it does not take into consideration the relatively large amounts of sway under EO conditions, even if the sway is further enlarged in the EC situation. Thus RQ could as evidenced here, mask a real difference in postural instability within each subject (Kilbreath, 1986), resulting in overlapping confidence intervals.

During the second testing day of the Deprenyl medication regimen, conflicting results were again observed for the dependent measure, lateral sway. Coefficient of variation values indicated that variability was greater in the EO condition than the EC condition. However the RQ value was greater than 100% indicating that vision actually improved performance. In this instance although greater variability may have been observed in the EO condition, overall performance was improved during this visual condition.

Eyes open confidence bandwidths were smaller than EC bandwidths for the dependent measure, area of sway. During the first testing day of the Sinemet medication regimen, and the Deprenyl medication regimen respectively, the RQ value was considerably larger than 100%. Coefficient of variation values on the other hand, were larger for the EO condition than the EC condition. This would suggest that although vision improved average performance, postural sway characteristics were more variable in the EO condition than
the EC condition.

Starkes et al. (1992) utilized a similar strategy as this study in a comparative analysis of the postural sway characteristics of children and a PD patient. It has been accepted that as a child develops, their postural sway characteristics improve. The authors indicated that height and weight explain a small but significant amount of between subject variability not readily explained by age or gender. In quiet standing, children sway less with increasing age in both anterior-posterior and lateral directions. Sway velocity also decreases with age and is sensitive to eye closure. Eye closure routinely increases velocity of sway.

If one compares the data obtained with the analysis of children's data, and those with PD, they may appear quite similar. One explanation used is that children are undergoing neurological "upgrading", while individuals with PD experience neurological impairment with accompanying deterioration in the vestibular, visual and proprioceptive systems (Starkes et al., 1991). However, this is where the comparison ends. PD subjects are far more variable in their performance. In the present study, this is evidenced by analyzing the individual plots over the course of the day.

With so many evaluative sessions during the day, the question of inter-rater and intra-rater reliability must be addressed. Inter-rater reliability measures the consistency
between examiners measuring the same subject; whereas intrarater reliability measures the consistency of repetitive evaluations of the same subject.

Kilbreath (1986) in examining PD individuals, found adequate intrasubject reliability with EO and also in the lateral direction with EC. In addition, it was found that the mean value of the third trial RMS a-p was less than that of the first trial. The author indicated (unlike LeClair & Riach), that this response was due to a familiarization of the test. This concept was not directly addressed in this study.

In recent study Hattori et al. (in press) demonstrated that normal subjects usually present with high inter-subject variability in performance. Intra subject performance of a-p, lat, velocity, and total excursion of sway however, are highly reliable throughout the course of the day. LeClair and Riach (1991) performed a study to compare six outcome measures for five test durations, two stance conditions, and two test times. They did not demonstrate any main effects for trials, indicating that subjects did not learn to improve their balance based on two practice sessions (LeClair & Riach, 1991).

In light of this variability the statistical method that has been incorporated in this study must be advocated. The individual analyses provided by the confidence intervals
over the course of a full day, clearly illustrate the behaviour of each subject under a large variety of conditions. In terms of rehabilitation, this method is quite applicable. As a Physiotherapist, the ability to assess performance and in essence function, is extremely important. By incorporating the confidence bandwidth analysis into the assessment and treatment regimen, one can monitor performance throughout the day, and observe when the client performs at his/her best or worst. One can also observe how the patient's function is affected by the administration of medication throughout the day. More importantly this method of analysis allows longitudinal changes in performance associated with disease progression to be examined. By the same token, this particular method does not lend itself well to generalization, particularly with such a heterogeneous population.

Fluctuations in postural sway characteristics and function, are evident throughout the day. These may be influenced by many factors such as diet, emotional status, and fatigue, time of day, time of last dose, drug interaction, exercise, stress and confidence in abilities (Factor & Weiner, 1988; Rozovski & Lurie, 1990; Starkes et al., 1991).

It has been suggested that a large amount of protein in the diet may alter the effect of L-dopa. As both protein and L-dopa contain amino acids, competition for absorption into the gut and across the blood brain barrier may occur
(Rozovski & Lurie, 1990). Based on this premise, an individual taking L-dopa may experience periods of ineffective dosages related to meal times.

A high incidence of depression has been documented with the PD patient (Selby, 1990). It can be interpreted as a reasonable reaction to a chronic progressive, disabling disease. Some degree of depression has to be anticipated in a patient with physical inertia, whose social contacts are limited by embarrassment over his tremor, by dependence on the help of others for even the most simple daily activities, and by a soft and slurred speech which listeners find difficult to understand (Selby, 1990). There is also the possibility of a relationship between depression and intellectual decline. Cognitive impairment and dementia are common in patients with PD, especially in the later stages of the disease, but the exact frequency with which they occur is unknown (Factor & Weiner, 1988; Rinne, Rummukainen, Paljarvi, & Rinne, 1989).

Many times subject FR performed worse after a meal, at the end of the eight hour (somewhat stressful session), and first thing in the morning after a long bus ride to the Centre. It is not inconceivable then that alterations in performance were either a direct or indirect result of any or all of the above factors.

Physiotherapeutic studies of PD patients, must be well designed. Problems of matching a treatment group to a control
group are quite common. Hence within-patient comparisons are advocated (Godwin-Austen, 1990; Franklyn & Stern, 1981; Starkes et al., 1991). Such studies have to allow for considerable diurnal, environmental and drug-related fluctuations in symptoms (Godwin-Austen, 1990).

Issue #3: Day to Day Changes in Individual Performance

Although significant variability exists for all subjects, no significant effects were detected across days for either subjects FR and JA and SR, (Tables 6, 7 and 8). Due to the fluctuating nature of PD, it is not surprising that consistent differences between days were not observed. It is not surprising that significant changes would not be observed between days due to the closeness of each testing session. An eight day interval was much too short to detect any changes that might have occurred as a result of disease progression. Increasing the length of time between sessions might have provided greater insight into the progressive nature of PD. Although subjects may have indicated that they were having a better or worse day, over a short period of time, significant change was not observed.
The results of this study provide information about the influence of vision on postural sway in Parkinson's disease. The possible existence of primary sensory alteration in PD has earlier been suggested (Bodis Wollner & Uotani, 1986). Reports of somatosensory and visual alteration in PD could be caused by either the abnormal functioning of the basal ganglia in sensory motor integration, or by the malfunction of dopaminergic systems at different levels of the sensory pathway. In this instance, as there was no analysis of the visual system, it is difficult to conclude that this was the cause. Nevertheless, these data did indicate that both individually and as a group, PD patients are greatly influenced by eye closure.

Recall for subject JA, that a two way interaction for visual condition X medication was observed for area of sway. Yet the Tukey post hoc analysis failed to declare any pairwise comparison of the means significant. Why the apparent contradiction? In actual fact, the F-test from the analysis of variance (ANOVA) is a more powerful and sensitive test procedure. The F-test not only compares the various pairs of means, it seeks to find differences that may be of a more complex form. The ANOVA is accounting for any linear combination of the means that might lead to a significant difference. Thus although its significance does say that the
groups are not a homogeneous set, this does not imply that at least one pairwise comparison of the means will be significant.

In a study comparing healthy subjects to 300 neurological patients, of which 47 were parkinsonian, Njokiktien and De Rijke, (1971) observed that PD patients' performance was worse than normal subjects when measured on amplitude or area of sway. They also indicated that this discrepancy increased when vision was eliminated.

One interesting note is that the influence of vision might be altered by foot position. Stribley, Albers, Tourtellotte, and Cockrell (1974) have suggested when the feet are parallel and touching, and eyes are closed, lateral steadiness (sway) is significantly greater than a-p steadiness.

Fernie et al. (1982) found the mean velocity of sway to be statistically significant when comparing the incidence of falls in geriatric subjects. It was suggested that both area and velocity of sway may be the most behaviorally important sway characteristics in the analysis of postural stability. This present study observed two, three way interactions of vision, medication and day, for subjects JA and SR respectively. Each of these interactions were observed for the dependent measure velocity of sway, which lends support to Fernie et al., (1982) suppositions.
In a comparison of scores obtained for normal subjects and very old subjects, differences were observed in how subjects use vision across age (Table 21) (Hattori et al., 1991).

Insert Table 21 about here

In the present study, subject FR performed worse than the elderly normal subjects, whereas both subjects JA and SH performed similarly to adult normal subjects.

Kilbreath (1986) found that male subjects between the ages of 70 - 89 years had values of: RMS lat eyes open = .52, eyes closed = .5, RMS a-p, eyes open = .58, eyes closed = .61. The youngest subject, FR, achieved scores higher than each of these values; both subjects JA and SR achieved values similar to those subjects in Kilbreath's study.

In many studies of the role of vision and postural sway, the Romberg Quotient (RQ) is used to test when subjects use vision the most (Kilbreath, 1986; Njolokikiten & Van Parys, 1976; Riach & Hayes, 1987; Starkes & Riachn. 1990; Vandervoort, Hayes, & Cape, 1985). Recall that RQ values for normal adults are generally greater than 100% indicating that vision improves postural stability. Kilbreath (1986) demonstrated RQ values of 113% for male parkinsonians aged 70-89 years for RMS lat and a score of 125% for RMS a-p. The
author indicated that RQ could actually mask a real difference in postural instability between parkinsonians and normals. Recall that RQ does not take into consideration the relatively large amounts of sway under the eyes open conditions, even if the sway is further enlarged in the eyes closed condition (Kilbreath, 1986). For example a normal subject might exhibit: RMS lat eyes open .33, eyes closed .35. A parkinsonian subject might exhibit: RMS lat eyes open .65, eyes closed .67. The RQ values for the normal subject would become 106%, and for the parkinsonian 103%.

Vandervoort et al. (1985) demonstrated that for normal adults males aged 70 - 89 years, RQ values for RMS a-p were 149%, and for the dependent measure RMS lat were 114%. In Starkes and Riach (1990) normal young adults demonstrated RQ's of approximately 108% for lateral sway, and 120% for a-p sway in normal eyes open stance. The RQ values for subject HR ranged from: RMS lat 91% - 133%, RMS a-p 103% - 133%; subject JA, RMS lat 93% - 143%, RMS a-p 93% -116%; subject SR, RMS lat 111% -138%, RMS a-p 100 - 119% respectively. With such a wide spectrum of RQ values, it is important to reconsider the idiosyncratic nature of PD. At times vision improves postural stability considerably, and at other times vision appears to impair performance.

The parkinsonian's dependence on vision as demonstrated by these results is an important consideration in
rehabilitation. In most instances, removal of vision caused a significant increase in postural sway. Holliday et al. (1978) have suggested that careful examination of the eyes and consideration of potential visual impairments should be considered when designing a rehabilitation program.

**Issue #5: The Effect of Medication on Postural Sway**

Although there have been major advances made in the pharmacological management of individuals with idiopathic PD, numerous functional deficits can still be observed in individuals with the disease. Upon analyzing the effect of medication on postural sway, no significant difference in any of the postural sway characteristics between either medication regimen for either subjects FR and SR were observed. Subject JA however, experienced a difference in performance with the introduction of Deprenyl to his medication regimen. This was observed for the dependent measures: RMS lat, area of sway and velocity of sway (Table 19). This subject started taking Deprenyl mid-way through the study, which allowed the author to observe changes in performance that may have been associated directly or indirectly with the administration of this medication. The other subjects were taking Deprenyl prior to their inclusion in the study.

Deprenyl has been suggested to delay progression of disease in de novo subjects. As each of these subjects had
been diagnosed at least five years previous to the commencement of this study, it is not unusual to find the results observed with subjects FR and SR. One study has evaluated the administration of Deprenyl to individuals on longstanding L-dopa therapy. This study concluded that one third to one half of the 200 subjects who participated in this study improved in function, if only temporarily (Elizan et al., 1989b). Subject JA would fit into this classification as he had been taking L-dopa therapy for at least ten years prior to commencing Deprenyl treatment.

Diurnal variation in response to L-dopa medication is commonly reported by patients. This often takes the form of attenuation of response doses taken later in the day. Patients describe shorter periods of response, or failure of response to afternoon and evening doses, and motor benefit is marred by a build-up of dyskinetic involuntary movements in the latter half of the day. These phenomena may be due to the effects of time of day, exercise, emotional status, time of last dose, drug interactions with meals (especially protein), loss of sleep, and changes in dopamine receptor sensitivity (Eriksson, Ganerus, Linde, Anders, & Carsson, 1988; Factor & Weiner, 1988; Frankel et al., 1990; Rajput & Duvoisin, 1990; Rozovski & Lurie, 1990). Although inconsistent, this trend was reflected in each of the subjects, especially subject FR.

In a study analyzing the relationship between motor
performance and sequential plasma and ventricular CSF levels of L-dopa, one researcher found that the time course of clinical improvement closely reflected CSF as opposed to plasma L-dopa levels. The onset of dyskinesia precisely correlated with peak CSF L-dopa levels. Peak plasma L-dopa levels occurred at .6 +/- .3 hours post-dose; peak CSF L-dopa levels were attained at 1.5 +/- .6 hours (Olanow, 1990).

The controversy currently exists as to the eight day "wash out" period for Deprenyl. Some investigators believe that only a few days are required for the system to rid itself of all traces (and side effects) of Deprenyl. Others disagree and suggest a longer period of "wash out", as Deprenyl remains at trace levels for at least two months. No studies to date have indicated that Deprenyl is effective at a subtherapeutic level. In light of this, the "wash out" period may have been quite appropriate, but confounding factors such as emotional status, may have masked the "true" effect of each medication.

All of the subjects were taking a number of medications, which again may have influenced that which was observed throughout the study. As medications were not controlled for, a clear picture of the role of Deprenyl, or Sinemet may not have been achieved. The data on whether L-dopa has a stabilizing effect on postural sway are unclear. Cernacek et al. (1973), and Folkerts and Njolokten (1972),
each demonstrated that L-dopa has a destabilizing influence on stable parkinsonians, and a stabilizing effect on unstable parkinsonians. Klawans (1986) suggested that L-dopa is ineffective in the treatment of postural instability.

The results of all subjects, especially subject JA lend support to the above studies. Performance on postural sway measures were enhanced with the introduction of Deprenyl into this individual's treatment regimen. The two other subjects who had been taking both Sinemet and Deprenyl prior to the commencement of this study, did not show statistically significant differences in performance between the two medication conditions. The question remains as to the duration of this positive effect of postural improvement that was observed in subject JA.

FUTURE RESEARCH

Postural stability is an important consideration, not only in various disease states, but also in normal aging. Knowledge of the nature of postural stability and its manifestations throughout the progression of Parkinson's disease, would assist in its assessment, treatment and management. From the prescription of medications, to the formation of individualized rehabilitative programs, insight into this unique area might produce modifications in the
existing approach to the administration of programs for the parkinsonian individual.

Although the goal of future research is to find a cure for this disease, current remediation as offered by medication must be evaluated. It is also important to determine (a) whether cardinal features of the disease are correlated with each other, (b) how are they differentially affected by medication, and (c) to what extent they affect function.

A number of significant and non-significant relationships were observed with the dependent measures of postural sway and the SPAF. In order to ascertain and confirm that there are significant relationships between all of the cardinal signs of PD, i.e. bradykinesia, rigidity, tremor and postural instability, correlational analyses incorporating these areas needs to be performed. In fact, both qualitative and quantitative analyses of each of the symptoms are required to validate any clinical assessment tool. To this end, a number of studies have been undertaken at the University of Alberta to further assess the SPAF.

To examine the functional effect(s) of medication on PD offers both the clinician and client, an opportunity to determine the effectiveness of each medication regimen; hence the development of reliable and functional clinical instruments is crucial.

It is also important to assess existing clinical tools
with new and innovative evaluative modalities. The development and implementation of disease specific, clinical assessment tools that are time efficient, valid and reliable would also greatly enhance current management protocols. Again a study is currently underway at the University of Alberta to assess the concurrent validity of the SPAF with the UPDRS.

Purdon Martin (1967) indicated that postural instability in PD occurred as a dysfunction of the globus pallidus. Many authors have tried to localize the areas in the basal ganglia that are directly responsible for disabling postural stability. The potential for the prediction of postural sway throughout the course of the disease may also provide an impetus for the development of educational and functional treatment programs specific to each stage of the disease.

As a clinician, it is important not only to understand the neurophysiology or neuropathology of this disease, but also to be able to assess and treat this disease. The subjective evaluation alone is not enough. When measurement of changes, no matter how subtle, are necessary, current clinical instruments lack sensitivity to even the most subtle changes. Ultimately, clinical tools could assist in the identification of the most subtle mechanisms that may effect the functional status of parkinsonians. Clinical tools need to be validated to justify inclusion as assessment tool. Too
often clinicians are limited by the lack of quantitative instruments to assist in the assessment and management of their clients, particularly those with chronic disabling illnesses. The development of reliable, valid and efficient clinical assessment tools may also diminish the necessity for the costly purchase of such quantitative instruments.

One of the results of this study supports the hypothesis that L-dopa may be less effective in the treatment of postural stability that other anti-parkinsonian medications. (Cernacek et al., 1972; Folkerts & Njokiktien, 1972; Kilbreath, 1986; Klawans, 1986). L-dopa treatment continues to be the medication regimen of choice in the treatment of parkinsonians. With the introduction of such medications as Deprenyl, Apomorphine and Sinemet CR, into daily medication regimens, it is most important to establish the efficacy of each medication not only for the population as a whole, but for each individual.

Longitudinal studies examining variables such as, stage of disease, medication, age, sex, date of onset of PD, and diet, need to be encouraged. Clinically relevant physiotherapeutic studies which address the behavioral aspects of disease must be more readily employed. The method of individual case analysis can be easily incorporated into patient assessment protocols to observe performance over one or more days and should be incorporated more often with
clinical research. Evaluative procedures that are disease specific and address the functional needs of the client must be incorporated into today's research.

CONCLUSIONS

1. Individual and group analyses established significant correlations between both the quantitative measures of postural stability, and the qualitative assessment tool.

2. Significant variability was evident upon the analysis of individual data plots. The employment of confidence intervals allows researchers to determine to what extent a subject's performance is similar and/or dissimilar to his/her own average response. Coefficients of variation confirm the extremely variable nature of this disease.

3. Although there was a high variance associated with each individual analysis, no statistically significant difference was observed for any subject, when measured from day to day.

4. The influence of vision was extremely variable for each subject. Most subjects found vision to be quite stabilizing, however it was not uncommon to observe the contrary for these same individuals in isolated sessions. Parkinsonian subjects
also appear to be different from age-matched normals, but appear similar to children under seven, and normal subjects over the age of eighty years (Hattori, Starkes & Takanashi, 1989; Kilbreath, 1986; Starkes & Riach, 1990; Starkes, Riach & Clarke, 1991; Vandervoort, 1985).

5. Although two of the subjects did not experience differences in postural sway profiles when altering medication, one subject (JA) did show improvements in postural sway measures once Deprenyl was introduced.
REFERENCES


Figure 1. Diagrammatic representation of pathways concerned with the execution and control of voluntary movement.

PLAN, PROGRAM

IDEA → SMA ← CORTEX ← THALAMUS → MOTOR CORTEX → MOVE

↑

LATERAL CEREBELLUM

INTERMEDIARY CEREBELLUM

SOMATO SENSORY

BASAL GANGLIA

ASSOCIATION

SMA-SUPPLEMENTARY MOTOR
<table>
<thead>
<tr>
<th>SUBJECT</th>
<th>AGE</th>
<th>AGE AT DIAGNOSIS</th>
<th>MEDICATION</th>
</tr>
</thead>
<tbody>
<tr>
<td>FR</td>
<td>60 years</td>
<td>46 years</td>
<td>Amitriptyline 37.5 mg/day</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Cogentin 2 mg/day</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Deprenyl 2.5 mg/day</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Sinemet 250/25</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>--6 capsules/day</td>
</tr>
<tr>
<td>JA</td>
<td>68 years</td>
<td>55 years</td>
<td>Amitriptyline 10 mg/day</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Bensylate 2 mg/day</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Sinemet 250/25</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>--2 tablets/day</td>
</tr>
<tr>
<td>SR</td>
<td>76 years</td>
<td>72 years</td>
<td>Deprenyl 5 mg/day</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Sinemet 100/10</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>--4.5 tablets/day</td>
</tr>
</tbody>
</table>
Table 2

Correlation of force platform dependent measures with the SPAF: group data, (N = 48).

<table>
<thead>
<tr>
<th>Dependent measure</th>
<th>Sinemet</th>
<th>Deprenyl</th>
</tr>
</thead>
<tbody>
<tr>
<td>Area of sway</td>
<td>.45*</td>
<td>.60*</td>
</tr>
<tr>
<td>Velocity of sway</td>
<td>.51*</td>
<td>.52*</td>
</tr>
</tbody>
</table>

*p<.05
Table 3

Correlation of force platform dependent measures with the
SPAF: individual subject data, (N = 16)

<table>
<thead>
<tr>
<th>Dependent measure</th>
<th>Sinemet time 1</th>
<th>Sinemet time 2</th>
<th>Deprenyl time 1</th>
<th>Deprenyl time 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>RMS lat</td>
<td>-.55*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Area of sway</td>
<td>-.87*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Velocity of sway</td>
<td>-.53*</td>
<td>-.52*</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*P < .05
### Table 4

**Coefficient of variation for subject FR**

<table>
<thead>
<tr>
<th>Dependent measure</th>
<th>Eyes open</th>
<th>Eyes closed</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Day 1 Sinemet</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RMS lat sway</td>
<td>.21</td>
<td>.30</td>
</tr>
<tr>
<td>RMS a-p sway</td>
<td>.31</td>
<td>.09</td>
</tr>
<tr>
<td>Area of sway</td>
<td>.44</td>
<td>.38</td>
</tr>
<tr>
<td>Velocity of sway</td>
<td>.30</td>
<td>.19</td>
</tr>
<tr>
<td><strong>Day 2 Sinemet</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RMS lat sway</td>
<td>.28</td>
<td>.12</td>
</tr>
<tr>
<td>RMS a-p sway</td>
<td>.22</td>
<td>.27</td>
</tr>
<tr>
<td>Area of sway</td>
<td>.35</td>
<td>.25</td>
</tr>
<tr>
<td>Velocity of sway</td>
<td>.24</td>
<td>.24</td>
</tr>
<tr>
<td><strong>Day 1 Deprenyl</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RMS lat sway</td>
<td>.37</td>
<td>.19</td>
</tr>
<tr>
<td>RMS a-p sway</td>
<td>.38</td>
<td>.44</td>
</tr>
<tr>
<td>Area of sway</td>
<td>.45</td>
<td>.30</td>
</tr>
<tr>
<td>Velocity of sway</td>
<td>.21</td>
<td>.17</td>
</tr>
<tr>
<td><strong>Day 2 Deprenyl</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RMS lat sway</td>
<td>.29</td>
<td>.20</td>
</tr>
<tr>
<td>RMS a-p sway</td>
<td>.22</td>
<td>.26</td>
</tr>
<tr>
<td>Area of sway</td>
<td>.33</td>
<td>.34</td>
</tr>
<tr>
<td>Velocity of sway</td>
<td>.22</td>
<td>.26</td>
</tr>
</tbody>
</table>
Table 5
Romberg quotients (%) for subject FR

<table>
<thead>
<tr>
<th></th>
<th>Day 1 Sinemet</th>
<th>Day 2 Sinemet</th>
<th>Day 1 Deprenyl</th>
<th>Day 2 Deprenyl</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>RMS lat sway</td>
<td>RMS a-p sway</td>
<td>RMS lat sway</td>
<td>RMS a-p sway</td>
</tr>
<tr>
<td></td>
<td>133</td>
<td>103</td>
<td>101</td>
<td>133</td>
</tr>
<tr>
<td></td>
<td>167</td>
<td>197</td>
<td>148</td>
<td>188</td>
</tr>
<tr>
<td></td>
<td>91</td>
<td>107</td>
<td>200</td>
<td>170</td>
</tr>
<tr>
<td>Area of sway</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Velocity of sway</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Table 6

Mean values and significance levels for the main effect of days: subject FR

<table>
<thead>
<tr>
<th>Dependent measure</th>
<th>Day 1</th>
<th>Day 2</th>
<th>Significance level</th>
</tr>
</thead>
<tbody>
<tr>
<td>RMS lat sway</td>
<td>0.83</td>
<td>0.86</td>
<td>F(1,9) = .02, nsd</td>
</tr>
<tr>
<td>RMS a-p sway</td>
<td>0.84</td>
<td>0.88</td>
<td>F(1,9) = .01, nsd</td>
</tr>
<tr>
<td>Area of sway</td>
<td>26.29</td>
<td>29.98</td>
<td>F(1,9) = 1.26, nsd</td>
</tr>
<tr>
<td>Velocity of sway</td>
<td>2.5</td>
<td>2.67</td>
<td>F(1,9) = 2.39, nsd</td>
</tr>
</tbody>
</table>
Table 7

Mean values and significance levels for the main effect of days: subject JA

<table>
<thead>
<tr>
<th>Dependent measure</th>
<th>Day 1</th>
<th>Day 2</th>
<th>Significance level</th>
</tr>
</thead>
<tbody>
<tr>
<td>RMS lat sway</td>
<td>0.45</td>
<td>0.49</td>
<td>$F(1,9) = .54$, nsd</td>
</tr>
<tr>
<td>RMS a-p sway</td>
<td>0.51</td>
<td>0.50</td>
<td>$F(1,9) = .27$, nsd</td>
</tr>
<tr>
<td>Area of sway</td>
<td>9.39</td>
<td>9.36</td>
<td>$F(1,9) = .01$, nsd</td>
</tr>
<tr>
<td>Velocity of sway</td>
<td>1.52</td>
<td>1.51</td>
<td>$F(1,9) = .001$, nsd</td>
</tr>
</tbody>
</table>
Table 8
Mean values and significance levels for the main effect of days: subject SR

<table>
<thead>
<tr>
<th>Dependent measure</th>
<th>Day 1</th>
<th>Day 2</th>
<th>Significance level</th>
</tr>
</thead>
<tbody>
<tr>
<td>RMS lat sway</td>
<td>0.53</td>
<td>0.56</td>
<td>$F(1,9) = 0.02$, nsd</td>
</tr>
<tr>
<td>RMS a-p sway</td>
<td>0.54</td>
<td>0.54</td>
<td>$F(1,9) = 0.04$, nsd</td>
</tr>
<tr>
<td>Area of sway</td>
<td>8.67</td>
<td>9.36</td>
<td>$F(1,9) = 0.43$, nsd</td>
</tr>
<tr>
<td>Velocity of sway</td>
<td>1.29</td>
<td>1.44</td>
<td>$F(1,9) = 1.18$, nsd</td>
</tr>
</tbody>
</table>
Table 9

*Visual condition X medication X days interaction. Mean values for RMS lat: subject FR*

<table>
<thead>
<tr>
<th>Medication condition</th>
<th>Eyes open</th>
<th>Eyes closed</th>
</tr>
</thead>
<tbody>
<tr>
<td>Day 1 Sinemet</td>
<td>.71</td>
<td>.95</td>
</tr>
<tr>
<td>Day 2 Sinemet</td>
<td>.87</td>
<td>.80</td>
</tr>
<tr>
<td>Day 1 Deprenyl</td>
<td>.78</td>
<td>.87</td>
</tr>
<tr>
<td>Day 2 Deprenyl</td>
<td>.82</td>
<td>.93</td>
</tr>
</tbody>
</table>
Table 10
Visual condition X medication interaction. Mean values for RMS a-p: subject FR

<table>
<thead>
<tr>
<th>Medication condition</th>
<th>Eyes open</th>
<th>Eyes closed</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sinemet</td>
<td>.85</td>
<td>.89</td>
</tr>
<tr>
<td>Deprenyl</td>
<td>.73</td>
<td>.96</td>
</tr>
</tbody>
</table>
Table 11
Mean values and significance levels for the main effect of vision: subject FR

<table>
<thead>
<tr>
<th>Dependent measure</th>
<th>Eyes open</th>
<th>Eyes closed</th>
<th>Significance level</th>
</tr>
</thead>
<tbody>
<tr>
<td>RMS lat sway</td>
<td>1.27</td>
<td>1.28</td>
<td>$F(1,9) = 1.45$, nsd</td>
</tr>
<tr>
<td>RMS a-p sway</td>
<td>1.11</td>
<td>1.74</td>
<td>$F(1,9) = 14.09$, $p&lt;.05$</td>
</tr>
<tr>
<td>Area of sway</td>
<td>34.91</td>
<td>67.61</td>
<td>$F(1,9) = 99.44$, $p&lt;.001$</td>
</tr>
<tr>
<td>Velocity of sway</td>
<td>3.31</td>
<td>3.37</td>
<td>$F(1,9) = 134.37$, $p&lt;.001$</td>
</tr>
</tbody>
</table>
Table 12

Visual condition x days interaction. Mean values for RMS lat: subject JA

<table>
<thead>
<tr>
<th>Condition</th>
<th>Eyes open</th>
<th>Eyes closed</th>
</tr>
</thead>
<tbody>
<tr>
<td>Day 1</td>
<td>0.39</td>
<td>0.53</td>
</tr>
<tr>
<td>Day 2</td>
<td>0.49</td>
<td>0.45</td>
</tr>
</tbody>
</table>
Table 13

Visual condition $\times$ medication interaction. Mean values for velocity of sway (cm/sec): subject JA

<table>
<thead>
<tr>
<th>Medication condition</th>
<th>Eyes open</th>
<th>Eyes closed</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sinemet</td>
<td>1.26</td>
<td>1.49</td>
</tr>
<tr>
<td>Deprenyl</td>
<td>1.24</td>
<td>1.75</td>
</tr>
</tbody>
</table>
### Table 14

**Visual condition X medication interaction. Mean values for area of sway (cm²): subject JA**

<table>
<thead>
<tr>
<th>Medication condition</th>
<th>Eyes open</th>
<th>Eyes closed</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sinemet</td>
<td>8.02</td>
<td>13.57</td>
</tr>
<tr>
<td>Deprenyl</td>
<td>6.85</td>
<td>9.08</td>
</tr>
</tbody>
</table>
Table 15

Mean values and significance levels for the main effect of vision: subject JA

<table>
<thead>
<tr>
<th>Dependent measure</th>
<th>Eyes open</th>
<th>Eyes closed</th>
<th>Significance level</th>
</tr>
</thead>
<tbody>
<tr>
<td>RMS lat sway</td>
<td>.43</td>
<td>.51</td>
<td>F(1,9) = 39.47, p&lt;.001</td>
</tr>
<tr>
<td>RMS a-p sway</td>
<td>.49</td>
<td>.52</td>
<td>F(1,9) = 2.30, nsd</td>
</tr>
<tr>
<td>Area of sway</td>
<td>7.43</td>
<td>11.32</td>
<td>F(1,9) = 33.98, p&lt;.001</td>
</tr>
<tr>
<td>Velocity of sway</td>
<td>1.26</td>
<td>1.78</td>
<td>F(1,9) = 154.50, p&lt;.001</td>
</tr>
</tbody>
</table>
Table 16

Visual condition X medication X days interaction. Mean values for velocity of sway (cm/sec): subject SR

<table>
<thead>
<tr>
<th>Medication condition</th>
<th>Eyes open</th>
<th>Eyes closed</th>
</tr>
</thead>
<tbody>
<tr>
<td>Day 1 Sinemet</td>
<td>1.24</td>
<td>1.32</td>
</tr>
<tr>
<td>Day 2 Sinemet</td>
<td>1.35</td>
<td>1.62</td>
</tr>
<tr>
<td>Day 1 Deprenyl</td>
<td>1.22</td>
<td>1.37</td>
</tr>
<tr>
<td>Day 2 Deprenyl</td>
<td>1.21</td>
<td>1.57</td>
</tr>
</tbody>
</table>
### Table 17

**Mean values and significance levels for the main effect of vision: subject SR**

<table>
<thead>
<tr>
<th>Dependent measure</th>
<th>Eyes open</th>
<th>Eyes closed</th>
<th>Significance level</th>
</tr>
</thead>
<tbody>
<tr>
<td>RMS lat sway</td>
<td>.48</td>
<td>.60</td>
<td>$F(1,9) = 50.43$, $p &lt; .001$</td>
</tr>
<tr>
<td>RMS a-p sway</td>
<td>.50</td>
<td>.57</td>
<td>$F(1,9) = 7.8$, $p &lt; .05$</td>
</tr>
<tr>
<td>Area of sway</td>
<td>7.61</td>
<td>10.97</td>
<td>$F(1,9) = 32.14$, $p &lt; .001$</td>
</tr>
<tr>
<td>Velocity of sway</td>
<td>1.25</td>
<td>1.47</td>
<td>$F(1,9) = 17.89$, $p &lt; .05$</td>
</tr>
</tbody>
</table>
Table 18

*Romberg quotients (%) for subject JA*

<table>
<thead>
<tr>
<th></th>
<th>Day 1 Sinemet</th>
<th>Day 2 Sinemet</th>
<th>Day 1 Deprenyl</th>
<th>Day 2 Deprenyl</th>
</tr>
</thead>
<tbody>
<tr>
<td>RMS lat sway</td>
<td>131</td>
<td>112</td>
<td>143</td>
<td>93</td>
</tr>
<tr>
<td>RMS a-p sway</td>
<td>116</td>
<td>116</td>
<td>94</td>
<td>104</td>
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<tr>
<td>Area of sway</td>
<td>174</td>
<td>165</td>
<td>139</td>
<td>126</td>
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<tr>
<td>Velocity of sway</td>
<td>148</td>
<td>144</td>
<td>136</td>
<td>134</td>
</tr>
</tbody>
</table>
Table 19  

**Romberg quotients (%) for subject SR**

### Day 1 Sinemet

<table>
<thead>
<tr>
<th>Metric</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>RMS lat sway</td>
<td>138</td>
</tr>
<tr>
<td>RMS a-p sway</td>
<td>100</td>
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<tr>
<td>Area of sway</td>
<td>121</td>
</tr>
<tr>
<td>Velocity of sway</td>
<td>106</td>
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### Day 2 Sinemet

<table>
<thead>
<tr>
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<th>Value</th>
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<tbody>
<tr>
<td>RMS lat sway</td>
<td>111</td>
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<tr>
<td>RMS a-p sway</td>
<td>119</td>
</tr>
<tr>
<td>Area of sway</td>
<td>152</td>
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<td>Velocity of sway</td>
<td>120</td>
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### Day 1 Deprenyl

<table>
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<th>Value</th>
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</thead>
<tbody>
<tr>
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<td>120</td>
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<tr>
<td>RMS a-p sway</td>
<td>117</td>
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<tr>
<td>Area of sway</td>
<td>138</td>
</tr>
<tr>
<td>Velocity of sway</td>
<td>112</td>
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</table>

### Day 2 Deprenyl

<table>
<thead>
<tr>
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<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>RMS lat sway</td>
<td>136</td>
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<tr>
<td>RMS a-p sway</td>
<td>114</td>
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<tr>
<td>Area of sway</td>
<td>165</td>
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<tr>
<td>Velocity of sway</td>
<td>129</td>
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</tbody>
</table>
Table 20

Mean values and significance levels for the main effect of medication: subject JA

<table>
<thead>
<tr>
<th>Dependent measure</th>
<th>Sinemet</th>
<th>Deprenyl</th>
<th>Significance level</th>
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<tbody>
<tr>
<td>RMS lat sway</td>
<td>.52</td>
<td>.43</td>
<td>F(1,9) = 9.87, p&lt;.05</td>
</tr>
<tr>
<td>RMS a-p sway</td>
<td>.53</td>
<td>.48</td>
<td>F(1,9) = 1.30, nsd</td>
</tr>
<tr>
<td>Area of sway</td>
<td>10.79</td>
<td>7.96</td>
<td>F(1,9) = 7.19, p&lt;.05</td>
</tr>
<tr>
<td>Velocity of sway</td>
<td>1.63</td>
<td>1.41</td>
<td>F(1,9) = 17.16, p&lt;.05</td>
</tr>
</tbody>
</table>
Table 21

Postural sway characteristics of normal young adults and elderly individuals

<table>
<thead>
<tr>
<th>Dependent measure</th>
<th>Eyes open Adults</th>
<th>Eyes open Elderly</th>
<th>Eyes closed Adults</th>
<th>Eyes closed Elderly</th>
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</thead>
<tbody>
<tr>
<td>RMS lat sway</td>
<td>.38</td>
<td>.66</td>
<td>.52</td>
<td>.89</td>
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<tr>
<td>RMS a-p sway</td>
<td>.45</td>
<td>.58</td>
<td>.60</td>
<td>.85</td>
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<tr>
<td>Maximum distance</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Excursion (cm)</td>
<td>22.60</td>
<td>34.30</td>
<td>31.80</td>
<td>62.40</td>
</tr>
<tr>
<td>Velocity of sway</td>
<td>1.03</td>
<td>1.05</td>
<td>2.10</td>
<td>4.20</td>
</tr>
</tbody>
</table>

APPENDIX I
THE SEARS PARKINSON’S ASSESSMENT FORM (SPAF)
POSTURAL STABILITY

NAME______________________ DATE______
ADDRESS____________________ TIME______
________________________________________
________________________________________
________________________________________
________________________________________
D.O.B.______ AGE AT DIAGNOSIS______

MEDICATIONS AND TIME TAKEN__________________
________________________________________
________________________________________

STATIC OPTICAL RIGHTING
DYNAMIC OPTICAL RIGHTING
STATIC LABYRINTHINE
DYNAMIC LABYRINTHINE

PROTECTIVE REACTION (ARM) (REO*) (LEO+) (REC-) (LEC+)
KNEEL STANDING (EO-) (EC+)
I/2 KNEEL STANDING (REO) (LEO) (REC) (LEC)
STANDING (EO) (EC)
DYNAMIC RIGHTING (REO) (LEO) (REC) (LEC)
PROTECTIVE REACTION (LEG) (REO) (LEO) (REC) (LEC)
ONE LEGGED STANDING (REO) (LEO) (REC) (LEC)

SCORE 2--PATIENT REQUIRES MODERATE ASSISTANCE AND/OR UNABLE
to COMPLETE TASK
SCORE 1--PATIENT REQUIRES MINIMAL ASSISTANCE AND/OR DELAYED
RESPONSE
SCORE 0--PATIENT IS ABLE TO COMPLETE TASK INDEPENDENTLY

KEY
* RIGHT EYES OPEN
+ LEFT EYES OPEN
x RIGHT EYES CLOSED
a LEFT EYES CLOSED
b EYES OPEN
c EYES CLOSED
FR: DAY 1 SINEMET
RMS LATERAL
S--SINEMET, C--COGENITIN

RMS LAT

<table>
<thead>
<tr>
<th>Time of Day</th>
<th>0</th>
<th>0.2</th>
<th>0.4</th>
<th>0.6</th>
<th>0.8</th>
<th>1.0</th>
<th>1.2</th>
<th>1.4</th>
<th>1.6</th>
<th>1.8</th>
</tr>
</thead>
<tbody>
<tr>
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<td></td>
</tr>
<tr>
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<td></td>
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</tr>
<tr>
<td>12.45</td>
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<td></td>
</tr>
<tr>
<td>1.40</td>
<td>sc</td>
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<td></td>
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<td></td>
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</tr>
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<td>3.05</td>
<td>sc</td>
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<td></td>
<td></td>
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</tr>
</tbody>
</table>

---

**5% EC CI**

**5% EO CI**
FR DAY 2 SINEMET  
RMS LATERAL  
S--SINEMET, C--COGENTIN  

TIME OF DAY
FR DAY 1 SINEMET
RMS A-P
S---SINEMET, C---COGENTIN

RMS A-P

TIME OF DAY
FR DAY 2 SINEMET
RMS A-P
S--SINEMET, C--COGENTIN

TIME OF DAY

RMS A-P

0.2 0.4 0.6 0.8 1.0 1.2 1.4 1.6 1.8 2.0

--- 5% EC CI

--- 5% EO CI

9.15 10.40 12.45 1.40 3.05
FR DAY 1 SINEMET
AREA OF SWAY (CM²)
S--SINEMET, C--COGENTIN.

AREA (CM²)

TIME OF DAY

5% EC CI
5% EO CI
FR: DAY 2 SINEMET

AREA OF SWAY (CM²)

S--SINEMET, C--COGENTIN

AREA (CM²)

TIME OF DAY

5% EC CI

5% EO CI
FR DAY 1 SINEMET
VELOCITY OF SWAY (CM/SEC)
S--SINEMET, C--COGENTIN
FR DAY 2 SINEMET
VELOCITY OF SWAY (CM/SEC)
S--SINEMET, C--COGENTIN

VELOCITY (CM/SEC)

TIME OF DAY

5% EC CI
5% EO CI
FR DAY 1 DEPRENYL
RMS LATERAL
S--SINEMET, C--COGENTIN, D--DEPRENYL

RMS LAT

TIME OF DAY

5% EC CI
5% EO CI
FR DAY 2 DEPRENYL
RMS LATERAL
S--SINEMET, C--COGENTIN, D--DEPRENYL

RMS LAT

TIME OF DAY

5% EC CI
5% EO CI
FR  DAY 1 DEPRENYL
RMS A-P
S--SINEMET, C--COGENTIN, D--DEPRENYL

RMS A-P

TIME OF DAY

SCD  10.40  12.45  1.40  3.05
9.15

5% EC CI
5% EO CI
FR DAY 2 DEPRENYL
RMS A-P
S--SINEMET, C--COGENTIN, D--DEPRENYL

RMS A-P

TIME OF DAY

5% EC CI
5% EO CI
FR DAY 1 DEPRENYL
AREA OF SWAY (CM²)
S--SINEMET, C--COGENTIN, D--DEPRENYL

AREA (CM²)

TIME OF DAY
FR  DAY 2 DEPRENYL
AREA OF SWAY (CM²)
S--SINEMET, C--COGENTIN, D--DEPRENYL

AREA (CM²)

TIME OF DAY

5% EC CI
5% EO CI

9.15 10.40 12.45 1.40 3.05
FR  DAY 1 DEPRENYL
VELOCITY OF SWAY (CM/SEC)
S--SINEMET, C--COGENTIN, D--DEPRENYL

VELOCITY (CM/SEC)

TIME OF DAY

5% EC CI

5% EO CI
FR DAY 2 DEPRENYL
VELOCITY OF SWAY (CM/SEC)
S--SINEMET, C--COGENTIN, D--DEPRENYL

VELOCITY (CM/SEC)
JM DAY 2 SINEMET
RMS LATERAL
S--SINEMET, B--BENSYLATE

RMS LAT

TIME OF DAY

5% EC CI
5% EO CI
JA  DAY 1  SINEMET
RMS  A-P
S--SINEMET, B--BENSYLATE

RMS A-P

TIME OF DAY

5% EC CI

5% EO CI
JA DAY 1 SINEMET

AREA OF SWAY (CM²)

S--SINEMET, B--BENSYLATE

AREA (CM²)

TIME OF DAY

5% EC CI

5% EO CI
JA DAY 1 SINEMET
VELOCITY OF SWAY (CM/SEC)
S--SINEMET, B--BENSYLATE

TIME OF DAY

VELOCITY (CM/SEC)

5
4.5
4
3.5
3
2.5
2
1.5
1
0.5
0

9.15 10.40 12.45 1.40 3.05

5% EC CI
5% EO CI
JA DAY 2 SINEMET
VELOCITY OF SWAY (CM/SEC)
S--SINEMET, B--BENSYLATE

VELOCITY (CM/SEC)

TIME OF DAY
JA DAY 1 DEPRENYL
RMS LATERAL
S--SINEMET, B--BENSYLATE, D--DEPRENYL

RMS LAT

TIME OF DAY

5% EC CI
5% EO CI
JA DAY 2 DEPRENYL
RMS LATERAL
S--SINEMET, B--BENSYLATE, D--DEPRENYL

RMS LAT

TIME OF DAY
JA  DAY 2 DEPRENYL
RMS A-P
S--SINEMET, B--BENSYLATE, D--DEPRENYL

RMS A-P

TIME OF DAY

5% EC CI
5% EO CI
JA DAY 1 DEPRENYL
AREA OF SWAY (CM²)
S--SINEMET, B--BENSYLATE, D--DEPRENYL

AREA (CM²)

5% EC CI
5% EO CI

TIME OF DAY
JA: DAY 2 DEPRENYL
AREA OF SWAY (CM²)
S--SINEMET; B--BENSYLATE, D--DEPRENYL

AREA (CM²)

TIME OF DAY
JA: DAY 1 DEPRENYL
VELOCITY OF SWAY (CM/SEC)
S--SINEMET; B--BENSYLATE, D--DEPRENYL

VELOCITY (CM/SEC)

TIME OF DAY
JA DAY 2 DEPRENYL

VELOCITY OF SWAY (CM/SEC)
S--SINEMET, B--BENSYLATE, D--DEPRENYL

VELOCITY (CM/SEC)

5

4.5

4

3.5

3

2.5

2

1.5

1

0.5

0

SBD
9.15

S
10.40

S
12.45

SD
1.40

3.05

TIME OF DAY

5% EC CI

5% EO CI
SR DAY 2 SINEMET
VELOCITY OF SWAY (CM/SEC)
S--SINEMET

VELOCITY (CM/SEC)

TIME OF DAY

5% EC CI

5% EO CI
SR DAY 1 DEPRENYL
RMS LATERAL
S--SINEMET, D--DEPRENYL

RMS LAT

TIME OF DAY

5% EC CI
5% EO CI
SR DAY 2 DEPRENYL
RMS LATERAL
S--SINEMET,  D--DEPRENYL

RMS LAT

TIME OF DAY

9.15 10.40 12.45 1.40 3.05

5% EC CI
5% EO CI
SR DAY 1 DEPRENYL
RMS A-P
S--SINEMET, D--DEPRENYL

RMS A-P

TIME OF DAY
SR DAY 1 DEPRENYL
AREA OF SWAY (CM²)
S--SINEMET, D--DEPRENYL

AREA (CM²)

TIME OF DAY

5% EC CI

5% EO CI
SR  DAY 2 DEPENYL
VELOCITY OF SWAY (CM/SEC)
S--SINEMET,  D--DEPENYL

VELOCITY (CM/SEC)

5
4.5
4
3.5
3
2.5
2
1.5
1
0.5
0

9.15  10.40  12.45  1.40  3.05

TIME OF DAY

5% EC CI
5% EO CI
<table>
<thead>
<tr>
<th>Dependent measure</th>
<th>Eyes open</th>
<th>Eyes closed</th>
</tr>
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<tbody>
<tr>
<td><strong>Day 1 Sinemet</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>RMS lat sway</td>
<td>.26</td>
<td>.14</td>
</tr>
<tr>
<td>RMS a-p sway</td>
<td>.31</td>
<td>.22</td>
</tr>
<tr>
<td>Area of sway</td>
<td>.25</td>
<td>.23</td>
</tr>
<tr>
<td>Velocity of sway</td>
<td>.13</td>
<td>.16</td>
</tr>
<tr>
<td><strong>Day 2 Sinemet</strong></td>
<td></td>
<td></td>
</tr>
<tr>
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<td>.17</td>
<td>.16</td>
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<td>RMS a-p sway</td>
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<td>.30</td>
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<td>Velocity of sway</td>
<td>.14</td>
<td>.19</td>
</tr>
<tr>
<td><strong>Day 1 Deprenyl</strong></td>
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<td></td>
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<tr>
<td>RMS lat sway</td>
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<td>.21</td>
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<td>.46</td>
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<td>Area of sway</td>
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<td>Velocity of sway</td>
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<td>.14</td>
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<td><strong>Day 2 Deprenyl</strong></td>
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<tr>
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Coefficient of variation for subject SR

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<tr>
<td>RMS lat sway</td>
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<tr>
<td>Area of sway</td>
<td>.49</td>
<td>.26</td>
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<td>Velocity of sway</td>
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<td><strong>Day 2 Sinemet</strong></td>
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</tr>
<tr>
<td>RMS lat sway</td>
<td>.16</td>
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<td>RMS a-p sway</td>
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<td>.28</td>
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<tr>
<td>Velocity of sway</td>
<td>.20</td>
<td>.16</td>
</tr>
<tr>
<td><strong>Day 1 Deprenyl</strong></td>
<td></td>
<td></td>
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<tr>
<td>RMS lat sway</td>
<td>.24</td>
<td>.29</td>
</tr>
<tr>
<td>RMS a-p sway</td>
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<tr>
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<td>.12</td>
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<td><strong>Day 2 Deprenyl</strong></td>
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<td>RMS lat sway</td>
<td>.19</td>
<td>.19</td>
</tr>
<tr>
<td>RMS a-p sway</td>
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<tr>
<td>Area of sway</td>
<td>.25</td>
<td>.45</td>
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<tr>
<td>Velocity of sway</td>
<td>.14</td>
<td>.30</td>
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