Intraindividual Variability as a Marker of Neurological Dysfunction:
A Comparison of Alzheimer's Disease and Parkinson's Disease

by

Catherine Louisa Burton
B.A., University of Saskatchewan, 2000

A Thesis Submitted in Partial Fulfillment of the
Requirements for the Degree of

MASTER OF ARTS

In the Department of Psychology

We accept this thesis as conforming
to the required standard

[redacted]
Dr. E. Strauss, Supervisor (Department of Psychology)

[redacted]
Dr. D. F. Hultsch, Departmental Member (Department of Psychology)

[redacted]
Dr. W. J. C. Walsh, Outside Member (Department of Educational Psychology and Leadership Studies)

[redacted]
Dr. J. O. Anderson, External Examiner (Department of Educational Psychology and Leadership Studies)

Supervisor: Dr. Esther Strauss

ABSTRACT

The study of intraindividual variability involves measuring fluctuations in an individual's performance across short intervals of time (e.g., minutes, day, weeks). Several researchers have suggested that intraindividual variability may be a behavioural marker of compromised neurobiological mechanisms associated with aging, disease, and injury (Bruhn & Parsons, 1977; Li & Lindenberger, 1999). For example, older adults and individuals with certain neurological conditions, such as traumatic brain injuries, epilepsy, and Alzheimer's disease (AD), have been shown to demonstrate greater intraindividual variability in cognitive and/or physical functioning than younger adults or healthy controls (Anstey, 1999; Bruhn & Parsons, 1977; Ferrandez, Durup, & Farioli, 1996; Hultsch, MacDonald, Hunter, Levy-Bencheton, & Strauss, 2000; Strauss, MacDonald, Hunter, Moll, & Hultsch, in press). The present study sought to investigate whether intraindividual variability is associated with general nervous system compromise or certain types of neurological disturbances by comparing 3 groups of older adults: healthy adults, and adults with either AD or Parkinson's disease (PD), two neurodegenerative disorders with differing neuropathology. Following an intake session, participants were assessed weekly on 4 separate occasions on a battery of cognitive (i.e., reaction-time, episodic memory), physical (i.e., grip strength, finger dexterity, blood pressure, pulse, respiratory functioning, gait), and affective/stress measures (i.e., affect, stress, pain, perceived competence, locus of control). Results indicated that on measures of cognitive functioning, the AD group demonstrated greater intraindividual variability in latency than the PD group, which in turn demonstrated greater variability than the healthy

group. On a measure of left finger dexterity, the AD group was significantly more variable than both the healthy and PD groups. However, intraindividual variability tended to be significantly correlated with severity of impairment and controlling for group differences in severity of impairment eliminated these significant group differences in intraindividual variability. Correlational analyses indicated that intraindividual variability was also related to variability on other tasks, as well as to level of performance on other tasks, particularly within the cognitive domain. These findings provide further support for an association between intraindividual variability and neurological dysfunction, in particular suggesting that intraindividual variability may be primarily associated with severity of neurological dysfunction, regardless of the nature of neurological compromise.

Examiners:

[redacted]

Dr. E. Strauss, Supervisor (Department of Psychology)

[redacted]

Dr. D. F. Hultsch, Departmental Member (Department of Psychology)

[redacted]

Dr. W. J. C. Walsh, Outside Member (Department of Educational Psychology and Leadership Studies)

[redacted]

Dr. J. O. Anderson, External Examiner (Department of Educational Psychology and Leadership Studies)

Table of Contents

List of Tables

List of Figures

Acknowledgements

I would like to express my sincere gratitude to my thesis supervisor, Dr. E. Strauss, and to Dr. D. F. Hultsch for the valuable guidance and support they have provided during the course of this research and in the preparation of this thesis. I would also like to thank Dr. A. Moll and Dr. M. Hunter, whose technical assistance was greatly appreciated. Thank you as well to my fellow graduate students and friends for their moral support and the occasional, much needed glass of wine.

Financial assistance from NSERC (PGS A) and from the University of Victoria is gratefully acknowledged.

In addition, I would like to thank my family, Ann, Rich, and Tom, for their continual support and encouragement, in addition to their patience, editorial comments, and comic relief. Finally, thanks to Scott Barron, who gave me the courage to pursue this degree and whose love, support, and understanding, has carried me through it.

Chapter 1

Introduction

Recent research has called into question the common practice of using a single measurement to represent an individual's cognitive status in a given domain. This caution stems from a number of studies that have found that, at least for some populations, an individual's cognitive performance fluctuates within a task on a single occasion and on the same task administered on multiple occasions over short intervals of time, e.g., hours, days, weeks (Fozard, Vercruyssen, Reynolds, Hancock, & Quilter, 1994; Stuss et al., 1989; Hultsch, MacDonald, Hunter, Levy-Bencheton, & Strauss, 2000). This phenomenon has been referred to as intraindividual variability.

Fiske and Rice (1955) defined intraindividual variability as the difference between an individual's two responses to the same stimuli, administered at two points in time under the same situation. They suggested that variability in the individual's response, given the same stimuli and situation, is necessarily determined by factors within the individual. According to Nesselroade (1991, 1992), intraindividual variability refers to relatively short-term, reversible changes or fluctuations, sometimes described as states, such as changes in mood or emotion. Intraindividual variability can be differentiated from intraindividual change, which refers to long-term changes that are more or less enduring, associated with, for example, development, learning, or changes in traits. Both forms of change are involved in determining one's behaviour at a given instance of measurement.

Nesselroade (1991) maintains that "intraindividual variability is not just random error or 'noise' but, rather, variability indicative of changes of state in the organism" (p.223). As such, intraindividual variability is believed to be a function of lawful but fluctuating influences on behaviour, independent of systematic effects associated with practice effects, learning to learn, and material effects (Hultsch et al., 2000). Recent research has shown that intraindividual variability is substantial in magnitude and can be reliably measured (e.g., Hertzog, Dixon, and Hultsch, 1992; Li, Aggen, Nesselroade, & Baltes, 2001). Furthermore, recent investigations have shown that intraindividual variability is a relatively stable characteristic of an individual, such that some individuals tend to be more variable across time than others (Hultsch et al., 2000; Li et al., 2001; Rabbitt, 2000; Rabbitt, Osman, Moore, & Stollery, 2001).

The concept of intraindividual variability has been investigated in a wide variety of domains, including cognitive functioning (e.g., Stuss et al., 1989), physical functioning (e.g., Anstey, 1999), affect (e.g., Eid & Diener, 1999), perceived control (Eizenmann, Nesselroade, Featherman, & Rowe, 1997), and worldviews and religious beliefs (Kim, Nesselroade, & Featherman, 1996). Intraindividual variability has even been investigated as a means of detecting malingering (Strauss, Hultsch, Hunter, Slick, & Patry, 2000). The findings from such studies indicate that coherent, systematic patterns of intraindividual variability can be found in many domains of functioning (Kim et al., 1996) and that intraindividual variability represents a potentially important phenomenon for study (Fiske & Rice, 1955; Li et al., 2001; Kim et al., 1996).

Several explanations for intraindividual variability have been proposed. For example, with respect to cognitive functioning, intraindividual variability on reaction

time tasks has been hypothesized to be related to frontal lobe functioning, specifically attentional abilities, with increased intraindividual variability reflecting an inability to sustain the "top-down" effort, or focused attention, required to maintain consistent performance (Stuss & Alexander, 2000; Stuss, Pogue, Buckle, & Bondar, 1994; Stuss et al., 1989). Similarly, Bunce, Warr, & Cochrane (1993) suggested that the intermittent occurrence of particularly slow responses on reaction time tasks represent occasional mental blocks that arise as a result of brief interruptions to an individual's ability to inhibit irrelevant information. Alternatively, Schultz, Kaye, & Hoyer (1984) proposed that intraindividual variability was highly related to flexibility, such that "the greater one's capacity to vary one's response, the greater the potential that exists to do so appropriately and adaptively" or, in other words, that more flexible people show greater intraindividual variability.

Of particular interest to the study at hand is the theory that intraindividual variability may be a behavioural marker of compromised neurobiological mechanisms associated with aging, disease, or injury (Bruhn & Parsons, 1977; Li & Lindenberger, 1999). For example, Eysenck (1982) and Hendrickson (1982) attributed intraindividual variability in reaction time to random errors in the transmission of neural signals in the central nervous system. Li and Lindenberger (1999) further suggested that an increase in random variability in the central nervous system, due to age-related decrements in the perceived competence of neural transmission, might result in the age-related increase in variability evidenced at the behavioural level. Consequently, in accordance with these theories, any neurological condition that potentially affects the fidelity of neural transmission should result in increased variability at the behavioural level. The limited

research conducted to date that has examined intraindividual variability at the behavioural level in the elderly and individuals with neurological conditions supports the theory that increased intraindividual variability may be indicative of compromised neurobiological mechanisms.

Intraindividual Variability in Older Adults and People with Neurological Conditions

A number of studies have investigated age differences in intraindividual variability. Anstey (1999) examined the performance of 180 women, ranging in age from 60 to 90, on reaction time tasks and found that intraindividual variability across trials increased with age. Salthouse (1993) also found an increase with age in intraindividual variability across trials in the reaction time performance of 784 participants, aged 18 to 87. Shammi, Bosman, and Stuss (1998) examined age differences in intraindividual variability on measures of choice reaction time, finger tapping, and a time estimation task administered on two separate occasions. They found that older adults were more variable than the younger adults on the finger-tapping task but not on the time estimation or choice reaction time tasks. Fozard et al. (1994) examined age differences in reaction time in 1265 participants, aged 17 to 96, who were tested on 3 to 5 separate occasions over 8 years. On the basis of cross-sectional analyses, they found that older participants showed greater intraindividual variability within a single occasion than younger participants. In addition, longitudinal analyses revealed that intraindividual variability increased as participants aged. The results of these studies indicate that intraindividual variability in reaction time increases with age.

Intraindividual variability in cognitive performance has also been investigated in individuals with neurological conditions. Bruhn and Parsons (1977) examined reaction

time variability in participants with epilepsy and participants with brain injuries. The participants with epilepsy and those with brain injuries showed significantly greater intraindividual variability within a single occasion than controls, but the two neurological groups did not differ from one another. Bruhn and Parsons (1977) concluded that the similarities in reaction time variability between the two groups suggest a basic disturbance of the central nervous system of both groups. Collins and Long (1996) examined variability in reaction time within a single occasion in traumatic brain injury (TBI) patients with cognitive impairment (indexed by the Halstead-Reitan Battery), TBI patients without cognitive impairment, and normal controls. They found that both TBI groups demonstrated significantly greater intraindividual variability than the controls and that variability positively correlated with impairment scores. In addition, intraindividual variation was better at discriminating between the non-cognitively impaired TBI group and the normal controls than mean reaction time performance.

In addition to observing greater intraindividual variability within a single testing session in individuals with neurological conditions, numerous studies have found evidence of increased intraindividual variability across multiple testing occasions. Stuss et al. (1989) examined the performance of individuals with head injuries on reaction time tasks, assessed across multiple sessions. Head injury patients showed significantly greater variability in performance, both within a session and across occasions, than the controls. In addition, greater variability was associated with a shorter interval since the time of injury. Bleiberg, Garmoe, Halpern, Reeves, and Nadler (1997) and Hetherington, Stuss, and Finlayson (1996) have also reported increased intraindividual variability across occasions in TBI patients. Furthermore, Hetherington et al. (1996) found that

intraindividual variability was greater in patients 5-years post-injury than normal controls and patients 10-years post injury, suggesting recovery in consistency of performance.

Evidence of greater intraindividual variability has also been found in individuals with dementia. Knotek, Bayles, and Kaszniak (1990) examined consistency of responses on a semantic memory task in a group of mildly and moderately impaired individuals with Alzheimer's disease (AD). Participants were administered the Peabody Picture Vocabulary Test twice with a 7-day intertest interval. Participants with AD were significantly more inconsistent in their responses than the normal control group. However, when the effects of guessing were taken into account, only the moderately impaired AD participants were significantly more inconsistent than the controls, with the mild AD participants showing a trend towards increased inconsistency.

Murtha, Cismaru, Waechter, and Chertkow (2002), examined intraindividual variability in individuals with frontal lobe dementia and individuals with AD on various reaction time measures. Participants with AD were found to be more variable than controls on a measure of choice reaction time. On a measure of simple reaction time, there was a tendency for the participants with frontal lobe dementia to be more variable than the controls and AD participants, however the differences were not significant, possibly due to small sample sizes (i.e., 5 participants with Frontal Lobe Dementia; 8 participants with AD).

In order to further investigate the issue of whether intraindividual variability represents a central nervous system phenomenon, Hultsch et al. (2000) compared individuals with neurological impairment (i.e., mild dementia) to individuals with arthritis, who were neurologically intact but experiencing somatic disturbance. The

purpose of including a comparison group of individuals with arthritis was to determine whether intraindividual variability is primarily a central nervous system phenomenon or the result of transient somatic conditions. Participants were tested weekly for 4 sessions on simple reaction time, choice reaction time, word recognition, and story recognition tasks. Hultsch et al. (2000) found that, controlling for mean level of performance and systematic changes associated with practice effects, learning to learn, and material effects, the dementia participants exhibited greater intraindividual variability in response latency than the arthritic group and the healthy controls, both across trials within a single occasion and across multiple occasions for all tasks. The arthritic group and the healthy controls did not differ significantly from one another, indicating neurological dysfunction as the source of increased intraindividual variability rather than somatic disturbances associated with general health problems such as arthritis. In addition, Hultsch et al. (2000) found that individuals who exhibited greater variability on one task were also more variable on other tasks and those who were more variable across trials within occasions were also more variable across occasions. According to Hultsch et al. (2000), these findings suggest that intraindividual variability is a relatively stable characteristic of a person, which is consistent with the hypothesis that intraindividual variability is mediated by neurological dysfunction, rather than other more transient sources, such as pain, stress, or fatigue.

As Strauss, MacDonald, Hunter, Moll, and Hultsch (in press) suggest, if variability in cognitive performance is associated with neurological dysfunction, then one would expect that individuals with neurological conditions would also demonstrate greater intraindividual variability in physical functioning as well. Physical functioning,

like cognitive functioning, is also dependent, at least partially, upon the integrity of the nervous system. Strauss et al. (in press) examined intraindividual variability in cognitive performance and physical status (i.e., balance/gait, peak expiratory flow, blood pressure, finger dexterity) in individuals with a neurological disturbance (mild dementia), individuals with arthritis (i.e., neurologically intact) and healthy controls. Participants with mild dementia showed greater intraindividual variability in physical performance than either of the neurologically intact groups. In addition, both cognitive and physical variability were found to be important predictors of neurological integrity, suggesting that measures of intraindividual variability in both cognitive functioning and physical functioning are behavioural markers of neurological integrity.

Further evidence in support of an association between increased intraindividual variability in physical status and neurological disturbance comes from a study conducted by Nakamura et al. (1997). They examined postural and gait disturbances in a group of individuals with AD, while walking 10 meters 3 times. Individuals with moderate and severe AD showed greater variability in postural sway and stride length compared to controls. In addition, increased variability was associated with a reduction of regional cerebral blood flow in the frontal lobe and basal ganglia. Goldstein, Bartzokis, Hance, and Shapiro (1998) examined the relationship between blood pressure and heart rate and MRI scans of hyperintensities. They found that individuals with more severe white matter lesions demonstrated greater variability in blood pressure while awake. In addition, individuals with more severe insular subcortical lesions exhibited greater heart rate variability while sleeping, again supporting a relationship between variability in physical functioning and neurological integrity.

Furthermore, Ferrandez, Durup, and Farioli (1996) have shown that intraindividual variability in sensorimotor performance also increases with age. They compared variability in gait in healthy elderly adults, between the ages of 61 and 80, to a group of younger adults, ranging in age from 25 to 48. The elderly participants demonstrated greater intraindividual variability in stride length than the younger group when walking at their preferred speed. In addition, Li et al. (2001) recently assessed elderly individuals, ranging in age from 64 to 86, on a variety of walking tasks, tested biweekly for approximately 7 weeks. The magnitude of intraindividual variability was found to positively correlate with age and intraindividual fluctuations were found to be a relatively stable individual attribute. Li et al. (2001) proposed that "one potential source of the behavioural fluctuations in elderly people's sensorimotor performance could be related to age-related declines in neurobiological mechanisms that reduce the fidelity of neural information processing" (p. 26).

In summary, the research conducted to date indicates that intraindividual variability increases with age and that individuals with neurological disturbances exhibit greater intraindividual variability than healthy controls.

Methodological Considerations and Relevance of Intraindividual Variability

Hultsch et al. (2000) discuss a number of issues to consider if intraindividual variability is to be regarded as a behavioural marker of compromised neurological mechanisms. To begin with, it must be shown that there is substantial intraindividual variability in cognitive performance that can be reliably measured independent of systematic effects such as practice effects, learning to learn and material effects. Hertzog et al. (1992) examined memory for structurally equivalent text passages in 7 healthy

elderly women (aged 67 to 83), assessed weekly for up to 2 years. They found that after accounting for material effects and occasion-specific trends, participants demonstrated substantial reliable intraindividual variation in performance. Hultsch et al. (2000) also found substantial variability in performance after removing systematic variation due to practice effects, learning to learn, and material effects. The results of these studies indicate that intraindividual variability can be reliably measured independent of systematic within-person variability.

Furthermore, in order to support the validity of intraindividual variability as a construct, Hultsch et al. (2000) recommended that group differences in mean level of performance be taken into account. For example, if groups differ in mean level of performance, one group may show greater intraindividual variability simply by virtue of having a higher mean level of performance (Hale, Myerson, Smith, & Poon, 1988). Consequently, it is important to rule out the possibility that group differences in intraindividual variability are simply a statistical artefact of average differences in performance. Hultsch et al. (2000) addressed this issue by controlling for mean level of performance and still found group differences in intraindividual variability.

Finally, with respect to the hypothesized relationship between intraindividual variability and central nervous system dysfunction, it is possible that inconsistency in cognitive performance across time may also be associated with fluctuations in affect and mood (Strauss et al., in press). To address this issue, Strauss et al. (in press) compared intraindividual variability in cognitive functioning, physical status, and affect and beliefs in individuals with a neurological disturbance (mild dementia) to individuals with arthritis and healthy controls. Controlling for mean level of performance and systematic

changes over time (e.g., practice effects), Strauss et al. (in press) found that intraindividual variability in cognitive performance for the dementia group was not consistently associated with variability in affective states or beliefs. In addition, variability in affect and beliefs did not make a significant independent contribution to predicting level of cognitive performance, nor was variability in affect (in contrast to physical and cognitive performance) helpful in differentiating neurologically impaired from neurologically intact individuals. According to Strauss et al. (in press), these findings support the hypothesis that variability in cognitive and physical functioning is an indicator of neurological integrity, rather than fluctuations in affect. Similarly, Hertzog et al. (1992) failed to find significant correlations between intraindividual variability in story recall and variability in measures of affective states.

An aggregation of findings from diverse areas of study on intraindividual variability, across areas of cognitive functioning and physical functioning, in populations ranging from elderly individuals to people with TBI, epilepsy, and AD, appears to converge towards the common conception of intraindividual variability as being more than mere measurement error or a statistical artefact. Rather, intraindividual variability appears to represent a stable characteristic of a person dependent upon the integrity of the central nervous system. The implications of these findings are two-fold. First, if an individual's performance is expected to fluctuate across time, a single administration of a task may not produce an accurate representation of the individual's true ability. Instead, multiple assessments may be necessary.

Secondly, intraindividual variability may also serve as a useful and sensitive measure of the degree of neurological disturbance. Based on the findings that

intraindividual variability (1) positively correlated with severity of cognitive impairment in individuals with TBI and (2) discriminated between normal controls and non-impaired TBI participants, Collins and Long (1996) suggested that using measures of intraindividual variability in addition to mean level of performance may provide useful information in clinical settings where distinctions need to made between levels of cognitive functioning in TBI. In addition, Stuss et al. (1989) and Hetherington et al. (1996) found that intraindividual variability in TBI decreased with a longer time since injury, suggesting that intraindividual variability may be sensitive to recovery of function. Finally, the finding of increased variability in individuals with mild dementia (Hultsch et al., 2000), suggests that intraindividual variability may be useful in the early detection of pathological or abnormal changes in the elderly (Hertzog et al., 1992).

Although the body of literature on intraindividual variability is growing, much is still left unknown. For example, Strauss et al. (in press) raise the issue of whether intraindividual variability is associated with general nervous system compromise or, rather, with specific types of neurological disturbances. Investigation of other neurological disorders will provide further insight into the nature of intraindividual variability in cognitive and physical functioning. One possible population with which to investigate this question is individuals suffering from Parkinson's disease (PD), which is an idiopathic, neurodegenerative disorder that affects both motor and cognitive functioning. Although AD and PD share some common clinical and neuropathological features (Perl, Olanow, & Calne, 1998), the two disorders primarily affect different areas of the brain and differ with respect to the presentation and evolution of symptoms (Gómez-Tortosa, Newell, Irizarry, & Hyman, 1998). Comparing the cognitive and

physical functioning of individuals with AD to individuals with PD may help to shed light on the issue of whether intraindividual variability is a marker for certain types of neurological compromise or an indication of general neurological disturbance.

Clinical Features and Pathophysiology of AD and PD

AD is a degenerative neurological disorder characterized by a progressive cortical dementia with memory, language, praxic, gnosic, attention, and executive function deficits (Nebes, 1992; Gómez-Tortosa et al., 1998). In contrast, PD is characterized primarily by various motor abnormalities, including rapid rhythmical shaking of limbs, jaw, and/or tongue that decrease(s) with voluntary movement (resting tremor); muscular rigidity; difficulties initiating movements (akinesia); motor slowing (bradykinesia); lack of facial expression and infrequent blinking (masked facies); slowed, shuffling gait; and postural reflex impairments (Gilroy, 2000; Lezak, 1995; Soukup & Adams, 1996). PD also results in progressive cognitive deterioration. Neuropsychological studies have identified numerous cognitive deficits associated with PD, including impairments in attention, memory and learning, language, visual-spatial abilities, executive function (concept formation and ability to shift sets), and speed of mental processing (Lezak, 1995; Soukup & Adams, 1996). Many individuals with PD eventually develop dementia, at a rate much greater than that of the general population (Soukup & Adams, 1996). According to Gilroy (2000), 50% of individuals with PD develop a progressive subcortical dementia, characterized by executive and visuospatial dysfunction (Gómez-Tortosa et al. 1998).

The predominant clinical feature of AD is progressive cortical dementia, whereas with PD, the primary features are motor abnormalities, with or without subcortical

dementia. These differences in clinical symptomatology are reflective of differences in neuropathology. AD is characterized by a diffuse cortical loss of neurons and synapses as well as the presence of neurofibrillary tangles and senile plaques in the hippocampal formation, entorhinal cortex and the association cortex of the temporal and parietal lobes (Bouras, Giannakopoulos, & Vallet, 1996; Terry, 1996). PD, on the other hand, is characterized by the presence of Lewy bodies in substantia nigra and brainstem nuclei (Gómez-Tortosa et al., 1998; Braak & Braak, 2000) in addition to severe cell loss in the substantia nigra pars compacta (McRitchie, Halliday, Cartwright, Hely, & Morris, 1996; Braak & Braak, 2000). The progressive degeneration of the pars compacta, which synthesizes and provides dopamine to the basal ganglia, causes a depletion of dopamine in the nigrostriatal pathway. The motor symptoms of PD are believed to result from dopamine deprivation in the basal ganglia, a component of the motor system which projects, by way of the thalamus, to the prefrontal motor cortex and other areas of the neocortex (Lezak, 1995). PD also results in cell loss in other areas of the brain, including the cerebral cortex, contributing to other symptoms associated with PD, such as cognitive impairments. The loss of dopamine in the nigrostriatal system is effectively treated, at least initially, with levodopa (LD) and other dopaminergic agonists (Kötter, 1999), with LD being the most commonly used.

Differences in the clinical and pathological features of AD and PD provide an opportunity to further explore whether increased intraindividual variability is specific to certain types of neurological disturbance or common to all neurological disturbances. If individuals with AD and PD show differences in intraindividual variability in cognitive

and motor functioning, these differences may be related to differences in neuropathophysiology.

Motor and Non-Motor Fluctuations in PD

The issue of fluctuations in cognitive and motor performance in individuals with PD has been the subject of study for many years. Research to date has shown that many individuals with PD experience a wide range of fluctuations in their symptoms. Among the various fluctuations experienced by PD patients, fluctuations in motor function are the most commonly researched and best described. Motor fluctuations refer to oscillations in motor function over the course of the day (Nutt & Holford, 1996). In assessing the prevalence of motor fluctuations in PD, Denny & Behari (1999) found that of 80 patients ranging in age from 28 to 78, 50% had developed motor fluctuations after a mean duration of 5 years of illness. In following 125 patients with PD, Caraceni, Scigliano, & Musicco (1991) found that 60% had developed motor fluctuations after 6 years of LD treatment. These findings are consistent with Shaw et al.'s (1980) estimate of a 10% annual incidence of motor fluctuations (as cited in Reardon, Shiff & Kempster, 1999).

According to Colosimo and De Michele (1999) and Nutt and Holford (1996), motor fluctuations can be classified into two groups. Predictable fluctuations, also called "wearing-off phenomenon" or "end-of-dose deterioration", refer to fluctuations in motor performance that are related to the timing of LD administration, such that motor symptoms increase as a result of decreased plasma concentration of LD. Unpredictable fluctuations, also called "on-off phenomenon" or "yo-yoing", are fluctuations in motor function that are not clearly related to LD dosing. With unpredictable fluctuations,

patients experience sudden changes from "on" (normal voluntary movement) to "off" (akinetic, rigid, and tremulous) and vice versa.

As the disease progresses and the duration of treatment increases, the fluctuations experienced by patients with PD tend to progress from predictable to unpredictable fluctuations. According to Quinn (1998), when LD therapy is initially started, patients have minor dose-related motor fluctuations unnoticeable to them. However with time, the response duration of LD decreases and patients begin to notice a gradual wearing off of the beneficial effect of their doses as plasma LD concentrations fall. Eventually, however, the wearing off becomes more abrupt and dramatic. Finally, patients develop an "all-or-none" response to individual doses, at which point a threshold level of dopaminergic stimulation develops. If below the threshold, patients are "off", but if above, they are "on". Fluctuations, thus, tend to evolve from predictable or "wearing off" fluctuations to unpredictable or "on-off" fluctuations.

Other types of motor fluctuations have also been described. Patients with PD commonly show diurnal motor fluctuations, whereby they have a better response to LD in the morning than later in the day (Nutt & Holford, 1996). For some patients this pattern is reversed (Quinn, 1998). Some individuals with PD experience sudden transient freezing (motor blocks) which are short duration motor fluctuations that last seconds to minutes (Quinn, 1998). Freezing usually occurs later in the course of the disease and consists of two distinct types. One type occurs when patients are "off" or wearing off and the other occurs independent of "on-off" status. Another type of motor fluctuation consists of sudden, brief relief of parkinsonian symptoms (paradoxic kinesis) often related to sudden

stress. Fluctuations in motor symptoms have also been reported in female patients with young-onset PD, who experience premenstrual worsening of symptoms (Quinn, 1998).

Various risk factors for developing motor fluctuations have been identified, including younger age of onset, longer duration of illness, greater severity of disease, longer duration of LD treatment, and shorter interval from disease onset to LD treatment initiation (Denny & Behari, 1999; Kostic, Przedborski, Flaster, & Sternic, 1991; Caraceni et al., 1991).

Not only do individuals with PD experience motor fluctuations, but they also experience cognitive, affective, sensory and autonomic response fluctuations (Colosimo & De Michele, 1999). However, non-motor fluctuations have not been studied as frequently, and most of the studies were conducted over a decade ago.

With respect to fluctuations in cognitive functioning, PD patients in the "off" phase, compared to the "on" phase, have been found to demonstrate general disinhibition of language, worsened memory, visual spatial problems (Delis, Direnfeld, Alexander, & Kaplan, 1982), deterioration in reasoning ability (Brown, Marsden, Quinn, & Wyke, 1984), and increased speed of memory scanning (Poewe, Berger, Benke, & Schelosky, 1991). Both Delis et al. (1982) and Brown et al. (1984) found that the fluctuations in cognitive performance experienced by the PD patients were relatively mild compared to the severity of their motor fluctuations. Other studies, however, have failed to find any evidence of cognitive fluctuations in comparing "on" versus "off" status, using measures of verbal memory, verbal fluency, visuospatial orientation, attention, associative conditional learning, conceptual ability, and behavioural regulation (Girotti et al., 1986; Gotham, Brown, & Marsden, 1988).

Fluctuations in mood have also been reported, with patients experiencing a more adverse state of affect and arousal in the "off" phase compared to the "on" phase (Gotham et al., 1988), as well as increases in depression and anxiety (Cantello, Gilli, Riccio, & Bergamasco, 1986; Gunal, Nurichalichi, Turner, Bekiroglu, & Aktan, 2002; Nissenbaum et al., 1987). Researchers have also reported that hallucinations, panic attacks, and moaning and screaming vary with motor fluctuations (Gunal et al., 2002; Riley & Lang, 1993). Some patients with PD experience fluctuations in autonomic symptoms, such as increased blood pressure during "off" phases compared to "on" (Calzetti, 1984), fluctuations in respiratory function (Ilson, Braun, & Fahn, 1983; Jankovic & Nour, 1986), sweating, urinary frequency, nausea, abdominal bloating, belching, facial flushing, salivation, difficulty swallowing, postural light-headedness, cough, and hunger (Gunal et al., 2002; Hillen & Sage, 1996). Furthermore, various sensory fluctuations have been reported, such as pain, paresthesias, akathesia, and restless legs (Gunal et al., 2002; Hillen & Sage, 1996; Riley & Lang, 1993). Although nonmotor fluctuations tend to coincide with motor fluctuations (i.e., they appear during "off" periods), they do not necessarily co-occur, particularly autonomic fluctuations which tend to show variable timing with respect to motor fluctuations (Gunal et al., 2002).

Aside from fluctuations caused by the rise and fall of plasma LD concentrations, the mechanisms underlying most other motor fluctuations are not well understood. A number of models have been proposed regarding the origins of motor fluctuations. One of the most widely accepted models is the presynaptic buffer model, which proposes that a progressive loss of dopaminergic neurons results in an increasing failure to store or buffer dopamine (Kötter, 1999). This model is particularly useful for conceptualizing wearing-

off phenomena, however, it fails to explain unpredictable motor fluctuations (i.e., fluctuations unrelated to drug-intake) (Kötter, 1999). Other models attribute motor fluctuations to postsynaptic mechanisms. For example, some investigators have suggested that motor fluctuations occur as a result of a downregulation or loss of responsiveness of post-synaptic dopamine receptors (Kötter, 1999). Although these models provide interesting hypotheses regarding the mechanisms underlying the origins of motor fluctuations, they are, as of yet, just hypotheses. No model has been proven conclusively and they all lack the ability to account for the entire spectrum of motor fluctuations seen in PD (Kötter, 1999). In addition, these models only pertain to motor fluctuations, failing to address the possible mechanisms responsible for the various cognitive, sensory and autonomic fluctuations associated with PD. Gunal et al. (2002) suggested that sensory fluctuations may be related to dopaminergic mechanisms whereas autonomic fluctuations may reflect the involvement of neuroanatomic structures beyond nigral degeneration. In short, the nature of motor and nonmotor fluctuations is poorly understood.

The prevalence of fluctuations in PD tends to increase with longer duration of LD therapy. This finding has lead many investigators to suggest that motor fluctuations may be the direct result of long-term LD therapy (Lesser et al., 1979; de Jong, Meerwaldt, & Schmitz, 1987). However, others claim that motor fluctuations arise as a result of the natural progression of the disease (Markham & Diamond, 1981; Caraceni, Scigliano, & Musicco, 1991; Denny & Behari, 1999; Reardon et al., 1999). Research comparing the relative contributions of duration of disease and duration of LD treatment on the development of motor fluctuations in PD has yielded conflicting results. However,

according to Cedarbaum, Gandy, and McDowell (1991), studies supporting the role of LD therapy in the development of motor fluctuations have been methodologically flawed (e.g., Lesser et al., 1979; de Jong et al., 1991), failing to control for the rate of disease progression.

For example, Lesser et al. (1979) compared two groups of PD patients, differing in duration of LD therapy, but matched for severity of disease at time of drug initiation and duration of disease at the time of study. They found that patients who had been on LD longer were at increased risk for developing motor fluctuations, consequently supporting the hypothesis that longer duration of LD therapy put patients at increased risk for motor fluctuations. However, as Cedarbaum et al. (1991) point out, Lesser et al. (1979) failed to account for the rate of progression of the disease. Because patients were matched for severity of disease at the time of LD initiation, the patients who began LD therapy sooner after diagnosis had a more rapidly progressing disease than those who started LD later after diagnosis. Consequently, it is possible that the early LD treatment group continued to progress at a more rapid rate than the later treatment group (those who had been on LD for a shorter period of time) following LD initiation. Therefore, it may have been the progression of the disease that put the early LD group at increased risk for developing motor fluctuations, rather than duration of treatment.

A number of studies have found the duration of disease to be the primary determinant of motor fluctuations (Markham & Diamond, 1981; Caraceni, Scigliano, & Musicco, 1991; Denny & Behari, 1999; Reardon et al., 1999). These studies, using prospective, longitudinal designs and controlling for the possible confound of rate of disease progression, have been methodologically stronger than studies finding otherwise.

In summary, many types of fluctuations in functioning have been described in the literature, some types directly relating to LD plasma concentrations (i.e., predictable fluctuations) and others not (i.e., unpredictable fluctuations). Furthermore, the evidence thus far supports the notion that unpredictable motor fluctuations are the result of the natural progression of the disease rather than LD therapy. With respect to intraindividual variability, the fluctuations of interest are those that occur as a result of the natural progression of the disease. Consequently, in order to examine intraindividual variability in individuals with PD, it is important to differentiate fluctuations associated with the disease process from those caused by plasma LD levels. Ideally, this task would be accomplished by assessing participants either when they are off their medication or when they are at their optimal level of medication.

Intraindividual Variability in Parkinson's Disease

Preliminary evidence of increased intraindividual variability in cognitive performance in PD is provided by a study conducted by Crawford, Goodrich, Henderson, and Kennard (1989), in which they examined the reaction time performance and coincidence timing performance (synchronizing a keypress with the onset of a visual signal following a warning signal) of 5 individuals with PD and 6 controls. The PD participants exhibited more variable latencies than the controls on both tasks. Unfortunately, Crawford et al. (1989) did not specify whether or not the PD participants were on their medications during the testing. Reed and Franks (1998) have also found evidence of increased intraindividual variability with increased severity of PD. They tested 12 individuals with PD, on their medications, and 3 controls on two simple motor tasks that involved moving a handle by hand, along a rod, to a target located to one side,

as quickly as possible upon the onset of an auditory signal. They found that the less severe patients tended to show less variability across trials in reaction time and movement time than more severe patients. Interestingly, less severe patients were more variable than more severe patients in terms of peak velocity and peak acceleration.

Findings pertaining to intraindividual variability in physical functioning in individuals with PD have yielded inconsistent findings. Teulings and Stelmach (1993) examined handwriting movements in individuals with PD, elderly adults and younger adults. The PD group demonstrated greater variability in stroke size across trials than the elderly and younger groups. However, Phillips, Stelmach, and Teasdale (1991) also examined handwriting movements in young adults, elderly adults and adults with PD, and failed to find significant differences between groups in intraindividual variability for stroke length or stroke duration.

Blin, Ferrandez, and Serratrice (1990) examined intraindividual variability in gait in individuals with PD and healthy elderly controls. They found that although the PD group did not differ significantly from the control group in terms of variability of stride duration, the PD group demonstrated greater variability in stride length. Conversely, in a study of gait in elderly individuals, Dobbs et al. (1990) did not find that individuals with PD were more variable in stride length than healthy elderly adults. In short, few studies have examined intraindividual variability in PD and those that have, have yielded contradictory results.

However, according to a recently proposed theory (Li and Lindenberger, 1999) one would predict that PD should be associated with an increase in intraindividual variability. Li and Lindenberger (1999) have proposed that catecholamines may play a

central role in age-related decrements in the perceived competence of neural transmission. According to Li and Lindenberger (1999), catecholamines serve as neuromodulators of information processing by enhancing the responsivity of neurons to incoming afferent signals. They suggest that age-related decreases in the concentration of catecholamines, found in the striatum and basal ganglia, may lead to "a noisier (or with a higher level of random variability) information processing system" (p.117). In formulating their theory, Li and Lindenberger (1999) drew upon studies such as that conducted by Schultz et al. (1989) in which decreases in nigrostriatal dopamine neurons in monkeys were found to have lead to increased variability in reaction time.

As previously discussed, PD is also associated with decreased dopamine in the nigrostriatal system. Therefore, as is consistent with Li and Lindenberger's theory (1999), the compromised efficacy of the dopaminergic system in PD should impinge upon the fidelity of neural transmission, consequently leading to increased variability at the level of the central nervous system, as well as the behavioural level.

In summary, increases in intraindividual variability, in both cognitive and physical functioning, have been found in the elderly and people suffering from neurological conditions. Research to date indicates that intraindividual variability is a stable characteristic of a person, possibly reflecting the integrity of the central nervous system. Comparing variability in cognitive function and physical status in individuals with PD and AD, two neurodegenerative disorders with differing clinical features and neuropathology, will provide an opportunity to begin to answer the question of whether increased intraindividual variability is associated with general nervous system compromise or is associated with certain types of neurological disturbances. A greater

understanding of the nature of intraindividual variability and the neurological mechanisms underlying intraindividual variability may have important implications for the clinical assessment of cognitive and physical functioning, for example in the early detection of neurological dysfunction, or simply in providing more accurate assessments of an individual's ability.

Research Questions

The present study sought to address several issues. First, in order to determine whether increased intraindividual variability is associated with general central nervous system dysfunction or specific neurological disturbances, healthy adults were compared to adults with mild dementia and adults with PD on various measures of cognitive and physical performance. If intraindividual variability is primarily a general central nervous system phenomenon, individuals with both neurological disorders should be more variable across measures of cognitive and physical status than healthy adults. However, if greater intraindividual variability is found to be associated with only one neurological disorder, rather than both, then intraindividual variability may be indicative of specific neurological disturbance.

A second question addresses whether neurological dysfunction is associated with greater intraindividual variability in both cognitive and physical performance. If intraindividual variability represents a marker for neurological dysfunction, neurologically impaired individuals should demonstrate greater variability in both domains of functioning than healthy controls. Whether or not both neurologically impaired groups demonstrate this pattern will help to clarify the issue of whether intraindividual variability represents general or specific neurological disturbance.

Thirdly, a related question is centred upon whether intraindividual variability primarily reflects brain-based endogenous mechanisms (ie., neurological dysfunction) or, rather, states that are more dependent on external environmental conditions such as shifts in affect, perceived competence, stress or pain. To the extent that intraindividual variability represents a behavioural marker for neurobiological functioning, variability in cognitive performance should be more strongly associated with variability in physical performance than variability in affective states or beliefs. Furthermore, mean level of cognitive performance should be more strongly associated with intraindividual variability in physical performance than intraindividual variability in affective states or beliefs.

A fourth question is whether intraindividual variability on one task is related to overall level of performance on that and other tasks (both within and across domains). These last two questions will also help to shed light on the nature of intraindividual variability. If intraindividual variability primarily represents brain-based endogenous mechanisms (i.e., neurological dysfunction) as opposed to exogenous influences (e.g., fluctuations in affect or pain), one would expect relatively stable individual differences in variability as well as relatively strong relationships between measures of intraindividual variability and level of performance.

A final question is focused on whether intraindividual variability is a relatively stable characteristic of an individual. Are some individuals consistently more variable than others, within the same domain of functioning and different domains of functioning? For example, are individuals who are more variable on one task also more variable on other tasks in the same domain of functioning as well as tasks in other domains of functioning?

Chapter 2

Method

Participants

Data from a total of 35 participants (18 women and 17 men), ranging in age from 54 to 86 ($\underline{x}$ = 73.09, $\underline{SD}$ = 7.67), were analyzed in the present study. Data collected for the purposes of the present study (i.e., the PD participants) were compared to data previously collected and reported by Hultsch et al. (2000) and Strauss et al. (in press; i.e., AD and healthy participants).

Participants were categorized into 3 groups on the basis of health status. The first group consisted of 10 individuals (4 women, 6 men) with idiopathic PD, diagnosed by a neurologist. Disease severity was assessed by the neurologist using the Hoehn and Yahr scale (Hoehn & Yahr, 1967): five of the PD participants were classified at Stage 1 (i.e., unilateral involvement only, usually with minimal or no functional impairment), four were classified as Stage 2 (i.e., bilateral or midline involvement, without impairment of balance), and one was classified as Stage 3 (i.e., impaired righting reflex, some functional impairment). Two of the 10 PD participants experienced motor/non-motor fluctuations. All participants were receiving dopamine replacement therapy, although, one participant did not commence drug therapy until after the third week of testing. Participants were tested during their normal medication cycle when they were optimally medicated, which was identified as the time of day that participants felt that their functioning, both physical and mental, was most stable. None of the PD participants showed signs of cognitive impairment, as assessed by the Mini Mental Status Examination (MMSE; Folstein,

Folstein, & McHugh, 1975), or depression, as assessed by the Geriatric Depression Scale (Yesavage et al., 1983).

The second group consisted of 10 individuals (4 women, 6 men) all of whom were diagnosed by their physician as having mild dementia according to the NINCDS-ADRDA diagnostic criteria for possible or probable AD (McKhann et al., 1984). The third group consisted of 15 healthy adults (10 women, 5 men) with no history of major medical, neurological, or psychiatric conditions.

Exclusionary criteria for all groups included a history of significant head injury (defined as loss of consciousness for more than five minutes), other neurological or major medical illnesses (e.g., heart disease, cancer), severe sensory impairment, extensive drug or alcohol abuse, inpatient psychiatry treatment, or a MMSE score less than 18 (dementia group) or 26 (PD and healthy groups).

All participants resided in the community. Participants diagnosed with mild dementia and PD were recruited from neurological and geriatric services. The healthy adults were recruited through newspaper and radio advertisements.

During an initial intake interview, participants provided demographic and self-reported health information, in which they were asked to rate their current state of health and their current state of health compared to other people their age, on a scale of 1 to 5, with 1 indicating very poor and 5 indicating very good. In addition, several benchmark cognitive measures were obtained including the MMSE, the Wechsler Adult Intelligence Scale-III (WAIS-III) Block Design and Vocabulary subtests (Psychological Corporation, 1997), and the North American Adult Reading Test (NAART; Blair & Spreen, 1989). Estimates of full-scale IQ (FSIQ) were computed based on the age-adjusted Block-

Design and Vocabulary subtests (Sattler & Ryan, 1999), and premorbid IQ based on the NAART (Blair & Spreen, 1989). Blair and Spreen's (1989) formula for estimating premorbid intellectual ability using the NAART is: $NAART_{estIQ} = 127.8 - .78 (NAART_{errors})$. This formula is based on the WAIS-R, whereas the present estimate of current IQ is based on the WAIS-III which is somewhat more difficult. Thus, the discrepancy between the NAART estimate and the WAIS-III estimate will, if anything, slightly overestimate cognitive decline.

Table 1 shows the age, education, self-reported health, and benchmark cognitive status of the participants as a function of group. Significant overall differences among the groups, using one-way ANOVAs were observed for Age: $F(2,32) = 8.86$, $p < .01$; Self-reported health: $F(2,32) = 6.93$, $p < .01$; Self-reported health compared to others: $F(2,32) = 6.11$, $p < .01$; MMSE: $F(2,32) = 30.65$, $p < .001$; NAART Errors: $F(2,32) = 4.88$, $p < .05$); WAIS-III Block Design: $F(2,32) = 21.00$, $p < .001$; WAIS-III Vocabulary: $F(2,32) = 11.55$, $p < .001$; NAARTIQ: $F(2,32) = 4.88$, $p < .05$; and Estimated Full-Scale IQ $F(2,32) = 25.33$, $p < .001$. Posthoc contrasts using Tukey's HSD ($p < .05$) indicated that the PD group was younger than the healthy and AD groups. The PD group rated their health in general and in comparison to others as poorer than the healthy and AD groups. For the cognitive benchmark variables, the AD group: obtained lower MMSE scores than the healthy and PD groups; made more errors on the NAART than the healthy group; had a lower Estimated NAART IQ than the healthy group; and performed more poorly on WAIS-III Vocabulary than the healthy group. With respect to the WAIS-III Scaled Block and the WAIS-III Estimated IQ, all three groups differed significantly from one another

with the AD group obtaining the lowest scores and the healthy group obtaining the highest scores.

Table 1

Demographic and Performance Characteristics as a Function of Group

	Group		
Variable	Healthy	AD	PD
Age			
M	**75.00**	**77.20**	**66.10**
SD	**5.01**	**5.20**	**8.76**
Years of Education			
M	15.00	11.90	13.40
SD	2.73	3.25	5.52
Self-Reported Health			
M	**4.47**	**4.40**	**3.50**
SD	**0.64**	**0.70**	**0.71**
Health Compared to Others			
M	**4.53**	**4.50**	**3.67**
SD	**0.74**	**0.53**	**0.50**
Chronic Illness [a]			
M	3.73	2.40	4.30
SD	2.74	1.51	1.06
Illness episodes [b]			
M	7.27	6.10	6.50
SD	7.64	5.11	3.54

(table continues)

Table 1 continued

Variable	Healthy	AD	PD
MMSE			
M	**28.93**	**24.20**	**29.40**
SD	**1.03**	**2.70**	**1.08**
WAIS-III Block Design			
M	**13.13**	**7.40**	**10.00**
SD	**2.41**	**2.41**	**1.49**
WAIS-III Vocabulary			
M	**14.53**	**9.10**	**11.90**
SD	**2.41**	**3.28**	**2.77**
Estimated WAIS-III FSIQ			
M	**122.13**	**89.80**	**105.50**
SD	**8.76**	**14.38**	**11.00**
NAART errors			
M	**14.07**	**27.30**	**18.50**
SD	**6.35**	**15.88**	**8.37**
Estimated NAART IQ			
M	**116.83**	**106.51**	**113.37**
SD	**4.95**	**12.38**	**6.53**

Note. [a]Chronic illness consists of self-reported presence of 16 chronic conditions during the past year. [b]Illness episodes consists of the number of self-reported visits to a physician during the past year.

Values in bold indicate significant group differences.

Procedure

The procedure followed in the present study replicated that described by Hultsch et al. (2000) and Strauss et al. (in press). Following an intake interview, participants were assessed on four separate sessions. Participants were tested weekly on a battery of cognitive tasks and state-like indicators of physical and emotional functioning. However, because of scheduling conflicts and holidays, more than 5 weeks were required to complete the assessments. To ensure that participants were tested at the same point in their medication cycle every week, participants were tested at the same time of day every week. To minimize the possible confound of day of week, an effort was made to distribute the testing sessions across days of the week, to the extent permitted by the individual's schedule. Most participants were tested in their homes, but a few individuals were tested at the university.

Cognitive Measures

The cognitive measures consisted of four tasks: two basic reaction time tasks and two recognition memory tasks. All of the tasks were administered on laptop computer, and measures of both latency (in ms) and accuracy (% correct) were obtained. Four alternate versions of each memory task were developed to minimize specific practice effects across occasions. Tasks were administered in a constant order across the four sessions. Hultsch et al. (2000) reported that latency was a more sensitive indicator of intraindividual variability than accuracy. Therefore for the purpose of the present study, only measures of latency were analyzed.

Reaction time. Simple reaction time (SRT) and two-choice reaction time (CRT) tasks were administered. The instructions emphasized speed of performance. In the SRT

task, participants were presented with a warning stimulus (•••) followed by a signal stimulus (+) in the middle of the screen. Participants were instructed to press a key with their preferred hand as quickly as possible when the signal stimulus appeared. A total of ten practice trials followed by 50 test trials were administered. Ten randomly arranged trials were presented at each of five intervals separating the warning and signal stimuli (500, 625, 750, 875, and 1000 ms). The measures used were the latencies of the 50 test trials.

For the CRT, participants received a warning signal consisting of two crosses presented to the left and right of the centre of the screen. After a delay of 1000 ms, one of the crosses changed into a square. The location of the square was randomly equalized across trials. Participants were instructed to press a key corresponding to the location of the square as quickly as possible. A total of 10 practice trials followed by 50 test trials were administered. The measures used were the latencies for the 50 test trials.

Episodic memory. Word and story recognition tasks were used. The instructions for these tasks emphasized accuracy. The word recognition task was based on prior exposure to an uncategorized list of 12 English words. Approximately 15 min after hearing and recalling the 12-word list, participants were presented with a list of 24 words consisting of the 12 previously presented words and 12 new words. The words were presented one at a time on the computer screen, and participants were asked to press one of two keys to indicate whether the word was old (on the previously presented list) or new (not on the previously presented list). Four lists with equivalent word characteristics and connectivity were developed. Words varied in length from 4 to 9 letters and all were above average in rated concreteness and imagery (Paivio, Yuille, & Madigan, 1968). Half

of the words in each list were higher (50+/million) and half were lower 1-49/million) in frequency of occurrence in English. Direct (associations) and indirect (mediated and converging) connections among words within and between each list were minimized using the approach developed by Nelson, Bennett, and Leibert (1997). The measures used were the latencies for all trials.

Story recognition was based on four narrative stories selected from a set of 25 structurally equivalent texts developed by Dixon, Hultsch, and Hertzog (1989). Each story was approximately 300 words and 160 propositions long and related events in the life (lives) of older adults. The stories were recorded on audiotape by a male professional actor. Immediately following presentation of the story, 24 statements about the story were presented one at a time on the computer screen. Participants were asked to press one of two keys to indicate whether each statement represented an idea that was contained in the story (statements presenting a correct idea from the story), or an idea that was not contained in the story (statements presenting an incorrect idea from the story, and ideas consistent with the theme of the story that were not mentioned). There were eight statements of each type. The measures used were the latencies for all trials.

Physical Measures

Physical performance was assessed by measures of balance/gait, fine motor dexterity, grip strength, blood pressure, and respiratory function. The measures were administered prior to the cognitive tasks. Order of administration was held constant across the four test sessions.

Turn 360 task. This measure of balance/gait evaluated the number of steps required to turn 360 degrees and return to the starting position.

Timed walk. The participant was asked to walk, as quickly as possible, a distance of 10 feet, turn around, and return to the starting point. The time to complete the walk was recorded.

Finger dexterity (dominant and nondominant). Fine motor dexterity was tested separately for each hand by having the person touch each of the fingers to their thumb, beginning with the little finger, as quickly as possible. The time to complete three entire sequences was recorded for each hand.

Grip strength. A single reading for each hand from a hand dynamometer was used to measure hand strength.

Blood pressure and pulse. Systolic and diastolic blood pressure and pulse were measured using an electronic, automatic monitor. The measures were taken from above the elbow of the right arm with the individual seated.

Peak expiratory flow. The participant was asked to blow as hard as possible into the mouthpiece of a peak flow meter. The volumes of three successive attempts were averaged and used as the measure.

Self-Perceived Affect and Beliefs Measures

Measures of positive and negative affect, self-perceived competence and control, daily stress, and pain were administered at each of four occasions. These measures were administered prior to the cognitive tasks in a constant order.

Positive and Negative Affect Schedule (PANAS). This questionnaire (Watson, Clark, & Tellegen, 1988) required participants to rate 10 positive and 10 negative affect descriptors (e.g., proud, hostile) on a five-point scale asking how they felt 'right now'. A positive affect score and a negative affect score were generated for each individual by

summing the responses to each of 10 positive affect descriptors and 10 negative affect descriptors, respectively, and dividing each by 10. Internal consistency and test-retest reliability are reported to be high for each scale. The two scales (positive and negative affect) are largely independent of each other and appear to be sensitive to fluctuations in mood (Watson et al., 1988).

Perceived competence and control. Scales of perceived competence (individual's perception of his or her ability to perform actions necessary to achieve desired outcomes) and locus of control (individual's beliefs about whether outcomes are contingent on his or her actions rather than chance, fate, or powerful others) from Eizenman, Nesselroade, Featherman, and Rowe (1997) were used. Participants were asked to respond to eight items (four for competence and four for control) on a 7-point adjective-anchored rating scale according to how they felt now. Higher scores indicate greater perceived competence and internal locus of control. Eizenman et al. (1997) have reported the items to show good test-retest reliability. Confirmatory factor analysis showed invariance of the two-factor model across multiple occasions of measurement spanning 25 weeks.

Daily stress. This questionnaire is a modified version of the Daily Stressors list used by Bolger, DeLongis, Kessley, and Schilling (1989) and it consists of a list of 24 possible stress-inducing events (e.g., a lot of work at job or school, problems with transportation). Participants were required to check off each event that they had experienced during the past 24 hours and rate how stressful the experience was according to a 5-point scale, ranging from not all stressed (1) to extremely stressed (5). Two different scores were generated for each individual: (1) the total number of stressful events encountered in the past 24 hours was calculated by summing the number of events

endorsed, and (2) the average degree of stress experienced in the past 24 hours was calculated by averaging the stress ratings associated with each event that was encountered in the past 24 hours.

Pain Questionnaire. Participants were asked to describe the pain or discomfort felt during the past week using a modified version of the Medical Outcomes Study Pain Measures (MOS; Sherbourne, 1992). The MOS assesses multiple dimensions of pain, including intensity, frequency, duration, and the effect of pain on behaviour/mood. The MOS provides an overall index of pain, as well as 3 subscales (severity, behavioural/mood effects of pain, and the number of days pain interfered with activities). Sherbourne (1992) reported that the subscales exhibited excellent internal-consistency reliabilities and were highly related to other health measures, supporting the construct validity of the scales. For the purposes of the present study, only two scores were utilized: (1) average pain currently experienced on a scale of 1 to 20, and (2) average pain experienced over the past week.

Data Preparation and Statistical Analyses

Following the procedures delineated by Hultsch et al. (2000), outliers are removed, prior to conducting statistical analyses, to eliminate the influence of extremely slow or fast responses (e.g., errors associated with accidental key press or distraction of the participant) in order to minimize error variance. For the cognitive measures, lower limits were set as follows: SRT – 150 ms, CRT – 150 ms, Word Recognition – 300 ms, and Story Recognition – 1,000 ms. Upper limits were established by computing the mean and standard deviation separately for each of the three groups for each occasion and dropping any trials exceeding the mean by three or more SDs.

One participant with PD failed to complete the Story Recognition task on Session 1 and the scales of perceived competence and locus of control on Session 3. To avoid statistical problems associated with missing data, values were imputed for the outlier trials by using a regression procedure in which missing value estimates are based on the relationships among responses across trials and occasions. This strategy will reduce intraindividual variability and therefore represents a conservative approach to examining the phenomenon.

Hultsch et al. (2000) and Strauss et al. (in press) describe a series of steps in the analysis of intraindividual variability. Intraindividual variability refers to within-person variation that is independent of both relatively durable changes (e.g., practice effects) over time and group differences in mean level of performance or report. Therefore, the first step in the analysis of intraindividual variability was the examination of overall level of cognitive performance, physical performance, and affect/stress across occasions. If differences in cognitive performance, physical performance, and/or affect/stress emerge as a function of group, occasion, and possibly their interaction, then it is necessary to purify the data of these systematic effects. To examine group differences in level of performance across occasions, separate Repeated Measures ANOVAs, with Group as a between subjects factor and Occasion varying within subjects, were performed on each of the measures.

The next step in the analysis of intraindividual variability was to statistically remove, or partial, any effects associated with group differences, occasion effects, or the interaction of the two, from the data by regressing each dependent measures on these two variables. This procedure produces residual scores that are uncontaminated by group

differences in cognitive ability, physical ability, or affect/stress. In addition, this procedure partials systematic variation across occasions due to influences such as practice, learning to learn, and material effects (Hultsch et al., 2000; Strauss et al., in press). These purified scores were then converted to T-scores to permit comparison of the tasks in the same metric.

The final step was to compute indices of intraindividual variability. Hultsch et al. (2000) and Strauss et al. (in press) used intraindividual standard deviations (ISDs) and the coefficient of variation (CV) as their indices of intraindividual variability. ISDs and CVs were computed on each of the cognitive, physical and affect/stress scores for each individual across the four occasions. The CV was computed by dividing each individual's ISD by his/her own mean score, which provides a measure of intraindividual variability relative to the individual's level of performance (Hultsch et al., 2000; Strauss et al., in press).

With respect to the present research questions, group differences in intraindividual variability across occasions were examined by Group X Task Repeated Measures ANOVAs conducted for each domain of functioning (i.e., cognitive functioning, physical performance, and affect/stress) on the ISD and CV scores. A series of correlational analyses, using the Pearson correlation coefficient, was performed to examine (1) the relationships among the various measures of intraindividual variability; (2) the relationships among mean levels of performance in one domain (e.g., cognitive performance) and variability in another domain (e.g., physical performance, affect/stress); and (3) relationships between mean levels of performance and intraindividual variability within each task and across tasks within the same domain. In

the interest of preserving power, Fisher's Least Square Difference (LDS) was used to examine posthoc group differences in level of performance.

Chapter 3

Results

Group Differences in Level of Performance Across Occasions

To examine group differences in overall level of performance/report, separate Repeated Measures ANOVAs were performed on each of the cognitive, physical, and stress/affective variables. The mean scores for the three groups are shown in Table 2. For each variable, covariates (i.e., key intake variables correlated with change across time) were identified by conducting Repeated Measures ANOVAs using each key intake variable individually as a covariate. Intake variables that produced significant Session x Covariate effects were then used as covariates in the analyses examining group differences in level of performance (see Table 3). The intake variables examined were: age, gender, total number of years of education, self-reported health, self-reported health compared to others, number of days in hospital in the past year, number of visits to the doctor in the past year, number of sick days in the past year, and number of chronic illnesses.

The analyses of the cognitive variables indicated that there were significant main effects associated with Group for all of the measures [SRT: $F(2,32) = 4.99$, $p < .05$, $\eta^2 = .24$; CRT: $F(2,32) = 8.77$, $p < .01$, $\eta^2 = .35$; Word Recognition: $F(2,30) = 10.97$, $p < .001$, $\eta^2 = .42$; Story Recognition: $F(2,31) = 21.30$, $p < .001$, $\eta^2 = .58$]. For all four cognitive variables, the AD group was slower than the PD group and the healthy group. For Story Recognition, the PD group was also slower than the healthy group. Occasion and Group x Occasion effects were not significant for any of the cognitive variables.

Table 2

Mean Level of Performance as a Function of Group

	Group		
Variable	Healthy	AD	PD
Cognitive			
SRT			
M	**381.43**	**501.59**	**388.63**
SD	**87.90**	**141.43**	**55.86**
CRT			
M	**491.69**	**693.66**	**524.63**
SD	**74.85**	**191.76**	**86.21**
Word Recognition			
M	**1568.97**	**2926.47**	**2135.55**
SD	**259.12**	**909.20**	**740.91**
Story Recognition			
M	**4183.77**	**7335.82**	**5245.25**
SD	**897.10**	**1540.03**	**933.02**
Physical			
Systolic BP			
M	**136.26**	**139.83**	**123.50**
SD	**3.97**	**3.33**	**5.37**
Diastolic BP			
M	79.62	86.28	72.83
SD	3.47	6.23	3.41

(table continues)

Table 2 continued

Variable	healthy	AD	PD
Pulse			
M	70.67	67.38	65.15
SD	3.09	2.57	1.77
Turn 360			
M	6.28	6.65	7.68
SD	.62	.45	2.22
Timed Walk			
M	7.90	8.20	6.77
SD	1.16	.58	.61
Right finger dexterity			
M	4.41	5.15	4.24
SD	.23	.37	.32
Left finger dexterity			
M	**4.15**	**5.89**	**4.52**
SD	**.13**	**.74**	**.44**
Peak expiratory flow			
M	360.29	279.07	425.38
SD	33.89	39.22	44.72
Right grip strength			
M	30.28	31.00	37.80
SD	3.16	3.96	4.39

(table continues)

Table 2 continued

Variable	Healthy	AD	PD
Left grip strength			
M	28.47	31.70	33.95
SD	3.05	4.01	3.86
Affect/Stress			
Positive affect			
M	**3.71**	**3.14**	**2.92**
SD	**.15**	**.19**	**.17**
Negative Affect			
M	1.21	1.11	1.18
SD	.06	.05	.05
Number of daily stressors			
M	4.77	2.03	5.00
SD	1.03	.65	.96
Average degree of daily stress			
M	.21	.08	.23
SD	.05	.04	.06
Locus of control			
M	5.72	5.18	5.15
SD	.28	.17	.24
Self-efficacy			
M	5.75	5.45	5.25
SD	.22	.23	.33

(table continues)

Table 2 continued

Variable	Healthy	AD	PD
Current pain			
M	1.73	1.18	1.75
SD	.88	.52	1.15
Average pain			
M	4.00	4.35	5.15
SD	1.11	1.19	.98

Note. Values in bold indicate significant group differences.

Table 3

Mean Level of Performance Covariates

Variable	Covariates
Cognitive	
SRT	--
CRT	--
Word Recognition	education
	doctor
Story Recognition	gender
Physical	
Systolic BP	education
Diastolic BP	--
Pulse	education
	# of days in hospital in past year
Turn 360	gender
	self-reported health compared to others
Timed Walk	--
Right finger dexterity	--
Left finger dexterity	--
Peak expiratory flow	age
Right grip strength	age
	# of chronic illnesses
	self-reported health compared to others
Left grip strength	--

(table continues)

Table 3 continued

Variable	Covariates
Affect/Stress	
Positive affect	age
	# of chronic illnesses
Negative Affect	age
Number of daily stressors	# of sick days
Average degree of daily stress	# of chronic illnesses
	gender
Locus of control	gender
	# of days in hospital in past year
Self-efficacy	--
Current pain	gender
Average pain	age

The analyses of the physical variables indicated that there were significant main effects associated with Group, Occasion, and Group x Occasion for some of the measures. Group differences emerged with systolic blood pressure, $\underline{F}$ (2,31) = 3.38, $\underline{p}$ < .05, η^2 = .18, such that the PD group had significantly lower systolic blood pressure than both the healthy and AD groups, who did not differ. A significant Occasion effect for systolic blood pressure, F (3,29) = 4.15, $\underline{p}$ < .05, η^2 = .30, indicated that systolic blood pressure on the first occasion was significantly greater than on the third and fourth occasions. Group differences also emerged on the left finger dexterity task, $\underline{F}$ (2,32) = 4.27, $\underline{p}$ < .05, η^2 = .21, in which the AD group was significantly slower than the healthy and PD groups, who did not differ. The Occasion, $\underline{F}$ (3,7.06) = 5.24, $\underline{p}$ < .01, η^2 = .14, and Occasion x Group effects, $\underline{F}$ (6, 3.05) = 2.26, $\underline{p}$ < .05, η^2 = .12, were also significant and indicated that between group differences were only significant for the second occasion. Occasion effects were also obtained for right finger dexterity, $\underline{F}$ (3,30) = 3.31, $\underline{p}$ < .05, η^2 = .25, and peak expiratory flow, $\underline{F}$ (3,93) = 2.83, $\underline{p}$ < .05, η^2 = .08. Participants were significantly slower on the right finger dexterity task on the second occasion compared to the fourth and their peak expiratory flow was significantly greater on the fourth occasion than the first. The Group x Occasion effect for the turn task was significant, $\underline{F}$ (6,54) = 2.70, $\underline{p}$ < .05, η^2 = .21. This interaction reflected a trend toward a decreasing number of steps to turn 360 degrees across occasions for the healthy and AD participants, but not for the PD participants.

The analyses of the affect and stress measures indicated that there were significant group differences for positive affect, $\underline{F}$ (2,30) = 9.51, $\underline{p}$ < .01, η^2 = .39. Healthy

individuals rated their mood more positively than the PD group. No other Group, Occasion, or Group x Occasion effects were significant.

These analyses indicate that systematic differences due to Group, Occasion, and Group x Occasion effects do exist, confirming the need to statistically remove these effects prior to examining group differences in intraindividual variability.

Group Differences in Intraindividual Variability

To examine group differences in intraindividual variability, separate Repeated Measures ANOVAs, with Group as a between subjects factor and task type as a within subjects factor, were performed for each domain of functioning (i.e., cognitive, physical, and affect/stress). Posthoc analyses were conducted using Fisher's Least Square Difference (LSD). Because the PD group was significantly younger than the other two groups and intraindividual variability has been found to increase with age (Anstey, 1999; Fozard et al., 1994; Salthouse, 1993), age was used as a covariate in the Repeated Measures ANOVAs. Figures 1, 2, and 3 present the mean ISDs for the various cognitive, physical, and affect/stress variables, respectively, for each group.

Analyses conducted on the ISD scores for the cognitive variables revealed a significant main effect associated with Group, $F\ (2,31) = 39.98$, $p < .001$, $\eta^2 = .72$; the Task Type effect and the Task Type x Group interaction were not significant. Post hoc analyses (i.e., LSD) indicated that the AD group was significantly more variable than the healthy and PD groups ($p < .001$) and the PD group was significantly more variable than the healthy group ($p < .05$). Analyses with the CV yielded the same results, $F\ (2,31) = 53.17$, $p < .001$, $\eta^2 = .77$. Figure 4 shows an example of the residual T scores for the

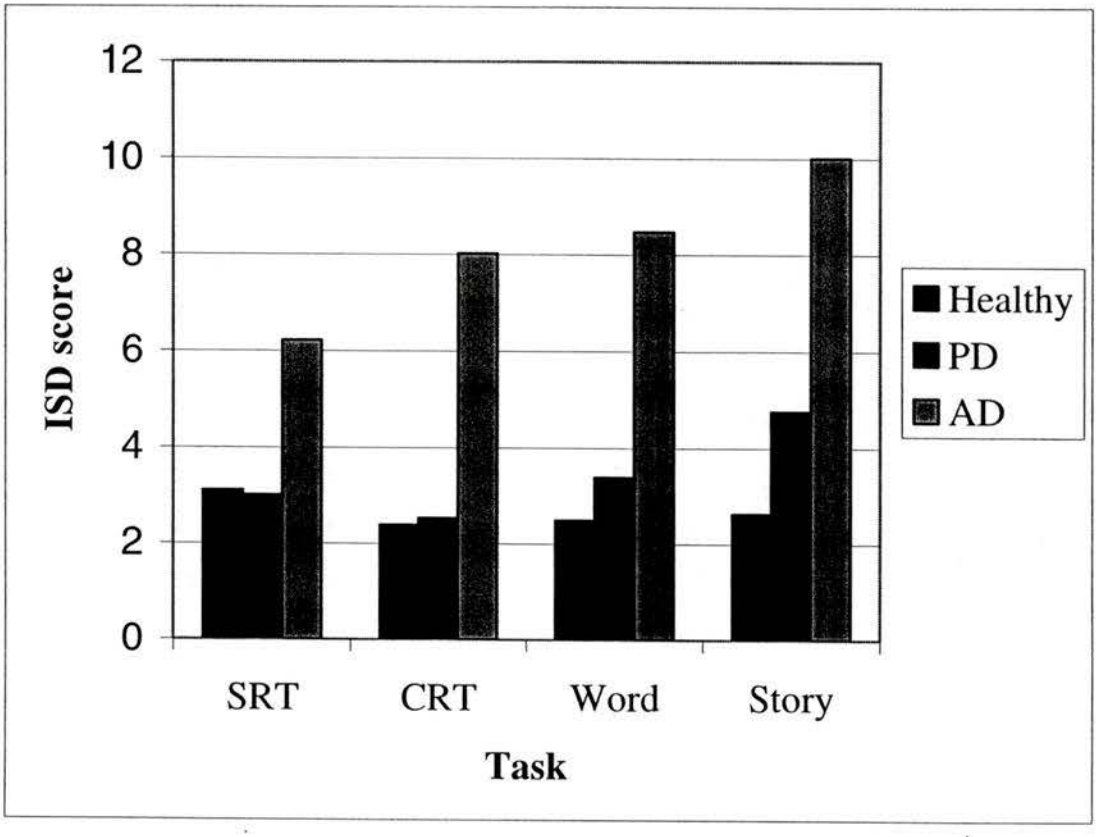

Figure 1. Mean intraindividual standard deviation (ISD) scores on cognitive tasks as a function of group. SRT = simple reaction-time; CRT = two-choice reaction-time task; Word = word recognition task; Story = story recognition task.

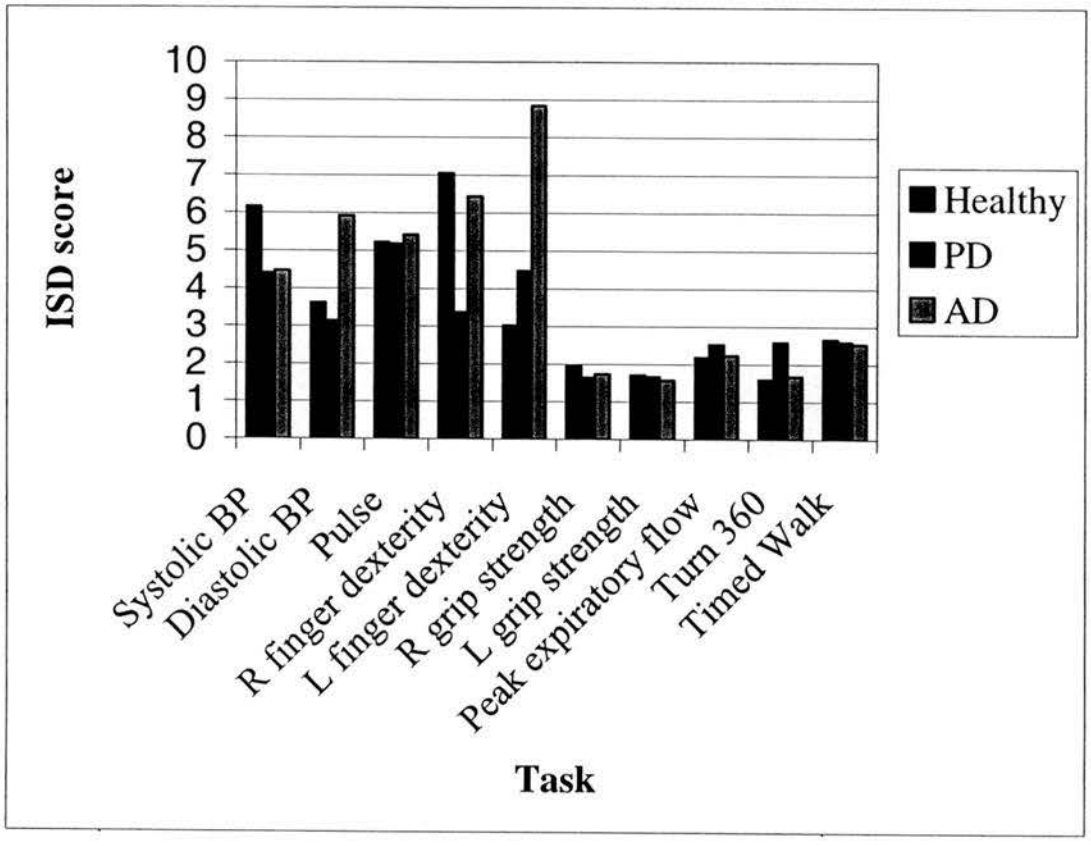

Figure 2. Mean intraindividual standard deviation (ISD) scores on physical tasks as a function of group.

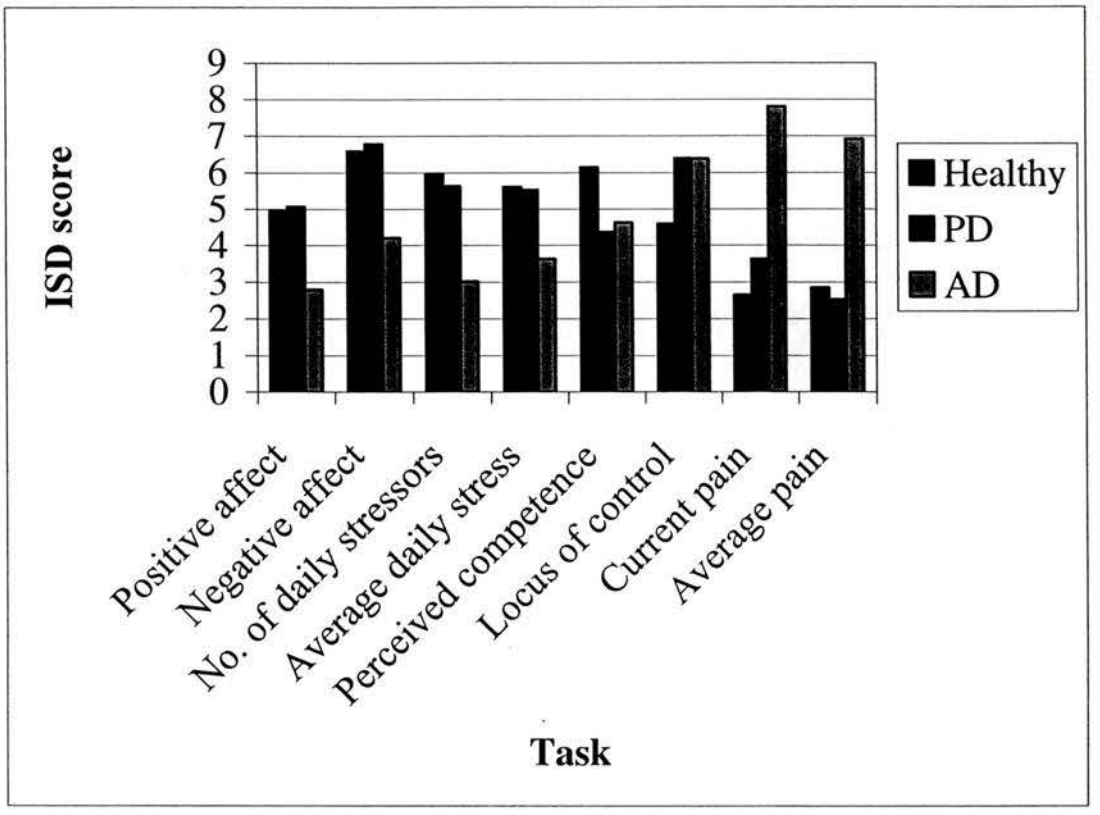

Figure 3. Mean intraindividual standard deviation (ISD) scores on affect/stress tasks as a function of group.

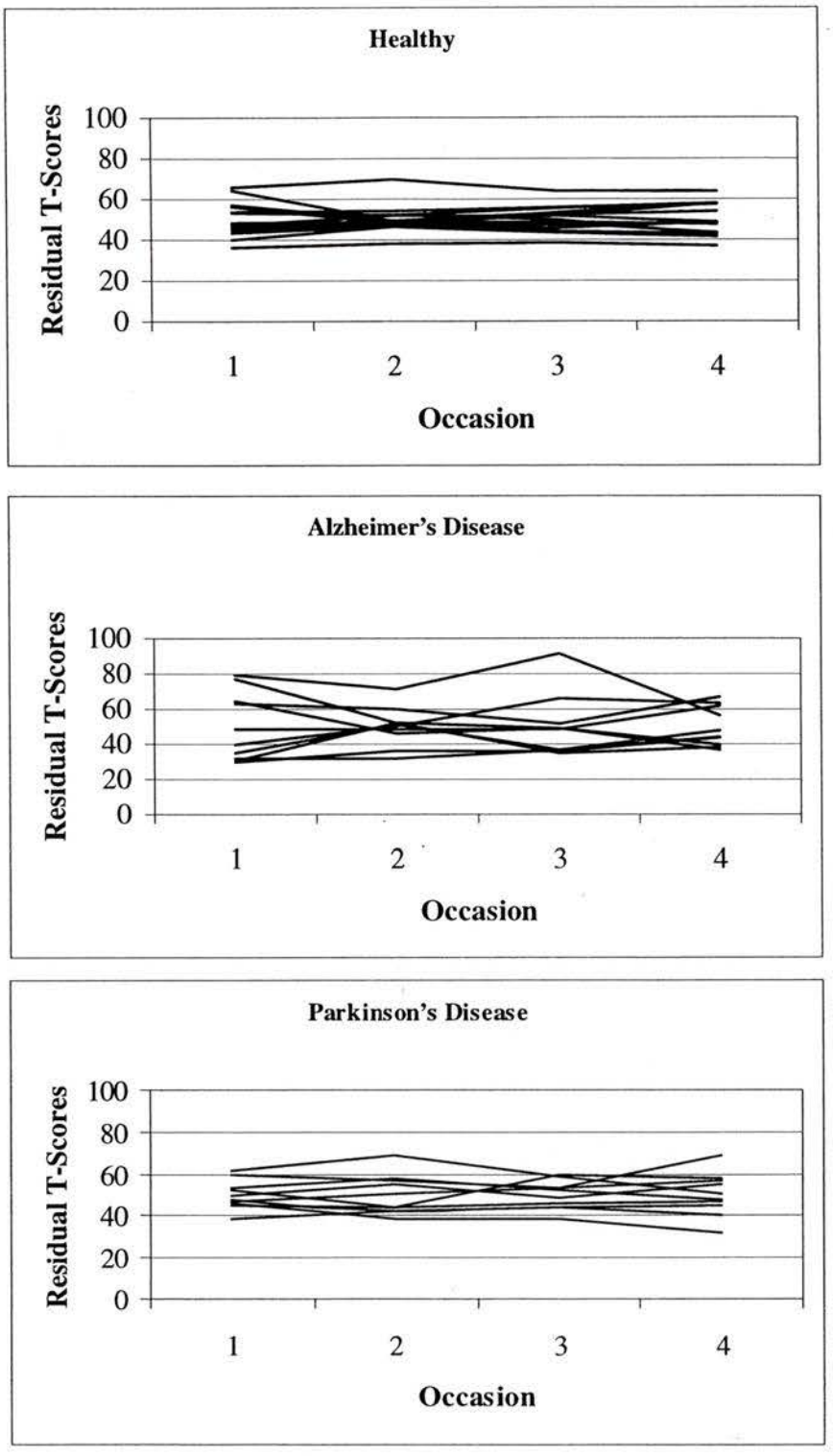

Figure 4. Word recognition residual T scores (purified for group, occasion, and group x occasion effects) for each participant, graphed separately by group.

word recognition task by occasion for each individual participant graphed separately by group. This figure demonstrates each individual's inconsistency across occasions.

To examine whether the increased variability in the PD group, in comparison to the healthy group, was driven by the inclusion of two participants classified as fluctuators, analyses were also repeated with the removal of these two PD participants. The results remained the same, indicating that the non-fluctuating participants were more variable than the healthy participants.

For the physical ISD scores, there were no significant main effects associated with Group or Task Type, but there was a significant Task Type x Group interaction, $\underline{F}$ (18,46) = 2.43, $\underline{p} < .01$, $\eta^2 = .49$, with univariate (i.e., ANOVA) and posthoc analyses (i.e., LSD, $\underline{p} < .05$) indicating that the AD group was significantly more variable than the healthy and PD participants on the left finger dexterity task, $\underline{F}$ (2,31) = 8.95, $\underline{p} < .01$, $\eta^2 = .37$. Analyses with the CV yielded the same results, $\underline{F}$ (18,46) = 2.25, $\underline{p} < .05$, $\eta^2 = .47$.

Analyses of the affect/stress ISD scores revealed a significant Task Type x Group interaction, $\underline{F}$ (14,50) = 2.66, $\underline{p} < .01$, $\eta^2 = .43$, but no significant main effects associated with Group or Task Type. ANOVA and posthoc analyses (i.e., LSD, $\underline{p} < .05$) indicated that the AD group was significantly more variable than the healthy and PD groups in terms of both their current pain and the average pain experienced over the past week, $\underline{F}$ (2,31) = 9.07, $\underline{p} < .01$, $\eta^2 = .37$, $\underline{F}$ (2,31) = 3.88, $\underline{p} < .05$, $\eta^2 = .20$, respectively. Analyses with the CV yielded similar results, with a significant Task Type x Group interaction, $\underline{F}$ (14,50) = 2.59, $\underline{p} < .01$, $\eta^2 = .42$. However, in addition to the AD group being more variable than the other two groups on current pain and average pain ratings, the healthy

group was more variable, as indexed by the CV, than the AD group in the number of experienced daily stressors ($p < .05$).

As previously described, significant group differences were found on a number of the cognitive benchmark variables in the initial analyses. For example, the AD group obtained lower scores on the MMSE and lower estimates of overall intellectual abilities (i.e., estimated FSIQ) than either the healthy or PD groups and the PD group obtained lower estimates of intellectual abilities than the healthy group. Such differences raise the question of whether differences in severity of neurological impairment are related to the group differences in intraindividual variability. To examine this issue, MMSE scores and Estimated FSIQ scores were correlated with all of the cognitive, physical, and affect/stress variables.

Both the MMSE scores and the Estimated FSIQ scores were significantly correlated with all of the cognitive variables, with correlations ranging from -.38 to -.59 (all $p < .05$), indicating that increasing severity of impairment is associated with increased intraindividual variability in cognitive functioning. The MMSE scores and Estimated FSIQ scores were then used as covariates in analyses investigating group differences in intraindividual variability (i.e., AD > PD > healthy on cognitive variables) to determine whether controlling for level of severity would eliminate group differences in intraindividual variability. With respect to the cognitive variables, controlling for differences in MMSE scores did not alter the results for either the ISD scores or the CV scores. Similarly, controlling for the estimated FSIQ produced identical results using the CV scores; however, with the ISD scores, the significant difference between the healthy group and the PD group was eliminated, resulting in a significant difference only between the AD group and the other two groups. These results suggest that differences in severity

of cognitive impairment, as measured by the Estimated FSIQ, may be responsible, at least partially, for group differences in intraindividual variability on cognitive measures.

The analyses of the physical variables indicated that the MMSE was significantly correlated with diastolic blood pressure ($r = -.45$, $p < .01$) and left finger dexterity ($r = -.52$, $p < .01$), and Estimated FSIQ score was significantly correlated with left finger dexterity (r -.54, $p < .01$), again suggesting that increased severity of impairment is associated with greater variability. The analyses of group differences in intraindividual variability were repeated using the MMSE and Estimated FSIQ as covariates. Covarying the MMSE eliminated the significant Type of Task x Group interaction previously found (i.e., greater intraindividual variability in AD group compared to the healthy and PD groups on left finger dexterity), using either the ISD scores or the CV scores. Using the Estimated FSIQ as the covariate also eliminated the significant Type of Task x Group interaction for the ISD scores and CV scores, and in addition, produced a significant Type of Task main effect when using the CV scores, $F(9,22) = 2.60$, $p < .05$, $\eta^2 = .52$. As with the cognitive variables, controlling for severity of impairment eliminated previously found group differences on a measure of physical functioning.

Correlations between MMSE scores and Estimated FSIQ scores and the affect/stress variables revealed significant associations with current pain (MMSE: $r = -.56$, $p < .01$; Estimated FSIQ: $r = -.59$, $p < .001$), average pain in the last week (MMSE: $r = -.66$, $p < .001$; Estimated FSIQ: $r = -.59$, $p < .001$), and locus of control (Estimated FSIQ: $r = -.41$, $p < .05$). Analyses of group differences in intraindividual variability controlling for differences in MMSE scores, eliminated the previous Type of Task x Group interaction (i.e., AD group was more variable than the healthy and PD groups in

terms of current and average pain experienced, and healthy group was more variable than the AD group in terms of number of daily stressors) and produced a significant Type of Task main effect instead [ISDs: $\underline{F}$ (7,24) = 2.73, $\underline{p} < .05$, $\eta^2 = .44$; CVs: $\underline{F}$ (7,24) = 2.97, $p < .05$, $\eta^2 = .46$]. Controlling for differences in Estimated FSIQ scores also eliminated the Type of Task x Group interaction and produced a significant Type of Task main effect [ISDs: $\underline{F}$ (7, 24) = 3.03, $\underline{p} < .05$, $\eta^2 = .47$; CVs: $\underline{F}$ (7,24) = 3.96, $\underline{p} < .01$, $\eta^2 = .54$] as well as a significant Group main effect [ISDs: $\underline{F}$ (2,30) = 4.83, $\eta^2 = .24$; CVs: $\underline{F}$ (2,30) = 4.05, $\underline{p} < .05$, $\eta^2 = .21$], with post hoc analyses (i.e., LSD, $\underline{p} < .05$) indicating that the healthy group was more variable than the AD group (ISDs and CVs) and the PD group (ISDs only). Controlling for differences in severity of impairment also eliminated the group differences in intraindividual variability previously found on affect/stress measures.

In summary, the impact of severity of impairment, as indexed by the MMSE and the Estimated FSIQ, consistently eliminated group differences in intraindividual variability obtained in the original analyses, suggesting that intraindividual variability is related to severity of impairment.

Correlational Analyses

A series of correlational analyses were then performed to examine (a) relationships among the different measures of intraindividual variability, and (b) relationships among mean level of performance and intraindividual variability.

Intercorrelations Between Measures of Intraindividual Variability.

Intercorrelations between the ISD scores for the various tasks within each domain of functioning are shown in Tables 4,5, and 6, respectively. It appears as though, for all three domains, increased variability on many of the tasks is associated with increased

Table 4

Intercorrelations Between Measures of Intraindividual Variability in Cognitive Functioning

Variable	SRT	CRT	Word	Story
SRT	--			
CRT	**.46****	--		
Word	.17	**.52****	--	
Story	.23	**.64****	**.80****	--

** $p < .05$

Table 5

Intercorrelations Between Measures of Intraindividual Variability in Physical Functioning

Variable	Systolic BP	Diastolic BP	Pulse	R finger dexterity	L finger dexterity	R grip	L grip	Peakflow	Turn 360	Timed walk
Systolic BP	--									
Diastolic BP	**.62****	--								
Pulse	.10	.15	--							
R finger dexterity	**.39***	.32	-.12	--						
L finger dexterity	-.03	.24	.10	.11	--					
R grip	.33	.08	-.01	.18	.16	--				
L grip	.02	.03	.24	-.08	.11	-.07	--			
Peakflow	**.43***	.20	.10	**.43****	.19	.09	.05	--		
Turn 360	.28	.21	-.19	.15	.11	**.49****	-.22	.20	--	
Timed walk	.05	.00	-.18	-.01	**.35***	**.41***	-.20	.12	**.61****	--

* $p < .05$, ** $p < .01$

Table 6

Intercorrelations Between Measures of Intraindividual Variability in Affect and Stress

Variable	Positive affect	Negative affect	No. of daily strssors	Average daily stress	Perceived competence	Locus of control	Average pain	Current pain
Positive affect	--							
Negative affect	.20	--						
No of daily stressors	.27	.11	--					
Average daily stress	-.01	**.41***	**.57****	--				
Perceived competence	.20	**.59****	-.13	.21	--			
Locus of control	.26	.24	.02	.14	.22	--		
Average pain	-.21	.17	-.27	.02	.06	.03	--	
Current pain	-.17	.27	-.21	-.10	.17	.03	**.57****	--

* $p < .05$, ** $p < .01$

variability on other tasks within the same domain. For example, Table 4 shows that the CRT, Word Recognition, and Story Recognition tasks were relatively strong predictors of variability on the other cognitive tasks. SRT was relatively a weak predictor, as it was only correlated with CRT. As shown in Table 5, for all of the physical measures, with the exception of pulse and left grip strength, greater intraindividual variability on a particular task was associated with increased variability on at least one other physical measure. Similarly, with the exceptions of positive affect and locus of control, intraindividual variability on each of the tasks within the domain of affective/stress is positively correlated with variability on at least one other task (Table 6).

Tables 7 and 8 show the cross-domain correlations between ISD scores for the cognitive, physical, and affect/stress variables. With respect to cognitive functioning, increased variability on SRT, Word and Story Recognition, was associated with greater variability in diastolic blood pressure and/or left finger dexterity (Table 7). The relationships between variability in cognitive functioning and variability in affect/stress were inconsistent, demonstrating both positive and negative associations between the two domains (Table 8). Of note, intraindividual variability in CRT was not predictive of variability in either the physical domain or affective/stress domain.

As shown in Table 8, intraindividual variability in physical functioning, specifically diastolic blood pressure, left finger dexterity, right and left grip strength, and timed walk, tended to be positively associated with variability on at least one of the affect/stress tasks.

Intercorrelations Between Mean Level of Performance and Variability. Correlations between individual mean level of performance and ISD scores for the

Table 7

Intercorrelations Between Measures of Intraindividual Variability in Physical Functioning and Cognitive Functioning and Affect/Stress

	Variability in Cognitive Functioning			
	SRT	CRT	Word	Story
Variability in Physical Status				
Systolic BP	-.11	-.14	.11	-.02
Diastolic BP	.02	.12	**.57****	**.44****
Pulse	-.01	-.01	.03	.02
R finger dexterity	.12	.11	.04	-.07
L finger dexterity	**.56****	.20	**.35***	**.38***
R grip	.12	-.13	-.11	-.30
L grip	.13	-.24	-.15	-.22
Peakflow	-.13	-.05	-.11	.01
Turn 360	.02	-.08	.16	.09
Timed walk	.28	.02	.01	-.02
Variability in Affect/Stress				
Positive affect	-.05	-.07	-.20	**-.38***
Negative affect	-.17	-.16	-.32	-.26
No. daily stressors	-.25	-.29	-.25	**-.34***
Average daily stress	-.25	-.30	-.28	-.31
Perceived competence	.21	.05	**-.34***	-.30
Locus of control	**.42***	.17	-.05	.07
Average pain	.28	.33	.30	.26
Current pain	.20	.29	.33	**.35***

* $p < .05$, ** $p < .01$

Table 8

Intercorrelations Between Measures of Intraindividual Variability in Cognitive Functioning and Affect/Stress

	Variability in Physical Functioning									
Variability in Affect/Stress	Systolic BP	Diastolic BP	Pulse	R finger dexterity	L finger dexterity	R grip	L grip	Peakflow	Turn 360	Timed walk
Positive affect	.14	-.13	.28	.03	-.18	.06	.29	.17	-.01	.07
Negative affect	.12	-.16	.15	-.13	-.11	.12	**.41***	.29	-.01	-.07
No. daily stressors	.29	.06	-.02	.26	-.21	.10	-.03	.26	.10	.10
Average daily stress	.11	-.10	-.27	.01	.09	.18	-.01	.18	.25	.15
Perceived competence	-.02	-.24	.08	-.11	-.02	**.38***	**.36***	.02	.04	.15
Locus of control	-.02	-.11	.26	-.12	**.41***	.07	-.01	.30	.11	**.36***
Average pain	-.14	.10	-.13	-.02	**.35***	.26	.00	-.09	.00	-.04
Current pain	-.02	**.35***	.24	-.07	.19	.07	.13	-.18	.03	.00

* $p < .05$, ** $p < .01$

cognitive variables, physical variables, and affect/stress variables are presented in Tables 9, 10, and 11, respectively. The tables illustrate, for each domain, the relationship between level of performance and variability for the same task as well as across different tasks within the same domain.

For all of the cognitive tasks (Table 9), greater intraindividual variability was associated with longer response latencies for the same task, as well as for all of the other cognitive tasks.

With respect to the physical domain (Table 10), the overall pattern of correlations was much less robust. Greater intraindividual variability was associated with poorer performance on the same task for the following measures: diastolic blood pressure, right finger dexterity, left finger dexterity, 360 turn, and timed walk. In addition, for a number of tasks, increased intraindividual variability was associated with poorer performance on other physical tasks, with the exception of right grip strength, in which increased variability in right grip strength was correlated with lower pulse.

With regard to the affective/stress domain (Table 11), increased intraindividual variability on many of the affective and stress variables (i.e., negative affect, number of daily stressors experienced, average daily stress, locus of control, and current pain) was associated with poorer functioning (i.e., increased negative affect, stress, pain, and decreased locus of control) on the same task and/or other tasks within the same domain.

In general, and in particular for the cognitive domain, the tables show that increased variability tends to be associated with poorer performance/report both within and across tasks, within the same domain.

Table 9

Intercorrelations Between Mean Level of Performance and Intraindividual Variability in Cognitive Functioning

	Mean Level of Performance			
ISD	SRT	CRT	Word	Story
SRT	**.79****	**.71****	**.43***	**.48****
CRT	**.47****	**.73****	**.35***	**.50****
Word	**.40***	**.50****	**.52****	**.58****
Story	**.43****	**.56****	**.63****	**.78****

* $p < .05$, ** $P < .01$

Table 10

Intercorrelations Between Mean Level of Performance and Intraindividual Variability in Physical Funtioning

	Mean Level of Performance									
ISD	Systolic BP	Diastolic BP	Pulse	R finger dexterity	L finger dexterity	R grip	L grip	Peakflow	Turn 360	Timed walk
Systolic BP	.18	.29	.15	.05	-.16	.17	.16	.12	.07	-.03
Diastolic BP	.24	**.54****	.18	.03	-.02	.02	.03	-.13	.00	-.02
Pulse	.24	**.36***	.26	-.09	.09	.29	.24	.26	-.22	-.10
R finger dexterity	.20	.14	.12	**.39***	.08	.00	-.02	-.20	.12	.03
L finger dexterity	.21	.22	-.05	**.50****	**.71****	.02	.00	-.19	.15	.30
R grip	-.23	-.28	**-.35***	.33	.13	.03	-.04	-.15	**.49****	.19
L grip	.13	.09	.08	-.05	.16	.21	.25	**.35***	-.28	-.17
Peakflow	.07	-.03	.28	**.35***	.22	.27	.24	.15	.18	.06
Turn 360	-.25	-.07	-.10	.02	-.02	-.19	-.23	-.25	**.86****	.26
Timed walk	-.16	-.16	.07	.30	.28	**-.43***	**-.50****	**-.43****	**.81****	**.77****

* $p < .05$, ** $p < .01$

Table 11

Intercorrelations Between Mean Level of Performance and Intraindividual Variability in Affect and Stress

	Mean Level of Performance							
ISD	Positive affect	Negative affect	No. of daily strssors	Average daily stress	Perceived competence	Locus of control	Average pain	Current pain
Positive affect	-.04	.14	.28	.14	-.01	-.08	-.05	.16
Negative affect	.09	**.80****	.32	**.42***	-.14	**-.43****	.05	.06
No of daily stressors	.09	.19	**.74****	**.48****	.22	.09	-.10	.03
Average daily stress	.16	**.39***	**.73****	**.87****	-.19	-.31	.06	-.02
Perceived competence	.24	.30	.05	.20	-.26	-.24	.12	.04
Locus of control	-.31	.23	.06	.11	-.31	**-.59****	-.04	.07
Average pain	.17	.12	-.15	-.06	-.05	-.24	.23	.04
Current pain	.25	.17	-.22	-.20	.12	-.18	**.49****	**.43***

* $p < .05$, ** $p < .01$

The cross-domain correlations between intraindividual variability and mean level of performance are shown in Tables 12, 13, and 14. As shown in Table 12, increased intraindividual variability on the cognitive tasks was associated with poorer functioning on a number of the physical tasks, specifically diastolic blood pressure, right and left finger dexterity, peak expiratory flow, and timed walk. With respect to the affect/stress domain, greater variability in cognitive performance tended to be associated with lower levels of negative affect and stress and lower perceived competence.

Table 13 shows that, out of all the physical measures, intraindividual variability in left finger dexterity was the only predictor of longer latencies on cognitive tasks. However, variability in a number of physical measures was predictive of poorer functioning in the affect/stress domain, suggesting that the cross-domain correlations between variability in physical functioning and mean level of affect/stress were stronger than those with level of cognitive performance.

As shown in Table 14, increased variability in locus of control was associated with longer response latencies in word and story recognition. Conversely, greater intraindividual variability in positive affect was predictive of faster response latencies in story recognition. With respect to physical functioning, greater variability in locus of control was associated with poorer performance on the right and left finger dexterity and timed walk tasks. Conversely, increases in variability in negative affect and pain were associated with better performance on pulse and right and left grip, respectively.

Table 12

Intercorrelations Between Intraindividual Variability in Cognitive Performance and Mean Level of Physical and Affect/Stress Status

	Variability in Cognitive Performance			
Mean Level of Performance	SRT	CRT	Word	Story
Physical Status				
Systolic BP	.11	.15	.32	.20
Diastolic BP	.02	.17	**.56****	**.44****
Pulse	.02	-.02	-.02	.06
R finger dexterity	**.44****	.16	-.01	.01
L finger dexterity	**.52****	.16	.02	.17
R grip	-.21	-.26	-.13	-.14
L grip	-.15	-.21	-.07	-.08
Peakflow	-.27	**-.47****	**-.40***	-.31
Turn 360	.17	.11	.08	.06
Timed walk	**.40***	.28	.05	.05
Affect/Stress Status				
Positive affect	-.17	-.16	-.07	-.26
Negative affect	-.14	-.20	**-.39***	**-.34***
No. of daily stressors	-.26	**-.35***	-.33	**-.43***
Average daily stress	-.28	**-.34***	-.32	**-.38***
Perceived competence	**-.38***	-.05	.07	-.13
Locus of control	-.31	-.09	.08	-.12
Average pain	.19	-.08	.07	.09
Current pain	.18	-.01	-.08	.03

* $p < .05$, ** $p < .01$

Table 13

Intercorrelations Between Intraindividual Variability in Physical Performance and Mean Level of Performance in Cognitive and Affect/Stress Status

	Variability in Physical Functioning									
Mean Level of Performance	Systolic BP	Diastolic BP	Pulse	R finger dexterity	L finger dexterity	R grip	L grip	Peakflow	Turn 360	Timed walk
Cognitive Status										
SRT	-.14	.03	.01	.08	**.42***	-.02	-.01	-.17	.03	.16
CRT	-.11	.10	.04	.19	.28	-.08	-.11	-.17	.01	.01
Word	-.12	.20	.19	.00	**.71****	-.15	.02	.20	-.04	.09
Story	-.12	.24	.08	.04	**.69****	-.16	-.08	.18	.07	.16
Affect/Stress Status										
Positive affect	-.09	-.10	-.03	-.13	-.27	.10	.00	**-.46****	-.15	-.05
Negative affect	.17	-.11	.07	.15	-.05	-.03	.21	.28	-.12	-.06
No. daily stressors	.28	.06	-.11	.33	-.05	.15	.15	**.37***	.15	.01
Average daily stress	.10	-.10	-.25	.11	-.04	.15	.09	.23	.17	.02
Perceived competence	-.07	-.12	.23	.02	**-.36***	-.12	-.20	-.31	**-.43****	**-.41***
Locus of control	-.11	-.09	-.02	.09	**-.36***	-.10	-.21	**-.37***	-.29	-.32
Average pain	-.07	.20	-.05	-.19	.32	-.01	.08	-.15	.09	**.35***
Current pain	-.17	-.04	-.01	-.18	.12	-.10	.03	-.18	.05	**.38***

* $p < .05$, ** $p < .01$

Table 14

Intercorrelations Between Intraindividual Variability in Affect/Stress and Mean Level of Performance in Cognitive and Physical Functioning

	Variability in Affect/Stress							
Mean Level of Performance	Positive affect	Negative affect	No. of daily strssors	Average daily stress	Perceived competence	Locus of control	Average pain	Current pain
Cognitive Status								
SRT	-.02	-.20	-.16	-.25	-.05	.30	.17	.09
CRT	-.07	-.25	-.20	-.29	-.03	.30	.21	.26
Word	-.16	.20	-.21	-.25	-.16	**.48****	.08	.02
Story	**-.33***	-.14	-.23	-.23	-.14	**.41***	.25	.18
Physical Status								
Systolic BP	.31	.01	.11	-.07	.01	-.02	.06	.03
Diastolic BP	.04	-.08	-.03	-.19	-.11	-.17	-.01	.33
Pulse	.15	-.12	.19	-.24	-.18	.02	**-.39***	-.01
R finger dexterity	-.16	-.18	.12	-.05	.01	**.35***	.22	.02
L finger dexterity	-.28	-.11	-.14	-.10	.10	**.46****	.22	.14
R grip	-.02	**.55****	.18	.28	.03	.04	.21	-.05
L grip	-.01	**.47****	.21	.25	-.03	-.05	.23	-.08
Peakflow	.08	.27	.21	.26	.01	-.10	-.17	-.31
Turn 360	.07	-.04	.008	.18	.15	.29	-.01	-.08
Timed walk	.25	-.08	.03	-.07	.17	**.39***	-.03	-.01

* $p < .05$, ** $p < .01$

Chapter 4

Discussion

The present study sought to explore the hypothesis that intraindividual variability represents a behavioural marker of compromised neurobiological mechanisms. In particular, the present study examined whether intraindividual variability is associated with general nervous system compromise or rather specific types of neurological dysfunction by comparing individuals with AD and PD, two neurodegenerative disorders with differing neuropathology.

Intraindividual variability refers to short-term fluctuations in an individual's functioning, independent of any systematic variation due to group differences in mean level of performance, practice effects, learning-to-learn, or material effects associated with alternate versions of the measures. Consequently, such systematic influences must be statistically removed prior to the analysis of intraindividual variability. Group differences in overall mean level of performance emerged on all four of the cognitive measures, revealing longer latencies for the AD group in comparison to the PD and healthy participants. In addition, on the story recognition task, the PD group was slower than the healthy group. Group differences in physical functioning, in addition to changes across testing sessions (in general reflecting improvement across time), and the interaction of the two effects, emerged on various physical measures. With respect to the affective and stress measures, the healthy group reported, on average, more positive affect than the PD group. Such systematic differences, although not unexpected, represent

potential confounds for the analysis of intraindividual variability, and therefore, were partialed from further analyses.

Using the individual standard deviation and the coefficient of variation as indices of intraindividual variability, the AD participants demonstrated greater intraindividual variability than the PD participants on measures of cognitive functioning, both of whom demonstrated greater intraindividual variability than the healthy participants. Such findings appear to provide preliminary support for the premise that intraindividual variability is primarily a general central nervous system phenomenon, as opposed to the result of specific neurological disturbance. However, greater intraindividual variability in both of the neurological groups, compared to the healthy group, was found only within the cognitive domain. The only significant group difference in physical functioning was found for left finger dexterity, in which the AD participants were more variable than the healthy and PD participants, who did not differ significantly. If intraindividual variability represents a marker for general neurological dysfunction, both neurologically impaired groups would be expected to demonstrate greater variability in physical functioning than the healthy controls. Consequently, these findings do not provide a clear answer as to whether or not intraindividual variability is associated with either general or specific types of neurological disturbance.

However, the finding of a relationship between intraindividual variability and severity of impairment shed further light on the nature of intraindividual variability. All of the measures for which significant group differences in variability were found, were found to correlate with indices of severity of impairment (i.e., MMSE scores and Estimated FSIQ). In addition, controlling for differences in severity of impairment

eliminated the previously obtained group differences. These findings suggest a dose-response relationship between neurological disturbance and intraindividual variability: the more severe the neurological disturbance, the greater the variability demonstrated, regardless of the nature of neurological dysfunction. Consequently, the key feature associated with increased inconsistency appears to be severity of impairment as opposed to the specific nature of the neurological disturbance.

Findings from studies of traumatic brain injuries also support an association between intraindividual variability and severity of neurological dysfunction. Collins and Long (1996) and Stuss et al. (1989) found that intraindividual variability in reaction time was related to measures of impairment and injury severity, such that individuals with greater impairment and more severe injuries demonstrated greater inconsistency. In a study of PD participants, Reed and Franks (1998) reported that, on a measure of horizontal arm extension, participants with less severe PD tended to demonstrate less intraindividual variability in reaction time and movement time than participants with more severe PD.

The present findings also provide further support for the more basic hypothesis relating intraindividual variability to neurological compromise, regardless of whether the dysfunction is general or specific. If variability represents a marker of neurological dysfunction, one would expect to observe increased intraindividual variability in both the cognitive and physical domains of functioning. In the present study, at least one of the neurologically impaired groups (i.e., AD) was found to be more variable than the controls on a measure of physical functioning, and both of the neurologically impaired groups were more variable than the controls on cognitive measures. These findings are consistent

with other studies that have found evidence of greater variability in individuals with AD and individuals with PD, compared to healthy controls, on measures of cognitive and physical functioning (e.g., Blin et al., 1990; Crawford et al., 1989; Hultsch et al., 2000; Knotek, 1990; Murtha et al., 2002; Nakamura et al., 1997; Reed & Franks, 1998; Strauss et al., in press; Teulings & Stelmach, 1993).

A set of correlational analyses conducted to shed further light on this issue provided modest support for the hypothesis that intraindividual variability primarily reflects neurological dysfunction as opposed to exogenous factors that are more dependent on environmental factors (e.g., affect, self-perceived competence, locus of control, stress, pain). One set of correlational analyses examined the relationships between different measures of intraindividual variability, with the assumption that, if intraindividual variability reflects neurological compromise, variability in cognitive functioning should be more strongly associated with variability in physical performance than variability in affective states or stress. The analyses revealed that increased variability on three of the cognitive measures was associated with increased variability on two of the physical measures, whereas increased variability in cognitive functioning was associated with either increased or decreased variability on the affective/stress measures, depending on the task. The findings suggest that the relationship between measures of variability may be slightly stronger between the cognitive and physical domains of functioning than between the cognitive and affective/stress domains of functioning. Strauss et al. (in press) also found that inconsistency in physical performance was related to inconsistency in cognitive performance, while inconsistency in cognitive performance was less consistently related to variability in affect/stress.

A second set of analyses, correlating mean level of performance with intraindividual variability, was conducted to examine whether mean level of cognitive performance was more strongly associated with variability in physical functioning than variability in affect/stress. Poorer performance on cognitive measures was associated with greater variability in left finger dexterity in the physical domain, and greater variability in internal locus of control, but less variability in positive affect, in the affective/stress domain. Whether these findings provide support for a stronger relationship between mean level of cognitive performance and variability in physical functioning compared to mean level of cognitive performance and variability in affect/stress is debatable.

Consequently, the analyses of group differences in intraindividual variability provide support for the hypothesis that variability reflects neurological dysfunction, while the correlational analyses provide only modest support, at best. The within-domain correlational analyses, on the other hand, provide additional support for the neurological basis of intraindividual variability. For all three domains of functioning, increased variability on many of the tasks was associated with increased variability on other tasks within the same domain. In addition, for all three domains of functioning, and in particular the cognitive domain, increased variability on a given measure was associated with poorer performance on the same and other measures within the same domain. Consistent with the findings of Hultsch et al. (2000) and Strauss et al. (in press), these results suggest that (1) intraindividual variability is a relatively stable characteristic of an individual (i.e., individuals who are more variable on one task tend to be more variable on other tasks), and (2) there is a relatively strong relationship between measures of

intraindividual variability and level of performance. These findings are to be expected if intraindividual variability reflects relatively stable endogenous mechanisms (i.e., neurological dysfunction) rather than exogenous influences (e.g., pain, fatigue, stress). For example, if levels of performance on cognitive and physical measures are considered to be indicators of neurological integrity, one would expect that individuals with poorer cognitive and physical functioning would be more variable than individuals with higher levels of functioning. In addition, our findings suggest that intra-individual variability reflects severity of CNS disturbance.

Limitations

Although the present study adds to the body of literature elucidating the nature of intraindividual variability, there are a number of limitations that should be considered. One of the most significant limitations of the present study involves the use of PD participants who were medicated at the time of testing. Individuals with PD have been shown to demonstrate fluctuations in their motor and cognitive functioning, as well as their mood, which are believed by some to occur as a natural result of the progression of the disease (e.g., Markham & Diamond, 1981; Caraceni et al., 1991, Denny & Behari, 1999; Reardon et al., 1980). However, individuals with PD also experience fluctuations in their functioning in direct relation to their LD plasma concentrations. In the examination of intraindividual variability, PD participants should, ideally, be tested before initiating LD therapy or, at least while off their medications, to examine variability associated with the disease process as opposed to inconsistency caused by medication levels. However, unless participants are obtained in the very earliest stages of the disease when they are still able to function fairly well without medications, requesting that people

refrain from taking their medications, and thereby causing significant disability, is unrealistic.

We attempted to address the potential problem of testing PD participants while medicated, by testing them while at their optimal level of medication, in order to hold LD plasma concentrations constant. However, the optimal level of medication was determined by self-report only, an indicator of unknown and potentially questionable reliability. Consequently, the present results are potentially confounded by level of LD. Even if participants' self-reports of optimal level of medication successfully held LD plasma concentrations constant, the present results do not reflect the natural disease process. Consequently if possible, testing PD participants in the earliest stages of the disease, before they initiate LD therapy, could provide more accurate information regarding the nature of inconsistency in PD.

A further possible complication of testing PD participants while medicated is apparent when one considers Li and Lindenberger's suggestion (1999) that increased intraindividual variability results from decreases in the concentration of catecholamines. As PD is associated with dopamine depletion, the PD participants were expected to demonstrate greater variability, in both cognitive and physical functioning, than the healthy controls. However, the lack of significant group differences in variability in the physical domain, between the PD group and the control group, is perhaps attributable to the PD participants' medications. It is possible that the dopamine replacement therapy was effectively compensating for the decrease in dopamine, masking any potential differences in variability between the PD participants and the healthy controls. Of note, the PD participants did not perform more poorly on any of the physical tasks compared to

the healthy controls. This finding is unexpected given the motor impairments associated with PD, suggesting that perhaps the medications were effectively compensating for the depletion of dopamine, eliminating differences in variability between the PD participants and the healthy controls. This possibility further reinforces the importance of examining the phenomenon of intraindividual variability in PD in the natural state, that is, while participants are medication-free.

An additional limitation of the present study is the lack of certainty regarding the diagnoses of the participants with AD and PD. The diagnosis of definite Alzheimer's disease requires neuropathological confirmation. Furthermore, there is evidence to suggest that, in some cases, there is overlap between AD and PD in terms of neuropathologic features (Perl et al., 1998). That is, on autopsy, some individuals with AD also show PD pathology, and vice versa. Consequently it is possible that the participants selected for the present study do not necessarily exhibit pure forms of AD or PD, with some participants possessing neuropathologic features associated with both disorders. In addition, Luis, Mittenberg, Gass, and Duara (1999) report that individuals with Dementia with Lewy bodies are often diagnosed with PD in the early stages of the disorder because they present with parkinsonism features, such as bradykinesia and rigidity. Consequently, it is also possible that some of the participants in the PD group may actually have Dementia with Lewy bodies rather than PD.

Future Directions

Given the present findings of a relationship between severity of impairment and intraindividual variability, it would be of interest to examine variability in different neurological groups matched for severity of neurological compromise in order to more

fully investigate the impact of nature of impairment on variability. In addition, longitudinal analyses could examine the trajectory of change in intraindividual variability in individuals in varying stages of disease severity to more fully examine the impact of severity of disease on intraindividual variability. Finally, given the present findings of cross-domain correlations between variability in cognitive and physical functioning, future investigations could also further explore whether the two covary systematically. Although the cross-domain relationships between variability in cognitive functioning and variability in affect and stress were less consistent, this does not rule out the possibility that more transient, exogenous factors, such as pain, fatigue, and affect, contribute to intraindividual variability in performance. Consequently, it would be of interest to also explore whether variability on measures of affect/stress covary systematically with variability in cognitive and physical functioning.

A number of other, more sophisticated, methodologies have been proposed for the study of intraindividual variability. For example, Jones and Nesselroade (1990) and Nesselroade (2001) recommended the use of P-technique factor analysis for studying intraindividual variability, which provides information about intraindividual variability both within and between individuals. This approach, involving factor analysis of longitudinal data, enables one to identify how multiple variables covary across time for a single individual (Jones & Nesselroade, 1990), portraying both concurrent and lagged relationships between sets of variables. Jones and Nesselroade (1990) explained that the factor pattern obtained from a single individual can then be compared with the patterns obtained from other individuals to assess the relative idiosyncrasy or generality of change processes.

Hierarchical linear models (HLM) have also been presented as an appropriate technique for studying intraindividual variability (Tate & Hokanson, 1993; Wu, 1996). This approach also provides information about change processes at both the individual and group level, and in addition, it permits the use of nonsynchronous longitudinal data with variable spacing of observations and unequal numbers of observations for each subject (Wu, 1996). HLM involves analyzing each individual's pattern of change over time and how this change is related to or affected by individual characteristics (Wu, 1996).

Techniques such as the P-technique factor analysis and HLM could potentially be of use in helping to elucidate the nature of intraindividual variability in neurological populations, to identify trajectories of change associated with the evolution of various neurological disorders, and to clarify the role of more transient, exogenous influences on inconsistency.

Conclusions

In conclusion, the present study provides additional information regarding the nature of intraindividual variability. The findings provide further support of an association between intraindividual variability and neurological dysfunction, suggesting that intraindividual variability may be primarily associated with the severity of nervous system dysfunction, regardless of the nature of neurological compromise. However, the present study is merely a starting point for uncovering the neurobiological nature of intraindividual variability, requiring continued research and the accumulation of findings from diverse neurological populations.

References

Anstey, K. J., (1999). Sensorimotor variables and forced expiratory volume as correlates of speed, accuracy, and variability in reaction time performance in late adulthood. Aging, Neuropsychology, and Cognition, 6, 84-95.

Blair, J. R., & Spreen, O. (1989). Predicting premorbid IQ: A revision of the National Adult Reading Test. The Clinical Neuropsychologist, 3, 129-136.

Bleiberg, J., Garmoe, W. S., Halpern, E. L., Reeves, D. L., & Nadler, J. D. (1997). Consistency of within-day and across-day performance after mild brain injury. Neuropsychiatry, Neuropsychology, and Behavioral Neurology, 10, 247-253.

Bolger, M., DeLongis, A., Kessley, R. C., & Schilling, E. A. (1989). Effects of daily stress on negative mood. Journal of Personality and Social Psychology, 57, 808-818.

Bouras, C., Giannakopoulos, P., Vallet, P. G. (1996). Regional distribution of neuropathological changes in Alzheimer's disease. In R. Becker, & E. Giacobini (Eds.), Alzheimer Disease: From Molecular Biology to Therapy (pp. 25-29). Boston: Birkhäuser.

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

Braak, H., & Braak, E. (2000). Pathoanatomy of Parkinson's disease. Journal of Neurology, 247 (Suppl 2), II/3-II/10.

Brown, R. G., Marsden, C. D., Quinn, N., & Wyke, M. A. (1984). Alterations in cognitive performance and affect-arousal during fluctuations in motor function in Parkinson's disease. Journal of Neurology, Neurosurgery, and Psychiatry, 47, 454-465.

Bruhn, P., & Parsons, O. A. (1977). Reaction time variability in epileptic and brain-damaged patients. Cortex, 13, 373-384.

Bunce, D. J., Warr, P. B., & Cochrane, T. (1993). Blocks in choice responding as a function of age and physical fitness. Psychology and Aging, 8, 26-33.

Calzetti, M. B. (1984). Fluctuation of arterial blood pressure during end-of-dose akinesia in Parkinson's disease. Journal of Neurology, Neurosurgery, and Psychiatry, 47, 1241-1243.

Cantello, R., Gilli, M., Riccio, A., & Bergamasco, B. (1986). Mood changes associated with "end-of-dose deterioration" in Parkinson's disease: A controlled study. Journal of Neurology, Neurosurgery, and Psychiatry, 49, 1182-1190.

Caraceni, T., Scigliano, G., & Musicco, M. (1991). The occurrence of motor fluctuations in parkinsonian patients treated long term with levodopa: Role of early treatment and disease progression. Neurology, 41, 380-384.

Cedarbaum, J. M., Gandy, S. E., & McDowell, F. H. (1991). "Early" initiation of levodopa treatment does not promote the development of motor response fluctuations, dyskinesias, or dementia in Parkinson's disease. Neurology, 41, 622-629.

Collins, L. F., & Long, C. J. (1996). Visual reaction time and its relationship to neuropsychological test performance. Archives of Clinical Neuropsychology, 11, 613-623.

Colosimo, C. & De Michele, M. (1999). Motor fluctuations in Parkinson's disease: Pathophysiology and treatment. European Journal of Neurology, 6, 1-21.

Crawford, T., Goodrich, S., Henderson, L., & Kennard, C. (1989). Predictive responses in Parkinson's disease: Manual keypresses and saccadic eye movements to regular stimulus events. Journal of Neurology, Neurosurgery, and Psychiatry, 52, 1033-1042.

Denny, A. P., & Behari, M. (1999). Motor fluctuations in Parkinson's disease. Journal of the Neurological Sciences, 165, 18-23.

de Jong, G. J., Meerwaldt, J. D., & Schmitz, P. I. M. (1987). Factors that influence the occurrence of response variations in Parkinson's disease. Annals of Neurology, 22, 4-7.

Delis, D., Direnfeld, L., Alexander, M. P., & Kaplan, E. (1982). Cognitive fluctuations associated with on-off phenomenon in Parkinson disease. Neurology, 32, 1049-1052.

Dixon, R. A., Hultsch, D. F., & Hertzog, C. (1989). A manual of 25 three-tiered structurally equivalent texts for use in aging research (CRGCA Technical Report No. 2). Victoria, BC: University of Victoria, Department of Psychology.

Dobbs, R. J., Charlett, A., Bowes, S. G., O'Neill, C. J. A., Weller, C., Hughes, J., Dobbs, S. M. (1993). Is this walk normal? Age and Ageing, 22, 27-30.

Eid, M., & Diener, E. (1999). Intraindividual variability in affect: Reliability, validity, and personality correlates. Journal of Personality and Social Psychology, 76, 662-676.

Eizenmann, D. R., Nesselroade, J. R., Featherman, D. L., & Rowe, J. W., (1997). Intraindividual variability in perceived control in an older sample: The MacArthur Successful Aging Studies. Psychology and Aging, 12, 489-502.

Eysenck, H. J. (1982). Introduction. In H. J. Eysenck (Ed.), A model for intelligence (pp. 151-196). Berlin: Springer-Verlag.

Ferrandez, A., Durup, M., & Farioli, F. (1996). Slowness, variability, and modulations of gait in healthy elderly. In A.-M. Ferrandez & N. Teasdale (Eds.), Changes in sensory motor behavior in aging (pp.53-88). New York: Elsevier Science.

Fiske, D. W., & Rice, L. (1955). Intra-individual response variability. Psychological Bulletin, 52, 217-250.

Folstein, M., Folstein, S., & McHugh, P. R. (1975). Mini-Mental State: A practical method for grading the cognitive state of patients for the clinician. Journal of Psychiatric Research, 12, 189-198.

Fozard, J. L., Vercruyssen, M., Reynolds, S. L., Hancock, P. A., & Quilter, R. E. (1994). Age differences and changes in reaction time: The Baltimore Longitudinal Study of Aging. Journal of Gerontology: Psychological Sciences, 49, P179-P189..

Gilford, J. (2000). Basic Neurology (3rd ed.). New York: McGraw-Hill.

Girotti, F., Carella, F., Grassi, M. P., Soliveri, P. Marano, R., & Caraceni, T. (1986). Motor and cognitive performances of Parkinsonian patients in the on and off phases of the disease. Journal of Neurology, Neurosurgery, and Psychiatry, 49, 657-660.

Goldstein, I. B., Bartzokis, G., Hance, D. B., & Shapiro, D. (1998). Relationship between blood pressure and subcortical lesions in healthy elderly people. Stroke, 29, 765-772.

Gómez-Tortosa, E., Newell, K., Irizarry, M., & Hyman, B. T. (1998). Clinical and neuropathological features of dementia with Lewy bodies. American Journal of Alzheimer's Disease, 13, 284-290.

Gotham, A. M., Brown, R. G., & Marsden, C. D. (1988). 'Frontal' cognitive function in patients with Parkinson's disease 'on' and 'off' levodopa. Brain, 111, 299-321.

Gunal, D. I., Nurichalichi, K., Tuncer, N., Bekiroglu, N., & Aktan, S. (2002). The clinical profile of nonmotor fluctuations in Parkinson's disease patients. The Canadian Journal of Neurological Sciences, 29, 61-64.

Hale, S., Myerson, J., Smith, G. A., & Poon, L. W. (1988). Age, variability, and speed: Between-subjects diversity. Psychology and Aging, 3, 407-410.

Hendrickson, A. E. (1982). The biological basis of intelligence Part 1: Theory. In H. J. Eysenck (Ed.), A model for intelligence (pp. 151-196). Berlin: Springer-Verlag.

Hertzog, C., Dixon, R. A., & Hultsch, D. F. (1992). Intraindividual change in text recall of the elderly. Brain and Language, 42, 248-269.

Hetherington, C. R., Stuss, D. T., & Finlayson, M. A. J. (1996). Reaction time and variability 5 and 10 years after traumatic brain injury. Brain Injury, 10, 473-486.

Hillen, M. E., & Sage, J. I. (1996). Nonmotor fluctuations in patients with Parkinson's disease. Neurology, 47, 1180-1183.

Hoehn, M. M., & Yahr, M. D. (1967). Parkinsonism. Onset, progression, and mortality. Neurology, 17, 427-442.

Hultsch, D. F., MacDonald, S. W. S., Hunter, M. A., Levy-Bencheton, J., & Strauss, E. (2000). Intraindividual variability in cognitive performance in the elderly:

Comparison of adults with mild dementia, adults with arthritis, and healthy adults. Neuropsychology,14, 588-598.

Ilson, J., Braun, N., & Fahn, S. (1983). Respiratory fluctuations in Parkinson's disease [abstract]. Neurology, 33 (Suppl 2), 113.

Jankovic, J., & Nour, F. (1986). Respiratory dyskinesia in Parkinson's disease [Letter to the editor]. Neurology, 36, 303-304.

Jones, C. J., & Nesselroade, J. R. (1990). Multivariate, replicated, single-subject, repeated measures designs and P-technique factor analysis: A review of intraindividual change studies. Experimental Aging Research, 16, 171-183.

Kim, J. E., Nesselroade, J. R., & Featherman, D. L. (1996). The state component in self-reported worldviews and religious beliefs of older adults: The MacArthur Successful Aging Studies. Psychology and Aging, 11, 396-407.

Knotek, P. C., Bayles, K. A., & Kaszniak, A. W. (1990). Response consistency on a semantic memory task in persons with dementia of the Alzheimer type. Brain and Language, 38, 465-475.

Kostic, V., Przedborski, S., Flaster, E., & Sternic N. (1991). Early developmental of levodopa-induced dyskinesias and response fluctuations in young-onset Parkinson's disease. Neurology, 41, 202-205.

Kötter, R. (1999). Motor fluctuations in Parkinson's disease: A postsynaptic mechanism derived from a striatal model. Progress in Brain Research, 121, 277-288.

Lesser, R. P., Fahn, S., Snider, S. R., Cote, L. J., Isgreen, W. P., & Barrett, R. E. (1979). Analysis of the clinical problems in parkinsonism and the complications of long-term levodopa therapy. Neurology, 29, 1253-1260.

Lezak, M. D. (1995). Neuropsychological Assessment (3rd ed.). New York: Oxford University Press.

Li, S.-C., Aggen, S. H., Nesselroade, J. R., & Baltes, P. B. (2001). Short-term fluctuations in elderly people's sensorimotor functioning predict text and spatial memory performance: The MacArthur Successful Aging Studies. Gerontology, 47, 100-116.

Li, S.-C., & Lindenberger, U. (1999). Cross-level unification: A computational exploration of the link between deterioration of neurotransmitter systems and dedifferentiation of cognitive abilities in old age. In L-G. Nilsson & H. Markowitsch (Eds.), Cognitive neuroscience and memory (pp.103-146). Toronto: Hogrefe & Huber.

Luis, C. A., Mittenberg, W., Gass, C. S., & Duara, R. (1999). Diffuse Lewy body disease: Clinical, pathological, and neuropsychological review. Neuropsychology Review, 9, 137-150.

Markham, C. H., & Diamond, S. G. (1981). Evidence to support early levodopa therapy in Parkinson disease. Neurology, 31, 125-131.

McKhann, G., Drachman, D., Folstein, M., Katzman, R., Price, D., & Stadlan, E. M. (1984). Clinical diagnosis of Alzheimer's disease: Report of the NINCDS-ADRDA Work Group under the auspices of Department of Health and Human Services Task Force on Alzheimer's Disease. Neurology, 34, 939-944.

McRitchie, D. A., Halliday, G. M., Hely, M. A., & Morris, J. G. L. (1996). Pattern of midbrain pathology in different parkinsonian syndromes. In C. Ohye, M. Kimura, & J. S. McKenzie (Eds.), The Basal Ganglia V (pp.441-444). New York: Plenum Press.

Murtha, S., Cismaru, R., Waechter, R., & Chertkow, H. (2002). Increased variability accompanies frontal lobe damage in dementia. Journal of the International Neuropsychological Society, 8, 360-372.

Nakamura, T., Meguro, K., Yamazaki, H., Okuzumi, H., Tanaka, A., Horikawa, A., Yamaguchi, K., Katsuyama, N., Nakano, M., Arai, H., & Sasaki, H. (1997). Postural and gait disturbance correlated with decreased frontal cerebral blood flow in Alzheimer disease. Alzheimer Disease and Associated Disorders, 11, 132-139.

Nebes, R. D. (1992). Cognitive dysfunction in Alzheimer's disease. In F. I. M. Craik & T. A. Salthouse (Eds.), The handbook of aging and cognition (pp.373-446). Hillsdale, NJ: Erlbaum.

Nelson, D. L., Bennett, D. J., & Leibert, T. W. (1997). One step is not enough: Making better use of association norms to predict cued recall. Memory & Cognition, 25, 785-796.

Nesselroade, J. R. (1991). The warp and the woof of the developmental fabric. In R. M. Downs, L. S. Liben, & D. S. Palermo (Eds.), Visions of aesthetics, the environment, and development: The legacy of Joachim F. Wohlwill (pp. 213-240). Hillsdale, NJ: Erlbaum.

Nesselroade, J. R. (1992). Interindividual differences in intraindividual change. In L. M. Collins & J. L. Horn (Eds.), Best methods for the analysis of change: Recent advances, unanswered questions, future directions (pp. 92-105). Washington, DC: American Psychological Association.

Nesselroade, J. R., (2001). Intraindividual variability in development within and between individuals. European Psychologist, 6, 187-193.

Nissenbaum, H., Quinn, N. P., Brown, R. G., Toone, B., Gotham, A. M., & Marsden, C. D. (1987). Mood swings associated with the "on-off" phenomenon in Parkinson's disease. Psychological Medicine, 17, 899-904.

Nutt, J. G., & Holford, N. H. G. (1996). The response to levodopa in Parkinson's disease: Imposing pharmacological law and order. Annals of Neurology, 39, 561-573.

Paivio, A., Yuille, J. C., & Madigan, S. A., (1968). Concreteness, imagery, and meaningfulness values for 925 nouns. Journal of Experimental Psychology Monograph Supplement, 76 (No.1, Part 2).

Perl, D. P., Olanow, W., & Calne, D. (1998). Alzheimer's disease and Parkinson's disease: Distinct entities or extremes of a spectrum of neurodegeneration? Annals of Neurology, 44 (Suppl 1), S19-S31.

Phillips, J. G., Stelmach, G. E., & Teasdale, N. (1991). What can indices of handwriting quality tell us about Parkinsonian handwriting? Human Movement Science, 10, 301-314.

Poewe, W., Berger, W., Benke, T., & Schelosky, L. (1991). High-speed memory scanning in Parkinson's disease: Adverse effects of levodopa. Annals of Neurology, 29, 670-673.

Psychological Corporation. (1997). WAIS-III Technical Manual. San Antonio, TX: Psychological Corporation.

Quinn, N. P. (1998). Classification of fluctuations in patients with Parkinson's disease. Neurology, 51 (Suppl 2), S25-S29.

Rabbitt, P. M. A. (2000). Measurement indices, functional characteristics and psychometric constructs in cognitive aging. In T. J. Perfect & E. A. Maylor (Eds.), Models of cognitive aging (pp. 160-187). New York: Oxford University Press.

Rabbitt, P., Osman, P., & Moore, B. (2001). There are stable individual differences in performance variability, both from moment to moment and from day to day. The Quarterly Journal of Experimental Psychology, 54A, 981-1003.

Reardon, K. A., Shiff, M., & Kempster, P. A. (1999). Evolution of motor fluctuations in Parkinson's disease: A longitudinal study over 6 years. Movement Disorders, 14, 605-611.

Reeds, C. L., & Franks, I. M. (1998). Evidence for movement preprogramming and on-line control in differentially impaired patients with Parkinson's disease. Cognitive Neuropsychology, 15, 723-745.

Riley, D. E., & Lang, A. E. (1993). The spectrum of levodopa-related fluctuations in Parkinson's disease. Neurology, 43, 1459-1464.

Salthouse, T. A. (1993). Attentional blocks are not responsible for age-related slowing. Journal of Gerontology: Psychological Sciences, 48, P263-P270.

Sattler, J. M., & Ryan, J. J. (1999). Assessment of children: Revised and updated Third Edition WAIS-III supplement. La Mesa, CA: Sattler.

Shammi, P., Bosman, E., & Stuss, D. T. (1998). Aging and variability in performance. Aging, Neuropsychology, and Cognition, 5, 1-13.

Schultz, N. R., Kaye, D. B., & Hoyer, W. J. (1984). Intraindividual variability in divergent and convergent thinking: Adult age differences. Educational Gerontology, 10, 109-118.

Schultz, W., Studer, A., Romo, R., Sundström, E., Jonsson, G., & Scarnati, E. (1989). Deficits in reaction times and movement times as correlates of hypokinesia in monkeys with MPTP-induced striatal dopamine depletion. Journal of Neurophysiology, 61, 651-668.

Sherbourne, C. D. (1992). Pain Measures. In A. L. Steward, & J. E., Ware, Jr. (Eds.), Measuring functioning and well-being: The Medical Outcomes Study approach (pp. 220-234). Durham, NC: Duke University Press.

Soukup, V. M., & Adams, R. L. (1996). Parkinson's disease. In R. L. Adams, O. A. Parsons, J. L. Aubertson, & S. L. Nixon (Eds.), Neuropsychology for clinical practice: Etiology, assessment, and treatment of common neuropsychological disorders (pp. 243-276). Washington, DC: American Psychological Association.

Strauss, E., Hultsch, D. F., Hunter, M., Slick, D. J., & Patry, B. (2000). Using intraindividual variability to detect malingering in cognitive performance. The Clinical Neuropsychologist, 14, 420-432.

Strauss, E., MacDonald, S. W. S., Hunter, M., Moll, A., & Hultsch, D. F. (in press). Intraindividual variability in cognitive performance in three groups of older adults: Cross-domain links to physical status and self-perceived affect and beliefs. Journal of the International Neuropsychological Society.

Stuss, D. T., & Alexander, M. P. (2000). Executive functions and the frontal lobes: A conceptual view. Psychological Research, 63, 289-298.

Stuss, D. T., Pogue, J., Buckle, L., & Bondar, J. (1994). Characterization of stability of performance in patients with traumatic brain injury: Variability and consistency on reaction time tasks. Neuropsychology, 8, 316-324.

Stuss, D. T., Stethem, L. L., Hugenholtz, H., Picton, T., Pivik, J., & Richard, M. T. (1989). Reaction time after head injury: Fatigue, divided and focused attention, and consistency of performance. Journal of Neurology, Neurosurgery, and Psychiatry, 52, 742-748.

Tate, R. L., Hokanson, J. E. (1993). Analyzing individual status and change with hierarchical linear models: Illustration with depression in college students. Journal of Personality, 61, 181-206.

Terry, R. D. (1996). Basis of structural Alzheimer disease and some pathogenic concepts. In R. Becker, & E. Giacobini (Eds.), Alzheimer Disease: From Molecular Biology to Therapy (pp. 25-29). Boston: Birkhäuser.

Teulings, H.-L., & Stelmach, G. E. (1993). Signal-to-noise ratio of handwriting size, force, and time: Cues to early markers of Parkinson's disease? In G. E. Stelmach & V. Hömberg (Eds.), Sensorimotor impairment in the elderly (pp.311-327). Dordrecht: Kluwer Academic Publishers.

Watson, D., Clark, L.A., & Tellegren, A. (1988). Development and validation of brief measures of positive and negative affect: The PANAS scales. Journal of Personality and Social Psychology, 54, 1063-1070.

Wu, Y.-W., B. (1996). An application of hierarchical linear models to longitudinal studies. Research in Nursing & Health, 19, 75-82.

Yesavage, J. A., Brink, T. L., Rose, T. L., Lum, O., Huang, V., Adey, M. B., & Leirer, V. O., (1983). Development and validation of a geriatric depression screening scale: A preliminary report. Journal of Psychiatric Research, 39, 37-49.

VITA

Surname: Burton Given Names: Catherine Louisa

Place of Birth: Saskatoon, Saskatchewan, Canada

Educational Institutions Attended:

University of Victoria	2000 to 2002
University of Saskatchewan	1995 to 2000

Degrees Awards:

B.A. (Honours)	University of Saskatchewan	2000

Honours and Awards:

Natural Sciences and Engineering Research Council Postgraduate Scholarship A (NSERC PGSA)	2000 to 2002
Natural Sciences and Engineering Research Council Undergraduate Research Scholarship	2000
University of Saskatchewan Undergraduate Award	1999
University of Saskatchewan Undergraduate Award	1998
Hantelman Humanities Award	1998

Publications:

Burton, C. L., Hultsch, D. F., Strauss, E., & Hunter, M. A. (in press). Intraindividual variability in physical and emotional functioning: Comparison of adults with traumatic brain injuries and healthy adults. The Clinical Neuropsychologist.

Sheerin, A., Burton, C. L., Elias, L. J., & Saucier, D. M. (2002). Free viewing perceptual asymmetries for judgment of brightness and quantity: Dependence on stimulus orientation. Brain & Cognition, 48, 347-351.

Conferences:

Burton, C., Strauss, E., Hultsch, E., & Hunter, M. (2002, February). Intraindividual variability in TBI: Physical and emotional functioning. Poster session presented at the annual meeting of the International Neuropsychological Society, Toronto, ON.

Crossley, M., & Burton, C. L. (2001, July). The Saskatchewan Mood Inventory for Individuals with Memory Loss: A clinical tool for early stage assessment. Paper presented at the annual meeting of PSAIGE, Oxford.

Elias, L. J., Burton, C. L., Sheerin, A., & Saucier, D. M. (2002, February). Viewing perceptual asymmetries for color judgments. Poster session presented at the annual meeting of the International Neuropsychological Society, Toronto, ON.

Elias, L. J., Saucer, D. M., Sheerin, A., Burton, C. L. (2001, June). Free viewing perceptual asymmetries for judgment of brightness and quantity: Dependence on stimulus orientation. Poster presentation at the annual meeting of TENNET, Montreal, QC.

UNIVERSITY OF VICTORIA PARTIAL COPYRIGHT LICENSE

Title of Thesis/Dissertation:

Intraindividual Variability as a Marker of Neurological Dysfunction: A Comparison of Alzheimer's Disease and Parkinson's Disease

Author [redacted]

Catherine Louisa Burton

September 6, 2002