FACTORS OF THE GERIATRIC DEPRESSION SCALE THAT MAY DISTINGUISH BETWEEN FOUR COGNITIVE DIAGNOSTIC GROUPS: NORMAL, MILD COGNITIVE IMPAIRMENT, DEMENTIA OF THE ALZHEIMER'S TYPE, AND VASCULAR DEMENTIA

Patricia F. Cornett, A.A.S., B.S., M.S.

Dissertation Prepared for the Degree of

DOCTOR OF PHILOSOPHY

UNIVERSITY OF NORTH TEXAS

December 2009

APPROVED:

James R. Hall, Major Professor
Clifton E. Watkins, Committee Member
Paul L. Lambert, Committee Member
Patricia A. Gwirtz, Committee Member
Vicki Campbell, Chair of the Department of Psychology
Michael Monticino, Dean of the Robert B. Toulouse School of Graduate Studies

Cornett, Patricia F. Factors of the Geriatric Depression Scale that may Distinguish between Four Cognitive Diagnostic Groups: Normal, Mild Cognitive Impairment, Dementia of the Alzheimer's Type, and Vascular Dementia. Doctor of Philosophy (Health Psychology/Behavioral Medicine), December 2009, 63 pp, 7 tables, 16 illustrations, references, 82 titles.

The purpose of the current study was to explore the relationship between cognitive status and depression in a sample of geriatric patients. Participants included 282 geriatric patients ranging in age from 65 to 96 years who were classified according to diagnosis as: DAT, VaD, MCI, and Norm. All were referred for neurocognitive testing from the Geriatric Assessment Program (GAP) at the University of North Texas Health Science Center (UNTHSC) in Fort Worth, Texas. This study sought to identify factor structures for two versions of the GDS using a geriatric sample of cognitively impaired and intact patients. It then compared these factors to each other to determine whether the GDS-15 is truly a shorter version of the GDS-30. These were then compared to a previously determined factor structure. This study explored whether the four-factors of the GDS-30 are able to differentiate cognitive diagnostic groups. Further, this study sought to identify whether the severity of cognitive decline impacted GDS factor score for each of the cognitively impaired groups. Results revealed a two-factor model of the GDS – 15 and a four-factor model with the GDS – 30. The GDS-15 factors did not differ from the first two factors of the GDS-30. Comparison between the GDS-30 factor structure and that reported by Hall and Davis (in press) revealed no significant differences despite the inclusion of a normal, non-demented group in the current study.

Comparisons of subscale scores revealed that DAT patients tended to score lower than the other groups on all but the cognitive impairment subscale. Severity level analyses indicated that as severity of deficits increases, awareness of deficits decreases. This study found that although the GDS-30 is a good screening tool for depression in geriatric patients, it is not particularly useful in differentiating cognitive status group. Also, the GDS-15 was not found to be a good substitute for the GDS-30.

TABLE OF CONTENTS

LIST OF TABLES

Page

LIST OF ILLUSTRATIONS

Page

INTRODUCTION

Americans are living longer, often into their 80s, 90s and 100s (Marson, 2002). With aging come physical and cognitive maladies that often affect quality of life and functional ability. Many of these individuals experience cognitive decline and depression (Korner et al., 2006; Wright & Persad, 2007). In fact, approximately 5 million older Americans are diagnosed with cognitive impairment (Alzheimer's Association, 2008; Caballero, Hitchcock, Beversdorf, Scharre, & Nabata, 2006; Xie & Tanzi, 2006). The prevalence of cognitive impairment is 2% of 65 year olds and doubles every five years afterwards (4% of 70, 8% of 75 year olds, etc.) (Alzheimer's Association, 2008). Depression often co-occurs with cognitive impairment in these individuals. The prevalence of depression in the elderly ranges between 1% and 25%, and is estimated that 25% to 50% of those with cognitive impairment experience depression symptoms (Barnes et al., 2006; Caballero et al., 2006; and Holtzer et al., 2005). Reportedly, 30% to 50 % of those with Alzheimer's disease experience depressive symptoms during the course of the disease process (Starkstein et al., 2005; Zubenko et al., 2003).

Depression is known to adversely affect cognitive functioning (Mehta et al., 2003) and executive functioning (Adams, 2001). Depression has also been reported to greatly affect patient functional ability which adversely impacts quality of life and often increases caregiver burden and caregiver stress (Nakaaki et al., 2007). Often, those with concomitant cognitive impairment and depression use health care services more often (Kunik et al., 2003), thus placing a strain on health care providers (Kaufer et al., 1998) and insurance companies. Depression has also been associated with increased mortality rates within those with mild dementia (Janzing et al., 1999) and was found to

be an independent predictor of mortality when severity, Mini Mental Status Exam (MMSE) score and vascular risk factors were controlled for (Suh et al., 2005).

Purpose and Hypotheses of the Study

The purpose of this study is to explore the relationship between cognitive status and depression in a sample of geriatric patients. The measure of depression that was used in the analyses is the Geriatric Depression Scale (GDS) (Yesavage et al., 1983). This study explored the factor structures of two versions of the GDS, the GDS-30 and the GDS-15. One goal of this study was to explore whether these factors differentiate or are common among certain cognitive diagnostic groups; normal, mild cognitive impairment (MCI), dementia of the Alzheimer's type (DAT), and vascular dementia (VaD). Many studies have been conducted to identify factors of the GDS. These studies have identified from 3 to 6 factors. In the current study, a factor analysis was conducted of both versions of the GDS and compare these factor structures to that found by Hall and Davis (in press). It was hypothesized that the current study would find a different factor structure due to the inclusion of a non-demented, normal comparison group in the analysis, unlike the work of Hall and Davis who studied only cognitively impaired individuals.

This study compared the factor structures of the GDS-15 and the GDS-30. It was hypothesized that the GDS-15 factor structure will be similar to that of the GDS-30 . Additionally, it was hypothesized that specific factors of the GDS-15 as well as factors of the GDS-30 will discriminate between the four participant groups of cognitive functioning.

It was hypothesized that the Apathy subscale, identified by Hall and Davis (in press), will discriminiate patients diagnosed with VaD; while the Dysphoria and Meaningless subscales (Hall & Davis, in press) will identify patients diagnosed with MCI and early to mild DAT, but not moderate DAT. It was hypothesized that the GDS will have little discriminate properties for non-demented, normal cognition patients.

Finally, this study identified individual items of the GDS-15 and the GDS-30 that are strongest for identifying depression in persons in specific cognitive diagnostic groups.

The current study focused on individuals who have been classified into four cognitive status groups: normal cognition, no dementia (NORM), mild cognitive impairment (MCI), Alzheimer's disease (DAT), and vascular dementia (VAD). An Analysis of Variance (ANOVA) was conducted to see if there is a relationship between cognitive status and total depression score. Analyses were conducted to determine the factor structure of the GDS using four cognitive groups for comparison. These factors were compared to the four-factor structure that Hall and Davis (in press) found in which they included only three diagnostic groups (MCI, DAT, and VaD). Analyses were also conducted to evaluate the ability of specific GDS items and/or factors to discriminate between types of cognitive status. This, in turn, may help to determine whether the GDS factors are useful in identifying type of depression, and in identifying the etiology of depression. It may also be found to be useful in identifying type of dementia. If the GDS factors are found to be good discriminators of type of dementia, then it could lead to more precipitous and more effective treatment.

Geriatric Depression Scale

The Geriatric Depression Scale (GDS) was developed specifically for use with geriatric patients (Yesavage, Brink, Rose, Lum, Huang, Adey, & Leirer, 1983) and has been found to effectively assess depressive symptoms regardless of the patient's level of cognitive functioning (Bentz & Hall, 2008), given the impairment is not too severe for the individual to comprehend the questions (Cotter et al., 2003; Yesavage et al., 1983). It is a 30–item questionnaire that requires a dichotomous response pattern, which can either be self-administered or administered by a trained professional (Cotter et al., 2003, Yesavage et al., 1983). Each question counts as one point, with a possible score of 0 – 30 points (Adams, Matto & Sanders, 2004), normal-0-9; mild depressives-10-19; severe depressives-20-30 (Yesavage et al., 1983). Many elderly individuals experience somatic maladies, so the inclusion of these types of items may produce false positives for depression in individuals who otherwise do not have depressive symptoms (Friedman, Heisel & Delavan, 2005; Wancata, Alexandrowicz, Marquart, Weiss & Friedrich, 2006). The GDS differs from some other commonly used depression scales such as the Beck Depression Scale in that it does not contain items concerning somatic complaints (Yesavage et al., 1983) or items of vegetative symptoms such as eating and sleeping behaviors (Bentz & Hall, 2008). The exclusion of these items is believed to make the GDS more appropriate to use with older individuals.

Shorter versions of the GDS have been developed in order to reduce administration time without compromising validity or reliability (Friedman et al., 2005; Wancata et al., 2006). The GDS-15 is one such version that exhibits good correlation with the 30 item GDS (Cotter et al., 2003). The GDS-15 is in the same dichotomous

format as the GDS-30 with questions of subjective depression symptoms experienced during the past week (Friedman, et al., 2005) that are taken from the original 30 – item version. The GDS-30 and GDS-15 reportedly exhibit moderate to strong sensitivity and specificity (Wancata et al., 2006).

The GDS-30 and GDS-15 provide total depression scores that are used to determine if a patient has no depression, mild depression, moderate depression, or severe depression (Yesavage et al, 1983). Merely obtaining a single depression score in order to diagnose depression or determine depressive symptoms is inadequate for effective diagnosis and treatment of these individuals. It is believed that looking at specific factor response patterns and individual item response patterns on this scale may lead to more effective identification and treatment of both cognitive maladies and depressive symptoms. Also, being able to determine the type of depression, affective type versus vascular depression greatly affects treatment considerations.

This study examined different response patterns on the GDS of four common cognitive groups. It is believed that persons with different cognitive status will report/endorse different depressive symptoms and thus will have different response patterns on individual items as well as on different factors of this depression scale.

Factor Analysis of the GDS

The original 30-item GDS was designed to provide a single score to identify depression in the elderly (Adams et al., 2004) although principal components analysis (PCA) and factor analyses have been conducted in order to identify specific depression type. One such analysis conducted by Adams et al. (2004) was conducted on a normal,

non-demented population and estimated a five-factor model of the GDS-30. The five factors that their analysis revealed included: dysphoric mood, hopelessness, withdrawal-apathy-vigor, worry, and cognitive (Adams et al., 2004). These authors proposed these five factors to be used as subscale scores of the GDS in order to specify type of dementia.

Another such analysis was conducted with Turkish elderly using the Turkish translation 30-item GDS (Ertan & Eker, 2000). This analysis originally found seven factors that loaded heavily on Factor 1 with the other six items sharing loading (Ertan & Eker, 2000). Due to this factor structure, these researchers conducted a subsequent factor analysis allowing for only two factors. This analysis resulted in 18 items loading on Factor 1 (depressive affect) and 11 loading on Factor 2 (items describing lack of interest, decrease in performing social and personal activities, and impairment in concentration, memory and decision-making ability) (Ertan & Eker, 2000). This factor structure was reportedly easier to interpret (Ertan & Eker, 2000) but lost the factors that identify individual depression types that may be valuable in identifying specific types of depression that are present in those who convert from MCI to DAT and those that are not.

Hall and Davis (in press) found a four-factor structure consisting of dysphoria, meaninglessness, apathy, and cognitive impairment factors in a sample of community dwelling cognitively impaired older individuals. Further, Hall, Davis, & Cornwell (in review) found that there was no difference in total GDS scores between four groups of older individuals with cognitive impairment, but did find that VaD patients differed from

DAT patients in that they scored significantly higher on the apathy, dysphoria and meaninglessness scales, but not higher than the MCI patients

The GDS-S, or the 15-item GDS, was designed like the original 30-item GDS, to provide a single score of depression in the elderly. As with the GDS, studies using factor analysis have been conducted in order to identify specific type of depression. One such study conducted an exploratory factor analysis that revealed a two-factor model (Friedman et al., 2005). Factor one (depression) included items of worthlessness, hopelessness, and boredom where factor two (positive affect) included items of happiness, good spirits, and life satisfaction (Friedman et al., 2005). This study was conducted with community dwelling elderly individuals who were cognitively intact (Friedman et al., 2005). This may have affected the factor structure to include only two factors.

Another study conducted a factor analysis using the 15-item GDS with a sample of cognitively intact elderly patients aged 65 and older who lived in diverse settings (Onishi et al., 2006). The factors included unhappiness, apathy and anxiety, loss of home and morale, and energy loss (Onishi et al., 2006).

Lai, Fung and Yuen (2005) conducted a factor analysis using the Chinese version of the 15-item GDS. Participants included Chinese community dwelling individuals age 55 of normal cognition and older living in Canada. This study found a four factor structure; negative mood, positive mood, inferiority and disinterested, and uncertainty (Lai, Fung, & Yuen, 2005).

As is evident, the factor structure of the GDS-15 and the GDS-30 has been extensively studied. Findings for both versions have been inconsistent between studies.

This may be due to methodological differences between studies. Some studies were conducted with elderly individuals with normal cognition, and some with cognitive disorder. The current study includes a combination of elderly individuals with normal cognition along with those with diagnosed cognitive disorder (i.e., MCI, VaD, and DAT).

Non-Demented (Normal Cognition for Age)

Persons who do not have cognitive impairment, determined by performance age specific norms on neuropsychological tests, are considered normal cognition for their age. These individuals may experience some simple forgetfulness that is common among this age group. As a person ages, their general physiological and neurological processes become less efficient, thus, leading to age-related cognitive changes. Therefore, cognition within these individuals is not the same as younger persons. Cognitive functioning is curvilinear; it increases from birth to around 30 years old, plateaus at approximately 80 years old and then declines (Rosenthal & Kavic, 2004). These changes lead to less efficient cognitive processing, but do not lead to memory dysfunction or the inability to carry out activities of daily living.

The prevalence of depression within the geriatric population varies considerably from 1% to 20% for major depression and up to 35% for those who experience depressive symptoms but do not fit the criteria for a diagnosis of depression. It is difficult to determine the cause of depression within this population. There is no evidence that aging causes depression, but there is evidence that psychosocial events (i.e., death of a spouse/child/close relative, retirement, lack of social support) and biological factors (i.e., vascular changes, especially in the central nervous system, endocrine dysfunction,

immune system activation, and nutritional deficiencies) may contribute to depression within this population (Lawhorn, 2005). Therefore, geriatric patients with normal cognition for age experience depressive symptoms as well as diagnosable depression. The etiology of depression is believed to determine patient response patterns on the GDS. Therefore, these patients, as a group, are expected to not establish a predictable response pattern on the GDS.

Mild Cognitive Impairment

Amnestic mild cognitive impairment, the most common type of MCI, is a state of cognitive decline, typically 1.5 standard deviation (SD) below age norms on memory tests (Petersen et al., 2001). It typically occurs when verifiable memory decline is more than normal aging but is less than that which meets the criteria for a dementia diagnosis (Cotter, Clark & Karlawish, 2003; Gauthier et al., 2006; Lyketsos, Lopez, Jones, Fitzpatrick, Breitner & DeKosky, 2002; Petersen et al., 1999, Petersen, 2004). Other areas of cognition are relatively spared (Cotter et al., 2003; Gauthier et al., 2006; Lyketsos et al., 2002; Petersen et al., 1999, Petersen et al., 2001; Petersen, 2004) leaving activities of daily living (ADLs) and independent activities of daily living (IADLs) relatively intact (Eibenstein et al., 2005). This diagnosis is not a diagnosis of dementia but is considered by some to be a pre-dementia state (Jicha et al., 2006; Petersen and Morris, 2005) or prodromal phase of DAT (Winblad et al., 2004). This diagnosis is somewhat difficult for clinicians to differentiate from normal age related forgetfulness (Petersen, 2004) and relies heavily on clinical judgment (Petersen, 2004). There are other types of MCI that do not have memory decline as the major clinical feature

(Petersen et al., 2001). For the purpose of the current study, the persons diagnosed with MCI have been grouped into one category due to the paucity of non-amnestic types.

Patients diagnosed with MCI, especially amnestic MCI (Petersen et al., 2001), are believed to be at a much higher risk of developing DAT than their normal cognition peers (Cotter et al., 2003; Gauthier et al., 2006). Persons diagnosed with MCI have been found to perform similarly to non-demented, normal cognition peers on tests of general cognition and more like DAT peers on tests of memory (Alladi, Arnold, Mitchell, Nestor, & Hodges, 2006; Petersen, et al, 1999). The rate of conversion from MCI to DAT ranges from 6% to 25% (Cotter et al., 2003; Petersen et al., 1999).

Elderly individuals who experience depressive symptoms have been found to be at a much higher risk of developing MCI than those who do not experience depressive symptoms (Barnes, Alexopoulos, Lopez, Williamson, & Yaffe, 2006; van Reekum, 2006) even in the absence of depressive illness. This minor depression may be associated with the magnitude of perceived functional impairment (Gabryelewicz et al., 2004), thus lending evidence for the hypothesis of depression as an affective disorder. Studies have reported that elderly individuals who express complaints of memory decline may be experiencing depression rather than actual cognitive decline (Bondi, 2004; Crowe, Andel, Wadley, Cook, Unverzagt, Marsiske, & Ball, 2006), thus, diagnosis may be difficult to distinguish.

MCI patients who experience depressive symptoms and have undergone treatment but do not experience improved cognition are at a much higher risk for converting to DAT (Adler, Chwalek, & Jajcevic, 2004; Gabryelewicz et al., 2004, Jicha et

al., 2006), because this may be an early sign of the early stages of neurodegeneration (Barnes et al., 2006) and/or vascular changes in the brain. MCI patients who later convert to DAT most likely have experienced neurodegeneration that is indicative of DAT (Jicha et al., 2006) in the absence of underlying vascular disease (Barnes et al., 2006). Therefore, identifying the type of depression in MCI patients may help to identify those at risk for developing DAT or VaD.

Dementia of the Alzheimer's Type

Persons who are diagnosed with dementia of the Alzheimer's type (DAT) experience neuropathology of the brain (Alzheimer's Association, 2007). This neurodegeneration gradually and progressively destroys brain matter. The result is a decline in cognitive functioning to include memory, starting with short-term memory, impairs executive functioning, leads to personality changes, and impairs a person's ability to carry out activities of daily living (Alzheimer's Association, 2007). Due to the progressive neurodegeneration of DAT, the severity of cognitive decline is also progressive and is associated with aging.

Persons diagnosed with probable DAT may experience depression, especially early in the disease process (Cotter et al., 2003; Holzer et al., 2005). In fact, 25% to 40% of these patients experience depressive symptoms (Caballero, Hitchcock, Beversdorf, Scharre, & Nahata, 2006). Research indicates that older adults who experience depressive symptoms are at higher risk for developing DAT, especially if they had a previous diagnosis of amnestic MCI (Modrego & Ferrandez, 2004).

Late-life depression has been reported as an early sign or prodromal phase of dementia (Barnes et al., 2006), especially DAT. It has also been found to place those with mild cognitive impairment at greater risk for advancing to Alzheimer's disease (Modrego & Ferrandez, 2004) and be at a higher risk for greater functional loss (Cotter, Clark, & Karlawish, 2003).

Some studies have found an association between patient awareness of declining cognition and the onset of depression (Cotter et al., 2003). Holzer and colleagues (2005) found a temporal relationship between diagnosis of DAT and the onset of depressive symptoms. They reported that patients diagnosed with mild DAT had higher rates of depression compared to themselves after they progressed to severe DAT, when depression rates dropped dramatically. They found that functional loss (such as perceived loss of independence) mediated the depressive symptoms, not awareness of their deficits. This change may be due to the inability of these more severely demented patients to express themselves adequately (Holzer et al., 2005; Muller-Thomsen, Arlt, Mann, MaB, & Ganzer, 2005).

Vascular Dementia

Persons diagnosed with vascular dementia experience cognitive decline that follows a stepwise progression. The etiology of VaD is due to the culmination of cardiovascular risk factors to include hypertension, hypercholesterolemia, obesity, sedentary lifestyle, glucose insufficiency, among others. Not all individuals experience cognitive decline following vascular ischemic changes in the brain, but many do, thus, precipitating the development of cognitive decline (Korczyn, 2005). Cognitive changes

that occur as a result of cerebrovascular disease vary with the location and quantity of damaged brain matter (Alzheimer's Association, 2007). For example, vascular dementia as a result of stroke in the cortex often have acute onset and result in deficits in specific neuropsychological domains accompanied by less pronounced deficits in other areas, while lesions found in the frontal lobe results in executive dysfunction, attention dysfunction, personality changes, changes in affect, and motor behavior changes (Alzheimer's Association, 2007).

Many individuals with vascular dementia exhibit behavioral disturbances, blunted affect, depressed mood, emotional withdrawal and anxiety (Ceccaldi, 2006). This late onset depression is believed to be precipitated by vascular changes in the brain (Alexopoulos, 2006; Alexopoulos, Meyers, Young, Kakuma, Silbersweig, & Charlson, 1997). The type of vascular depression (subcortical ischemic depression versus depression-executive dysfunction syndrome) is determined by the etiology of vascular changes in the brain (Alexopoulos, 2006).

Depression

Many factors can contribute to depression in the geriatric population, such as stressful changes in lifestyle resulting from retirement, change in social support (illness or death of spouse), and coexisting medical illnesses (i.e., thyroid disorder, Parkinson's disease, cerebrovascular disorders, myocardial infarction, and chronic obstructive pulmonary disease), and their medicinal treatment (Alzheimer's Association, 2007). Depression is also commonly associated with cognitive decline (Amore, Tagariello, Laterza, & Savoia, 2007a; Amore, Tagariello, Laterza, & Savoia, 2007b; Chinello,

Grumelli, Perrone, & Annoni, 2007; Fountoulakis et al., 2003; Kessels, Ruis, & Kappelle, 2007; Stickle & Onedera, 2006). Two major hypotheses of depression within this population that will be discussed include depression as an affective disorder and the vascular depression hypothesis. Many individuals who become aware that they are experiencing cognitive decline that is not considered "normal" for their age may experience depressed mood as a result (Amore et al., 2007a; Amore et al., 2007b; Chinello et al., 2007; Stickle & Onedera, 2006). This type of depression is considered to be linked with the experience of cognitive decline. This awareness of one's deficits is believed to decline with the progression of the disease process that is typically seen in individuals with cognitive decline.

Research suggests that depression is related to negative affect in reaction to being aware of one's cognitive decline (Amore et al., 2007a; Amore et al., 2007b; Chinello et al., 2007; Heun & Hein, 2005; Stickle & Onedera, 2006). It is often an affective response to the negative life events surrounding the experience of cognitive decline such as perceived functional losses, cognitive impairment, and frustration associated with the disease (Aalten, van Valen, Clare, Kenny & Verhey, 2005; Lawhorn, 2005). This indicates that individuals exhibit depressive symptoms or endorse items on depression assessment tools as a result of experiencing negative affect. Negative affect experienced due to awareness of deficits relies on an individual's ability to be cognitively aware of their own mental state. Holzer and colleagues (2005) found that some experience depression due to functional loss (such as perceived loss of independence), not awareness of deficits. This may be related to the inability of the more severely impaired patients to express themselves adequately (Bierman et al.,

2007; Holzer et al., 2005; Muller-Thomsen et al., 2005). This decline in symptom reporting may be related to decreased awareness of one's own cognitive status (Aalten et al., 2006). Others report no difference in depressive symptoms in those with more severe dementia (Verkiak et al., 2007; Wilson et al., 2008). Often depressive symptoms mimic dementia symptoms, thus making diagnosis and treatment difficult at times (Persad & Giordani, 2007).

Research suggests that prolonged depression in younger individuals may predispose them for, and thus place them at a higher risk for developing Alzheimer's disease and other dementing disorders later in life (Geerlings et al., 2008; Kessing & Anderson, 2004). Steffens & Potter report that the same regions that are associated with depression are also associated with dementia, providing some evidence that there is a common biological link between the two disorders (2008). Some suggest that dementia and early onset depression have a common immunologic response (Leonard, 2007; Maes, 1999; Lanquillon, 2000; and Owen, 2001) which could explain this predisposition.

Many neurobiological changes are related to the aging process, such as neurodegeneration, cardiovascular diseases, and brain atrophy, all of which can lead to cognitive decline and possibly dementia (Adler, Chwalek & Jajcevic, 2004; Barnes et al., 2006; Gabryelewicz et al., 2004; and Jicha et al., 2006). Some specific cardiovascular risk factors that may increase risk for dementia include: hyperlipidemia, hypertension, type II diabetes, inactivity, obesity, and other related factors. A vast amount of literature has recognized the link between these cardiovascular risk factors and depression (Alexopoulos et al., 1997; Alexopoulos, 2003; Mast, Azar, & Murrel, 2005; Mast,

Yochim, MacNeill, & Lichtenberg, 2004; Thomas, Kalaria, & O'Brien, 2004). Much literature also recognizes that those with increased cardiovascular risk factors are at a higher risk for developing dementias, especially vascular dementia (Alexopoulos, 2003; Alexopoulos, 2006; Barnes et al., 2006; Ceccaldi, 2006; Korczyn, 2005; Konstantinos et al., 2003).

The vascular depression hypothesis posits that depression within this population may be the result of cerebrovascular changes such as ischemia that occur due to aging, vascular disease, and/or due to the physiological changes that accompany neural degeneration (Fountoulakis et al., 2003). Since depression is often present in individuals diagnosed with dementia (Cotter et al., 2003; Ritter, Depres, Monsch & Manning, 2006), one questions whether depression is a symptom of vascular changes or a result of negative affect in response to cognitive decline, or a combination of the two. The former hypothesis indicates that depression and executive dysfunction are related, with the common denominator being microvascular disease (Yochim, MacNeill, & Lichtenberg, 2006) that occur with late-life brain changes and possibly due to brain changes associated with DAT. This is referred to as the vascular depression hypothesis (Fountoulakis et al., 2003; Yochim et al., 2006).

The vascular depression hypothesis (Alexopoulos et al, 1997) identified the clinical presentation of subcortical ischemic depression as prominent psychomotor retardation, apathy and pronounced disability (Alexopoulos, 2006; Krishnan et al., 2004). These patients have poor outcomes in that they usually have persistent and unstable depressive symptoms and increased risk for dementia (Alexopoulos, Kiosses, Choi, Murphy, & Lim, 2002; Taylor, Steffens, & Krishnan, 2006; Thomas, Kalaria, &

O'Brien, 2004) and mortality (Alexopoulos, 2006). These individuals usually do not respond well to psychopharmacological treatment for depression (Alexopoulos, 2006). Patients who have had vascular incidents such as stroke, myocardial infarct, and coronary artery disease are at greater risk for developing subcortical ischemic depression (Alexopoulos, 2003), especially if they are 85 years old and beyond (Mast, Azar, & Murrell, 2005). There is evidence that the relationship between vascular maladies and subcortical ischemic depression is bi-directional (Mast et al., 2005; Thomas et al., 2004).

Those identified with depression-executive dysfunction syndrome present with psychomotor retardation, lack of interest, limited depressive ideation and lack of insight, and prominent disability (Alexopoulos et al., 2002). This type of vascular depression may be caused by aging-related changes, degenerative brain disease, or an accumulation of both of these factors (Alexopoulos, 2006).

Patients with vascular depression will endorse items consistent with type of depression. Those with predominantly subcortical ischemic depression will endorse apathy items on the GDS where those with predominantly depression-executive dysfunction syndrome will endorse dysphoria items on the GDS.

It is apparent that the etiology of cognitive impairment and depression are related in some way. This study strives to explore this relationship by looking at the difference in cognitive group status members' response patterns on the GDS. If differences in response patterns exist, then it may be presumed that etiology is the culprit. Therefore, treatment of concomitant depression and cognitive impairment can be more specific to etiology. Also, if one experiences either depression or dementia first, then more

precipitous and more specific treatments may help to prevent the other from occurring, or at least prolong its onset.

The purpose of the current study was to explore the relationship between cognitive status and depression. It also identified the factor structure of the GDS-30 and the GDS-15 and explored the factor's ability to differentiate cognitive status group. These factor structures were compared with each other and then to the factor structure identified by Hall and Davis (in press). The hypotheses of this study included: 1). Expect to find a different factor structure than Hall and Davis (in press) due to the inclusion of a normal group; 2) the factor structure of the GDS-15 will be similar to the factor structure of the GDS-30; the GDS-15 and GDS-30 factors will discriminate between the four cognitive status groups; patients diagnosed with VaD will endorse the Apathy subscale score more than the other cognitive status groups; early and mild DAT and MCI patients will endorse items on the dysphoria and meaninglessness subscales more than the other cognitive status groups; and the GDS factors will not discriminate the normal group.

METHODS

Participants

Participants consist of 282 geriatric patients who had been referred for neuropsychological testing from the GAP (Geriatric Assessment Program) clinic at the University of North Texas Health Science Center, in Ft. Worth, Texas. Participants consist of 82 male and 200 female participants ranging in age from 65 to 96 (*M* = 79.11, *SD* = 7.29) and include 131 participants diagnosed with Alzheimer disease (DAT), 74 with vascular dementia (VaD), 51 with mild cognitive impairment (MCI), and 26 with no dementia diagnosis (NORM). The data were previously collected from a retrospective chart review. As this sample was taken from the same sample pool as that of Hall and Davis (in press), the patients in the DAT, VaD, and MCI cognitive groups are the same. Diagnosis was determined based on a consensus diagnosis given by a team consisting of geriatricians and neuropsychologists. Diagnosis of Alzheimer's dementia was determined using the NINCDS-ADRDA (McKhann et al., 1984), vascular dementia was determined using the NINCDS-AIREN criteria (Roman et al., 1993) and MCI was determined using the criteria of the International Working Group on Mild Cognitive Impairment (Winblad et al., 2004).

Materials

A data collection form was used to collect patient demographic information along with scores on several neurocognitive scales (see Appendix A). Two versions of the Geriatric Depression Scale were included in the analyses, the GDS-30 (see Appendix B) and the GDS-15 (see Appendix C). All participants had been administered the GDS-

30. Since all of the items of the GDS-15 are included in the GDS-30, GDS-15 items were extracted and analyzed.

Statistical Analyses

Factor analyses were conducted to identify the factor structure of two versions of the GDS. This provided information used for the comparison between the two versions of this test and for comparison with the factor structure that Hall and Davis (in press) found.

Multinomial logistic regression analysis was conducted in order to identify which factors of the GDS-15 discriminate between the four cognitive groups. Multinomial logistic regression was also conducted in order to identify which factors of the GDS-30 discriminate between the four cognitive groups.

Finally, an exploratory factor analysis was conducted in order to identify the individual items of both the GDS-15 and GDS-30 to find the strongest items for identifying persons in specific cognitive diagnostic groups.

RESULTS

One-way Analysis of Variance

A one-way analysis of variance (ANOVA) was conducted to compare cognitive status with total depression score. Results indicate that there is no significant relationship between these two variables, F (25,256) = 1.12, p = .32.

Principal Components Analyses

A principal components analysis (PCA) with varimax rotation as conducted by Hall and Davis (in press), was conducted using SPSS version 11.5 in order to determine the factor structure of the GDS-30 using the four cognitive groups dementia of the Alzheimer's type (DAT), vascular dementia (VaD), mild cognitive impairment (MCI), and normal (NORM) for comparison. This study differed from Hall & Davis (in press) by including a normal group in addition to the three diagnostic groups (DAT, VaD, and MCI) they included (see Table 1). The PCA revealed a four-factor structure model of the GDS-30. See Table 2 for factor loadings. The Contentment subscale contained 9 items with factor loadings ranging from .625 to .368, the Discontentment subscale contained 11 items with factor loadings ranging from .626 to .346, the Apathy subscale contained 6 items with factor loadings ranging from .579 to .437, the Cognitive Impairment subscale contained 4 items with factor loadings ranging from .588 to .459 (see Table 1). Cronbach's alpha reliability coefficients revealed that two subscales (Contentment and Discontentment) exhibited good alpha coefficient reliabilities (.74 and .79) while two subscales (Cognitive Impairment and Apathy) exhibited poor reliability (.60 and .45).

A PCA was performed using SPSS 11.5 in order to determine the factor structure of the GDS-15. As described above, the PCA performed used the same inclusion criteria as that used by Hall and Davis (in press). The PCA revealed a two-factor structure model of the GDS-15. The Contentment (CONT) subscale contained 7 items with factor loadings ranging from .622 to .487 while the Discontentment (DISCON) subscale contained 7 items with factor loadings ranging from .753 to .280 (See Table 3). One item on the GDS-15 had poor loading on both subscales, Question 10, which is a question of memory impairment. Its factor loading with Factor 1 was .054 and with Factor 2 was .050. Cronbach's alpha reliability coefficients for the two subscales exhibited high alpha coefficient reliabilities (.66, and .64 respectively).

Factor Structures of the GDS-30 of the Current Study and of Hall & Davis (in press) using Receiver Operating Characteristic Curve

The receiver operating characteristic (ROC) curve was designed to evaluate the performance of a classification tool, the Geriatric Depression Scale, in this study. It provides information on the tool's specificity and selectivity. The information derived from ROC curve was the area under the curve and the confidence interval. The ROC curve can provide four outcomes which are formulated on a 2x2 contingency table. The upper left corner represents a true positive, or 100% sensitivity and 100% specificity. This is expressed as coordinates (0,1) or 1. The lower right corner represents a true negative, or 0% sensitivity and 0% negativity. This is expressed as coordinates (1,0) or -1. The area of random guessing, or 50% selectivity and 50% specificity or .5 is graphed as a diagonal line running from low left to upper right. Therefore, the closer the area under the curve is to 1, the more specific and selective it is. If the area under the curve

is closer to .5, the tool is not considered to have specificity or selectivity. It the area under the curve is closer to -1, and then the tool is considered to not have specificity or selectivity (Fawcett, 2006).

Receiver operating characteristic (ROC) curves of the GDS-30 four-factor structure identified by Hall and Davis (in press) and GDS-30 four-factor structure identified by the current study were performed to explore if the two differ in their ability to measure depression in a geriatric sample. The extraction method of the current study matches the extraction method used by Hall and Davis (in press). The GDS-30 subscales identified by Hall and Davis (in press) revealed poor ability to identify depression (dysphoria subscale area = .475, p = .69, CI = .357 - .593; meaninglessness subscale area = .515, p = .813, CI = .392 - .638; apathy subscale area = .430, p = .26, CI = .304 - .556; and cognitive impairment subscale area = .583, p = .18, CI = .455 - .712). The GDS-30 subscales identified by the current study revealed similar poor specificity (contentment subscale area = .470, p = .64, CI = .330 - .611; discontentment subscale = .467, p = .60, CI = .351 - .584; apathy subscale area = .579, p = .21, CI = .453 - .705; and cognitive impairment subscale area = .579, p = .22, CI = .46, CI - .457 - .696). Due to this lack of difference between these two subscale structures, further analyses were conducted using the four-factor structure subscales identified by Hall and Davis (in press).

Factor Structure of the GDS-30 and of the GDS-15 using ROC curve

Receiver operator characteristic (ROC) curves of the GDS-30 total score and subscale scores and GDS-15 total score and subscale scores were performed to

measure the ability to measure depression in a geriatric sample. This comparison was conducted to see if the shorter version of the GDS is able to identify depression (using total GDS score) and type of depressive symptoms (using subscale scores) as effectively as the GDS-30. Results indicate that neither the GDS-30 total score (area = .491, *p* = .89, CI = .369 - .614) nor the GDS-15 total score (area = .487, *p* = .83, CI = .366 - .608) discriminates depression in patients with Alzheimer's disease compared to normal cognition patients. Comparisons of the GDS-30 subscale scores and the GDS-15 subscale scores revealed that the individual subscales of the GDS-30 dysphoria subscale area = .475, *p* = .69, CI = .357 - .593; meaninglessness subscale area = .515, *p* = .813, CI = .392 - .638; apathy subscale area = .430, *p* = .26, CI = .304 - .556; and cognitive impairment subscale area = .583, *p* = .18, CI = .455 - .712) and of the GDS-15 contentment subscale area = .452, *p* = .44, CI = .324 - .581; and discontentment subscale area = .471, *p* = .62, CI = .360 - .583) do not identify depression in patients with Alzheimer's disease compared to normal cognition patients. The GDS-15 provides information on contentment or positive response items, and discontentment or negative response items (see Table 3). Given this, it appears that these items do not impact total depression scores, but specific information concerning depressive symptoms is lost when using the GDS-15. Thus there is no gain in using the GDS-15 over the GDS-30. Since the GDS-15 subscales were found to be statistically not different than the GDS-30 dysphoria subscale and meaninglessness subscale, no additional analyses were conducted with the GDS-15.

Multinomial Logistic Regression

Multinomial logistic regression was conducted to explore the discriminative properties of the GDS-30 subscales on cognitive group status (DAT, VaD, MCI, and Norm). A relationship was found between the GDS-30 and cognitive group, X^2 (12) = 29.58, $p < .01$, although the relationship was small (Nagelkerke R^2 = .109). The chi square test provides information on the relationship between the dependent variable and the independent variable and R^2 provides information on the amount of variance in the dependent variable accounted for by the independent variable. (Grimm & Yarnold, 1995). The proportional by chance accuracy criteria was 40.71%. Correct classification rates were 87.8% for DAT, 25.7% for VaD, 3.9% for MCI and 0% for Norm; the overall correct classification rate was 48.2%. The correct classification rate criterion of 25% greater than the proportional by chance accuracy was satisfied (Grimm & Yarnold, 1995).

The log-likelihood test revealed a relationship between the GDS-30 dysphoria subscale and cognitive diagnosis (X^2 (3) = 8.17, p = .043) and cognitive impairment subscale and cognitive diagnosis (X^2 (3) = 9.37, p = .03). This test can be used to test the overall model. It can be used to determine whether any predictor coefficient differs from zero (Grimm & Yarnold, 1995). No relationship between the GDS-30 meaninglessness subscale and cognitive status (X^2 (3) = 2.178, p = .54) or apathy subscale and cognitive status (X^2 (3) = 7.11, p = .07) was found.

The Wald criterion, which is used to test for effect size (Grimm & Yarnold, 1995) was used to evaluate whether the GDS-30 dysphoria and cognitive impairment subscales are significant in differentiating between diagnostic groups. Results indicate

that the dysphoria subscale was not significant in distinguishing DAT (Wald = .202, *p* = .653, CI = .746 - 1.201), VaD (Wald = .457, *p* = .499, CI = .853 - 1.385) or MCI (Wald = 1.937, *p* = 1.64, CI = .929 – 1.540). The cognitive impairment subscale was significant in distinguishing DAT (Wald = 4.326, *p* = .038, CI = 1.022 – 2.062) but not VaD (Wald = .408, *p* = .523, CI = .783 – 1.620) or MCI (Wald = .114, *p* = .735, CI = .726 – 1.573).

Comparison of Diagnostic Group Total GDS Scores

A one-way ANOVA was conducted comparing cognitive group status and total GDS score to see if differences exist in between persons diagnosed with DAT, VaD, MCI, or Normal cognition for age. Results indicate a significant difference between cognitive groups on GDS score, *F*(3, 278) = 3.68, *p* = .01. Tukey post hoc analysis found that persons diagnosed with DAT (M = 6.18, SD = 4.58) scored significantly different than those with VaD (M = 8.42, SD = 5.88), *p* = .02. No other differences of GDS total scores were found.

Comparison of Diagnostic Group Subscale Scores

A one-way ANOVA was conducted using cognitive group status and the factor scores from the four-factor model generated by Hall and Davis (in press) to compare subscale scores on the GDS-30 and the different cognitive groups. Results indicate significant difference between cognitive groups on the dysphoria subscale, *F*(3, 278) = 4.53, *p* = .004, the meaninglessness subscale, *F*(3, 278) = 3.091, *p* = .03, and the apathy subscale, *F*(3, 278) = 3.25, *p* = .02, but not the cognitive impairment subscale, *F*(3, 298) = .79, *p* = .50. Tukey post hoc analysis found DAT differed in GDS subscale

scores from VaD on the dysphoria, the meaninglessness, and the apathy subscales and DAT differed from MCI on the dysphoria subscale. Similar subscale scores were found between all other groups (see Table 4).

Comparison of Diagnostic Group by Severity Level

A one-way ANOVA was conducted comparing the four-factor (subscale score) structure of Hall and Davis (in press) and severity level with three cognitive groups included in the analysis (DAT, VaD, and MCI) excluding the normal group. Results indicate no significant differences of subscale scores with severity level on dysphoria, (see Table 4).

Comparison of Diagnostic Group GDS-30 Subscale Scores by Severity Level

A one-way ANOVA was conducted comparing persons with early severity level and the GDS-30 subscale scores. Results indicate significant differences of subscale scores on the dysphoria subscale, $F(2, 148) = 4.24$, $p = .02$, the meaninglessness subscale, $F(2, 148) = 3.62$, $p = .03$, and the apathy subscale, $F(2, 148) = 3.55$, $p = .03$, but not on the cognitive impairment subscale, $F(2, 148) = 1.08$, $p = .34$. Tukey post hoc analyses indicate different subscale scores between DAT and VaD on the dysphoria subscale, $p = .04$, and between DAT and VaD on the meaninglessness subscale, $p = .03$, between DAT and VaD on the apathy subscale, $p = .03$, but no difference in subscale scores were found between DAT and VaD on the cognitive impairment subscale score, $p = .72$ (see Table 5). Differences on the dysphoria subscale were also found between DAT and MCI, $p = .04$, but no differences were found the

meaninglessness subscale, p = .18, the apathy subscale p = .25, or on the cognitive impairment subscale, p = .71 (See Table 5). No differences were found between VaD and MCI on the dysphoria subscale, p = .99, the meaninglessness subscale p = .65, the apathy subscale p = .52, and the cognitive impairment subscale, p = .31 (see Table 4 and see Figure 1).

A one-way ANOVA was conducted comparing persons with mild (DAT and VaD) severity level and the GDS-30 subscale scores. Results indicate no differences for the Dysphoria subscale, $F(1, 88) = 2.23$, p = .11, the Meaninglessness subscale, $F(1, 88) = 1.95$, p = .15, or the Cognitive Impairment subscale, $F(1, 88) = .97$, p = .38. Interestingly, subscale scores for the apathy subscale were found to approach significance between DAT (M = 1.33, SD = 1.28) and VaD (M = 1.73, SD = 1.20), $F(1, 88) = 3.02$, p = .054 (see Table 6 and Figure 1).

A one-way ANOVA was conducted comparing persons with moderate impairments and the GDS-30 subscale scores (Hall & Davis, in press). Results indicate no significant differences on the Dysphoria subscale, $F(1, 14) = .36$, p = .56, the Meaninglessness subscale, $F(1, 13) = .01$, p = .94, the Apathy subscale, $F(1, 13) = .97$, p = .34, and on the Cognitive Impairment subscale, $F(1, 13) = .60$, p = .45 (see Table 7 and Figure 1).

Factorial Analysis of Variance of DAT and VaD and Severity Levels by Subscale Scores

Factorial ANOVAs were conducted comparing persons with DAT and VaD and severity level on the GDS-30 subscale scores. These analyses helped determine whether differences exist between these diagnostic groups and severity levels. They will

also help determine what those differences are. The analyses were conducted comparing diagnostic group and severity level on each subscale score separately.

A factorial ANOVA was conducted comparing DAT and VaD patients and severity level on the Dysthymia subscale of the GDS-30. Results indicate that there was no difference of subscale score between persons with DAT (M = 1.90, SD = .29) (see Figure 3) and VaD (M = 2.52, SD = .47) (see Figure 4), $F(1, 204) = 1.27$, $p = .26$. No difference was found between early ($M = 2.53$, $SD = .26$), mild ($M = 2.78$, $SD = .28$) and moderate ($M = 1.32$, $SD = .73$) severity levels, $F(2, 204) = 1.76$, $p = .18$ (see graph 2). Analysis revealed no interaction effect, $F(2, 204) = .81$, $p = .45$.

A factorial ANOVA was conducted comparing DAT and VaD patients and severity level on the Meaninglessness subscale of the GDS-30. Results indicate that there was no significant difference of subscale score between persons with DAT (M = .79, SD = .16) (see Figure 3) and VaD (M =1.21, SD = .25) (see Figure 4), $F(1, 204) = 1.96$, $p = .16$. A significant difference was found between early (M = 1.06, SD = .14), mild (M = 1.43, SD = .15) and moderate (M = .52, SD = .39) severity levels, $F(1, 204) = 3.21$, $p = .04$ with those with moderate severity scoring lower on this subscale than early and mild severity levels (see Figure 2). Analysis revealed no interaction effect, $F(2, 204) = .38$, $p = .68$.

A factorial ANOVA was conducted comparing DAT and VaD patients and severity level on the apathy subscale of the GDS-30. Results indicate that there was a significant difference found between DAT ($M = 1.37$, $SD = .16$) (see Figure 3) and VaD (M = 1.99, SD = .24) (see Figure 4), $F(1, 204) = 4.62$, $p = .03$. No significant difference was found between early (M = 1.59, SD = .14), mild (M = 1.53, SD = .15), and moderate

(M = 1.90, SD = .39) severity levels, $F(2, 204) = .39$, $p = .68$ (see Figure 2). No interaction effect was found, $F(2, 204) = .41$, $p = .66$.

A factorial ANOVA was conducted comparing DAT and VaD patients and severity level on the cognitive impairment subscale of the GDS-30. Results indicate that there was no significant difference between DAT ($M = 2.01$, $SD = .17$) (see Figure 3) and VaD (M = 1.73, SD = .27) (see Figure 4), $F(1, 204) = .80$, $p = .37$. No significant difference was found between early ($M = 2.18$, $SD = .15$), mild ($M = 1.85$, $SD = .16$) and moderate ($M = 1.58$, $SD = .43$), $F(2, 204) = 1.67$, $p = .19$ (see Figure 2). No interaction effect was found, $F(2, 204) = 1.37$, $p = .26$.

DISCUSSION

Many Americans 65 years old and older have dementia with concomitant depression. Due to this prevalence, it was believed that there is a relationship between endorsement of depression symptoms on a measure of depression and type of dementia. This study sought to explore the comorbidity between subscale scores (Dysphoria, Meaninglessness, Apathy, and Cognitive Impairment) of the Geriatric Depression Scale (GDS) that was defined by Hall and Davis (in press) with four cognitive groups dementia of the Alzheimer's type (DAT), vascular dementia (VaD), mild cognitive impairment (MCI), and normal (NORM). It was believed that the GDS subscales would prove useful in identifying type of depressive symptoms and possibly in identifying type of cognitive impairment.

Many studies sought to identify subscales of the GDS using either a clinical sample or a normal sample. This study differs in that it included both. Comparisons were made between the GDS-30 and GDS-15 to see whether the shorter version provided the same information as the longer version with the bonus of saving time. Analyses were conducted to explore differences of subscale scores between the cognitive status groups. This information may help clinicians by providing collateral information to help validate type of dementia. Finally, analyses were conducted to explore differences of subscale scores between those with diagnosed dementia across severity levels.

A principle components analysis (PCA) was conducted with the GDS-30 and four cognitive diagnostic groups, unlike Hall and Davis (in press) who did not include a normal cognition group. Comparison analyses revealed that the four-factor structure of

the current study and that of Hall and Davis (in press) were virtually no different. Therefore, the factor structure reported by Hall and Davis (in press) was used for all subsequent analyses.

A PCA was conducted with the GDS-15 and four cognitive diagnostic groups. This analysis revealed a two-factor structure. Unfortunately, analyses indicate that these two factors are not statistically different from the Dysphoria and Meaninglessness subscales of the GDS-30 and there was no overlap with the Apathy or Cognitive Impairment subscales. In fact, only one item of the cognitive impairment subscale was found on the GDS-15. This finding refutes the hypothesis that the GDS-15 will reveal the same factor structure as the GDS-30 and thus indicates that the shorter version is not simply a shorter version of the same test.

When looking at the factor structures of both versions of the GDS, it is apparent why these versions' total scores do not differ. The Dysphoria and Meaninglessness subscales are heavily weighted in that they comprise 18 of the GDS-30 items and all 15 of the GDS-15 items. Thus, if a clinician is interested in assessing mood, then the GDS-15 may be useful, but specific symptom information is lost when using this shorter version. Also, the amount of time for completion between the GDS-15 and GDS-30 from an evaluative point of view is quite minimal, so there is no gain in using the shorter version. No difference of total depression score was found between the two versions. This provides evidence that the shorter version provides the same general information as the GDS-30. The classic method of GDS administration is self-administration. In the current study, the GDS-30 was administered orally by a clinician. It is believed that this method increases the utility of the GDS-30 by providing valuable information through

behavioral observations during administration. Since there is virtually no difference between the GDS-15 subscales and the Dysphoria and Meaninglessness subscales of the GDS-30, no further analyses using the GDS-15 were conducted.

Another goal of the current study was to explore whether the GDS-30 subscales are useful in distinguishing between cognitive status groups. This was based on the possibility that etiological differences between the cognitive status groups would be reflected in GDS subscale scores. The etiology of Alzheimer's disease is neurodegeneration of brain tissue with the presence of ammyloid plaques and neurofibrillary tangles in the cortical structure of the brain. More specifically, this destruction of cortical brain tissue typically strikes the hippocampus and the prefrontal cortex early in the disease process. The hippocampus is the brain structure that is responsible for encoding and converting short-term memory into long-term memory. Destruction of this structure early in the disease process typically leads to early signs of short-term memory impairment and a declining ability to interpret events around them. The prefrontal cortex, which is located anterior to the hippocampus, is also destroyed early in the disease process, which leads to labile mood and decreased inhibition. As a result of the progressive destruction of these structures, DAT patients tend to experience sadness. In fact, the hallmark depressive symptom of DAT is dysphoria.

Vascular dementia has quite a different clinical picture. VaD etiology is a combination of cardiovascular risk factors such as hyperlipidemia, hypertension, obesity, glucose intolerance, inactivity, and others. These risk factors increase the possibility of microvascular changes and often lead to transient ischemic accidents and cerebrovascular accident. VaD affects the basal ganglia in the sub-cortical area of the

brain. This structure is responsible for initiation of behavior and maintenance of behavioral interactions with the outside world. The prefrontal cortex is also affected in VaD patients. Destruction of this structure typically leads to personality change and apathy. In fact, apathy is the hallmark depressive symptom of VaD patients. Therefore, differences would be expected in depressive symptoms between DAT and VaD patients. Patients with mild cognitive impairment are not classified as having dementia, but are experiencing cognitive decline that is more than expected for normal aging, but is less than that expected of those with dementia. Different types of MCI have been identified, but all participants in the current study have been grouped into one category of MCI. Amnestic MCI has been reported as being a precursor to DAT. Therefore, the presumption that MCI patients would perform similarly to mild DAT patients was made.

A result of a multinomial logistic regression indicated only one subscale (cognitive impairment) was found to differentiate DAT patients. This finding indicates that the GDS is not appropriate to use for diagnosing or differentiating cognitive impairment group. In order for the GDS subscales to have been found to be good at differentiating type of dementia, one would have expected to find that the other subscales would have differentiated type of cognitive status. Although these subscales are not good for distinguishing cognitive status, the information that is reaped may provide valuable information that may help clinicians in identifying and possibly treating cognitive impairment.

The possibility that differences of subscale scores would be found based on etiological differences of cognitive status when not taking severity into account were inconsistent. Results indicate that patients scored differently on the Dysphoria subscale

(a reflection of emotional mood), the Meaninglessness subscale (which relies on the patient's ability to express their sense of worth), and the Apathy subscale (a reflection of symptoms of apathy) but not the Cognitive Impairment subscale (reflects patient worry about cognitive decline). This does not indicate any difference of responding based on etiology of cognitive status.

Further analyses looking at the diagnostic groups revealed that DAT patients differed from VaD patients on the Dysphoria, Meaninglessness, and Apathy subscales. VaD patients tended to have higher scores on these subscales than DAT patients. This lends some evidence to the belief that DAT patients have decreased awareness of their cognitive status and emotional state early in the disease process. However, the hypothesis that DAT and VaD patients would differ based on etiological differences was not upheld. It was assumed that DAT and VaD patients' scores would differ on the Dysphoria and Apathy subscales based on hallmark symptoms. The fact that they also differ on the Meaninglessness subscale is not consistent with this hypothesis.

DAT patients differed from MCI on the Dysphoria subscale. This finding counters the hypothesis that MCI patients would score similarly to DAT patients. This hypothesis was based on the premise that MCI patients are at high risk of converting to DAT and are in a prodromal stage of DAT. The fact that these two diagnostic groups scored differently further supports the fact that the GDS subscales are not good for differentiating type of dementia. Also, since the hallmark depressive symptom of DAT is dysphoria, this difference counters this argument. This result may be due to participant dynamics of the MCI group. Peterson and colleagues (1999, 2001, 2004, & 2005) delineated four types of MCI. Most of the participants in the current study were

diagnosed with the amnestic type of MCI. Due to the paucity of participants who fall into the other three subtypes of MCI, all patients with an MCI diagnosis were grouped together.

This study found that VaD patients endorse apathy symptoms more often than DAT patients, supporting the hypothesis based on etiological differences. This may suggest that apathy is an early symptom of VaD. It may be inferred that persons who score higher on the Apathy subscale may be at a higher risk for vascular dementia, or at least indicate that risk factors need to be reviewed. Therefore, identifying those with this hallmark symptom before the onset of VaD may help clinicians identify those who may be at high risk for vascular dementia. Early detection and treatment of vascular risk factors may prevent these patients from falling victim to this believed to be preventable form of dementia.

The final goal of the current study was to explore the effect that severity level (early, mild, or moderate) of cognitive decline has on patient GDS-30 total score and subscale scores. Analyses revealed no difference between cognitive status group (DAT, VaD, and MCI) on total score or subscale scores when participants were grouped together and analyzed at different severity levels. But, when analyses explored subscale scores at different severity level, differences were found. Individuals in the early and mild levels of impairment did not differ in the number of endorsed items on the Dysphoria subscale but those at the moderate level endorsed significantly fewer items. Scores on the Meaninglessness subscale were highest for those with mild severity and lowest for those with moderate severity. These findings support the hypothesis that persons with increasing severity of Cognitive Impairment also have decreasing

awareness of their own deficits. Thus, these individuals report more depressive symptoms at earlier severity level than those at the moderate severity level.

Apathy subscale scores were highest for those with moderate severity and lowest for those in the early severity level. This contradicts the hypothesis that with increasing severity of cognitive impairment, endorsement of items would decrease. This finding is indicative of past reports of a positive relationship between apathy and level of cognitive impairment. As severity increases, apathetic symptoms also increase.

The Cognitive Impairment subscale scores did not differ between patients at different severity levels. This finding was not expected. One would expect to find an increase in Cognitive Impairment subscale scores with early severity due to the awareness of one's own deficits. These results may have been affected by the large number of DAT patients who typically lack awareness.

Comparisons between DAT, VaD, and MCI patients revealed some differences based on severity level and subscale scores conducted separately. DAT patients, in the early phase of cognitive decline were found to endorse fewer subscale items than VaD patients on the Dysphoria, Meaninglessness, and Apathy subscales. DAT and VaD patients in the mild severity group did not differ on subscale scores, but their scores approached difference on the Apathy subscale. VaD patients tended to have higher subscale scores than DAT patients. Patients with moderate severity of impairment did not differ on subscale scores. The results of these comparisons support the premise that persons with more severe cognitive impairment have less awareness of their affective state.

Comparisons to determine whether differences in subscale scores between DAT

and VaD were conducted examining whether differences are related to etiological differences or severity level. The findings were mixed. Dysphoria subscale scores did not differ. This finding indicates that there is no difference on this subscale based on etiology or awareness of deficits.

Interestingly, a difference in subscale scores was found for the Meaninglessness subscale. Patients with moderate impairment endorsed fewer items on this subscale than those with early or mild severity. This indicates that awareness may play a part in reporting of meaninglessness. Since this subscale is based on the ability of patients to be able to describe their self-worth, this decline may indicate in a patient's ability to express themselves.

VaD patients endorsed more apathy items than DAT patients. There were no differences found for severity level. This finding lends evidence that etiological difference may be the culprit. It also supports reports that VaD patients tend to experience apathy symptoms early in the disease process, while DAT patients typically experience more affective types of depression (Alzheimer's Association, 2008).

Finally, no differences were found for diagnostic group or severity level on the Cognitive Impairment subscale. This finding was rather surprising in that one would believe that individuals who are aware of their own cognitive decline would express concern for this decline, especially in the earlier stages of the disease process. This was not found. A possible explanation for this finding is that the participants in the current study may be attributing their cognitive decline on the aging process. A chi square analysis comparing question #30 (Is your mind as clear as it used to be?) and cognitive group score revealed low endorsement of this item among all groups. This

may be due to patients attributing normal decline due to aging and not pathology as being the cause of one's cognitive decline.

In conclusion, this study found that using the GDS for identifying cognitive status is not feasible. The GDS is a good geriatric depression screening tool. The four-factor structure identified by Hall and Davis (in press) provides a very useful set of subscales. The subscale scores can provide clinicians information about patient's specific depressive symptoms, which provides increased utility for the GDS. Clinicians can benefit from this added information by being able to be more specific in their treatment of their patient's depressive symptoms. The GDS-15 was found to not be a good substitute for the GDS-30. It was formulated with the intent of providing the same information as the GDS-30 with the benefit of saving time. This study found that 14 of the 15 items are from the Dysphoria and Meaninglessness subscales of the GDS-30, the Apathy and Cognitive Impairment subscales are not represented. Only one item from the Cognitive Impairment subscale was included on the GDS-15. Therefore, specific depressive symptoms are missed with this version.

ROC curve analyses revealed that the Hall and Davis factor structure and the factor structure of the current study were no different. This finding is in direct contrast to hypotheses of this study. Therefore, the factor structure identified by Hall and Davis (in press) was found to be stable even when a non-dementia group was added into the analyses. The four-factor structure by Hall and Davis (in press) is sufficient to be used to identify four specific depressive symptoms with geriatric patients with and without dementia.

The current study had a sufficient number of patients, but the groups were not even. Alzheimer's patients were overrepresented and normal patients were underrepresented. This may have affected the results found in this study. It is possible that with even groups, the findings might be different.

Table 1

GDS-30 Factor Structure that Hall and Davis (in press) Found with Inclusion of Three Groups

Item #	DYS	MNGLS	APATHY	COGIMP
1	-.248	**-.591**	-.216	.221
2	**.403**	.183	.206	.050
3	.452	**.537**	.136	-.043
4	**.626**	.020	.134	-.013
5	-.042	**-.569**	-.172	-.003
6	**.517**	-.058	.014	.327
7	**-.616**	-.294	-.176	.062
8	.271	.375	.006	**.421**
9	**-.714**	-.225	-.129	-.053
10	**.384**	.341	.074	.202
11	**.315**	-.137	.298	.201
12	-.050	.171	**.702**	.140
13	.273	.256	-.113	**.433**
14	.005	-.297	.283	**.431**
15	-.083	-.298	**-.437**	.276
16	**.703**	.127	.246	.020
17	**.508**	.321	.013	.227
18	.105	**.496**	-.088	.270
19	-.267	**-.417**	-.368	-.193
20	.041	.078	**.573**	.356
21	-.135	-.156	**-.399**	-.174
22	-.007	**.724**	.204	.049
23	.183	**.419**	-.064	-.088
24	**.459**	.112	.148	.079
25	**.622**	.074	-.123	-.104
26	.017	-.012	.093	**.688**
27	-.253	-.068	**-.479**	-.016
28	.184	-.057	**.619**	-.024
29	-.088	-.002	-.052	**-.311**
30	.184	-.012	-.155	**-.456**

Note. Dys = Dysphoria; MNGLS=Meaninglessness; CogImp=Cognitive impairment.

Table 2

GDS-30 Factor Structure of Current Study with Three Groups and Normal Cognition Group

Item #	CONT	DISCT	COGIMP	APATHY
1	**.592**	.170	.187	-.076
2	**.368**	.395	.111	-.008*
3	.337	**.395**	.446	-.008*
4	.316	**.452**	-.007	.228
5	.373	-.099	.039	**.500**
6	.132	**.652**	.133	-.005
7	**.531**	.339	-.171	.080*
8	.327	**.462**	.144	-.208
9	**.537**	.492	-.003	.048*
10	**.430**	.273	.052	.237*
11	-.052	**.392**	.220	.233
12	.082	-.066	**.437**	.385*
13	.306	**.346**	.226	-.068
14	-.094	.144	**.583**	-.064
15	**.519**	.008	.001	.206
16	.380	**.626**	-.033	.215
17	.330	**.397**	**.**103	.353
18	-.086	**.406**	.057	.389*
19	**.527**	.159	.345	.179
20	.271	.128	**.530**	.160*
21	**.625**	-.049	.233	-.155
22	.361	.007	-.025	**.588**
23	-.103	.173	-.058	**.579**
24	.075	**.580**	.209	.010
25	.013	**.611**	.006	.149
26	.018	.165	**.579**	-.027
27	**.539**	.045	.130	-.108
28	.046	.170	.253	**.459**
29	.042	.152	**.543**	.125
30	.174	-.129	**.553**	-.050

Note. *Signifies items that are the same between the factor structure of the GDS-30 of Hall & Davis (in press) and current study. CONT=Contentment; DISCT=Discontentment.

Table 3

Comparison of GDS-30 and GDS-15 Subscales

	GDS-30					GDS-15	
Item #	CONT	DISCT	COG	APATHY	Item #	CONT	DISCT
1	**.592**	.170	.187	-.076	1	**.487**	.376*
2	**.368**	.395	.111	-.008	2	**.427**	.175*
3	.337	**.395**	.446	-.008	3	.255	**.657***
4	.316	**.452**	-.007	.228	4	.386	**.403***
5	.373	-.099	.039	**.500**			
6	.132	**.652**	.133	-.005			
7	**.531**	.339	-.171	.080	5	**.573**	.298*
8	.327	**.462**	.144	-.208	6	**.597**	.007
9	**.537**	.492	-.003	.048	7	**.622**	.329*
10	**.430**	.273	.052	.237	8	.405	**.452**
11	-.052	**.392**	.220	.233			
12	.082	-.066	**.437**	.385	9	.066	**.280**
13	.306	**.346**	.226	-.068			
14	-.094	.144	**.583**	-.064	10	.054	.050
15	**.519**	.008	.001	.206	11	**.488**	.161*
16	.380	**.626**	-.033	.215			
17	.330	**.397**	.103	.353	12	.384	**.497***
18	-.086	**.406**	.057	.389			
19	**.527**	.159	.345	.179			
20	.271	.128	**.530**	.160			
21	**.625**	-.049	.233	-.155	13	**.617**	-.073*
22	.361	.007	-.025	**.588**	14	.174	**.581**
23	-.103	.173	-.058	**.579**	15	-.293	**.753**
24	.075	**.580**	.209	.010			
25	.013	**.611**	.006	.149			
26	.018	.165	**.579**	-.027			
27	**.539**	.045	.130	-.108			
28	.046	.170	.253	**.459**			
29	.042	.152	**.543**	.125			
30	.174	-.129	**.553**	-.050			

Note. * indicates items loading on similar factors. CONT = Contentment; DISCT = Discontentment; COG=Cognitive impairment.

Table 4

Comparison of Diagnostic Groups on GDS-30 Subscale Scores

		Mean	*SD*		Mean	*SD*	*p*<
Dysphoria	DAT	2.00	2.26	VaD	3.15	2.91	**.01**
				MCI	3.12	2.75	**.04**
				Norm	2.12	2.25	.99
	VaD	3.15	2.91	MCI	3.12	2.74	.99
				Norm	2.12	2.25	.28
	MCI	3.12	2.74	Norm	2.12	2.25	.30
Meaninglessness	DAT	.89	1.21	VaD	1.49	1.57	**.02**
				MCI	1.16	1.58	.66
				Norm	.89	1.31	.99
	VaD	1.49	1.57	MCI	1.57	1.58	.57
				Norm	.89	1.31	.23
	MCI	1.16	1.58	Norm	.89	1.31	.85
Apathy	DAT	1.30	1.28	VaD	1.89	1.37	**.02**
				MCI	1.65	1.49	.42
				Norm	1.77	1.70	.39
	VaD	1.89	1.37	MCI	1.65	1.49	.77
				Norm	1.77	1.70	.98
	MCI	1.65	1.49	Norm	1.77	1.70	.98
Cognitive Impairment	DAT	2.05	1.35	VaD	1.97	1.64	.98
				MCI	1.84	1.40	.81
				Norm	1.62	1.39	.49
	VaD	1.97	1.64	MCI	1.97	1.64	.96
				Norm	1.62	1.39	.70
	MCI	1.97	1.64	Norm	1.62	1.39	.91

Note. DAT = Dementia of the Alzheimer's type; VaD = Vascular dementia; MCI = Mild cognitive impairment; Norm = No dementia, normal.

Table 5

Comparison between DAT, VaD, and MCI at Early Severity Level

		Mean	*SD*		Mean	*SD*	*p*<
Dysphoria	DAT	1.87	2.27	VaD	3.20	2.07	.**04**
				MCI	3.12	2.72	.**04**
	VaD	3.20	2.07	MCI	3.12	2.72	.99
Meaninglessness	DAT	.72	.92	VaD	1.40	1.37	.**03**
				MCI	1.16	1.58	.18
	VaD	1.40	1.37	MCI	1.16	1.58	.65
Apathy	DAT	1.22	1.29	VaD	1.98	1.51	**.03**
				MCI	1.65	1.49	.25
	VaD	1.98	1.51	MCI	1.65	1.49	.52
Cognitive Impairment	DAT	2.07	1.41	VaD	2.30	1.65	.72
				MCI	1.84	1.40	.71
	VaD	2.30	1.65	MCI	1.84	1.40	.31

Note. DAT = Dementia of the Alzheimer's type; VaD = Vascular dementia; MCI = Mild cognitive impairment; Norm = No dementia, normal.

Table 6

Comparison between DAT and VaD at Mild Severity Level

		Mean	*SD*		Mean	*SD*	*p*<
Dysphoria	DAT	2.20	2.31	VaD	3.37	2.79	.11
Meaninglessness	DAT	1.13	1.47	VaD	1.73	1.84	.15
Apathy	DAT	1.33	1.47	VaD	1.73	1.20	.38
Cognitive Impairment	DAT	2.07	1.31	VaD	1.63	1.54	.054

Note. DAT = Dementia of the Alzheimer's type; VaD = Vascular dementia; MCI = Mild cognitive impairment; Norm = No dementia, normal.

Table 7

Comparison between DAT and VaD Subscale Scores at Moderate Severity Level

		Mean	*SD*		Mean	*SD*	*p*<
Dysphoria	DAT	1.64	1.96	VaD	1.00	1.15	.56
Meaninglessness	DAT	.55	.93	VaD	.50	1.00	.94
Apathy	DAT	1.55	1.21	VaD	2.25	1.25	.34
Cognitive Impairment	DAT	1.91	1.30	VaD	1.25	1.44	.45

Note. DAT = Dementia of the Alzheimer's type; VaD = Vascular dementia; MCI = Mild cognitive impairment; Norm = No dementia, normal.

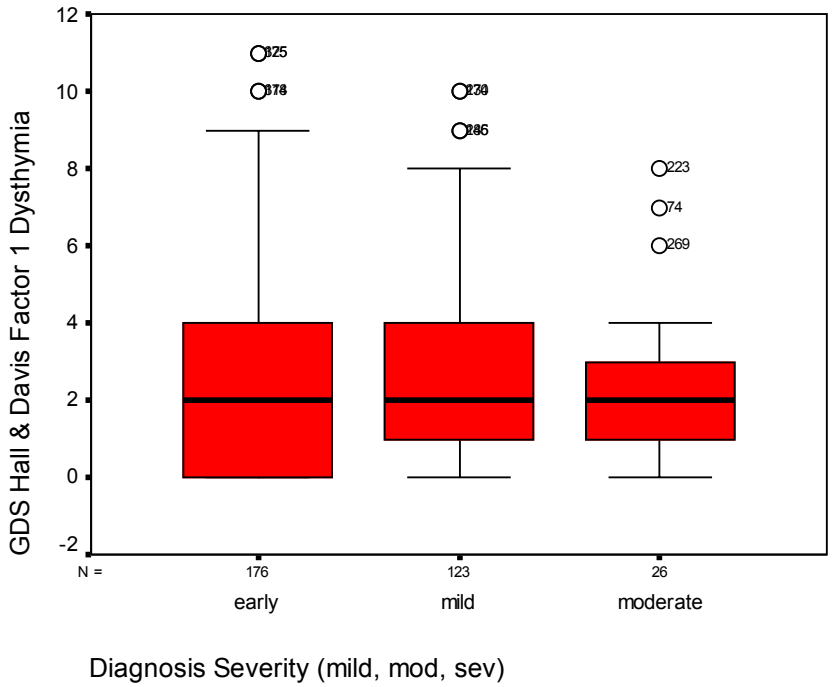

Figure 1. Box plot graph comparing diagnosis severity with Factor 1 of the Geriatric Depression Scale-30.

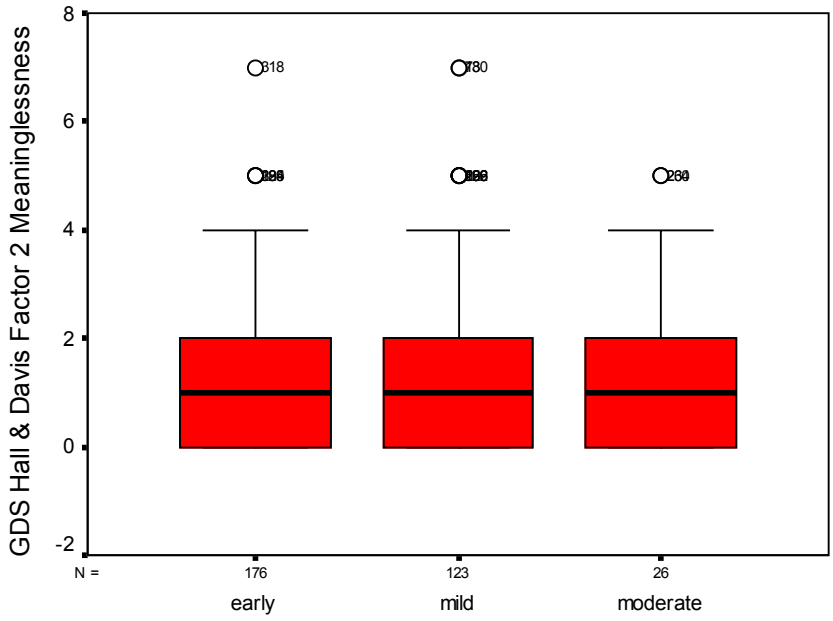

Figure 2. Box plot graph comparing diagnosis severity with Factor 2 of the Geriatric Depression Scale-30.

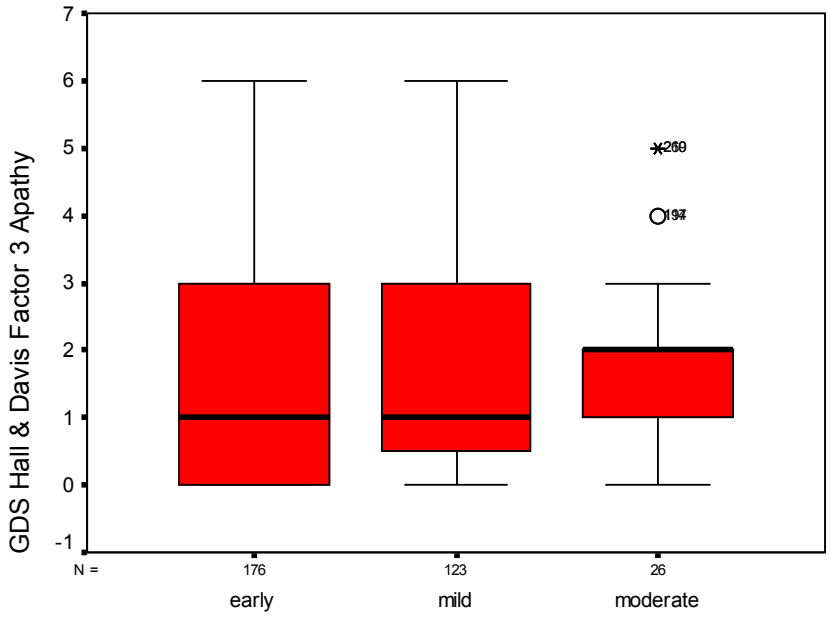

Figure 3. Box plot graph comparing diagnosis severity with Factor 3 of the Geriatric Depression Scale-30.

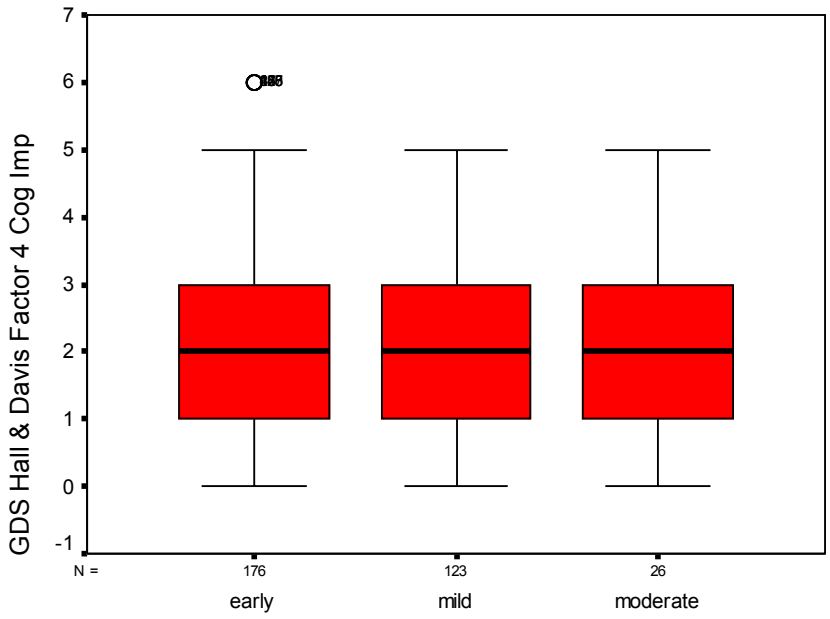

Figure 4. Box plot graph comparing diagnosis severity with Factor 4 of the Geriatric Depression Scale-30.

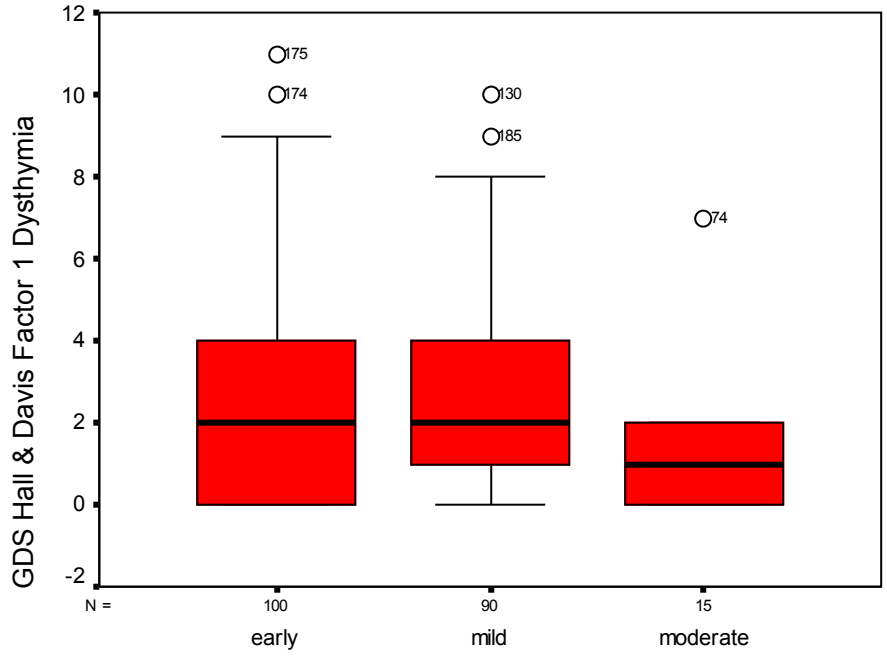

Figure 5. Box plot graph comparing DAT and VaD patients with Factor 1 of the Geriatric Depression Scale-30.

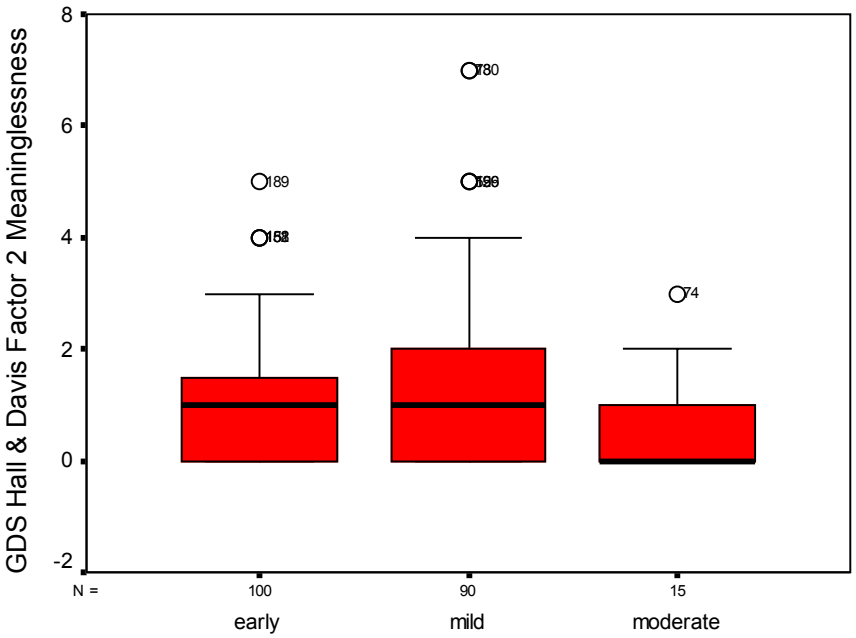

Figure 6. Box plot graph comparing DAT and VaD patients with Factor 2 of the Geriatric Depression Scale-30.

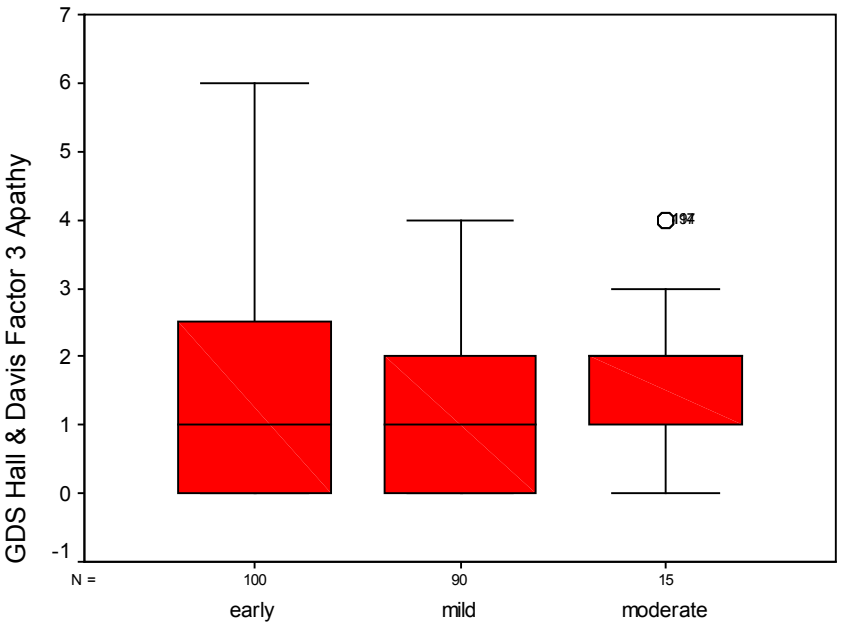

Figure 7. Box plot graph comparing DAT and VaD patients with Factor 3 of the Geriatric Depression Scale-30.

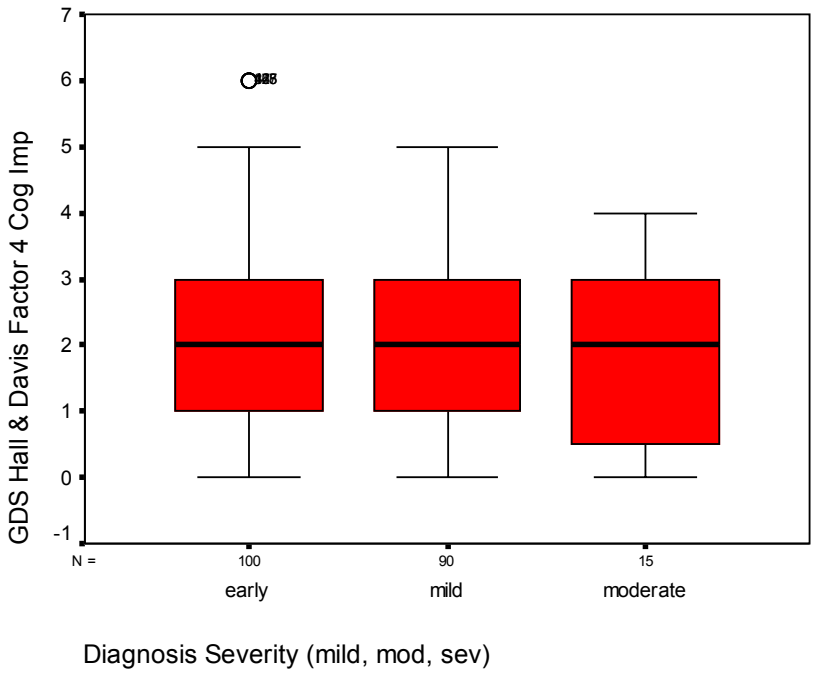

Figure 8. Box plot graph comparing DAT and VaD patients with Factor 4 of the Geriatric Depression Scale-30.

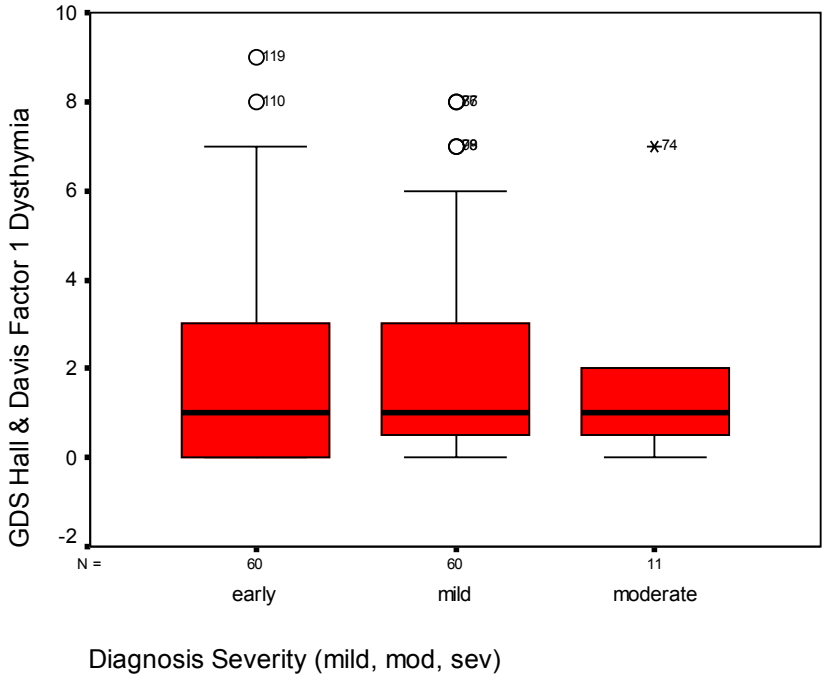

Figure 9. Box plot graph comparing DAT patients with Factor 1 of the Geriatric Depression Scale-30.

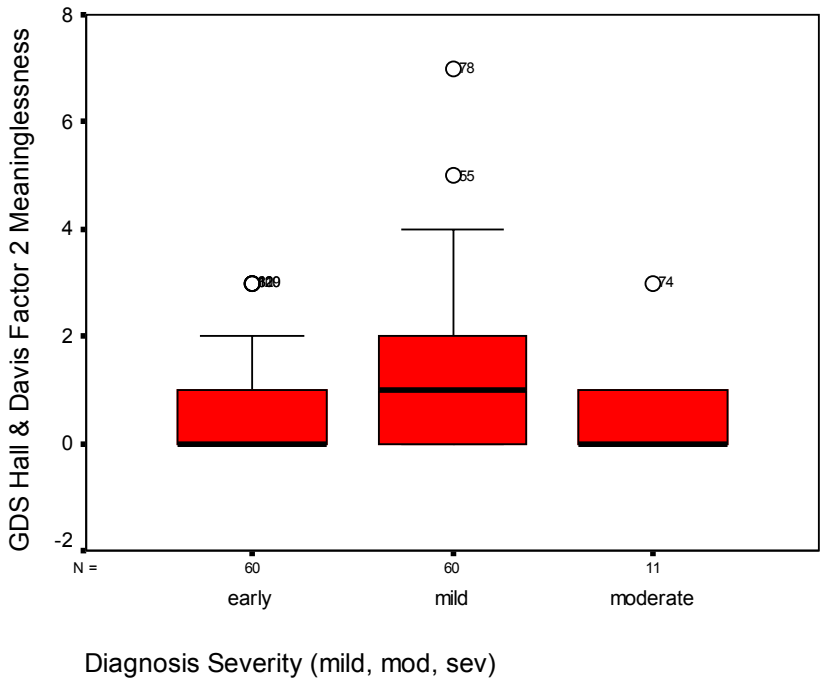

Figure 10. Box plot graph comparing DAT patients with Factor 2 of the Geriatric Depression Scale-30.

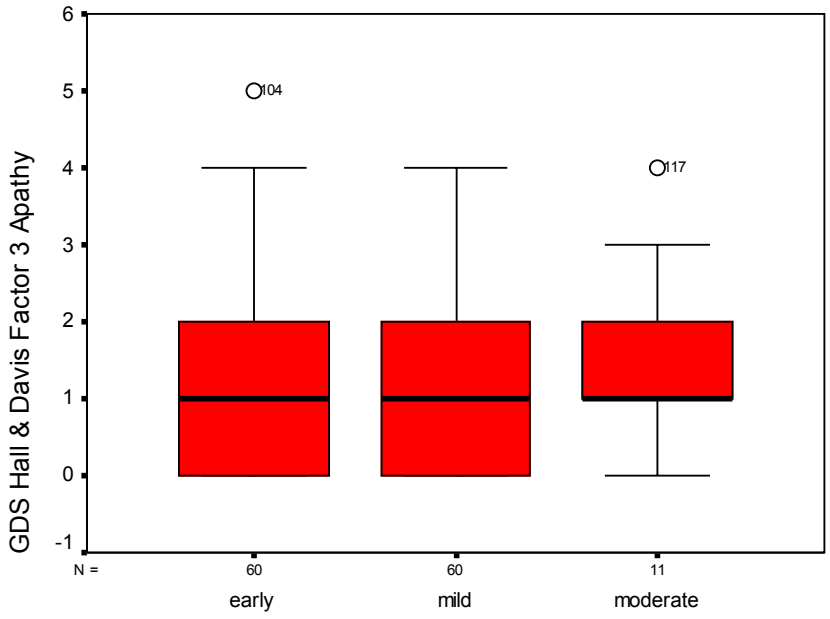

Figure 11. Box plot graph comparing DAT patients with Factor 3 of the Geriatric Depression Scale-30.

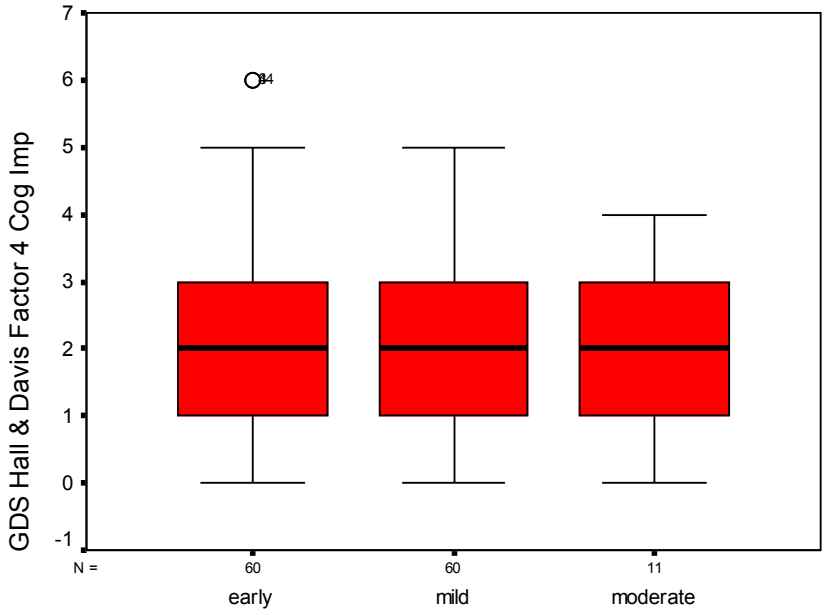

Figure 12. Box plot graph comparing DAT patients with Factor 4 of the Geriatric Depression Scale-30.

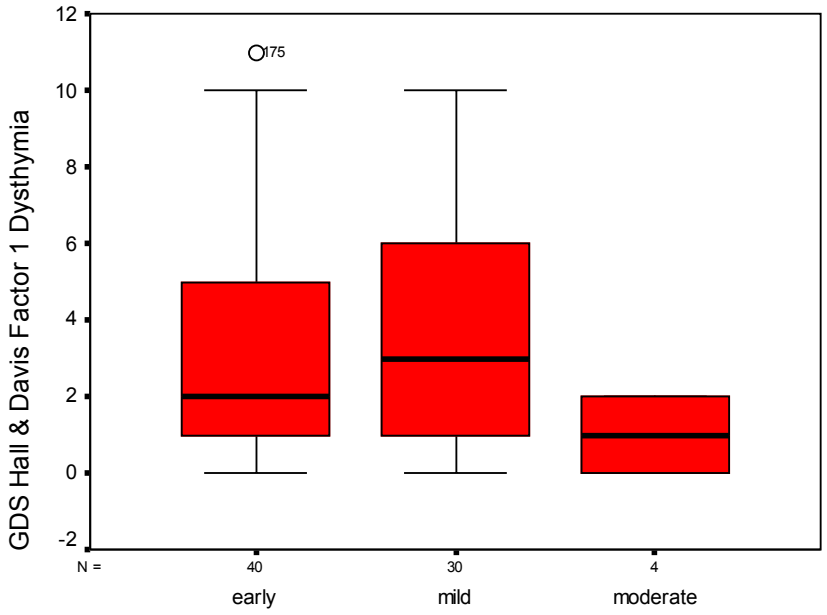

Figure 13. Box plot graph comparing VaD patients with Factor 1 of the Geriatric Depression Scale-30.

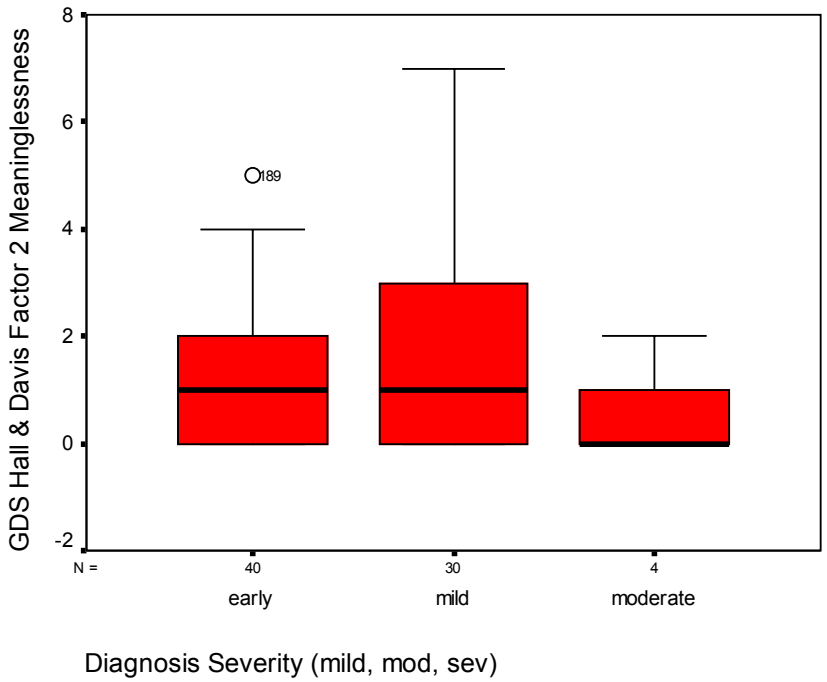

Figure 14. Box plot graph comparing VaD patients with Factor 2 of the Geriatric Depression Scale-30.

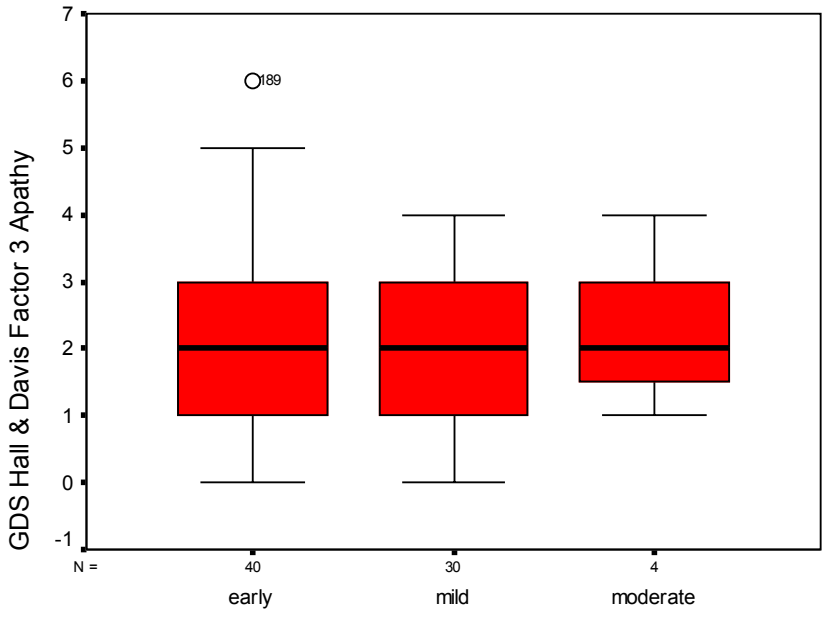

Figure 15. Box plot graph comparing VaD patients with Factor 3 of the Geriatric Depression Scale-30.

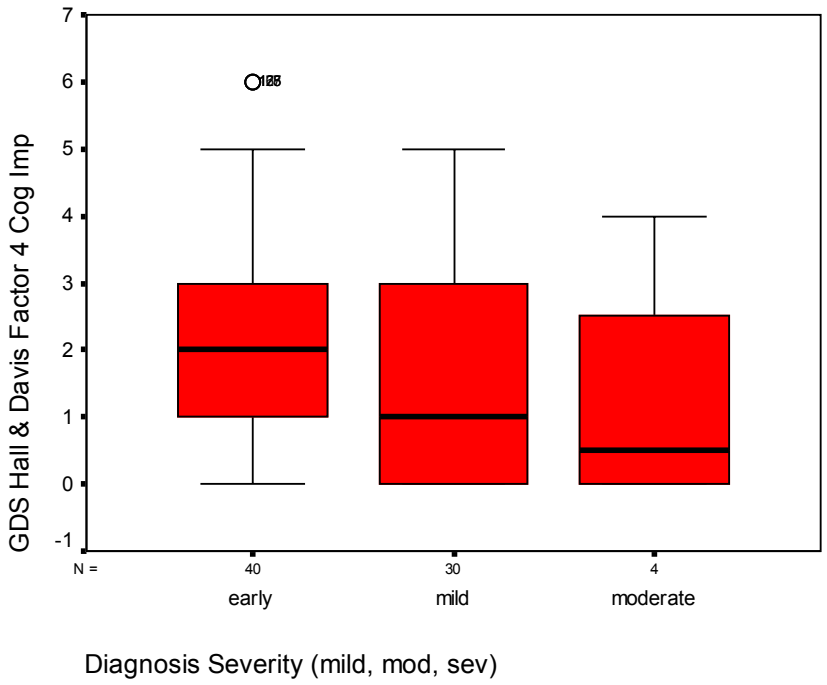

Figure 16. Box plot graph comparing VaD patients with Factor 4 of the Geriatric Depression Scale-30.

APPENDIX

DATA COLLECTION FORM

NEUROCOGNITIVE DATABASE WORKSHEET

Name: Date of Birth: Date of Exam:

Sex: Ethnicity: Previous Exam(s)? If yes, date(s) _____

WMS: ___
Extended Orientation:

Logical Memory I: Total A____ Total B_1 ____ Total B2 ___ Total B1 + B2 ___
LM I Raw Score ___ LM I Scale Score ___ LM I Slope ___

Visual Reproduction I: A ___ B ___ C ___ D ___ E ___ Total ___ Scale ___

LNS Total ___ LNS Scale ___ Spatial Span For ___ Spatial Scan Back ___
Spatial Scan For Scale ___ Spatial Scan Back Scale ___
Spatial Span Total ___ Spatial Scan Total Scale ___

Mental Control: (Individual items). MC Total ___ MC Scale ___
Digit Span For ___ DS Back ___ DS Total ___ DS Scale ___

Logical Memory II: Total A____ Total B ____
LM II Raw Score ___ LM II Scale Score ___ LM I Retention ___
Logical Memory Recognition: (Individual items) Total A ___ Total B ___ Total A+B ___

Visual Reproduction II: A ___ B ___ C ___ D ___ E ___ Total ___ Scale ___
Retention Total ___ Ret Scale ___ Recognition: (Individual items) Total ___ Scale ___

CERAD:
Verbal Fluency 0-15 sec ___ 16-30 sec ___ 31-45 sec ___ 45-60 sec ___ Total ___

BNT: High ___ Medium ___ Low ___ Total ___

MMSE: (Individual items) Total ___ T Score: ___

Word List Recognition: Trial 1 ___ Trial 2 ___ Trial 3 ___ Total ___ (Individual items)

Constructional Praxis: Circle ___ Diamond ___ Rectangles ___ Box ___ Total ___
Word List Recall: _____
Word List Recognition: # Yes Correct ___ # No Correct ___ Total Correct ___

Hooper: Total Score ___ T Score ___ (Individual items)

BDS: Total ___ (Individual items)

GDS: Total ___ (Individual items)

Trails A: Time ___ Scale Score ___ T Score ___
Trails B: Time ___ Scale Score ___ T Score ___
Clock: Circle ___ Numbers ___ Spacing ___ Time ___ Total ___
Traffic Sign Naming: _____
ADAS Cog Conversion Score: ___

Dx: _____ **Dx Severity** ____ **CDRS:**___

REFERENCES

Aalten, P., van Valen,E. Clare, L., Kenny G. & Verhey, F. (2005). Awareness in dementia: A review of clinical correlates. *Aging & Mental Health, 9* (5), 414-422.

Adams, K. B. (2001). Depressive symptoms, depletion, or developmental change? Withdrawal, apathy, and lack of vigor in the Geriatric Depression Scale. *Gerontologist, 41*, 768-777.

Adams, K.B., Matto, H.C. & Sanders, S. (2004). Confirmatory factor analysis of the Geriatric Depression Scale. *Gerontologist, 44* (6)*,* 818-826.

Adler, G., Chwalek, K., & Jajcevic, A. (2004). Six-month course of mild cognitive impairment and affective symptoms in late-life depression. *European Psychiatry, 19*, 502-505.

Alexopoulos, G. (2003). Vascular disease, depression, and dementia. *Journal of the American Geriatrics Society, 51*, 1178-1180.

Alexopoulos, G. (2006). The vascular depression hypothesis: 10 years later. *Biological Psychiatry, 60*, 1304-1305.

Alexopoulos, G., Kiosses, D., Choi, S., Murphy, C., & Lim, K. (2002). Frontal white matter microstructure and treatment response of late-life depression: A preliminary study. *American Journal of Psychiatry, 159*, 1929-1932.

Alexopoulos, G., Meyers, B., Young, R., Kakuma, T., Silbersweig, D., & Charlson, M. (1997). Clinically defined vascular depression. *American Journal of Psychiatry, 154*, 562-565.

Alladi, S., Arnold, R., Mitchell, J., Nestor, P., & Hodges, J. (2006). Mild cognitive impairment: Applicability of research criteria in a memory clinic and characterization of cognitive profile. *Psychological Medicine, 36*, 507-515.

Alzheimer's Association (2008). 2008 Alzheimer's disease facts and figures. *Alzheimer's & Dementia, 4* (2). Retrieved June 3, 2008, from http://www.alz.org/national/documents/report_alzfactsfigures2008.pdf

Amore, M., Tagariello, P., Laterza, C., & Savoia, E. (2007a). Beyond, Nosography of depression in elderly. *Archives of Gerontological Geriatrics, Supplement 1*, 13-22.

Amore, M., Tagariello, P., Laterza, C., & Savoia, E. (2007b). Subtypes of depression in dementia. *Archives of Gerontological Geriatrics, Supplement 1*, 23-33.

Alzheimer's Association (2008). *Alzheimer's disease facts and figures*. Retrieved May 1, 2008, from http://www.alz.org/national/documents/report_alzfactsfigures2008.pdf

Barnes, D., Alexopoulos, G., Lopez, O., Williamson, J., & Yaffe, K. (2006). Depressive symptoms,vascular disease, and mild cognitive impairment – findings from the cardiovascular health study. *Archives of General Psychiatry*, *63*, 273-280.

Bentz, B. & Hall, J. (2008). Assessment of depression in a geriatric inpatient cohort: A comparison of the BDI and GDS. *International Journal of Clinical and Health Psychology, 8* (1), 93-104.

Bierman, E.J., Comijs, H.C., Jonker, C., Beekman, A.T. (2007). Symptoms of anxiety and depression in the course of cognitive decline. *Dementia, Geriatric Cognition & Disorders, 24* (3)*,* 213-219.

Bondi, M. (2004). Subjective memory deterioration in elderly people is associated with future dementia. Evidence *Based Mental Health, 8*, 47.

Caballero, J., Hitchcock, M., Beversdorf, D., Scharre, D., & Nahata, M. (2006). Long-term effects of antidepressants on cognition in patients with Alzheimer's disease. *Journal of Clinical Pharmacy and Therapeutics, 31*, 593-598.

Ceccaldi, M. (2006). Vascular dementia: From concepts to clinical practice. *Psychogeriatrics, 6*, S38-S41.

Chinello, A., Grumelli, B., Perrone, C., & Annoni, G. (2007). Prevalence of major depressive disorder and dementia in psychogeriatric outpatients*. Archives of Gerontological Geriatrics, Suppliment 1*, 101-104.

Cotter, V., Clark, C. & Karlawish, J. (2003). Cognitive function assessment in individuals at risk for Alzheimer's disease. *Journal of the American Academy of Nurse Practitioners, 15*, 79-86.

Crowe, M., Andel, R., Wadley, V., Cook, S., Unverzagt, F., Marsiske, M., & Ball, K. Subjective cognitive function and decline among older adults with psychometrically defined amnestic MCI. *International Journal of Geriatric Psychiatry, 21*, 1187-1192.

Eibenstein, A., Fioretti, A., Simaskou, M., Sucapane, P., Mearelli, S., Mina, C., Amabile, G. & Fusetti, M. (2005). Olfactory screening test in mild cognitive impairment. *Neurological Sciences, 26*, 156-160.

Ertan, T & Eker, E. (2000). Depression, reliability, validity, and factor structure of the Geriatric Depression Scale in Turkish elderly: Are there different factor structures for different cultures? *Internationa Psychogeriatrics, 12* (2), 163-172.

Fawcett, T. (2006). An introduction to ROC analysis. *Pattern Recognition Letters*, 27, 861-874

Fountoulakis, K., O'Hara, R., Iacovides, A., Camilleri, C., Kaprinis, S., Kaprinis, G., & Yesavage, J. (2003). Review: Unipolar late-onset depression: A comprehensive review. *Annals of General Hospital Psychiatry, 2*, retrieved June 14 2007 at http://www.annals-general-psychiatry.com/content/pdf/1475-2832-2-11.pdf

Friedman, B., Heisel, M.J. & Delavan, R.L. (2005). Psychometric properties of the 15-item Geriatric Depression Scale in functionally impaired, cognitively intact, community-dwelling elderly primary care patients. *Journal of the American Geriatrics Society, 53,* 1570-1576.

Gabryelewicz, T., Styczynska, M., Pfeffer, A., Wasiak, B., Barczak, A., Luczywek, E., Androsiuk, W., & Barcikowska, M. (2004). Prevalence of major and minor depression in elderly persons with mild cognitive impairment – MADRS factor analysis. *International Journal of Geriatric Psychiatry, 19*, 1168-1172.

Gauthier, S., Reisberg, B., Zaudig, M., Petersen, R., Ritchie, K., Broich, K., Belleville, S., Brodaty, H., Bennet, D., Chertkow, H., Cummings, J., de Leon, M., Feldman, H., Ganguli, M., Hampel, H., Scheltens, P., Tierney, M. Whitehouse, P. & Winblad, B. (2006). Mild cognitive impairment. *Lancet, 367*, 1262-1270.

Geerlings, M.I., den Heijer, T., Koudstaal, P.J., Hofman, A. & Breteler, M.M. (2008). History of depression, depressive symptoms, and medical temporal lobe atrophy and the risk of Alzheimer's disease. *Neurology, 70* (15), 1258-1264

Grimm, L.G. & Yarnold, P.R. (1995). *Reading and understanding multivariate statistics.* Washington, DC: American Psychological Association.

Hall, J. & Davis, T. (in press). Factor structure of the Geriatric Depression Scale in cognitively impaired older adults. *Clinical Gerontologist*.

Hall, J., Davis, T., & Cornwell, S. (in review). *Components of geriatric depression and types of cognitive impairment.*

Heun, R. & Hein, S. (2005). Risk factors of major depression in the elderly. *European Psychiatry, 20,*199-204.

Holtzer, R., Scarmeas, N., Wegesin, D., Albert, M., Brandt, J., Dubois, B., Hadjigeorgiou, G., & Stern, Y. (2005). Depressive symptoms in Alzheimer's disease: Natural course and temporal relation to function and cognitive status. *Journal of the American Geriatrics Society, 53*, 2083-2089.

Janzing, J.G., Bouwens, J.M., Teunisse, R.J., Van't Hof M.A. & Zitman, F. G. (1999). The relationship between depression and mortality in elderly subjects with less severe dementia. *Psychological Medicine, 29* (4), 979-983.

Jicha, G., Parisi, J., Dickson, D., Johnson, K., Cha, R., Ivnik, R., Tangalos, E., Boeve, B., Knopman, D., Braak, H., Petersen, R. (2006). Neuropathologic outcome of mild cognitive impairment following progression to clinical dementia. *Archives of Neurology, 63*, 674-681.

Kaufer, D.I., Cummings, J.L., Christine, D., Bray, T., Castellon, S., Masterman, D., MacMillan, A., Ketchel, P. & DeKosky, S.T. (1998). Assessing the impact of neuropsychiatric symptoms in Alzheimer's disease: The Neuropsychiatric Inventory Caregiver Distress Scale. *Journal of American Geriatric Society, 46* (2)*,* 210-215.

Kessels, R., Ruis, C., & Kappelle, L. (2007). The impact of self-reported depressive symptoms on memory function in neurological patients. *Clinical Neurology and Neurosurgery, 109*, 323-326.

Kessing, L.V. & Anderson, P.K. (2004). Does the risk of developing dementia increase with the number of episodes in patients with depressive disorder and in patients with bipolar disorder? *Journal of Neurology and Neurogurgery Psychiatry, 75*(12), 1662-1666.

Korczyn, A. (2005). The underdiagnosis of the vascular contribution to dementia. *Journal of the Neurological Sciences, 229-230*, 3-6.

Korner, A., Lauritzen, L., Abelskov, K., Gulmann, N., Brodersen, A.M., Wedervang-Jensen, T., & Kjeldgaard, K.M. (2006). The Geriatric Depression Scale and the Cornell Scale for Depression in Dementia. A validity study. *Nordic Journal of Psychiatry, 60*, 360-364.

Krishnan, K., Taylor, W., McQuoid, D., MacFall, J., Payne, M., Provenzale, J., & Steffens, D. (2004). Clinical characteristics of magnetic resonance imaging-defined subcortical ischemic depression. *Biological Psychiatry, 55*, 390-397.

Kunik, M.E., Snow, A.L., Molinari, V.A., Menke, T.J., Souchek, J., Sullivan, G. & Ashton, C.M. (2003). Health care utilization in dementia patients with psychiatric comorbidity. *Gerontologist, 43* (1), 86-91.

Lai, D., Fung, T.S. & Yuen, C.T.Y. (2005). The factor structure of a Chinese version of the Geriatric Depression Scale. *International Journal of Psychiatry in Medicine, 35* (2)*,* 137-148.

Lanquillon, S., Krieg, J.C., Bening-Abu-Shach, U. & Vedder, H. (2000). Cytokine production and treatment response in major depressive disorder. *Neuropsychopharmacology, 22* (4)*,* 370-379.

Lawhorn, L. (2005). Depression in the older adult. *Primary Care: Clinics in Office Practice, 32*, 777-792.

Leonard, B.E. (2007). Inflammation, depression and dementia: Are they connected? *Neurochemical Research, 32,* 1749-1756.

Lyketsos, C., Lopez, O., Jones, B., Fitzpatrick, A., Breitner, J., & DeKosky, S. (2002). Prevalence of neuropsychiatric symptoms in dementia ad mild cognitive impairment. *Journal of American Medical Association, 288*, 1475-1483.

Maes, M., DeVos, N., Wauters, A., Demedts, P., Maurits, V.W., Neels, H., Bosmans, E., Altamura, C., Lin, A., Song, C., Vandenbroucke, M. & Scharpe, S. (1999). Inflammatory markers in younger vs. elderly normal volunteers and in patients with Alzheimer's disease. *Journal of Psychiatric Research, 33* (5), 397-405.

Marson, D. (2002). Competency assessment and research in an aging society. *Mental Health and Mental Illness in Later Life, 26,* 99-103.

Mast, B., Azar, A., & Murell, S. (2005). The vascular depression hypothesis: The influence of age on the relationship between cerebrovascular risk factors and depressive symptoms in community dwelling elders. *Aging and Mental Health, 9*, 146-152.

Mast, B.T., Yochim, B., MacNeill, S.E. & Lichtenberg, P.A. (2004). Risk factors for geriatric depression: The importance of executive functioning within the vascular depression hypothesis. *Journals of Gerontology: Series A: Biological Sciences and Medical Sciences, 59A* (12), 1290-1294.

Mehta, K.M., Yaffe, K., Langa, K.M., Sands, L., Whooley, M.A. & Covinsky, K.E. (2003). Additive effects of cognitive function and depressive symptoms on mortality in elderly community-livinig adults. *Journals of Gerontology: Series A: Biological Sciences and Medical Sciences, 58A* (5), 461-467.

Mondrego, P., & Ferrandez, J. (2004). Depression in patients with mild cognitive impairment increases the risk of developing dementia of Alzheimer type. *Archives of Neurology, 61*, 1290-1293.

Muller-Thomsen, T., Arlt, S., Mann, U, MaB, R & Ganzer, S. (2005). Detecting depression in Alzheimer's disease: Evaluation of four different scales. *Archives of Clinical Neuropsychology, 20*, 271-276.

Nakaaki, S., Murata, Y., Sato, J., Shinagawa, Y., Tatsumi, H., Hirono, N. & Furukawa, T. (2007). Greater impairment of ability in the divided attention task is seen in Alzheimer's disease patients with depression than in those without depression. *Dementia and Geriatric Cognitive Disorders, 23,* 231-240.

Onishi, J., Suzuki, Y., Umegaki, H., Endo, H., Kawamura, T., & Iguchi, A. (2006). A comparison of depressive mood of older adults in a community, nursing homes, and a geriatric hospital: Factor analysis of geriatric depression scale. *Journal of Geriatric Psychiatry & Neurology, 19* (1)*,* 26-32.

Owen, B.M. (2001). Raised plasma IL-6 beta in major and minor post viral depression. *Acta Psychiatrica Scandanavia, 103,* 226-228.

Persad, C., & Giordani, B. (2007). Neuropsychological advances in the differential diagnosis and treatment of neurocognitive deficits in older individuals. *Journal of Geriatric Psychiatry and Neurology, 20* (4), 187-188.

Petersen, R., Doody, R., Kurz, A., Mohs, R., Morris, J., Rabins, P., Ritchie, K., Rossor, M., Thal, L., & Winblad, B. (2001). Current concepts in mild cognitive impairment. *Archives of Neurology, 58*, 1985-1992.

Petersen, R. (2004). Mild cognitive impairment as a diagnostic entity. *Journal of Internal Medicine, 256*, 183-194.

Petersen, R., & Morris, J. (2005). Mild cognitive impairment as a clinical entity and treatment target. *Archives of Neurology, 62*, 1160 – 1163.

Petersen, R., Smith, G., Waring, S., Ivnik, R., Tangalos, E., & Kokmen, E. (1999). Mild cognitive impairment: Clinical characterization and outcome. *Archives of Neurology, 56*, 303-308.

Ritter, E., Despres, O., Monsch, A. & Manning L. (2006). Topographical recognition memory sensitive to amnestic mild cognitive impairment but not to depression. *International Journal of Geriatric Psychiatry, 21*, 924-929.

Rosenthal, R. & Kavic, S. (2004). Assessment and management of the geriatric patient. *Critical Care Medicine, 32* (4 supplement), S92-S105.

Starkstein, S.E., Jorge, R., Mizrahi, R. & Robinson, R.G. (2005). The construct of minor and major depression in Alzheimer's disease. *American Journal of Psychiatry, 162* (11), 2086-2093.

Steffens, D.C. & Potter, G.G. (2008). Geriatric depression and cognitive impairment. *Psychological Medicine, 38,* 163-175.

Stickle, F., & Onedera, J. (2006). Depression in older adults. *Adultspan Journal, 5*, 36-44.

Suh, G.H., Kil Yeon, B., Shah, A. & Lee, J.Y. (2005). Mortality in Alzheimer's diseas: A comparative prospective Korean study in the community and nursing homes. *International Journal of Geriatric Psychiatry, 20* (1), 26-34.

Taylor, W., Steffens, D., & Krishnan, K. (2006). Psychiatric disease in the twenty-first century: The case for subcortical ischemic depression. *Biological Psychiatry, 60*, 1299-1303.

Thomas, A., Kalaria, R., & O'Brien, J. (2004). Depression and vascular disease: What is the relationship? *Journal of Affective Disorders, 79*, 81-95.

van Reekum, R. (2006). Depressive symptoms in elderly people are associated with an increased risk of developing mild cognitive impairment independently of vascular disease. *Evidence Based Mental Health, 9*, 111.

Verkiak, R., Nuyen, J., Schellevis, F. & Francke, A. (2007). The relationship between severity of Alzheimer's disease and prevalence of comorbid depressive symptoms and depression: A systematic review. *International Journal of Geriatric Psychiatry, 22* (11), 1063-1086.

Wancata, J., Alexandrowicz, R., Marquart, B., Weiss, M. & Friedrich, F. (2006). The criterion validity of the geriatric depression scale: A systematic review. *Acta Psychiatrica Scandinavica, 114*, 398-410.

Wilson, R.S., Arnold, S.E., Beck, T.L., Bienias, J.L. & Bennett, D.A. (2008). Change in depressive symptoms during the prodromal phase of Alzheimer's disease. *Archives of General Psychiatry, 65* (4), 439-445.

Winblad, B., Palmer, K., Kivipelto, M., Jelic, V., Fratiglioni, L., Wahlund, L., Nordberg, A., Backman, L., Albert, M, Almkvist, O., Arai, H., Basun, H., Blennow, K. DeLeon, M., Decarli, C., Erkinjuntti, T., Giacobini, E., Graff, C., Hardy, J., Jack, C., Jorm, A., Ritchie, K., VanDuijn, C., Visser, P. & Petersen, R. (2004). Mild cognitive impairment – beyond controversies, towards a consensus: Report of the International Working Group on Mild Cognitive Impairment*. Journal of Internal Medicine, 256*, 240-246.

Wright, S., & Persad, C. (2007). Distinguishing between depression and dementia in older persons: Neuropsychological and neuropathological correlates. *Journal of Geriatric Psychiatry and Neurology, 20* (4), 189-198.

Xie, Z. & Tanzi, R.E. (2006). Alzheimer's disease and post-operative cognitive dysfunction. *Experimental Gerontology, 41,* 346-359.

Yesavage, J.A., Brink, T.L., Rose, T.L., Lum, O., Huang, V., Adey, M. & Leirer, V.O. (1983). Development and validation of a geriatric depression screening scale: A preliminary report. *Journal of Psychiatric Research, 17* (1), 37-49.

Yochim, B., MacNeill, S & Lichtenberg, P. (2006). "Vascular depression" predicts verbal fluency in older adults*. Journal of Clinical and Experimental Neuropsychology, 28*, 495-508.

Zubenko, G.S., Zubenko, W.N., McPherson, S., Spoor, E., Marin, D.B., Farlow, M.R., Smith, G.E., Geda, Y.E., Cummings, J.L., Petersen, R.C., and Sunderland, T. (2003). A collaborative study of the emergence and clinical features of the major depressive syndrome of Alzheimer's disease. *American Journal of Psychiatry, 160* (5)*.* 857-866.