

Recognition Memory and Response Bias in Elderly Demented,
Depressed, and Demented/Depressed Patients

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ABSTRACT

Elderly depressed patients are often referred for a neuropsychological examination to evaluate their cognitive abilities and to assist in making a differential diagnosis of dementia vs. depression when the etiology of presenting symptoms is unclear. Indices of discrimination (i.e., the ability to identify target from distractor items during recognition memory testing) and response bias (i.e., the probability of saying "yes" to an item when uncertain if it is a target or a distractor) have been suggested as useful aids when making such a differential diagnosis. Prior research indicated that discrimination abilities were significantly better in depressed elderly as compared to demented elderly and that depressed patients used the most conservative response strategy (i.e., they tended to say "no" more often when uncertain) while demented patients had a more liberal response style (i.e., they tended to say "yes" more often). However, little is known about the performance of patients with dementia complicated by depression.

This study attempted to address the nature of discrimination ability and response bias in patients with dementia complicated by depression, as compared to demented patients without affective symptoms and depressed patients without cognitive impairment. Results indicated that

demented and demented/depressed groups had poorer discrimination abilities than the depressed group. Discrimination abilities of the demented and the demented/depressed groups were similar. Pattern of responses, however, reflected a significantly more conservative response bias in the demented/depressed group as compared to the demented group. The demented/depressed group were also more conservative than the depressed group, but this comparison did not reach statistical significance.

These results suggest that the combined effects of dementia and depression have a "conservatizing" effect on response bias in demented/depressed patients. This "conservatizing" effect may provide useful information when considering if an underlying depression exists in a demented patient.

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DEDICATION

To my family

"It is the very best of gifts God gave,
You take it with you from the cradle to the
grave;
It is a family tie,
It is a family tie that binds."

Maura O'Connell
"A Family Tie"

I. INTRODUCTION

Major depression complicates unequivocal dementia in 10-20% of demented patients and significant depressive features occur in 40-50% of demented patients (Wragg, 1989). The diagnosis of depression in demented patients, however, is often confounded by the overlap of symptoms of depression and dementia (e.g., psychomotor changes, appetite and sleep disturbances, diminished interest). These symptoms, commonly associated with depression, are also often part of the dementia syndrome in patients who do not have depression (Greenwald, Mather, Mohs, Levy, Johns, and Davis, 1986). Therefore, a common clinical circumstance is the demented patient who is suspected of having depression, but wherein the clinician cannot confidently discriminate the diagnosis of depression.

Conversely, depression in the elderly is often accompanied by depression-dependent cognitive impairment that is reversible following antidepressant therapy. This condition has been referred to as "pseudodementia" (Kiloh, 1961), "dementia syndrome of depression" (Folstein and McHugh, 1978), and more recently "depression with cognitive impairment" (Folstein and Rabins, 1991). Diagnosis of depression in demented individuals is critical since the development of depression superimposed upon a dementia may be accompanied by a reversible depression-dependent cognitive impairment that worsens the overall cognitive

functioning of the patient (Breen, Larson, Reifler, Vitaliano, and Lawrence, 1984; Greenwald, Kramer, Ginsberg, Marin, Hermann, Mohs, and Davis, 1989; McAllister and Price, 1982; Rovner, Broadhead, Spencer, Carson, and Folstein, 1989; Shraberg, 1978; Snow and Wells, 1981). Somatic antidepressant treatments have been shown to improve mood (Reifler, Larson, Teri, and Poulson, 1986; Reifler, Teri, Raskind, Veith, Barnes, White, and McLean, 1989; Reynolds, Perel, Kupfer, Zimmer, Stack, and Hoch, 1987; Snow and Wells, 1981) and cognitive status (Greenwald, et al., 1989; McAllister and Price, 1982; Shraberg, 1978) in demented patients with a superimposed depression. Cognitive functioning improves presumably on the basis of remission of depression and alleviation of depression-mediated cognitive deficits. Therefore, successful identification and treatment of depression in demented patients may represent the only "improvable" component in an otherwise unremitting cognitive deterioration. The establishment of a neuropsychological tool to aid the identification of depression in diagnostically enigmatic demented patients would be an important contribution to differential diagnosis. At present, however, little is known about the performance of demented patients with depression on neuropsychological measures.

The current study was designed to further investigate the cognitive functioning of demented patients with

depression and to attempt to identify distinguishing characteristics that may assist with differential diagnosis. Specifically, demented/depressed patients were compared to non-depressed demented and non-demented depressed patient groups on a forced-choice recognition memory task, which yields indices of discrimination ability and response bias. Response bias reflects the respondent's tendency to say "yes" or "no" when uncertain if a given stimulus is a target or a distractor item. Results of prior research indicated that discrimination abilities were better in depressed patients than in demented patients and that depressed patients manifested a conservative response bias (i.e., they tended to say "no" more often when uncertain) while demented patients showed a more liberal response bias (i.e., they tended to say "yes" more often) (Brandt, Corwin, and Krafft, 1992; Corwin, Peselow, Feenan, Rotrosen, and Fieve, 1990; Hart, Smith, and Swash, 1985; Larner, 1977; Massman, Delis, Butters, Levin, and Salmon, 1990; Miller and Lewis, 1977; Niederehe, 1986; Snodgrass and Corwin, 1988). This study will attempt to replicate these findings and newly address the nature of discrimination ability and response bias in demented patients with depression. By identifying discrete cognitive characteristics of this diagnostically enigmatic group, demented patients with depression may be more readily identified.

In the following review the prevalence and presentation of dementia and depression in the elderly will be discussed, followed by a description of current approaches used in making the differential diagnosis of dementia versus depression. The prevalence and presentation of symptoms in persons with both dementia and depression will then be reviewed, followed by a description of prior attempts to define distinguishing characteristics of this group.

II. LITERATURE REVIEW

A. DEFINITION AND PREVALENCE OF DEMENTIA

Dementia may be broadly defined as an unusual loss of intellectual function that is beyond what would be expected due to normal aging (Joynt and Shoulson, 1985). Cummings, Benson, and LoVerme (1980) defined dementia more specifically as an acquired persistent loss of intellectual function with compromised abilities in at least three of the following five areas: cognition (i.e., abstraction, calculation, judgement), memory, visuospatial skills, language, and personality.

In an attempt to gain some uniformity in the diagnosis of dementia, the National Institute of Neurology and Communication Disorders and Stroke (NINCDS) and the Alzheimer's Disease and Related Disorders Association (ADRDA) (McKhann, Drachman, Folstein, Katzman, Price, and Stadlan, 1984), outlined criteria for the diagnosis, which included "...the decline of memory and other cognitive functions in comparison with the patient's previous level of functioning as determined by history of decline in performance and by abnormalities noted from clinical examination and neuropsychological tests." The diagnosis cannot be made when consciousness is impaired. The NINCDS-ADRDA report further pointed out that dementia is a diagnosis based on behavior and cannot be determined by

computed tomography, electroencephalography, or other laboratory instruments (although causes of dementia may be discovered with the aid of these techniques).

The definition of dementia was operationalized to a somewhat greater extent in the Diagnostic and Statistical Manual of Mental Disorders (Third Edition - Revised) (DSM III-R; American Psychiatric Association, 1987). The DSM III-R defines dementia as an overall decline in intellectual functioning; the essential feature is impairment in short and long term memory, and is associated with at least one of the following: 1) impairment in abstract thinking (e.g., difficulty with novel tasks), 2) impaired judgement (e.g., inappropriate joking, neglect of personal appearance), 3) other disturbances of higher cortical function (e.g., aphasia, apraxia, agnosia), or 4) personality change (i.e., either an alternation or an accentuation of premorbid traits). These disturbances must be significant enough to interfere with work, usual social activities, or relationships with others and must occur when consciousness is intact. In addition, DSM III-R requires evidence of an organic etiologic factor related to the disturbance (from history, physical examination, or laboratory tests); if an organic basis to the disturbance cannot be determined, it may be presumed if the presenting symptoms cannot be accounted for by a "nonorganic mental disorder", such as Major Depression. DSM III-R divides dementia into general

diagnostic entities, based on presumed or proven etiology including Primary Degenerative Dementia of the Alzheimer Type, Multi-Infarct Dementia, and Dementia Not Otherwise Specified.

Current estimates suggest that approximately six percent of persons over age 65 have severe dementia and an additional 10-15% have mild to moderate dementia (Cummings and Benson, 1992). The prevalence of dementia doubles approximately every five years after age 65 (Jorm, Korten, and Henderson, 1987). It is expected that this population group will triple by the year 2050 due to a bulk of the so-called "baby-boomer" generation entering their senior years; in addition, improved health care is helping many individuals in this age group live longer lives.

Dementia is a symptom of many different disorders and not a disease unto itself. Causes of dementia include structural, metabolic, toxic, and psychiatric abnormalities.

The most common cause of dementia is Alzheimer's disease (AD), which has been found to account for 50 - 70 % of older patients with dementia seen in psychiatric practice (Kaszniak, 1986). Cummings and Benson (1992) cautioned, however, that these figures may be overestimates; that is, since the diagnosis of AD is one of exclusion based on the absence of other identifiable causes of dementia, such a category is likely to include unrecognized causes of dementia in addition to true cases of AD.

The clinical features of AD include memory loss, language abnormalities, visuospatial impairment, impaired abstraction and calculation, personal and behavioral changes, and intact motor function (Cummings, 1987). AD has an insidious onset and progresses gradually, often leading to death in 6-12 years.

As mentioned, the diagnosis of AD is one of exclusion; that is, other systemic or brain diseases which may account for the cognitive deterioration must first be ruled out. The NINCDS-ADRDA outlined criteria for possible, probable, and definite AD (McKhann et al., 1984). A diagnosis of definite AD requires histopathological evidence, obtained from biopsy or autopsy, of an excess number of neurofibrillary tangles and senile plaques in the cerebral cortex. The hippocampus and amygdala are usually most severely affected, with marked cortical involvement of the frontal and temporal lobes and less involvement of the parietal and occipital areas (Tomlinson, 1977).

The severity of dementia was shown to correlate with the number of senile plaques in the cerebral cortex (Blessed, Tomlinson, and Roth, 1968). The density of plaques was also shown to correlate with the reduction of choline acetyltransferase (ChAT, the enzyme responsible for the synthesis of acetylcholine), which was also shown to correlate with the severity of the dementia (Perry, Tomlinson, Blessed, Bergman, Gibson, and Perry, 1978). The

reduction in ChAT activity in AD is related to a loss of cholinergic neurons in the basal forebrain and septal areas, which have widespread projections to the cerebral cortex and hippocampus (Mesulam and Van Hoesen, 1976). Reductions in the noradrenergic and serotonergic systems have also been reported (Bondareff, Mountjoy, and Roth, 1981; Mann, Lincoln, Yates, Stamp and Toper, 1980; Mann and Yates, 1983).

Multiple cerebral infarctions are the second most common cause of dementia (multi-infarct dementia, MID), accounting for approximately 15 - 25% of the cases (Kazniak, 1986). MID is characterized by an abrupt onset, stepwise deterioration, a fluctuating course, nocturnal confusion, relative preservation of personality, depression, somatic complaints, emotional lability, evidence of associated atherosclerosis, a history of hypertension, stroke, or transient neurologic symptoms, and focal neurologic signs on physical examination (Cummings, 1987). The clinical features depend on the location and size of the infarcts.

Dementia has also been associated with degenerative diseases of the subcortical systems, such as Parkinson's disease and Huntington's disease. The cognitive impairments associated with subcortical dysfunction include deficits in arousal, attention, motivation, and a slowed rate of information processing. Memory disturbances include forgetfulness, characterized by difficulty spontaneously

retrieving information, which improves when given clues and structure. Associated mood disturbances (commonly depression and more rarely mania) have been described (Cummings and Benson, 1992).

Dementia may also result from potentially reversible causes, such as metabolic deficiencies (e.g., a B12 deficiency), toxic conditions (e.g., misuse of medication), or treatable diseases (e.g., pulmonary or cardiac insufficiency, or endocrinopathy) (Benson, 1982). Potentially reversible causes of dementia were found to account for 13.2% of patients presenting with cognitive impairment (Clairfield, 1988).

A common potentially reversible cause of cognitive impairment in the elderly is an underlying psychiatric disturbance, which is most often a severe form of depression. Cognitive impairment due to depression has been termed "pseudodementia" (Kiloh, 1961), the "dementia syndrome of depression" (Folstein and McHugh, 1978), and more recently "depression with cognitive impairment" (Folstein and Rabins, 1992). In such cases cognitive symptoms remit following appropriate antidepressant treatment. In a recent series of studies depression was found to account for 4.5% of nearly 3000 patients presenting with cognitive difficulties (Clairfield, 1988).

A review of the literature by Stoudmire, Hill, Gulley, and Morris (1989) indicated that while the majority of

depressed patients do not exhibit cognitive difficulties, a predominantly elderly subgroup of patients does show signs of depression-related cognitive dysfunction. The cognitive difficulties that accompany depression are most often related to attention, memory, and speed of processing, while disorders of cortically-mediated functions (e.g., aphasia, apraxia, agnosia) are usually absent (Miller, 1975; Caine, 1981, 1986). The pattern of deficits is similar to that associated with dementia of a subcortical origin, such as Huntington's disease (Massman, Delis, Butters, DuPont, and Gillin, 1992).

Determining if presenting cognitive deficits are related to an underlying depression or to a dementing disorder is often difficult. Issues related to differential diagnosis will be addressed in later sections.

B. DEFINITION AND PREVALENCE OF DEPRESSION IN THE ELDERLY

The essential feature of a Major Depressive episode, as defined by DSM III-R, is either a depressed mood or a loss of interest or pleasure in most activities. In the elderly this may manifest as a sense of hopelessness or intense irritability. These symptoms represent a change from previous functioning, occur most of the day nearly every day for a period of at least two weeks, and are associated with appetite and/or sleep disturbances, weight changes, psychomotor retardation or agitation, decreased energy,

feelings of worthlessness or excessive/inappropriate guilt, difficulty thinking/concentrating, and recurrent thoughts of death, suicide, or suicide attempts. Deficits in catecholaminergic functioning, particularly norepinephrine, have been implicated as the biochemical basis to depression (Schildkraut, 1965).

Depressive disorders are the most common psychopathologic disturbances found in the elderly (Butler, Lewis, and Sunderland, 1991). Data reported by the National Institute of Health (NIH; 1991) indicated that depressive symptoms occur in approximately 15% of community residents over the age of 65 and a major depressive syndrome occurs in approximately 3%. In addition, the rate of completed suicides for older people is higher than that of the general population. For example, in 1988 the suicide rate in the general population of the United States was 12.4/100,000, while the rate in the 80-84 year old age group was 26.5/100,000. Elderly white men were at greatest risk for successfully completing a suicide.

Diagnosis of depression in the elderly can be a difficult task. Sleep and appetite changes occur in normal aging (Butler et al., 1991; Dement, Miles, and Carskadon, 1982) and fatigue may be related to health problems as often as to depression (Salzman and Shader, 1979). Kazniak and Allender (1985) pointed out that patients with occult carcinoma may demonstrate depressive-like symptoms,

including fatigue, poor appetite and weight loss, and sleep disturbances; patients with cardiac disease also frequently complain of disturbed sleep, fatigue, and difficulties with concentration. Thus, somatic symptoms in the elderly must be interpreted more cautiously than when they present in younger persons. Somatic symptoms in the elderly should be attributed to depression only after a thorough physical evaluation (Gallagher, 1986).

Diagnosis and treatment of late-life depression is complicated further by the frequently occurring admixture, overlap, and interaction of cognitive deficits and depressive symptomatology. Some authors have noted that depressed patients' complaints about impaired cognitive functioning, particularly memory, tend to overestimate the extent of their actual difficulty, as measured by psychometric tests (Kahn, Zarit, Hilbert, Niederehe, 1975). It has been suggested, however, that while such disparity may exist in mildly or moderately depressed patients, severely depressed patients are more likely to experience significant cognitive difficulties (Raskin, 1986). Henry, Weingartner, and Murphy (1973) found that the intensity of depression was inversely correlated with how much could be learned and remembered. As mentioned previously, a predominantly elderly subgroup of depressed patients are especially vulnerable to depression-related cognitive dysfunction, characterized most often by deficits in

attention, memory, and speed of processing (Miller, 1975; Caine, 1981, 1986).

Both qualitative and quantitative changes in cognition occur in depression, leading to changes in the efficiency and effectiveness of information processing. Weingartner and Silberman (1984) reviewed theories that infer that mood state serves to bias cognitive processing. For example, Beck (1972) argued that dysfunctional cognitions define the depressed state. That is, in depression a negative view of the self, world, and future, lowered self-esteem, and decreased motivation serve as a cognitive filter through which all other experiences are perceived. Beck's theory of depression was reformulated in the "learned helplessness" model (Maier and Seligman, 1976). This model contends that depressed patients expect unfavorable events to occur, feel they have no control over these negative consequences, and as a result demonstrate passivity and a negative cognitive set. It has been argued that such a negative cognitive set affects not only one's perception of ongoing events, but also accessibility and retrieval of information already in memory (Weingartner, Miller, and Murphy, 1977). For example, it was found that in depression negative past experiences were more likely to be remembered than positive experiences (Breslow, Kocis, & Belkin, 1981; Lishman, 1972) and negative adjectives that had been selected previously as self-descriptive were more easily recalled than positive

adjectives (Zuroff, Colussy, and Wielgus, 1983).

Given that the intensity of depression was shown to correlate with cognitive deficits (Henry et al., 1973), it seems plausible that the qualitative changes in cognition, which have been used to define mood state by the authors cited above, may be related to the quantitative cognitive losses experienced in depression. Weingartner and colleagues (Cohen, Weingartner, Smallberg, Pickar, & Murphy, 1982; Weingartner, Cohen, Murphy, Martello, & Gerdt, 1981; Weingartner et al., 1977) suggested that such qualitative changes contribute to changes in attention, arousal, and motivation. They proposed a "central motivational deficit" in depression based on the observation that depressed patients showed memory deficits only when effortful processing (i.e., requiring active and sustained cognitive activity), as opposed to automatic processing (i.e., somewhat more passive and requiring less cognitive activity) was required. They proposed that changes in motivational state in depression manifest as quantitative deficits in learning and memory. This theory will be described in more detail when discussing qualitative differences between the performances of depressed and demented patients on memory measures.

The overlap in cognitive and depressive symptoms in elderly affective disturbances often makes it difficult to determine the primary etiology of presenting complaints.

The quantitative cognitive deficits seen in severe depression may be of a similar magnitude to those associated with the presence of dementia, thus leading to diagnostic confusion. It is the qualitative characteristics of cognitive impairment in depression that become essential when trying to determine if presenting cognitive symptoms are due to depression, to a dementing process, or to a combination of both. Issues of differential diagnosis will be addressed in the next two sections.

C. DIFFERENTIAL DIAGNOSIS OF DEMENTIA VERSUS DEPRESSION

Misdiagnosis of dementia in the elderly is not uncommon. Estimates of false-positive identification range from 31% to 57% (Nott and Fleminger, 1975; Ron, Toone, Garralda, and Lishman, 1979). Retrospective analyses indicated that the majority of misidentified cases represented psychiatric disorders, the most common of which was depression (Haward, 1977; Marsden and Harrison, 1972; Nott and Fleminger, 1975; Ron et al., 1979).

Elderly patients are often referred for a neuropsychological examination to evaluate their cognitive abilities and to assist in making a differential diagnosis of depression vs. dementia when the etiology of presenting symptoms is unclear. Much emphasis has been placed on discovering characteristics of cognitive performance that discriminate depression from dementia due to the

reversibility of cognitive impairment associated with depression in comparison to the bleak, progressive course associated with a dementing process. Many aspects of the neuropsychological evaluation are used in making this distinction.

1. Historical and Clinical Variables

Historical and clinical variables have been useful in helping to discriminate patients who are primarily depressed from those who are suffering from a progressive dementing process. In a clinical case review, Wells (1979) summarized his own observations, and those of others, in an attempt to outline characteristics that distinguished the two disorders. He reported that in depression the onset of cognitive symptoms tended to be somewhat abrupt, could be dated with some precision, progressed rapidly, and usually followed the onset of depressive symptoms. Depressed patients tended to complain of cognitive loss and described it in some detail, while their behavior was often incongruent with the extent of reported cognitive losses. In dementia, the onset of cognitive symptoms was usually slow and insidious with a gradual worsening. A history of prior psychiatric dysfunction was unusual, patients complained little of cognitive loss, and were often unaware of the extent of their cognitive difficulties. During formal evaluation, depressed patients appeared to put forth little effort and responded "don't know" to questions with some

frequency. In contrast, demented patients usually struggled to do their best and provided "near miss" answers. Demented patients usually performed poorly on most tasks, while it was the depressed patients' "inconsistent performance from test to test that argued most strongly against attributing their clinical dysfunction to primarily organic disease."

Rabins, Merchant, and Nestadt (1984) conducted a two year follow-up study of patients initially diagnosed as either irreversibly demented or suffering cognitive loss due to a major depressive episode. They found that a past history of affective disorder, subacute onset, a persistently depressed or dysphoric mood, a history of poor appetite and weight loss, delusions of self-blame, hopelessness, and physical ill-health identified those patients suffering from cognitive impairment caused by depression. The two-year follow-up confirmed the initial diagnosis with 92% (12/13) of those patients diagnosed as irreversibly demented evidencing deterioration in cognitive functioning, while 88% (14/16) of those diagnosed as depressed evidenced improvement in cognitive functioning to within the normal range.

An assessment of overall level of cognitive functioning may also assist with the differential diagnosis of dementia vs. depression. Folstein, Folstein, and McHugh (1975) reported that patients with a diagnosis of progressive dementing illness performed most poorly ($M = 9.7$, $S.D. = 5.8$

out of a maximum of 30 points) on the Mini-Mental State Exam (MMSE; a brief screening of various cognitive functions including orientation, attention, memory, and constructional abilities). Patients with depression and cognitive impairment performed significantly better but scores were still in the impaired range ($M = 19.0$, $S.D. = 6.6$). Patients with uncomplicated depression performed best ($M = 25.1$, $S.D. = 5.4$) and were not significantly different from a normal elderly control group ($M = 27.6$, $S.D. = 1.7$). An analysis with age-matched samples showed a similar pattern of results. Further, scores of depressed patients with cognitive impairment improved significantly following treatment with antidepressant medication. Scores of the uncomplicated depressed group also improved following treatment, albeit to a smaller but still statistically significant degree. There was no change in the demented group over the same one month time interval.

While overall level of cognitive functioning is an important variable, some global measures of functioning yield a high rate of false-negative diagnostic errors, particularly in individuals in the early stages of dementia (i.e., patients in the early stages of dementia may be misclassified as cognitively intact). The results of such examinations should therefore be interpreted cautiously and diagnoses determined only when results are used in conjunction with a more extensive assessment of cognitive

abilities.

2. Neuropsychological Variables

Certain patterns of performance on various neuropsychological tests have been proposed as characteristic of demented and depressed populations.

a. Pattern of Performance on the Wechsler Scales: Fuld (1984) found that a specific profile of subtest scores on the Wechsler Adult Intelligence Scale (WAIS) identified 44% of the patients in the study with AD and only 7% of a non-AD dementia contrast groups (i.e., MID and "other dementias"). The profile is $A > B > C \leq D$, $A > D$, where A = mean of age-corrected scaled scores on Information and Vocabulary, B = mean of Similarities and Digit Span, C = mean of Digit Symbol and Block Design, and D = Object Assembly scaled score. Brinkman and Braun (1984) found this profile in 56% of AD patients and only 5% of MID patients. Further work with the Wechsler scales also supported the low prevalence rate of this pattern in normal elderly groups (Satz, Hynd, D'Elia, Daniel, Van Gorp, and Conner, 1990; Satz, Van Gorp, Soper, and Mitrushina, 1987; Tuokko and Crockett, 1987) and elderly psychiatric patients (Tuokko, Crockett, Hurwitz, and Kozak, 1988). Bornstein, Termeer, Longbrake, Heger, and North (1989) found this profile in only 16% of depressed elderly and Marcopulos and Graves (1990) found it in only 3.7% (2/54) of their elderly depressed sample. Fuld's pattern

thus appears to have some value in discriminating AD patients from depressed patients, however its use in discriminating depressed and non-AD demented patients has not been investigated as of yet.

b. Language: Differences in verbal output have been noted in depressed and demented patients. Weingartner and Silverman (1984) reported that on verbal fluency tasks depressed patients performed within normal limits under both phonetically and semantically-cued conditions, and were relatively better when provided with semantic cues. Demented patients, however, were significantly less productive on both tasks than depressed or control groups, and were particularly poor when provided with semantic cues.

c. Memory: Impaired memory is common in both demented and depressed elderly patients. Memory consists of three fundamental cognitive processes: 1) acquisition/encoding (i.e., getting new information into memory), 2) retention (i.e., maintaining or keeping that information in memory for a prolonged period of time), and 3) retrieval (i.e., recalling the information at a later time) (Caine, 1986). It has been hypothesized that demented and depressed individuals do poorly on memory tasks for different reasons; that is, that deficiencies in different neuropsychological mechanisms affect the acquisition/encoding, retention, and retrieval abilities of these groups.

Weingartner and colleagues performed a series of

studies in an attempt to delineate qualitative features that distinguish memory impairment in depression from memory impairment in progressive dementia. Their work suggested deficiencies in different mechanisms underlying the acquisition/encoding process.

Weingartner, Cohen, et al. (1981) found that depressed patients demonstrated learning and memory failures on tasks that lacked an obvious internal structure, and on tasks where the patient was required to impose organization independently for effective learning and memory to take place. For example, depressed patients and controls matched for age and education were similar in their ability to freely recall a list of organized, semantically-related words; however, depressed patients performed significantly more poorly when asked to recall a list of unorganized, unrelated words. The authors interpreted this finding as reflecting the depressed patients' ability to take advantage of cues and organization to aid the encoding process. In the absence of such external cues, however, the task became more effortful and the depressives' performance deteriorated. Further work by Cohen et al. (1982) demonstrated that memory and motor impairments in depression were most apparent on tasks that required sustained effort.

In contrast to the above findings, Weingartner, Kaye, Smallberg, Ebert, Gillin, and Sitaram (1981) found that patients suffering from a progressive idiopathic dementia

(probably of an Alzheimer type) could not use externally provided organization and semantic cueing to aid encoding. Recall of semantically related words and random words were both poor in comparison to a healthy control group. Dementia patients remembered about the same number of random and related words after a single presentation and after repeated presentations. In these patients, encoding was not facilitated by organization, semantic cues, or repetition. The authors contended that the information was weakly encoded and therefore easily forgotten under all conditions.

Weingartner and colleagues (Cohen, et al., 1982; Weingartner et al., 1977; Weingartner, et al., 1984; Weingartner, Cohen, et al., 1981; Weingartner, Kaye, et al., 1981) attributed the differences in performance in depressed and demented patients to dysfunction of different cognitive mechanisms. They proposed a "central motivational deficit" in depression to explain the memory dysfunction experienced by these patients. Cognitive impairment in depressed patients was less evident on "easier" cognitive tasks that involved relatively automatic, effortless, or passive operations, and was more evident on tasks requiring effortful encoding and retrieval activities. Dementia patients, however, failed to process information effectively on all tasks despite the presence of cues and organizational aids, and their apparent motivation to do well and sustained effort when completing tasks. The memory impairment in

dementia was attributed to decreased access to semantic knowledge, thereby prohibiting effective organization and encoding of information (Weingartner, Grafman, Boutelle, Kaye, and Martin, 1983). It was proposed that differences in the cognitive determinants of memory dysfunction in depression, as contrasted to dementia, may be due in part to involvement of different neuroanatomical structures (i.e., more cortical involvement in dementia and more limbic-subcortical involvement in depression), as well as to a relatively greater degree of disruption in different neurotransmitter systems (e.g., a relatively more prominent disruption of cholinergic activity in AD and relatively greater disruption of catecholaminergic systems in depression). These hypotheses have yet to be tested.

Hart, Kwentus, Taylor, and Harkins (1987) similarly proposed that motivation and attention deficits in depressed patients would be expected to have maximum impact on acquisition of new information rather than on retention of well-learned material, whereas both acquisition and retention would be poor in demented patients. This theory was confirmed in their study examining rate of forgetting of line drawings of common objects. Depressed and mildly demented (probable Alzheimer's disease) patients both demonstrated learning impairment; however, after being matched for acquisition abilities only the demented group showed rapid forgetting in the first ten minutes after

learning to criterion. That is, while depressed patients showed acquisition deficits, demented patients had difficulty with both acquisition and retention. These results were consistent with earlier reports that showed depressed patients had relatively greater deficits in immediate recall than in delayed recall (Croholm & Ottosson, 1961; Sternberg & Jarvik, 1976).

Mattis (1976) had also proposed that depressed patients may perform poorly on free recall serial-list learning tasks due to an underlying motivational deficit, rather than due to a memory deficit per se. This theory was based on the observation that depressed patients performed significantly better on more passive recognition memory tasks as compared to free recall tasks, which presumably take more effort. Demented patients performed poorly on both tasks. Such an observation suggested a retrieval deficit in depression due to the necessity of employing somewhat more effortful search processes in a free recall task, as compared to the more passive task of simply recognizing previously presented information. Depressives' ability to improve their performance on a recognition task indicated that the information was initially encoded at some level to allow correct recognition, though perhaps not sufficiently enough to allow easy retrieval during free recall. Demented patients' poor recall and recognition suggested that the information was not adequately encoded initially, thus

leading to difficulties in even simply recognizing information they had been presented with previously. Better recognition memory in depressed patients as compared to demented patients has been reported by many authors (Larner, 1977; Massman et al., 1992; Miller and Lewis, 1977; Niederehe, 1986) and will be discussed in more detail in the following section.

d. Discrimination and Response Bias Indices: The study of recognition memory in depressed and demented patients has led not only to the discovery of differential improvement in memory ability when information is presented in a forced-choice format, but also to the discovery of differences in decision criteria, or response bias, employed by these groups when forced to make a choice. Response bias, defined as the tendency to say "yes" or "no" when uncertain if a given stimulus is a target or a distractor item during recognition memory testing, has been shown to differ in demented and depressed patients. It is this measure, as well as discrimination abilities, that will be the focus of the present investigation.

Miller and Lewis (1977) were among the first to use the recognition memory paradigm to study discrimination ability and response bias in elderly depressed and demented individuals. They hypothesized that depressed patients do poorly on memory tasks because they adopt a conservative response strategy, rather than due to a true failure of

memory processes. That is, they hypothesized that depressed patients require a "higher level of internal or subjective certainty before they respond"; they would adopt a highly cautious decision strategy when in a state of uncertainty. Specifically, on a forced choice "yes-no" recognition memory task (i.e., respond "yes" if the item was seen before or "no" if it was not) depressed patients would need a high level of certainty before they would respond "yes" and thus would respond "no" more often. They also proposed that recognition accuracy, or discriminability, would be normal in depressed patients but impaired in the demented. Elderly depressed, demented, and healthy controls (patients suspected of having mixed symptoms of depression and dementia were excluded) were administered a recognition memory task involving geometric and nonsense figures (Kimura Recurring Figures; Kimura, 1963). Both hypotheses were supported: 1) discrimination abilities were similar in depressed patients and controls, and were significantly poorer in demented patients, and 2) depressives used the most conservative response strategy (i.e., they tended to respond "no" more often) while demented patients tended to make more false positive errors (i.e., they tended to respond "yes" more often). The authors interpreted this finding as suggesting that depressives had an extremely low willingness to guess, perhaps due to lowered motivation or to the depressives' perception of guessing and being wrong

as highly aversive, and thus they tended to avoid such aversive stimuli.

Similar results have been found by other investigators. Larner (1977) studied recognition memory abilities and response bias in depressed, demented, and physically ill elderly patients using high frequency and low frequency words as stimuli. Discrimination abilities were poorest in the demented group and were within normal limits in the depressed and physically ill groups. These latter two groups adopted a conservative response strategy, while the demented group displayed more liberal criteria (i.e., they responded "yes" more often), especially when recognizing high frequency words.

Niederehe (1986) found that on a recognition memory task for high frequency/high imagery words discrimination abilities were not significantly different in young (aged 20-45) and old (aged 55-80) depressed groups as compared to healthy controls. Discrimination abilities of a mild to moderately demented group were significantly poorer than both the depressed and control groups. Young and old depressed groups manifested conservative decision criteria (i.e., tended to make more false-negative errors) whereas the normal control group and the demented group employed less stringent decision-making criteria (i.e., made more false-positive errors). The tendency toward depressive cautiousness appeared greater among the older patients.

Recent work on response bias has been carried out by Corwin and colleagues (Corwin, Peselow, Feenan, Rotrosen, and Fieve, 1990; Snodgrass and Corwin, 1988). In a series of investigations they have attempted to more clearly delineate the cognitive and neurochemical mechanisms underlying response bias in demented and depressed patients.

Snodgrass and Corwin (1988) studied discrimination and response bias indices in a young, normal control group (101 undergraduate students) and a moderately to moderately-severely demented group (nine probable AD, two Parkinsonian dementia patients) using line drawings of common objects (e.g., asparagus, sailboat) as stimuli. There were two study-immediate recognition trials followed by a half-hour delayed recognition trial (ten targets were interspersed with ten distractor items). Results for the young, normal group indicated improved discrimination abilities across trials with no drop off in memory after a delay; response bias was slightly conservative on trial one, but neutralized on subsequent trials. As would be expected, discrimination abilities of the demented group were significantly poorer on all trials than those of the young, normal group; response bias in the demented group was liberal on trial one and increased in liberality across trials. These results were similar to those of Hart, Smith, and Swash (1985) who found that patients with AD made more false positive errors than a healthy elderly control group when recognizing both verbal

(single nouns) and non-verbal (geometric shapes, histology slides) stimuli.

Snodgrass and Corwin (1988) went on to analyze discrimination and response bias indices in patients with amnesia, Huntington's disease (HD), and an age-matched healthy control group. These data were derived from Butters, Wolfe, Martone, Granholm, and Cermak (1985). Unrelated words served as stimuli in five study-immediate recognition trials and a twenty minute delayed trial. Results showed that discrimination abilities were best in the control group, worst in the amnesics, and intermediate in the HD group. On the delay trial the amnesics forgot the most, the HD patients forgot a little, and the control group did not forget at all. Response bias was slightly conservative in controls and amnesics, while HD patients showed a much more liberal response style. Bias became more liberal across trials in all groups. A liberal response bias in HD patients and Parkinson's disease patients was also found by Massman, Delis, Butters, Levin, and Salmon (1990), although HD patients were found to be not as liberal as AD patients (Brandt, Corwin, and Krafft, 1992).

The results of these investigations suggested that a liberal response bias was not solely the result of poor memory skills, since amnesics, who had the worst memory, actually showed a slightly conservative response bias. Demented patients, regardless of etiology, displayed liberal

responding. Snodgrass and Corwin suggested that there must be some property in dementia that causes new (distractor) items to appear familiar (like targets), as if they had been seen before. A cognitive property and a neurochemical theory were offered as possible explanations:

1) Cognitive: Snodgrass and Corwin suggested that a liberal response bias in dementia may be related to semantic memory deficits. "Recognition" takes place via pattern matching between features of the test stimuli and stored active representations of the "old" (target) items. If a match or near match is found, the subject responds "yes"; otherwise, the subject responds "no". An increasing number of features in common between the test items and internal representations (either from the target set or from semantic memory) yields a parallel increase in subjective familiarity. Thus, the subject might have high levels of familiarity for both old (target) and new (distractor) items if stimuli are not encoded distinctly at presentation and semantic memory representations have been inappropriately activated by prior target and distractor items. Therefore, stimuli that are not encoded distinctly at presentation will adequately match stored representations of similarly poorly encoded target items or very familiar items in semantic memory, leading to a general increase in familiarity and "yes" responses to both old and new items.

Prior reports of disorders in semantic memory in AD

(Martin & Fedio, 1983; Nebes, Boller, and Holland, 1986), in combination with the findings cited above, led Snodgrass and Corwin to suggest that the liberal response bias displayed in dementia may be related to deficits in semantic memory. Such deficits lead to difficulty defining distinctive features of new information, which leads to a more general sense of familiarity with both old and new items and an increased probability that the demented subject will respond "yes" that an item had been seen before, even if it had not. Deficits in semantic memory have not been reported in amnesia, and thus may account, at least in part, for the disparate findings between demented and amnesic groups noted above.

2) Neurochemical: An abnormally liberal response bias on recognition testing and intrusion errors on recall tasks have been associated with cortical acetylcholine deficits in AD (Fuld, Katzman, Davies, and Terry, 1982; Mohs and Davis, 1982). In addition, liberality was reduced following physostigmine treatment (which enhances synaptic acetylcholine availability) in patients with AD (Mohs & Davis, 1982). These findings suggested that the presence of a liberal response bias in AD patients was related to decreased acetylcholine activity. This is not a complete explanation, however, since HD patients, in whom acetylcholinergic deficits are not prominent, also displayed a liberal response bias. The neurochemical basis of response

bias in dementia requires further investigation.

The underlying mechanism of response bias in depression was also investigated by Corwin and colleagues (1990). This study consisted of two parts: 1) an investigation of discrimination and response bias in depressed and manic patients both before and after treatment, and 2) an investigation of discrimination and response bias in psychiatrically normal hypertensives when on and off propranolol (a beta-adrenergic blocker).

In the first part of the study, 50 patients meeting criteria for major depression, 28 bipolar patients in a manic state, and 21 psychiatrically normal controls were administered three immediate and one delayed trial of a yes-no recognition memory task involving high imagery (e.g., apple) and low imagery (e.g., freedom) words. Testing was completed both before and one month after administration of appropriate medication (imipramine or lithium). The control group was tested at the same points in time. Following treatment, patients were defined as responders or non-responders as determined by at least a 50% reduction in their score on a rating scale of depression or mania. Results at baseline testing indicated: 1) discrimination abilities were poor only when symptoms of mania or depression were severe, 2) response bias was liberal in mania and conservative in depression, regardless of severity of illness, and 3) bias deficits were more pronounced (i.e.,

either more liberal in manic patients or more conservative in depressed patients) for the more difficult low imagery words. Following treatment results showed: 1) discrimination abilities remained poor in the severely affected patients who did not respond to treatment, and 2) response bias remained abnormal only in manic non-responders; bias "normalized" in both manic and depressed responders, and in depressed non-responders (i.e., treatment with imipramine seemingly altered response bias in those depressed patients who became less depressed and even in those who did not).

The finding that response bias deficits, but not discrimination deficits, were present even when symptoms of depression or mania were mild, suggested that bias abnormalities were more primary to each disorder than discrimination difficulties. The authors suggested that the bias deficits may be related to dysfunction of noradrenalin systems, based on the seemingly parallel finding that bias was liberal in mania, which has been associated with elevated levels of noradrenalin, and was conservative in depression, which has been associated with lowered levels of noradrenalin. The depressed patients' less conservative, more liberal, response bias following treatment with imipramine, which increases the availability of noradrenalin, gave further support to this claim.

Corwin et al. (1990) further investigated the relationship between noradrenergic functioning and response

bias. Psychiatrically normal mild to moderate hypertensives were administered a "yes-no" digit recognition memory task at baseline, and four, eight, and twelve weeks after treatment with propranolol (a beta-adrenergic blocker, thus decreasing the availability of noradrenalin). Results indicated that propranolol did not affect discrimination abilities. Response bias, however, became significantly more conservative on the most difficult task. Thus, response bias became more conservative when noradrenergic functioning was decreased. This finding was taken as support for the hypothesis that noradrenergic functioning affects response bias. Specifically, a decrease in noradrenalin is associated with conservative responding (based on findings with depressed patients and conservative responding after treatment with propranolol) and an increase in noradrenalin is associated with liberal responding (based on liberal responding in mania and an increase in liberality following imipramine treatment).

In summary, the work of Corwin and her colleagues supports the claim that memory deficits in depression are not primary, but may be a function of conservative decision rules, as originally suggested by Miller and Lewis (1977). These decision rules appear to be affected by deficits in noradrenergic functioning in depression. In dementia, particularly AD, memory deficits and liberal response bias tendencies appear related to abnormalities in semantic

memory, which may be associated with acetylcholine deficits. These explanations are not complete, however, since decreased noradrenergic functioning has also been reported in AD patients (Bondareff, et al., 1981; Mann, et al., 1980; Mann and Yates, 1983), who show a liberal response bias. In addition, liberal responding has also been found in manic patients, a disorder associated with elevated levels of noradrenalin and no known acetylcholine deficits. Thus, it appears that the neurochemical basis of response bias in dementia and depression is not fully understood at the present time.

3. Summary

The foregoing discussion reviewed many of the current approaches used when making the differential diagnosis of dementia vs. depression. Historical and clinical variables, as well as measures of overall level of cognitive ability, and patterns of performance on the Wechsler scales and verbal fluency tasks have all been useful in making this distinction. Performance on memory tasks has been widely studied and appears to offer the most clinical utility when discriminating dementia from depression. Specifically:

- 1) In depression performance on recognition memory tasks is relatively better than performance on free recall tasks and cued recall is better than free recall. Findings suggest that acquisition and retrieval of new information are

impaired, while retention is intact. A conservative response bias is displayed.

2) In dementia both recognition and free recall are impaired and cues do not improve free recall. Findings indicate that acquisition, retrieval, and retention are deficient. Liberal responding is evidenced.

Weingartner proposed a central motivational deficit in depression to account for the cognitive deficits depressed patients experience, while others suggested that these deficits may be due to conservative response strategies. Most investigators agree that the memory impairment present in dementia, particularly AD, is most likely associated with dysfunction of semantic memory.

The previous studies have focused on dementia and depression as distinct clinical entities. Patients suspected of having coexisting syndromes of both dementia and depression were excluded from these analyses. These patients are perhaps the most clinically confusing, and thus warrant further investigation.

D. COEXISTENT DEMENTIA AND DEPRESSION: PRIOR ATTEMPTS AT DIAGNOSIS

As the previous discussion reflects, much emphasis has been placed on discovering characteristics of cognitive performance that discriminate dementia from depression. However, little is known about the pattern of cognitive

deficits present in patients with coexistent irreversible dementia and superimposed depression. Many authors (Devanand and Nelson, 1985; McAllister and Price, 1982; Reifler, Larson, and Hanley, 1982; Shraberg, 1978) have cautioned that an undue emphasis on distinguishing depression from dementia may prevent recognition of coexisting conditions, such that potentially helpful antidepressant therapy is not prescribed in patients suffering from both disorders.

Kaszniak (1987) emphasized the importance of diagnosing and treating coexisting syndromes due to the occurrence of suicide and suicide attempts in these patients, as well as indirect self-destructive behavior, which may serve as an alternate form of suicidal behavior.

In Alzheimer's original case study (1907) of the disease which now bears his name, he described a 51 year old woman with memory loss, disorientation, apraxia, delusions, and depression (cited in Wragg, 1989). Later, English (1942) noted the coexistence of dementia and depressive symptoms in five of seven case descriptions. A recent review (Wragg, 1989) indicated that symptoms of depressed mood were reported in 40-50% of patients with AD, while the actual clinical syndrome of depression occurred in 10-20% of AD patients. Similar rates have been reported for mixed samples of patients with dementia of various origins (Greenwald, Kramer, Ginsberg, Marin, Hermann, Mohs, and Davis, 1989; Liston, 1979; Reding, Haycox, and Blass, 1985; Reifler, et

al., 1982).

The mechanism underlying the development of depression in demented patients is not well understood. A postmortem study of cytopathologic features in various brain regions of demented patients with or without major depression indicated that patients with major depression had significantly more degenerative findings in the locus coeruleus and substantia nigra than demented patients who were not depressed, suggesting that development of depression in demented individuals is associated with degeneration of these nuclei (Zubenko and Moosey, 1988). Cummings (1989) noted that the reported neurobiological bases of depression and AD overlap. That is, in depression dysfunction of noradrenergic and serotonergic systems has been reported and the source nuclei of both these transmitter systems (locus coeruleus and raphe nuclei, respectively) are also involved in AD. These shared features could explain, at least in part, the development of depression in some AD patients. At present, however, the underlying mechanisms causing the depression remain unknown.

Depressive symptoms have been reported as most common in the early stages of dementia (Busse, 1975). However, depression has also been observed in patients with severe dementia (Demuth and Rand, 1980). Reifler et al. (1982) found the rate of coexisting depression to decrease significantly with greater severity of cognitive impairment, while Kaszniak (1985) did not find any significant

relationship between severity of depressive symptoms and severity of cognitive impairment. Kaszniak (1987) pointed out that given these discrepant findings it is probably most prudent to conclude that depressive symptoms can occur at any point in a dementing illness.

Depressive symptoms in demented patients may present as maladaptive behavior, aggression, or changes in motor activity (Cummings, 1989). Kaszniak (1985) and Lazarus et al. (1987) found that vegetative symptoms were less common than depressed mood, slowing of thoughts and actions, suspiciousness, and loss of insight in these patients. Such an atypical presentation can make it difficult to identify depression in patients with dementia.

Depression may exacerbate cognitive and behavioral dysfunction in patients with dementia, making the presence of a depressive component even less apparent, as described in various case reports (McAllister and Price, 1982; Shraberg, 1978; Snow and Wells, 1981) and systematic investigations (Breen, Larson, Reifler, Vitaliano, and Lawrence, 1984; Greenwald et al., 1989; Rovner, Broadhead, Spencer, Carson, and Folstein, 1989). Rovner et al. (1989) and Greenwald et al. (1989) found that demented patients who also met criteria for major depression scored significantly more poorly than patients with uncomplicated dementia on the MMSE and on measures of self-care ability. Others (Breen et al., 1984; Raskin, 1986; Reifler, Larson, Teri, and Poulson,

1986) did not find any significant differences on the MMSE in more mildly demented groups. However, Breen et al.'s work showed that the presence of depression in their mildly demented patients was associated with increased intellectual impairment, as reflected by lower full scale WAIS IQ scores.

It appears that depression-dependent cognitive impairment may exacerbate existing cognitive difficulties in demented patients. Some authors have found that somatic antidepressant treatments improve cognitive status in demented patients with a superimposed depression, presumably on the basis of remission of depression and alleviation of depression-mediated cognitive impairment (Greenwald, et al., 1989; McAllister and Price, 1982; Shraberg, 1978). Although patients remained demented, performance on tests of cognitive ability and daily functioning improved. Other investigators (Reifler et al., 1986; Reifler, Teri, Raskind, Veith, Barnes, White, and McLean, 1989; Reynolds, Perel, Kupfer, Zimmer, Stack, and Hoch, 1987) did not find a significant change in demented/depressed patients on global measures of cognitive ability following treatment with antidepressant medication, however, these patients showed clear evidence of improvement in mood, vegetative signs, and activities of daily living. While the impact of antidepressant therapies on cognitive test performance remains unclear, these results suggest that demented/depressed patients do respond favorably to

treatment. Successful identification and treatment of depression in demented patients may represent the only "improvable" component in an otherwise unremitting cognitive deterioration.

Clinical tools to help identify patients with coexistent dementia and depression are essential. Previous attempts to discover factors that discriminate this group include metabolic, sleep electroencephalographic, and neuropsychological techniques. Results have been mixed.

1. Metabolic Studies

The dexamethasone suppression test (DST) has been shown to have a high specificity and sensitivity for endogenous depression, and has been suggested as potentially useful in discriminating patients with dementia and depression from those with dementia alone (McAllister, Ferrell, Price, and Neville, 1982). This hypothesis has not been confirmed by most investigators. Positive DST results were found in 50-73% of non-depressed AD and MID samples (Balldin, Gottfries, Karlsson, Linstedt, Langstrom, and Walinder, 1983; Greenwald, Mathe, Mohs, Levy, Johns, and Davis, 1986). Shrimankar, Soni, and McMurray (1989) found a high incidence of positive DST results in elderly patients diagnosed with major depression, dementia, and dementia with depression. DST results were unrelated to presence or severity of affective symptoms. These results thus cast doubt on the utility of the DST for discriminating patients with dementia

and depression from patients with dementia alone.

2. Sleep Encephalographic Studies

Sleep electroencephalograms (EEG's) have also been used to study dementia and depression syndromes. Such studies are rarely conducted for purposes of diagnosis due to practical considerations such as time, expense, and need for an extended period of patient cooperation, but Reynolds and colleagues (Reynolds, Kupfer, Houck, Hoch, Stack, Berman, and Zimmer, 1988) have discovered that they have some, though limited, utility in clarifying diagnostic questions involving dementia and depression. They found that use of four EEG parameters (early morning awakening, REM sleep latency, percent of REM sleep, and percent indeterminate non-REM sleep) correctly classified 64% of patients with mixed symptoms of dementia and depression into either a primarily depressed group with secondary cognitive impairment or a primarily demented group with secondary depression. The majority of misclassified patients were demented patients misclassified as depressed. The authors acknowledged that a 64% classification rate reflects significant limitations of this technique's utility in clinical practice, but it also represents an improvement over the 50% chance of identifying such patients by chance alone.

Further work by this group (Buysse, Reynolds, Kupfer, Houck, Hoch, Stack, and Berman, 1988) suggested that

patients with primary depression and secondary cognitive impairment were significantly different from primarily demented patients with depression in baseline clinical and REM (rapid eye movement) sleep measures after one night of sleep deprivation. Specifically, primarily depressed patients showed 1) less severe symptoms of dementia at baseline, 2) a more predictable decrease in depressive symptoms, as measured by the Hamilton Depression Rating Scale, after one night of sleep deprivation, 3) higher REM sleep percent and phasic REM activity/intensity at baseline, and 4) a longer first REM period on recovery night two following sleep deprivation. The authors pointed out that these groups differed from each other in the expected direction; that is, patients with primary depression with cognitive impairment had REM sleep characteristics similar to those of uncomplicated depressives, whereas patients with primary dementia with depression had REM sleep similar to that of uncomplicated demented patients.

3. Neuropsychological Studies

Differences in overall level of cognitive functioning between demented and demented/depressed groups have been investigated by many authors, as described above. However, only two studies, to the present author's knowledge, have investigated possible differences in patterns of performance on neuropsychological tests.

Lopez, Boller, Becker, Miller, and Reynolds (1990)

studied mildly demented patients with probable AD who also met criteria for major depression and compared them with mildly impaired AD patients who were not depressed on five domains of cognitive functioning, including attention, language, memory, learning, and visuospatial functions. Depressed and non-depressed AD patients could not be differentiated by their neuropsychological test results or by rate of decline over a one year time interval. Both groups were significantly impaired on all domains and showed a general deterioration in abilities over time.

In contrast, Raskin (1986) reported that the presence of depression in a mildly demented group of elderly patients resulted in poorer immediate and delayed recall as compared to a non-depressed demented group. They noted, however, that these differences were apparent only on more difficult tasks (e.g. significant differences emerged on a supraspan ten-digit repetition task, but not on the repetition task involving only five to seven digits).

Of the diagnostic measures noted above, sleep EEG's and rigorous neuropsychological assessment appear to offer the most help in determining if coexisting dementia and depression are present. Sleep EEG's are less than ideal as a diagnostic tool due to the somewhat cumbersome nature and cost of the procedure. Neuropsychological assessment may be a more viable option, but as of yet relatively little is

known about the performance of demented/depressed patients on neuropsychological measures. The need for a relatively simple, sensitive clinical aid for identifying the patient with dementia and depression still exists.

E. PURPOSE OF THE PRESENT STUDY

The purpose of the present study was twofold: 1) to further elucidate differences in memory ability, specifically recognition memory or discriminability, that might exist between demented/depressed patients and uncomplicated demented or depressed patients, and 2) to investigate if response bias of patients with both dementia and depression is different from patients with uncomplicated dementia or depression. Literature documenting the different response bias tendencies of depressed only and demented only patients suggests that response bias is an index that is differentially sensitive to both syndromes and as such may be an effective diagnostic aid when coexistent diagnoses are suspected.

III. HYPOTHESES

1. It was hypothesized that recognition memory ability, or discriminability, would be different in depressed, demented and demented/depressed patient groups. Specifically:

a. based on prior recognition memory studies with depressed and demented patients (Corwin et al., 1990; Larner, 1977; Massman et al., 1992; Miller and Lewis, 1977; Niederehe, 1986; Snodgrass and Corwin, 1988), it was predicted that discrimination abilities would be better in depressed patients than in demented or demented/depressed patients;

b. differences in discrimination ability between demented and demented/depressed patients would be explored. Differences in memory abilities between these two groups remains unclear at present, with some authors reporting no difference in performance (Lopez et al., 1990) and others (Raskin, 1986) suggesting that demented/depressed patients perform relatively more poorly than uncomplicated demented patients on more difficult tasks.

2. It was hypothesized that response bias would be different in depressed, demented, and demented/depressed groups. Specifically, it was predicted that:

a. based on prior research (Brandt et al., 1992; Corwin et al., 1990; Hart et al., 1985; Larner, 1977; Massman et

al., 1990; Miller and Lewis, 1977; Niederehe, 1986; Snodgrass and Corwin, 1988), response bias would be more conservative in the depressed group and would be more liberal in the demented group;

b. the index of response bias in demented/depressed patients would be influenced by the depressive process, such that response bias would be relatively conservative as compared to demented patients without depression;

c. conversely, response bias would also be influenced by the dementing process in the demented/depressed group, such that response bias would be relatively liberal in this group as compared to patients with depression without cognitive impairment.

3. Exploratory analyses would determine if indices of discrimination and response bias were related to either level of cognitive ability or severity of depressive symptoms. Specifically, it was hypothesized that:

a. discrimination ability would correlate positively with level of cognitive ability (i.e., discrimination abilities would improve as level of cognitive ability increased);

b. discrimination ability would correlate negatively with severity of depression (i.e., discrimination abilities would worsen as severity of depression increased);

c. response bias would correlate negatively with level

of cognitive ability (i.e., response bias would become more liberal as level of cognitive ability decreased);

d. response bias would correlate negatively with degree of depression (i.e., response bias would become more conservative as severity of depression increased);

e. cognitive impairment and depression would make independent contributions to discrimination and response bias.

IV. METHODS

A. SUBJECTS

Subjects were recruited from inpatient and outpatient geriatric psychiatry and neurology services at Long Island Jewish-Hillside Medical Center in Glen Oaks, New York. The following criteria were employed for preliminary subject selection: 1) at least 65 years of age; 2) presented with symptoms of cognitive impairment and/or depression, 3) acute medical conditions, including toxic and/or metabolic disturbances, that may have been contributing to the clinical presentation were ruled out by a standard medical/laboratory work-up; 4) no history of prior neurologic disease (other than cerebral vascular accidents or dementia), head injury with prolonged unconsciousness, and/or substance abuse in order to rule out possible contributors to presenting symptoms other than depression and/or dementia; and 5) patients were alert and able to participate in testing.

Appropriate participants who consented (if their ability to provide consent was questionable, it was also obtained from a family member) were then included in the study sample if they met criteria for one of the three diagnostic groups of interest. Diagnostic grouping was based on historical information (obtained from a reliable informant, i.e., the patient or a significant other, and/or

the medical record), a clinical interview, and performance on cognitive and affective screening measures:

- 1) Demented (DEM): Seventeen patients met DSM III-R criteria for Dementia, had no other Axis I disorder, had scores of 24 or below on the Mini-Mental State Exam (MMSE; Folstein, 1975) and scores below 123 on the Dementia Rating Scale (DRS; Mattis, 1976). Significant depressive symptoms were absent as indicated by a score of 10 or below on the Hamilton Depression Rating Scale (HDRS; Hamilton, 1960, 1967) and/or a score of 2 or below on the Clinical Global Impression for Depression (CGI; Guy, 1976).
- 2) Depressed (DEP): Seventeen patients met DSM III-R criteria for a Major Depressive episode, unipolar type, had no other Axis I disorder, had scores greater than 24 on the MMSE and scores equal to or greater than 123 on the DRS. Depressive symptoms were in the moderate to severe range as indicated by scores of at least 16 on the HDRS and/or a CGI of at least 4.
- 3) Demented/Depressed (DEM/DEP): Twelve patients met DSM III-R criteria for Dementia with depression, had no other Axis I disorder, had scores of 24 or below on the MMSE and scores below 123 on the DRS. Depressive symptoms were in the moderate to severe range as indicated by a HDRS score of at least 16 and/or a CGI of at least 4.

B. PROCEDURE

Possible study participants were identified by either a licensed psychiatrist or psychologist who made the initial clinical diagnosis of dementia, depression, or dementia with depression.

If these candidates consented to participate, scores on affective and cognitive screening measures, given by staff trained in their administration, were used to determine if they met criteria for one the three diagnostic groups described above. These screening measures included: 1) MMSE (Folstein, 1975), a brief screening of various cognitive functions, including orientation, attention, memory and constructional abilities; scores may range from 0 to 30 with higher scores reflecting better performance; 2) DRS (Mattis, 1976), a somewhat more detailed assessment of cognitive abilities which yields a composite score based on performance in five ability areas including attention, initiation/perseveration, construction, conceptualization, and memory; scores may range from 0 to 144 with higher scores reflecting better performance; 3) HDRS (Hamilton, 1960, 1967), an observer rated scale that assesses severity of depressive symptoms based on responses to 21 inquiries regarding mood, activity/interest level, anxiety, appetite and weight changes, sleep disturbances, insight, and thought disturbances; scores of 12-15 indicate mild depressive symptoms, 16-22 moderate symptoms, and scores of 23 and

greater indicate the presence of severe depression; 4) CGI (Guy, 1976), an observer based rating which estimates severity of depressive symptoms depending on the rater's overall impression of the patient's affect, behavior, and mood; scores range from 1 (no symptoms of depression present) to 7 (very severe depression). An estimate of each participant's premorbid level of intellectual abilities was also determined through the use of a prediction equation based on demographic variables devised by Barona, Reynolds, and Chastain (1984).

After the initial screening instruments were completed, recognition memory and response bias were assessed using Kimura's Recurring Figures test (KRF; Kimura, 1963). KRF was used by Miller and Lewis in their original work on discrimination and response bias in depression and dementia (1977). This visual recognition memory test consisted of 20 target stimuli (10 geometric figures and 10 nonsense figures on 3X5 inch cards; see examples in Appendix A) that were initially presented for three seconds each. Instructions to the patient were as follows: "I am going to show you some designs one at a time, and I want you to look at each one carefully and try to remember it." The study trial was immediately followed by a "yes-no" recognition trial consisting of seven blocks with 20 items (8 targets, 12 distractors) in each block. The 8 targets (4 geometric, 4 nonsense) recur once in each test block of twenty cards. The

distractors do not recur. On the recognition trial the patient was told: "Now I am going to show you some of those designs again, along with some new ones that you have not seen before. Each time I show you a card now, I would like you to say 'yes' if I have shown the picture to you before, and 'no' if you have not seen it before."

The total number of correct "yes" responses (hits) and total number of incorrect "yes" responses (false alarms) were used to compute indices of discriminability (P_r) and response bias (B_r) using the Two-High Threshold model described by Snodgrass and Corwin (1988). The index of discriminability, P_r , was computed by the following formula: $\text{Hits} - \text{False Alarms}$. The index of response bias, B_r , was computed by: $\text{False Alarms} / 1 - (\text{Hits} - \text{False Alarms})$. The raw number of hits and false alarms were transformed into conditionalized rates, such that $\text{Hits} = \text{number of hits} + 0.5 / \text{number of targets} + 1$; $\text{False Alarms} = (\text{number of false alarms} + 0.5) / (\text{number of targets} + 1)$. The use of conditionalized rates rather than raw number of hits and false alarms is important when the number of targets and distractors are not equal (as is the case in KRF) for two reasons: 1) raw numbers may yield unequal discrimination scores for subjects whose discrimination abilities are actually equivalent (e.g., a subject may respond "yes" to all items resulting in 56 hits and 84 false alarms, yielding a net raw score of -28, while another subject may respond

"no" to all items resulting in 0 hits and 0 false alarms, yielding a net raw score of 0; resulting net raw scores are different, but discrimination ability is the same for each of these subjects); 2) subjects tend to try to match presentation probabilities (e.g., if more distractors are presented than targets, subjects will tend to respond "no" more often when uncertain).

In the Two-High Threshold model, discriminability, Pr , may range from -0.99 (all wrong responses) to 0.99 (all correct responses); that is, higher scores reflect better discrimination ability. Response bias, Br , may range from 0.01 (all "no" responses) to 0.99 (all "yes" responses); that is, higher scores reflect a greater tendency to respond "yes" (a more liberal response bias), and lower scores reflect a greater tendency to respond "no" (a more conservative response bias). Scoring was facilitated by a computerized method designed by Capruso and Barr (1992), which yields discrimination and response bias indices for nonsense and geometric figures combined, as well as for each condition separately.

Four subjects (one demented, two depressed, one demented/depressed) were administered a short form of KRF, which consisted of five blocks of 20 cards during the recognition trial, rather than seven. Pr and Br scores were computed in the same manner as for the long form score. Due to the small number of subjects who received the short form,

it was not possible to determine statistically if these scores were significantly different from long form scores, however scores for each of the subjects noted above were within one and one half standard deviations of their respective group means and were thus combined with scores of their respective groups for later analyses.

C. DATA ANALYSIS

Analysis of variance (ANOVA) and when appropriate, multivariate analysis of variance (MANOVA), were used to investigate differences among the three diagnostic groups (demented, depressed, and demented/depressed) on demographic variables, measures of cognitive and affective status, and indices of discrimination and response bias. Pre-planned pairwise comparisons between groups were conducted to more clearly delineate the source of differences between groups. Adjustments for the increased possibility of Type I error due to multiple comparisons were made using the Tukey-B method. Pearson product-moment correlation coefficients were computed to explore potential associations between measures of cognitive ability, depression severity and indices of discrimination and response bias. Linear regression analysis was employed to investigate the relative contributions of the presence of dementia and depression to discrimination ability and response bias.

Results of analyses involving demographic variables and

cognitive and affective screening measures will be presented first, followed by analyses of discrimination ability and then response bias. Finally, results of correlational studies and the linear regression analysis will be presented.

V. RESULTS

A. DEMOGRAPHICS AND CLINICAL VARIABLES

Forty-six patients participated: 17 (9 male, 8 female) met criteria for the demented group, 17 (8 male, 9 female) met criteria for the depressed group, and 12 (5 male, 7 female) met criteria for the demented/depressed group.

In the total sample, age ranged from 66 to 90 years, with a mean of 76.04 years (S.D. = 5.73). Education ranged from 7 to 19 years, with a mean of 11.65 years (S.D. = 2.92). Estimated premorbid Full Scale IQ scores ranged from 86.71 to 121.13, with a mean of 104.33 (S.D. = 9.00). Descriptive demographic information for each group is presented in Table 1. Preliminary ANOVA's revealed no significant differences among the groups on these variables.

Scores on the depression rating scales, HDRS and CGI, are presented in Table 2. Depression ratings were lowest in the demented group (HDRS: M = 5.0, S.D. = 2.85; CGI: M = 1.35, S.D. = 0.61) and were higher in the depressed group (HDRS: M = 21.53, S.D. = 5.08; CGI: M = 4.35, S.D. = 0.61) and demented/depressed group (HDRS: M = 18.33, S.D. = 4.33; CGI: M = 4.08, S.D. = 0.29). This pattern of scores was expected, given the inclusion criteria described above. ANOVA's with diagnostic group as the independent variable and HDRS and CGI scores as dependent variables, revealed that scores on these measures were significantly different

Table 1.
Demographic Data for Each Diagnostic Group*

	DEM	DEP	DEM/DEP	TOTAL
N	17	17	121	46
MALE	9	8	5	22
FEMALE	8	9	7	24
AGE (S. D.)	76.88 (5.44)	74.00 (4.65)	77.75 (7.00)	76.04 (5.73)
YEARS OF EDUCATION	10.94 (2.22)	12.53 (2.92)	11.42 (3.63)	11.65 (2.92)
ESTIMATED PREMORBID FSIQ	104.03 (6.03)	106.31 (10.52)	101.96 (10.26)	104.33 (9.00)

*DEM = Demented
 DEP = Depressed
 DEM/DEP = Demented/Depressed

Table 2.

Depression Ratings for Each Diagnostic Group

	DEM	DEP	DEM/DEP
Hamilton Depression Rating Scale (HDRS)	5.00* (2.85)	21.53 (5.08)	18.33 (4.33)
Clinical Global Impression for Depression (CGI)	1.35* (0.61)	4.35 (0.61)	4.08 (0.29)

* Significantly lower than the other two groups, $p < .05$

DEM = Demented

DEP = Depressed

DEM/DEP = Demented/Depressed

among groups (HDRS: $F [2,43] = 73.21, p < .001$; CGI: $F [2,43] = 152.46, p < .001$). Pairwise comparisons revealed that HDRS and CGI scores were significantly lower in the demented group than in the depressed group and the demented/depressed group ($p < .05$). The depressed and demented/depressed groups were not significantly different from each other on ratings of depression severity ($p > .05$).

Scores on measures of cognitive ability are presented in Table 3. Overall level of cognitive ability, as measured by the MMSE and DRS, was highest in the depressed group (MMSE: $M = 28.06, S.D. = 1.43$; DRS: $M = 132.82, S.D. = 5.35$) and lower in the demented group (MMSE: $M = 19.12, S.D. = 2.15$; DRS: $M = 102.82, S.D. = 13.04$) and demented/depressed group (MMSE: $M = 20.08, S.D. = 5.07$; DRS: $M = 104.00, S.D. = 21.37$). This pattern of results was once again expected, given the inclusion criteria described previously. ANOVA's with diagnostic group as the independent variable and MMSE and the DRS scores as dependent variables revealed that scores on these measures were significantly different among groups (MMSE: $F [2,43] = 43.51, p < .001$; DRS: $F [2,43] = 24.49, p < .001$). Pairwise comparisons revealed that scores were significantly higher in the depressed group than in the demented group and the demented/depressed group ($p < .05$). The demented and demented/depressed groups were not significantly different from each other on measures of overall cognitive ability ($p > .05$).

Table 3.

Scores on Measures of Cognitive Ability in Each Diagnostic Group

	DEM	DEP	DEM/DEP
Mini-Mental State Exam (MMSE)	19.12 (2.15)	28.06* (1.43)	20.08 (5.07)
Dementia Rating Scale (DRS)	102.82 (13.04)	132.82* (5.35)	104.00 (21.37)

* Significantly higher than the other two groups, $p < .05$

DEM = Demented

DEP = Depressed

DEM/DEP = Demented/Depressed

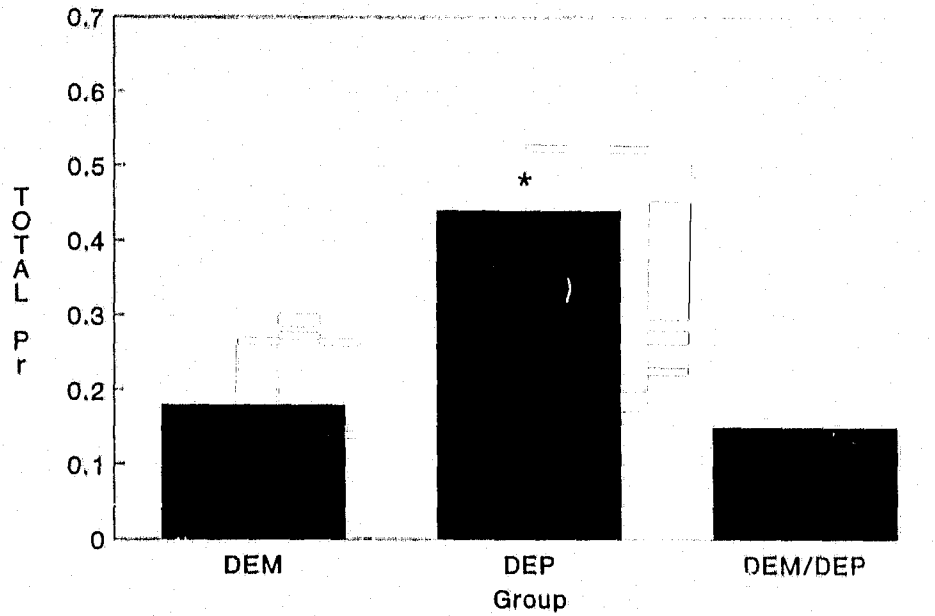
These results indicate that the level of depression in the depressed and demented/depressed groups was similar, and in the moderate to severe range. Significant depressive symptoms were not observed in the demented group. Level of cognitive impairment was similar in the demented and demented/depressed groups, and was in the mild to moderate range. The depressed group scored within the normal range on measures of overall cognitive ability.

B. DISCRIMINATION ABILITIES: HYPOTHESIS 1

Discrimination scores for total figures (TPr; geometric and nonsense combined) were highest in the depressed group ($M = 0.44$, $S.D. = 0.20$) and lower in the demented ($M = 0.18$, $S.D. = 0.18$) and demented/depressed groups ($M = 0.15$, $S.D. = 0.11$) (see Figure 1). An ANOVA of TPr scores revealed a significant group effect ($F [2,43] = 13.55$, $p < .001$). Use of contrasts for planned comparisons indicated that discrimination abilities were significantly better in the depressed group than in the demented group and the demented/depressed group ($p < .05$). TPr scores from the demented and demented/depressed groups were not significantly different from each other ($p > .05$).

Differences among the groups in discrimination of geometric figures (GPr) as compared to nonsense figures (NPr) were then investigated. In general, the pattern of results was similar to that found with TPr. That is, GPr and

Figure 1.
TPr by Group



* p < .05

TPr = Discrimination of Total figures

DEM = Demented

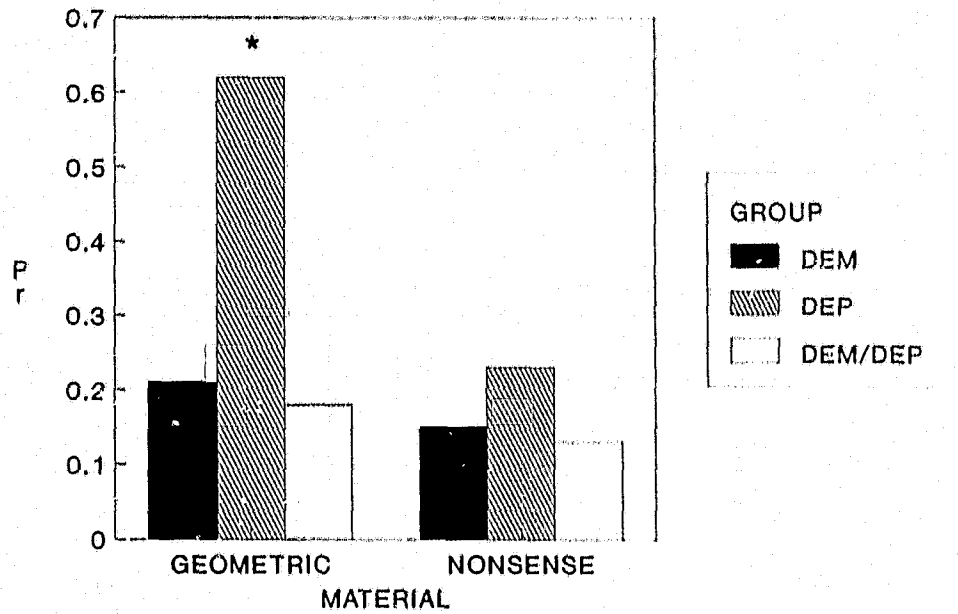
DEP = Depressed

DEM/DEP = Demented/Depressed

NPr scores were highest in the depressed group (GPr: $M = 0.62$, $S.D. = 0.24$; NPr: $M = 0.25$, $S.D. = 0.21$) and lower in the demented (GPr: $M = 0.21$, $S.D. = 0.24$; NPr: $M = 0.14$, $S.D. = 0.14$) and demented/depressed groups (GPr: $M = 0.18$, $S.D. = 0.17$; NPr: $M = 0.13$, $S.D. = 0.15$) (see Figure 2). MANOVA with group (demented, depressed, demented/depressed) as a between subjects factor and material (geometric versus nonsense) as a within subjects factor revealed a significant main effect for group ($F [2,43] = 13.41$, $p < .001$), consistent with the above findings for overall discrimination abilities. Results also revealed a significant effect for material due to overall lower scores for the nonsense figures ($F [1,43] = 27.24$, $p < .001$), and a significant group by material interaction ($F [2,43] = 11.63$, $p < .001$). In order to assess what factors were contributing to this overall significance, ANOVA's of GPr and NPr were conducted separately. Results showed that discrimination of geometric figures was significantly different among the groups ($F [2,43] = 19.15$, $p < .001$), while discrimination of nonsense figures was not ($F [2,43] = 2.32$, $p > .05$).

Use of contrasts for planned comparisons indicated that discrimination of geometric figures, GPr, was significantly better in the depressed group than in the demented group and the demented/depressed group ($p < .05$). The demented and demented/depressed groups were not significantly different from each other on this measure ($p > .05$) (see Figure 2). An

Figure 2.
GPr and NPr by Group



* $p < .05$

GPr = Discrimination of Geometric figures
NPr = Discrimination of Nonsense Figures

DEM = Demented
DEP = Depressed
DEM/DEP = Demented/Depressed

inspection of Figure 2 revealed that the same pattern of scores among groups resulted for NPR scores, but the differences were not significant, as noted above. A floor effect for NPR may have limited variability among groups on this index.

The hypothesis that discrimination abilities would be better in the depressed group than in either demented group was supported. The demented and the demented/depressed groups were not significantly different from each other on any of the discrimination indices examined. Analyses revealed that the significant differences that were found among groups were due to different performances when presented with geometric figures, rather than nonsense figures. The same pattern of scores resulted with nonsense figures, but all groups performed more poorly with them, suggesting that this condition was difficult for all the elderly subjects involved in the study and any differences that may have existed between them were obscured by the level of task difficulty. Variability among the groups in discrimination of nonsense figures appeared to be limited by the presence of a floor effect.

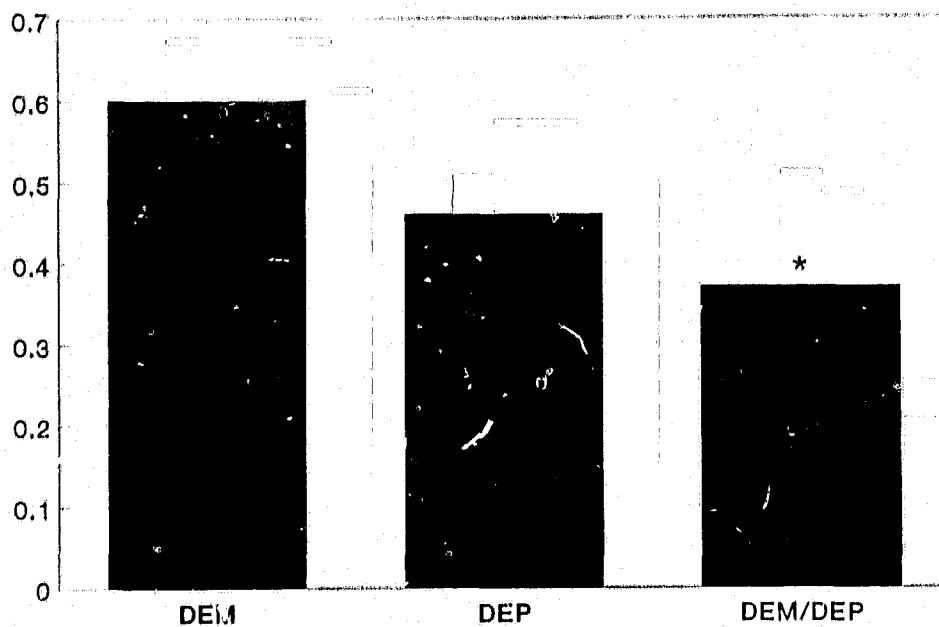
C. RESPONSE BIAS: HYPOTHESIS 2

Response bias scores for total figures (TBr; geometric and nonsense combined) were highest (most liberal) in the demented group ($M = 0.60$, $S.D. = 0.21$), lower (more

conservative) in the depressed group ($M = 0.46$, $S.D. = 0.28$), and lowest (most conservative) in the demented/depressed group ($M = 0.37$, $S.D. = 0.22$) (see Figure 3). An ANOVA of TBr scores revealed a significant group effect ($F [2,43] = 3.68$, $p = .03$). Use of contrasts for planned comparisons indicated that TBr was significantly more conservative in the demented/depressed group than in the demented group ($p < .05$). No other comparisons were significant, however, the resulting pattern of scores reflected the presence of a more liberal response bias in the demented group as compared to the depressed group and a more liberal response bias in the depressed group as compared to the demented/depressed group (see Figure 3).

Differences in response bias when presented with either geometric (GBr) or nonsense figures (NBr) was then investigated. The pattern of results for GBr scores was similar to that found for TBr scores. That is, GBr was highest in the demented group ($M = 0.64$, $S.D. = 0.32$), lower in the depressed group ($M = 0.43$, $S.D. = 0.34$), and lowest in the demented/depressed group ($M = 0.21$, $S.D. = 0.25$). NBr scores were similar in all three groups (DEM: $M = 0.55$, $S.D. = 0.32$; DEP: $M = 0.49$, $S.D. = 0.33$; DEM/DEP: $M = 0.51$, $S.D. = 0.31$). (See Figure 4.) MANOVA with group as a between subjects factor and material (geometric vs. nonsense) as a within subjects factor revealed a significant main effect for group ($F [2,43] = 4.51$, $p = .02$), consistent with the

Figure 3.
TBr by Group



*Significantly different from DEM, $p < .05$

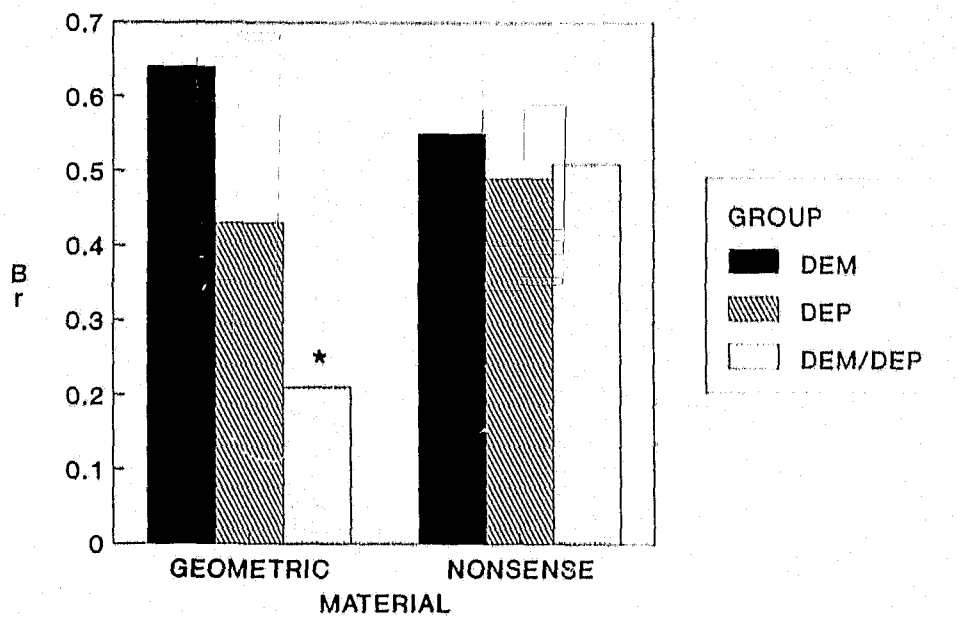
TBr = Response Bias for Total figures

DEM = Demented

DEP = Depressed

DEM/DEP = Demented/Depressed

Figure 4.
GBr and NBr by Group



*Significantly different from DEM, $p < 0.05$

GBr = Response Bias for Geometric figures
NBr = Response Bias for Nonsense Figures

DEM = Demented
DEP = Depressed
DEM/DEP = Demented/Depressed

previous analysis for overall response bias. Type of material (nonsense vs. geometric) did not significantly affect response bias ($F [1,43] = 1.72, p = .20$). There was also no group by material interaction ($F [2,43] = 2.38, p = .10$), but inspection of Figure 4 indicated that the effect was actually due to differences when responding to geometric figures. GBr and NBr scores were explored separately with ANOVA's. Results showed that GBr was significantly different among the groups ($F [2,43] = 6.61, p = .003$), but NBr was not ($F [2,43] = 2.32, p > .05$).

Use of contrasts for planned comparisons revealed that GBr was significantly lower (i.e., reflecting a more conservative response bias) in the demented/depressed group than in the demented group ($p < .05$). No other comparisons were significant, but the resulting pattern of scores reflected a more liberal response bias in the demented group as compared to the depressed group and a more liberal response bias in the depressed group, as compared to the demented/depressed group (see Figure 4).

The hypothesis that response bias would be different in demented, depressed, and demented/depressed patients was partially supported. Demented/depressed patients had a significantly more conservative response bias than the demented group. Significant differences were not found between the demented group and the depressed group, or between the depressed group and the demented/depressed

group, but the pattern of scores reflected a more liberal response bias in the demented group as compared to the depressed group and a more liberal bias in the depressed group as compared to the demented/depressed group. These differences appeared to be due to differences when responding to geometric figures. No such differences or patterns appeared when presented with nonsense figures.

D. ASSOCIATIONS BETWEEN INDICES OF DISCRIMINATION AND RESPONSE BIAS AND MEASURES OF DEMENTIA AND DEPRESSION:

HYPOTHESIS 3

Pearson product-moment correlation coefficients were computed to explore associations that might exist between indices of discrimination (TPr, GPr, NPr) and response bias (TBr, GBr, NBr) and performance on measures of overall cognitive ability (MMSE, DRS) and ratings of depression severity (HDRS, CGI).

Analyses using scores from the total sample revealed a strong, positive association between level of cognitive ability and discriminability (see Table 4). That is, higher levels of cognitive ability were associated with better discrimination scores. These effects were strong for correlations between TPr and MMSE scores ($r = .60, p < .001$), TPr and DRS scores ($r = .64, p < .001$), GPr and MMSE scores ($r = .62, p < .001$), and GPr and DRS scores ($r = .62, p < .001$). Relatively weaker, though still statistically

Table 4.

**Correlations Between Measures of Cognitive Ability and
Discriminability
(N = 46)**

	TPr	GPr	NPr
MMSE	.60**	.62**	.38*
DRS	.64**	.62**	.47*

* p < .009

** p < .001

MMSE = Mini-Mental State Exam

DRS = Dementia Rating Scale

TPr = Discrimination of Total Figures

GPr = Discrimination of Geometric Figures

NPr = Discrimination of Nonsense Figures

significant, correlations were seen between NPr and MMSE scores ($r = .38$, $p = .009$) and NPr and DRS scores ($r = .47$, $p = .001$).

Associations between ratings of depression severity (HDRS, CGI) and indices of discriminability were also explored through the use of correlations. Results revealed a moderate positive association between most of these variables, indicating that more severe depressive symptoms were associated with better discrimination abilities (see Table 5). Specifically, moderate associations were evident between TPr and HDRS ($r = .36$, $p = .02$), TPr and CGI ($r = .30$, $p = .04$), GPr and HDRS ($r = .37$, $p = .01$), and GPr and CGI ($r = .35$, $p = .02$). Correlations between NPr scores and depression ratings resulted in weak, non-significant correlations ($r = .12$ to $.22$, $p > .14$). These results appear to be somewhat paradoxical; that is, more severe depressive symptomatology was associated with better discrimination ability. However, it is likely that this pattern of results is related to the criteria used for subject selection; that is, depressed patients were required to have depressive symptoms in the moderate to severe range and be cognitively intact, while demented patients were required to show minimal, if any, symptoms of depression and be cognitively impaired. So, by definition those patients with higher depression ratings would be expected to have better discrimination scores because they were more cognitively

Table 5.
Correlations Between Ratings of Depression Severity and
Discriminability
(N = 46)

	TPr	GPr	NPr
HDRS	.35*	.37*	.22
CGI	.30*	.35*	.12

* $p < .05$

HDRS = Hamilton Depression Rating Scale
CGI = Clinical Global Impression

TPr = Discrimination of Total figures
GPr = Discrimination of Geometric figures
NPr = Discrimination of Nonsense figures

intact. The demented/depressed group was the exception in that they had moderate to severe depressive symptoms and were cognitively impaired. To explore the association between depression severity and discrimination ability further, correlations between these variables were computed in each diagnostic group separately (see Table 6). In the demented group and the depressed group correlations were small and non-significant ($r = -.19$ to $.11$, $p > .47$), indicating that in these two groups there is little association between depression severity and discrimination ability. In the demented/depressed group stronger, though not statistically significant, correlations resulted between TPr and CGI scores ($r = -.42$, $p = .18$), GPr and HDRS scores ($r = -.51$, $p = .09$), and NPR and HDRS scores ($r = .43$, $p = .16$). In this group more severe depression was associated with poorer discrimination overall, poorer discrimination of geometric figures, and better discrimination of nonsense figures.

Pearson product-moment correlations were computed to explore associations that might exist between performance on measures of overall cognitive ability and indices of response bias (TBr, GBr, NBr). No associations between MMSE scores and any index of response bias, nor between DRS scores and any index of response bias, were evident (see Table 7). These results indicated that level of cognitive ability, as assessed by these measures, did not

Table 6.

**Correlations Between Ratings of Depression Severity and
Discriminability in Each Diagnostic Group**

		HDRS	CGI
	TPr	-.17	-.02
DEMENTED (n=17)	GPr	-.15	-.08
	NPr	-.18	.08
	TPr	.11	-.18
DEPRESSED (n=17)	GPr	.07	-.14
	NPr	.11	-.19
	TPr	-.09	-.42
DEMENTED/ DEPRESSED (n=12)	GPr	-.51	-.37
	NPr	.43	-.18

p >.05 for all correlations

HDRS = Hamilton Depression Rating Scale
CGI = Clinical Global Impression

TPr = Discrimination for Total figures
GPr = Discrimination for Geometric figures
NPr = Discrimination for Nonsense figures

Table 7.

Correlations Between Measures of Cognitive Ability and
Response Bias
(N = 46)

	TBr	GBr	NBr
MMSE	-.04	-.12	.08
DRS	-.05	-.12	.05

p >.05 for all correlations

MMSE = Mini-Mental State Exam

DRS = Dementia Rating Scale

TBr = Response Bias for Total figures

GBr = Response Bias for Geometric figures

NBr = Response Bias for Nonsense figures

significantly affect response bias.

Associations between ratings of depression severity and response bias were then explored. Resulting correlations suggested that as depressive symptoms became more severe, response bias became more conservative (see Table 8). Moderate, negative associations appeared between CGI and TBr scores ($r = -.33$, $p = .02$) and between CGI and GBr scores ($r = -.40$, $p = .006$). Weaker associations were seen between HDRS and TBr ($r = -.22$, $p = .15$) and HDRS and GBr ($r = -.28$, $p = .06$). NBr scores were not associated with depression ratings, as assessed with these measures ($r = -.08$ to $-.04$, $p > .60$).

To explore the hypothesis that dementia and depression make independent contributions to discrimination ability and response bias, a linear regression model was utilized wherein the presence of dementia and depression were entered as separate predictors. Results revealed that the presence of dementia made the most significant contribution to discrimination scores (see Table 9). Resulting regression coefficients indicated that the presence of dementia lowered TPr scores by -0.29 (Standard Error = $.06$) ($t [43] = -4.46$, $p < .001$), and GPr scores by -0.44 (S.E. = $.08$) ($t [43] = -5.20$, $p < .001$). The presence of depression lowered TPr scores by only -0.03 (S.E. = $.06$) ($t [43] = -.044$, $p = .66$) and GPr scores also by only -0.03 (S.E. = $.08$) ($t [43] = -0.34$, $p = .74$). Neither dementia nor depression made

Table 8.
Correlations Between Ratings of Depression Severity and
Response Bias
(N = 46)

	TBr	GBr	NBr
HDRS	-.22	-.28	-.05
CGI	-.33*	-.40**	-.08

* p < .05

** p < .02

HDRS = Hamilton Depression Rating Scale

CGI = Clinical Global Impression

TBr = Response Bias for Total figures

GBr = Response Bias for Geometric figures

NBr = Response Bias for Nonsense Figures

Table 9.

Regression Coefficients for Discrimination Indices

	TPr	GPr	NPr
PRESENCE OF DEPRESSION (S.E.)	-.03 (.06)	-.03 (.08)	-.02 (.06)
PRESENCE OF DEMENTIA	-.29* (.06)	-.44* (.08)	-.12 (.06)

* p < .001

TPr = Discrimination for Total figures
 GPr = Discrimination for Geometric figures
 NPr = Discrimination for Nonsense figures

significant contributions to NPr scores.

In contrast to the results above, the presence of depression made the most significant contribution to response bias (see Table 10). Regression coefficients indicated that the presence of depression lowered TBr scores by -0.24 (S.E. = $.09$) (t [43] = -2.65 , $p = .01$) and GBr scores by -0.43 (S.E. = $.12$) (t [43] = -3.62 , $p < .001$). Presence of dementia lowered TBr scores by only -0.10 (S.E. = $.09$) (t [43] = -1.07 , $p = .29$) and GBr scores by -0.21 (S.E. = $.12$) (t [43] = -1.80 , $p = .08$). Neither dementia nor depression made significant contributions to NBr scores.

Table 10.

Regression Coefficients for Response Bias Indices

	TBr	GBr	NBr
PRESENCE OF DEPRESSION (S. E.)	-.24* (.09)	-.43** (.12)	-.04 (.12)
PRESENCE OF DEMENTIA	-.10 (.09)	-.21 (.12)	.02 (.12)

*p = .01

**p < .001

TBr = Response Bias for Total figures
 GBr = Response Bias for Geometric figures
 NBr = Response Bias for Nonsense figures

VI. DISCUSSION

Results pertaining to each hypothesis will be discussed separately, followed by a general discussion of the study's limitations and implications.

A. SUMMARY OF HYPOTHESES

1. Discrimination Abilities/Hypothesis 1: The hypothesis that discrimination abilities would be better in depressed patients than in demented or demented/depressed patients was supported. This was true for discrimination of nonsense and geometric figures combined (TPr), discrimination of geometric figures alone (GPr), and discrimination of nonsense figures alone (NPr), although differences with nonsense figures did not reach statistical significance. Both the demented and the demented/depressed patient groups performed more poorly than the depressed group, but they were not significantly different from each other on any of the discrimination indices examined.

All groups performed more poorly with the nonsense figures, suggesting that this condition was more difficult than the geometric figures for all the elderly patients involved in this study, regardless of diagnosis. Variability in NPr scores may have been limited by a floor effect.

a. Better discrimination abilities in depressed

patients as compared to demented patients is consistent with results of previous investigations (Larner, 1977; Miller and Lewis, 1977; Niederehe, 1986; Massman et al., 1992). It appears that depressed patients' ability to encode and store information is superior to that of demented patients.

Although free recall was not assessed in this study, these results indirectly support the theory of Weingartner and colleagues (Cohen, et al., 1982; Weingartner et al., 1977; Weingartner, et al., 1984; Weingartner, Cohen, et al., 1981; Weingartner, Kaye, et al., 1981) of a "central motivational deficit" in depression, in that depressed patients performed relatively well on a passive task of recognition memory which presumably takes less effort than a more active, effortful task of free recall, on which depressed patients have been reported to do relatively poorly. The depressed patients' poorer performance with nonsense figures, which appeared to be more difficult and presumably took more effort to remember, also supports this view.

Poor performance by demented patients on this passive recognition memory task may be explained by Weingartner's (Weingartner, Kaye, et al., 1981) theory of deficient encoding processes in dementia. That is, demented patients perform poorly on memory tasks because information is not encoded well initially and is thus easily forgotten, resulting in poor performance on effortful, as well as relatively effortless tasks.

b. The present finding of no difference in discrimination ability between demented and demented/depressed groups is consistent with the results of Lopez et al. (1990) who found no difference in memory ability, or other cognitive areas (i.e., attention, language, visuospatial functions) in probable AD patients with or without depression. Present results are in contrast to those of Raskin (1986) who found that the presence of depression in demented patients resulted in poorer immediate and delayed recall on the more difficult tasks of their battery. The nonsense figures appeared to be the more difficult condition in this study. However, as mentioned previously, variability between groups on this measure was most likely limited due to a floor effect. It is possible that significant differences may have been found with a measure allowing for greater variability in performance at lower levels. It may be of interest to address this issue in future research.

In summary, present results confirm prior research that documented better recognition memory abilities in depressed patients as compared to demented patients and also confirm research that has suggested that the presence of depression in demented patients does not exacerbate their cognitive difficulties. Differences on the more difficult task of this study were most likely obscured by the presence of a floor

effect, however. Future research with measures more sensitive to differences in cognitive ability at lower levels is needed to further investigate potential differences between demented and demented/depressed patients.

2. Response Bias/Hypothesis 2: The hypothesis that response bias would be different in demented, depressed, and demented/depressed patients was partially supported. Demented/depressed patients had a significantly more conservative response bias than the demented group. No other group comparisons were significant, but the pattern of scores reflected a more liberal response bias in the demented group as compared to the depressed group, as well as a more liberal bias in the depressed group as compared to the demented/depressed group. That is, the demented group had the most liberal response bias, the depressed group was more conservative, and the demented/depressed group was the most conservative. This pattern was apparent when responding to geometric and nonsense figures combined (TBr), and when responding to just geometric figures (GBr), but no such differences appeared when responding to nonsense figures.

a. While response bias was not significantly different in the demented group and the depressed group, the pattern of results when responding to total figures and geometric

figures (i.e., relatively more liberal responding in the demented group and relatively more conservative responding in the depressed group) was consistent with results of previous investigations (Brandt et al., 1992; Corwin et al., 1990; Hart et al., 1985; Larner, 1977; Massman et al., 1990; Miller and Lewis, 1977; Niederehe, 1980; Snodgrass and Corwin, 1988). It is likely that these differences would reach statistical significance with a larger sample size.

b. The finding of a more conservative response bias in demented patients with depression as compared to non-depressed demented patients is consistent with the hypothesis that the depressive process influenced response bias in demented/depressed patients such that they were more likely to say "no" when uncertain if they had seen an item before or not. That is, the presence of depression in demented patients had a "conservatizing" effect on the typically liberal response tendencies of demented patients. These different response tendencies in demented and demented/depressed patients suggest that assessment of response bias may provide useful information when considering if an individual with dementia is also depressed.

Present results support Snodgrass and Corwin's (1988) theory that liberal responding is not simply due to poor memory abilities. Both demented and demented/depressed groups had similarly poor discrimination abilities, but

their response biases were very different. That is, the demented group tended to respond "yes", while the demented/depressed group tended to respond "no". In Snodgrass and Corwin's study the amnesic group had the poorest discrimination abilities, but their response bias was relatively conservative as compared to demented patients. The authors suggested that the demented patients responded liberally due to a deficit in semantic memory, which is not present in amnesics. They hypothesized that a semantic memory deficit would lead to encoding deficits and a general increase in familiarity among targets and distractors due to difficulties encoding distinguishing characteristics of stimuli at presentation. This increase in familiarity would then lead to a greater tendency to respond "yes" that an item had been seen before, even if it had not. In this study it seems that the depressive process and its associated negative frame of reference outweighed the "liberalizing", perhaps "familiarizing" effect of dementia, resulting in the use of more conservative decision criteria in demented/depressed patients.

One possible explanation for this finding is suggested by Miller and Lewis' (1977) contention that the presence of depression is associated with a need for a "higher level of internal or subjective certainty before responding". It appears that the possibly "familiarizing" effect of dementia did not make items appear familiar enough to give the sense

of certainty needed by these demented/depressed patients, and thus they responded "no" more often when in a state of uncertainty.

Although the material and group by material interaction of the response bias analyses were not significant, an inspection of Figure 4 indicated that demented/depressed patients responded more liberally to the relatively more difficult nonsense figures. This result suggests that these items may have been more difficult to distinguish from one another and thus both targets and distractors appeared familiar enough to cross the level of certainty needed, leading to an increase in "yes" responses in this condition.

Another possible explanation for the generally more conservative response bias of demented/depressed patients as compared to non-depressed demented patients is suggested by cognitive-behavioral theorists, such as Beck (1972) and Maier and Seligman (1976) who define depression in terms of a negative cognitive set through which life is perceived and responded to. That is, the presence of depression in demented patients may predispose them towards saying "no" rather than "yes" under any condition. This argument, however, does not explain the relatively more liberal responding of demented/depressed patients when presented with nonsense figures.

c. The hypothesis that the dementing process in demented/depressed patients would "liberalize" response bias

in comparison to non-demented depressed patients was not supported. In contrast to the expected findings, response bias of the demented/depressed group was actually more conservative than the non-demented depressed group, although this difference was not statistically significant. Based on the theory of Miller and Lewis (1977) described above, it is suggested that the presence of dementia, in addition to moderate to severe depressive symptoms, may have made demented/depressed patients experience a greater sense of uncertainty regarding their responses as compared to non-demented depressed patients, thus increasing the tendency to respond "no" in the demented/depressed group. That is, a greater sense of uncertainty in demented/depressed patients (due to the presence of cognitive impairment) may have exacerbated the depressive tendency to respond "no" (due to the need for a higher level of certainty before responding "yes"), leading to an even more conservative response bias in demented/depressed patients as compared to non-demented depressed patients.

In summary, response bias of the demented/depressed group was significantly more conservative than in the demented group. Response bias of the demented/depressed group was even more conservative than in the non-demented depressed group, who were more conservative than the non-demented demented group, but these differences were not statistically significant. Differences in response bias of

the demented and demented/depressed groups suggest that evaluation of response bias may offer useful diagnostic information when assessing a demented patient for the presence of depression. Differences in response bias were explained in terms of a greater need for internal certainty in demented/depressed patients (due to the presence of depression) as compared to non-depressed demented patients, and an exceedingly low level of certainty in demented/depressed patients (due to the presence of cognitive impairment) as compared to non-demented depressed patients.

3. Associations Between Indices of Discrimination and Response Bias and Measures of Dementia and

Depression/Hypothesis 3: Results of these exploratory correlational analyses should be interpreted cautiously due to the relatively small sample size ($n = 46$) studied. These analyses confirmed some of the apriori hypotheses and refuted others. Specifically:

a. As expected, higher levels of cognitive ability were associated with better discrimination scores, particularly TPr and GPr. That is, more cognitively intact patients tended to perform better than more cognitively impaired patients on measures of recognition memory. Approximately 36 to 41% of the variance in TPr and GPr scores were accounted

for by performance on the MMSE and DRS. Correlations between performance on these measures and NPr scores were statistically significant, but they accounted for only 14 to 22% of the variance in NPr, suggesting that little association exists between level of cognitive ability and discrimination of nonsense figures. This result is most likely due to the restricted range of NPr scores, with all groups performing near the floor of this measure.

b. The hypothesis that discrimination abilities would worsen as depression severity increased was not substantiated when scores from the total sample were analyzed. Only 9 to 14% of the variance in TPr and GPr scores, and only 1 to 5% of the variance in NPr scores were accounted for by ratings from the CGI and HDRS. In addition, these modest correlations were positive, indicating that more severe depressive symptoms were associated with better discrimination abilities. This somewhat counter-intuitive result was most likely related to the criteria used for subject selection; that is, depressed patients were required to have depressive symptoms in the moderate to severe range and be cognitively intact, while demented patients were required to show minimal, if any, depressive symptoms and be cognitively impaired. So, by definition, those patients with higher depression ratings would be expected to have better discrimination scores because they were cognitively more intact. The demented/depressed group was the exception in

that they had moderate to severe depressive symptoms and were cognitively impaired. In an attempt to resolve this issue, correlations between indices of discrimination and ratings of depression severity were computed in each diagnostic group separately. Results indicated that there was no association between these measures in the demented group or the depressed group, most likely due to the restricted range of scores. In the demented/depressed group, however, there was a suggestion (though not significant most likely due to the small sample size) that more severe depressive symptoms were associated with poorer discrimination overall and poorer discrimination of geometric figures. This result confirms earlier research that found that the intensity of depression was inversely correlated with performance on cognitive tasks (Henry et al., 1973). However, in the demented/depressed group more severe depressive symptoms were also associated with better discrimination of nonsense figures. This result is difficult to explain given the current knowledge of depression and its effect on memory. Further research with demented/depressed patients on difficult cognitive tasks is needed to clarify the meaningfulness of this result.

c. The hypothesis that level of cognitive ability would correlate with response bias was not supported. Results indicated that level of cognitive ability, as assessed by the MMSE and the DRS, did not significantly affect response

bias, as measured by TBr, GBr, and NBr. While results discussed previously indicated that response bias was different among the three diagnostic groups, these results suggest that the differences in response bias were not associated with level of cognitive ability. This lack of association was most likely due to the presence of a conservative response bias in the demented/depressed group, who were equivalent in cognitive ability to the more liberally responding demented group.

d. The hypothesis that severity of depression would correlate with response bias indices was supported, but to only a modest degree. Resulting correlations suggested that as depressive symptoms became more severe, response bias became more conservative. CGI ratings accounted for 11 to 16% of the variance in TBr and GBr scores, respectively, while HDRS ratings accounted for only 5 to 8% of the variance in these measures. CGI ratings appear to be slightly more sensitive to differences in response bias. No associations between NBr and depression ratings were evident. These correlations are consistent with previous results indicating generally more conservative responding of the depressed and demented/depressed groups as compared to the more liberal responding in the non-depressed demented group. However, since correlations were only modest they suggest that severity of depression was not a sensitive indicator of response bias, as assessed in the present

study. It is possible that correlations involving more subjects would more firmly support the proposed hypothesis.

e. The hypothesis that dementia and depression would make independent contributions to discrimination and response bias was not supported. Rather, results indicated that the presence of dementia made a significant contribution to discrimination scores as indicated by TPr and GPr, but presence of depression did not contribute a significant amount to these scores. In contrast, presence of depression made a significant contribution to response bias as assessed by TBr and GBr, but the presence of dementia did not. Neither dementia nor depression contributed significantly to NPr or NBr.

These results indicated that the dementing process had the most significant impact on cognitive tasks, while the depressive process had the most significant impact on response tendencies. These results are perhaps not surprising if the prominent feature of each disease process is considered. That is, the prominent feature of dementia is the presence of cognitive deficits, particularly memory deficits; thus it might be expected that dementia would make the most significant contribution to performance on a memory task. The prominent feature of depression is a disorder of mood characterized by a negative cognitive set; thus it might be expected that depression would make the most significant contribution to measures of tendencies to

respond in a positive or negative way. For demented/depressed patients such differential contributions of each disease process led to the discovery of a distinguishing characteristic of this group as compared to the non-depressed demented group. That is, the presence of depression in this group of demented patients seemed to "conservatize" their response bias, making it distinct from the liberal tendencies of the non-depressed demented group.

In summary, exploratory analyses revealed that higher levels of cognitive ability were associated with better performance on measures of recognition memory. However, cognitive ability was not associated with response bias tendencies. Ratings of depression severity were not related to discrimination scores in the demented group or the depressed group, however in the demented/depressed group there was a suggestion that more severe depressive symptoms were associated with poorer discrimination scores on most measures, although paradoxically, they were associated with better discrimination scores on a more difficult task. Correlations between depression severity and response bias suggested that more severe depressive symptoms were associated with a more conservative response bias, but correlations were modest, indicating that the severity of depression was not a sensitive indicator of differences in response bias, as assessed here. Presence of dementia made a

significant contribution to discrimination scores, while presence of depression did not. Presence of depression made a significant contribution to response bias, while presence of dementia did not. The presence of depression in demented/depressed patients appeared to have a "conservatizing" effect on response bias, making it distinct from the liberal tendencies in the non-depressed demented group.

B. GENERAL DISCUSSION

The purpose of this study was twofold: 1) to further elucidate differences in memory ability, specifically recognition memory or discrimination ability, that might exist between elderly demented, depressed and demented/depressed patients, and 2) to investigate differences in response bias of elderly demented, depressed, and demented/depressed patients. Results indicated that discrimination abilities of depressed patients were significantly better than those of demented and demented/depressed patients, who performed at similarly low levels. These results supported previous research indicating that the presence of depression in demented patients did not exacerbate their cognitive deficits (Lopez, et al., 1990; Reifler, et al., 1986). Others (Greenwald, et al., 1989; Raskin, 1986; Rovner, et al., 1989), however, have reported poorer performance on cognitive measures, as well as on

ratings of self-care ability in demented patients with depression. While the issue of increased cognitive deficit in these patients is debatable, research has shown improvement in cognitive ability (Greenwald, et al., 1989; McAllister and Price, 1982; Shraberg, 1978) and mood (Reifler et al., 1986; Reifler, et al., 1989; Reynolds, et al., 1987; Snow and Wells, 1981) in these patients following appropriate antidepressant therapy. It is therefore essential to identify if a depressive process exists in demented patients so that they may be offered potentially helpful treatment.

Identifying depression in demented patients, however, is often difficult due to the overlap of symptoms in both disorders and the sometimes atypical manifestations of depression in demented patients. Results of this study indicated that response bias may be a useful aid when trying to determine if a demented patient is also depressed. That is, response bias tendencies of demented/depressed patients were significantly more conservative than the response bias tendencies of non-depressed demented patients. The presence of depression had a "conservatizing" effect on the typically liberal response tendencies of demented patients.

Demented/depressed patients were also more conservative than non-demented depressed patients, who were more conservative than non-depressed demented patients, but these differences did not reach statistical significance. It is

likely that with a larger sample these differences would be significant. It was suggested that the demented/depressed group displayed the most conservative response bias due to the combined effects of dementia and depression. That is, according to Miller and Lewis (1977) the presence of depression would increase the likelihood of responding "no" in these patients due to depressed patients' need for a higher level of certainty before they respond. It was hypothesized that the presence of dementia would exacerbate the demented/depressed patients' sense of uncertainty about their responses, and thus increase their tendency to say "no" beyond what would be expected due to depression alone. Further research is needed to substantiate this claim.

It is important to note that the significant differences among groups noted above occurred on measures of overall discrimination ability and response bias (TPr, TBr) and on measures of discrimination ability and response bias when responding to geometric figures (GPr, GBr). Analyses with nonsense figures did not reveal significant differences among the groups. Discrimination scores for nonsense figures were generally lower, suggesting that this condition was more difficult and variability among groups may have been limited by a floor effect. It appears that administration of only the geometric figures of KRF may be sufficient to assess discrimination and response bias in demented, depressed, and demented/depressed elderly

patients.

This study is limited in its ability to infer causality for differences among groups in terms of neuroanatomical, neuropathological, or neurochemical factors, due to the inclusion of etiologically heterogeneous demented patients in both the demented and the demented/depressed groups (including Primary Degenerative Dementia of the Alzheimer type, MID, and Dementia, NOS). This was not considered a major obstacle, however, since the main focus of the study was to investigate differences between demented and demented/depressed groups, rather than between dementias of specific etiologies. Based on the findings of Zubenko and Moossy (1988), it may be inferred that there was a relatively greater loss of cells in the locus coeruleus and substantia nigra of the demented/depressed group as compared to the non-depressed demented group. The loss of cells in these areas may be related to the relatively more conservative response bias of the demented/depressed group, but such a suggestion is only speculative at present and would require further investigation to be conclusive. If such a relationship were established it would be consistent with the findings of Corwin et al. (1990), who suggested that the conservative response bias seen in depression is associated with decreased noradrenergic functioning, the source nuclei of which are in the locus coeruleus.

Relatively greater involvement of the locus coeruleus

and substantia nigra did not seem to exacerbate the cognitive deficits of the demented/depressed group as compared to the non-depressed demented group. However, it must be emphasized that the neuropathology of many of the demented patients was not known, so distinctions between groups based on neuropathological differences are only speculative.

Continued research in this area would help clarify the nature of differences between demented, depressed, and demented/depressed patients. Suggestions for future investigations include:

1. using a larger sample in each diagnostic group so that the patterns suggested by this study (i.e., liberal responding in the demented group, more conservative responding in the depressed group, and most conservative responding in the demented/depressed group) may be more firmly established;
2. when assessing differences in cognitive ability between demented and demented/depressed groups more difficult tasks with a greater range of scores should be included, allowing for more variability between groups so that potential differences may be more readily discernible;
3. when assessing response bias, patients may be asked to rate the certainty of their responses (e.g., on a scale of 0 to 5, where 0 is equivalent to not at all certain and 5 is equivalent to absolutely certain) so that it may be

determined (a) if depressed patients require a higher level of certainty before responding "yes", as suggested by Miller and Lewis (1977); and (b) if the presence of depression in demented patients exacerbates their level of uncertainty leading to more "no" responses as compared to both depressed and demented groups, as suggested by the present study;

4. using more homogeneous demented groups (e.g., all AD patients) and obtaining an assessment of neuropathological involvement in demented, depressed, and demented/depressed patients (e.g., through the use of magnetic resonance imaging, positron emission tomography, or single photon emission computerized tomography), so that differences in cognitive functioning among these groups may be understood in terms of potentially different underlying neural substrates.

In summary, results of this study indicate that while both demented and demented/depressed patients performed poorly on a recognition memory task, their errors were very different. The non-depressed demented group tended to respond "yes" more often when uncertain, while the demented/depressed group tended to respond "no" more often. These results suggest that the combined effects of dementia and depression have a "conservatizing" effect on response bias in demented/depressed patients. This "conservatizing" effect may provide useful information when considering if a demented patient is also depressed.

REFERENCES

- American Psychiatric Association (1987). Diagnostic and Statistical Manual of Mental Disorders (Third Edition - Revised). American Psychiatric Association: Washington, D.C.
- Balldin, J., Gottfries, C.G., Karlsson, I., Lindstedt, G., Langstrom, G., and Walinder, J. (1983). Dexamethasone suppression test and serum prolactin in dementia disorders. British Journal of Psychiatry, 143, 277-281.
- Barona A., Reynolds, C.J., and Chastain, R. (1984). A demographically based index of premorbid intelligence for the WAIS-R. Journal of Consulting and Clinical Psychology, 52, 885-887.
- Beck, A.T. (1972). Depression: Causes and Treatment. Philadelphia: University of Pennsylvania Press.
- Benson, D.F. (1982). Treatable dementias. In D.F. Benson and D. Blumer (Eds.), Psychiatric Aspects of Neurological Disease (Volume II) (pp. 123-148). New York: Grune and Stratton.
- Blessed, G., Tomlinson, B.E., and Roth, M. (1968). The association between quantitative measurements of dementia and in senile changes in the cerebral gray matter of elderly subjects. British Journal of Psychiatry, 14, 797-811.
- Bondareff, W., Mountjoy, C.Q., and Roth, M. (1981). selective loss of neurones of origin of adrenergic projection to the cerebral cortex (nucleus locus coeruleus) in senile dementia. Lancet, 2, 783-784.
- Bornstein, R.A., Termeer, J., Longbrake, K., Heger, M., and North, R. (1989). WAIS-R cholinergic deficit profile in depression. Psychological Assessment: Journal of Clinical and Consulting Psychology, 1, 342-344.
- Brandt, J., Corwin, J., and Krafft, L. (1992). Is verbal recognition memory really different in Huntington's and Alzheimer's disease? Journal of Clinical and Experimental Neuropsychology, 14, 773-784.
- Breen, A.R., Larson, E.B., Reifler, B.V., Vitaliano, P.P., and Lawrence, G.L. (1984). Cognitive performance and functional competence in coexisting dementia and depression. Journal of the American Geriatric Society, 32, 132-137.

- Breslow, R., Kocsis, J., and Belkin, B. (1981). Contributions of the depressive perspective to memory function in depression. American Journal of Psychiatry, 138, 227-230.
- Brinkman, S.D. and Braun, P. (1984). Classification of dementia patients by a WAIS profile related to central cholinergic deficiencies. Journal of Clinical Neuropsychology, 6, 393-400.
- Busse, E.W. (1975). Aging and psychiatric diseases of late life. In S. Aireti (Ed.), American Handbook of Psychiatry (2nd Edition), Volume IV: Organic Disorders and Psychosomatic Medicine (pp. 67-89). New York: Basic Books.
- Butler, R. N., Lewis, M.I., and Sunderland, T. (1991). Aging and Mental Health (Fourth Edition). New York: Macmillian Publishing Company.
- Butters, N., Wolfe, J., Martone, M., Granholm, E., and Cermak, L.S. (1985). Memory disorders associated with Huntington's disease: Verbal recall, verbal recognition, and procedural memory. Neuropsychologia, 23, 729-743.
- Buyse, D.J., Reynolds, C.F., Kupfer, D.J., Houck, P.R., Hoch, C.C., Stack, J.A., and Berman, S.R. (1988). Electroencephalographic sleep in depressed pseudodementia. Archives of General Psychiatry, 45, 568-575.
- Caine, E.D. (1981). Pseudodementia. Archives of General Psychiatry, 38, 1359-1364.
- Caine, E.D. (1986). The neuropsychology of depression: The pseudodementia syndrome. In I. Grant and K.M. Adams (Eds.), Neuropsychological Assessment of Neuropsychiatric Disorders (pp.221-243). New York: Oxford University Press.
- Capruso, D.X. and Barr, W.B. (1992). Automated Kimura Scoring. Unpublished.
- Clairfield, A.M. (1988). The reversible dementias: Do they reverse? Annals of Internal Medicine, 109, 476-486.
- Cohen, R.M., Weingartner, H., Smallberg, S.A., Pickar, D., and Murphy, D.L. (1982). Effort and cognition in depression. Archives of General Psychiatry, 39, 593-597.

- Corwin, J., Peselow, E., Feenan, K., Rotrosen, J., and Fieve, R. (1990). disorders of decision in affective disease: An effect of Beta-adrenergic dysfunction? Biological Psychiatry, 27, 813-833.
- Cronholm, B. and Ottosson, J.O. (1961). Memory functions in endogenous depression. Archives of General Psychiatry, 5, 193-199.
- Cummings, J.L. (1987). Dementia syndromes: Neurobehavioral and neuropsychiatric features. Journal of Clinical Psychiatry, 48, 3-8.
- Cummings, J.L. (1989). Dementia and depression: An evolving enigma. Journal of Neuropsychiatry and Clinical Neurosciences, 1, 236-242.
- Cummings, J.L. and Benson, D.F. (1992). Dementia: A Clinical Approach (2nd Edition). Boston: Butterworth-Heinemann.
- Cummings, J.L., Benson, D.F., and LoVerme, S. (1980). Reversible dementia. Journal of the American Medical Association, 243, 2434-2439.
- Dement, W.C., Miles, L.E., and Carskadon, M.A. (1982). The white paper on sleep and aging. Journal of the American Geriatrics Society, 30, 26-50.
- DeMuth, G.W. and Rand, B.S. (1980). Atypical major depression in a patient with severe primary degenerative dementia. American Journal of Psychiatry, 137, 1609-1610.
- Devanand, D.P. and Nelson, J.C. (1985). Concurrent depression and dementia: implications for diagnosis and treatment. Journal of Clinical Psychiatry, 46, 389-392.
- English, W.H. (1942). Alzheimer's disease: Its incidence and recognition. Psychiatric Quarterly, 16, 91-94.
- Folstein, M.F., Folstein, S.E. and McHugh, P.R. (1975). "Mini-Mental State": A practical guide for grading the cognitive state of patients for the clinician. Journal of Psychiatric Research, 12, 189-198.
- Folstein, M.F. and McHugh, P.R. (1978). Dementia syndrome of depression. In R. Katzman, R.D. Terry, and K.L. Bick (Eds.), Alzheimer's Disease: Senile Dementia and Related Disorders (pp. 87-92). New York: Raven Press.

- Folstein, M.F. and Rabins, P.V. (1991). Replacing pseudodementia. Neuropsychiatry, Neuropsychology, and Behavioral Neurology, 4, 36-40.
- Fuld, P.A. (1984). Test profile of cholinergic dysfunction and of Alzheimer-type dementia. Journal of Clinical Neuropsychology, 6, 380-392.
- Fuld, P.A., Katzman, R., Davies, P., and Terry, R.D. (1982). Intrusions as a sign of Alzheimer's disease: Chemical and pathological verification. Annals of Neurology, 11, 155-159.
- Gallagher, D. (1986). Assessment of depression by interview methods and psychiatric rating scales. In L.W. Poon (Ed.), Handbook for Clinical Memory Assessment of Older Adults (pp. 202-212). Washington, D.C.: American Psychological Association.
- Greenwald, B.S., Kramer-Ginsberg, E., Marin, D.B., Laitman, L.B., Hermann, C.K., Mohs, R.C., and Davis, K.L. (1989). Dementia with coexistent major depression. American Journal of Psychiatry, 146, 1472-1478.
- Greenwald, B.S., Mathe, A.A., Mohs, R.C., Levy, M.I., Johns, C.A., and Davis, K.L. (1986). Cortisol and Alzheimer's Disease, II. Dexamethasone suppression, dementia severity, and affective symptoms. American Journal of Psychiatry, 143, 442-446.
- Guy, W. (Ed.). (1976). ECDEU Assessment Manual for Psychopharmacology: Publication ADM 76-338. Washington D.C., Department of Health, Education and Welfare, 534-537.
- Hamilton, M. (1960). A rating scale for depression. Journal of Neurology, Neurosurgery, and Psychiatry, 23, 56-62.
- Hamilton, M. (1967). Development of a rating scale for primary depressive illness. British Journal of Social and Clinical Psychology, 6, 278-296.
- Hart, R.P., Kwentus, J.A., Taylor, J.R., and Harkins, S. (1987). Rate of forgetting in dementia and depression. Journal of Clinical and Consulting Psychology, 55, 101-105.
- Hart, S.A., Smith, C.M., and Swash, M. (1985). Recognition memory in Alzheimer's disease. Neurobiology of Aging, 6, 287-292.

- Haward, L.R.C. (1977). Cognition in dementia presenilis. In W.L. Smith and M. Kinsbourne (Eds.), Aging and Dementia (pp. 189-202). New York: Spectrum Publishers.
- Henry, G.M., Weingartner, H., and Murphy, D.L. (1973). Influences of affective states and psychoactive drugs on verbal learning and memory. American Journal of Psychiatry, 130, 966-971.
- Jorm, A.F., Korten, A.E., and Henderson, A.S. (1987). The prevalence of dementia: A quantitative integration of the literature. Acta Psychiatrica Scandinavica, 76, 465-479.
- Joynt, R.J. and Shoulson, I. (1985). Dementia. In K.M. Heilman and E. Valenstein (Eds.), Clinical Neuropsychology (pp. 453-479). New York: Oxford University Press.
- Kahn, R.L., Zarit, S.H., Hilbert, N.M., and Niederehe, G. (1975). Memory complaint and impairment in the aged. Archives of General Psychiatry, 32, 1569-1573.
- Kaszniak, A.W. (1985). Personality and emotional change in dementia of the Alzheimer type: Issues in the investigation of individual differences. Journal of Clinical and Experimental Neuropsychology, 7, 611 (Abstract).
- Kaszniak, A.W. (1986). The neuropsychology of dementia. In I. Grant and K.M. Adams (Eds.), Neuropsychological Assessment of Neuropsychiatric Disorders (pp. 172-219). New York: Oxford University Press.
- Kaszniak, A.W. (1987). Neuropsychological consultation to geriatricians: Issues in the assessment of memory complaints. The Clinical Neuropsychologist, 1, 35-46.
- Kaszniak, A.W. and Allender, J.A. (1985). Psychological assessment of depression in older adults. In G.M. Chaisson-Stewart (Ed.), Depression in the Elderly: An Interdisciplinary Approach (pp. 107-160). New York: John Wiley and Sons.
- Kiloh, L.G. (1961). Pseudodementia. Acta Psychiatrica Scandinavica, 37, 336-351.
- Kimura, D. (1963). Right temporal lobe damage. Archives of Neurology (Chicago), 8, 264-271.

- Larner, S. (1977). Encoding in senile dementia and elderly depressives: A preliminary study. British Journal of Social and Clinical Psychology, 16, 379-390.
- Lazarus, L.W., Newton, N., Cohler, B., Lesser, J., and Schweon, C. (1987). Frequency and presentation of depressive symptoms in patients with PDD. American Journal of Psychiatry, 144, 41-45.
- Lishman, W.A. (1972). Selective factors in memory. Part 2: Affective disorder. Psychological Medicine, 2, 248-253.
- Liston, E.H. (1979). Clinical findings in presenile dementia: A report of 50 cases. Journal of Nervous and Mental Disease, 167, 337-342.
- Lopez, O.I., Boller, F., Becker, J.T., Miller, M., and Reynolds, C.F. (1990). Alzheimer's disease and depression: Neuropsychological Impairment and Progression of the Illness. American Journal of Psychiatry, 147, 855-860.
- Maier, S.F. and Seligman, M.E.P. (1976). Learned Helplessness: Theory and evidence. Journal of Experimental Psychology:General, 105, 3-46.
- Mann, D.M.A., Lincoln, J., Yates, P.O., Stamp, J.E., and Toper, S. (1980). Changes in the monoamine containing neurons of the human CNS in senile dementia. British Journal of Psychiatry, 136, 533-541.
- Mann, D.M.A. and Yates, P.O. (1983). Serotonin nerve cells in Alzheimer's disease. Journal of Neurology, Neurosurgery, and Psychiatry, 46, 96.
- Marcopulos, B. and Graves, R.E. (1990). Antidepressant effect on memory in depressed older persons. Journal of Clinical and Experimental Neuropsychology, 12, 655-663.
- Marsden, C.D. and Harrison, M.G. (1977). Outcome of investigation of patients with presenile dementia. British Medical Journal, 2, 249-252.
- Martin, A. and Fedio, P. (1983). Word production and comprehension in Alzheimer's disease: The breakdown of semantic knowledge. Brain and Language, 19, 124-141.

- Massman, P.J., Delis, D.C., Butters, N., Levin, B.E., and Salmon, D.P. (1990). Are all subcortical dementias alike? Verbal learning and memory in Parkinson's disease and Huntington's disease patients. Journal of Clinical and Experimental Neuropsychology, 12, 729-744.
- Massman, P.J., Delis, D.C., Butters, N., DuPont, R.M., and Gillin, J.C. (1992). The subcortical dysfunction hypothesis of memory deficits in depression: Neuropsychological validation in a subgroup of patients. Journal of Experimental and Clinical Neuropsychology, 14, 687-706.
- Mattis, S. (1976). Mental status examination for organic mental syndrome in the elderly patient. In L. Bellack and T.B. Karasu (Eds.), Geriatric Psychiatry (pp. 77-121). New York: Grune and Stratton.
- McAllister, T.W., Ferrell, R.B., Price, T., and Neville, M.B. (1982). The dexamethasone suppression test in two patients with severe pseudodementia. American Journal of Psychiatry, 139, 479-481.
- McAllister, T.W. and Price, T. (1982). Severe depressive pseudodementia with and without dementia. American Journal of Psychiatry, 131, 626-629.
- McKhann, G., Drachman, D., Folstein, M., Katzman, R., Price, D., and Stadlan, E.M. (1984). Clinical diagnosis of Alzheimer's disease: Report of the NINCDS-ADRDA workgroup under the auspices of the Department of Health and Human Services task force on Alzheimer's disease. Neurology, 34, 939-944.
- Mesulam, M.M. and VanHoesen, G.W. (1976). Acetylcholinesterase-rich projections from the basal forebrain of the rhesus monkey to neocortex. Brain Research, 109, 152-157.
- Miller, W.R. (1975). Psychological deficit in depression. Psychological Bulletin, 82, 238-260.
- Miller, E. and Lewis, P. (1977). Recognition memory in elderly patients with depression and dementia. A signal detection analysis. Journal of Abnormal Psychology, 86, 84-86.
- Mohs, R.C. and Davis, K.L. (1982). A signal detectability of the effect of physostigmine on memory in patients with Alzheimer's disease. Neurobiology of Aging, 3, 105-110.

- National Institute of Health (1991). Diagnosis and treatment of depression in late life. Reprinted from NIH Consensus Development Conference Consensus Statement, November 4-6: 2 (3).
- Nebes, R.D., Boller, F., and Holland, A. (1986). Use of semantic context by patients with Alzheimer's disease. Psychology and Aging, 3, 261-269.
- Niederehe, G. (1986). Depression and memory impairment in the aged. In L.W. Poon (Ed.), Handbook for Clinical Memory Assessment of Older Adults (pp.226-237). Washington, D.C.: American Psychological Association.
- Nott, P.N. and Fleminger, J.J. (1977). Presenile dementia: The difficulty of early diagnosis. Acta Psychiatrica Scandinavia, 51, 210-217.
- Perry, E.K., Tomlinson, B.E., Blessed, G., Bergman, K., Gibson, P.H., and Perry, R.H. (1978). Correlations of cholinergic abnormalities with senile plaques and mental test scores in senile dementia. British Medical Journal, 2, 1457-1459.
- Rabins, P.V., Merchant, A., and Nestadt, G. (1984). Criteria for diagnosing dementia caused by depression: Validation of a two-year follow-up. British Journal of Psychiatry, 144, 488-492.
- Raskin, A. (1986). Partialing out the effects of depression and age on cognitive functioning: Experimental data and methodological issues. In L.W. Poon (Ed.), Handbook for Clinical Memory Assessment of Older Adults (pp.244-256). Washington, D.C.: American Psychological Association.
- Reding, M., Haycox, J., and Blass, J. (1985). Depression in patients referred to a dementia clinic. Archives of Neurology, 42, 894-896.
- Reifler, B.V., Larson, E., and Hanley, R. (1982). Coexistence of cognitive impairment and depression in geriatric outpatients. American Journal of Psychiatry, 139, 623-626.
- Reifler, B.V., Larson, E., Teri, L.T., and Poulson, M. (1986). Dementia of the Alzheimer type and depression. Journal of the American Geriatric Society, 34, 855-859.

- Reifler, B.V., Teri, L.T., Raskind, M.R., Veith, R., Barnes, R., White, E., and McLean, P. (1989). Double-blind trial of imipramine in Alzheimer's disease patients with and without depression. American Journal of Psychiatry, 146, 45-49.
- Reynolds, C.F., Kupfer, D.J., Houck, P.R., Hoch, C.C., Stack, J.A., Berman, S.R., and Zimmer, B. (1988). Reliable discrimination of elderly depressed and demented patients by electroencephalographic sleep data. Archives of General Psychiatry, 45, 258-264.
- Reynolds, C.F., Perel, J.M., Kupfer, D.J., Zimmer, B., Stack, J.A., and Hoch, C.C. (1987). Open-trial response to antidepressant treatment in elderly patients with mixed depression and cognitive impairment. Psychiatry Research, 21, 111-122.
- Ron, M.A., Toone, B.K., Garralda, M.E., and Lishman, W.A. (1979). Diagnostic accuracy in presenile dementia. British Journal of Psychiatry, 134, 161-168.
- Rovner, B.W., Broadhead, J., Spencer, M., Carson, K., and Folstein, M.F. (1989). Depression and Alzheimer's disease. American Journal of Psychiatry, 146, 350-353.
- Salzman, C. and Shader, R.I. (1979). Clinical evaluation of depression in the elderly. In A. Raskin and L.F. Jarvik (Eds.), Psychiatric Symptoms and Cognitive Losses in the Elderly (pp. 39-72). New York: John Wiley and Sons.
- Satz, P., Hynd, G.W., D'Elia, L., Daniel, M.H., Van Gorp, W., and Conner, R. (1990). A WAIS-R marker for accelerated aging and Dementia, Alzheimer type: Base rates of the Fuld formula in the WAIS-R standardization sample. Journal of Clinical and Experimental Neuropsychology, 12, 759-765.
- Satz, P., Van Gorp, W., Soper, H.V., and Mitrushina, M. (1987). WAIS-R marker for dementia of the Alzheimer type? An empirical and statistical induction test. Journal of Clinical and Experimental Neuropsychology, 9, 767-774.
- Schildkraut, J.J. (1965). The catecholamine hypothesis of affective disorders: A review of supporting evidence. American Journal of Psychiatry, 122, 509-522.
- Shrager, D. (1978). The myth of pseudodementia: Depression and the aging brain. American Journal of Psychiatry, 135, 601-603.

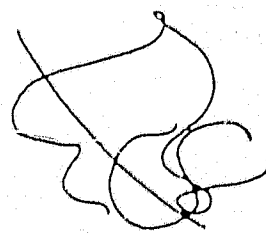
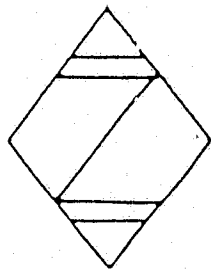
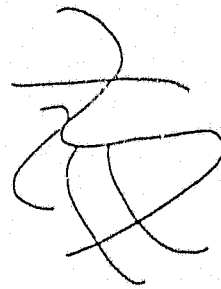
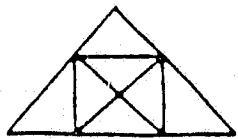
- Shrimanker, J., Soni, S.D., and McMurray, J. (1989). Dexamethasone suppression test in dementia and depression: Clinical and biological correlates. British Journal of Psychiatry, 154, 372-377.
- Snodgrass, J.G. and Corwin, J. (1988). Pragmatics of measuring recognition memory: Applications to dementia and amnesia. Journal of Experimental Psychology: General, 117, 34-50.
- Snow, S.S. and Wells, C.E. (1981). Case studies in neuropsychiatry: Diagnosis and treatment of coexistent dementia and depression. Journal of Clinical Psychiatry, 42, 439-441.
- Sternberg, D.E. and Jarvik, M.E. (1976). Memory functions in depression: Improvements with antidepressant medication. Archives of General Psychiatry, 33, 219-224.
- Stoudemire, A., Hill, C., Gulley, L.R., and Morris, R. (1989). Neuropsychological and biomedical assessment of depression-dementia syndromes. Journal of Neuropsychiatry, and Clinical Neurosciences, 1, 347-361.
- Tomlinson, B.E. (1977). The pathology of dementia. In C.E. Wells (Ed.), Dementia (pp.113-153). Philadelphia: F.A. Davis.
- Tuokko, H. and Crockett, D.J. (1987). Central cholinergic deficiency WAIS profiles in a nondemented age sample. Journal of Clinical and Experimental Neuropsychology, 9, 225-227.
- Tuokko, H., Crockett, D.J., Hurwitz, T., and Kozak, J. (1988). An examination of the clinical utility of Fuld's formula in mixed samples. Paper presented at the International Neuropsychological Society meeting, New Orleans, LA.
- Weingartner, H., Cohen, R.M., Murphy, D.L., Martello, J., and Gerdt, C. (1981). Cognitive processes in depression. Archives of General Psychiatry, 38, 42-47.
- Weingartner, H., Grafman, W., Boutelle, W., Kaye, W., and Martin, R.P. (1983). Forms of memory. Science, 221, 380-382.

- Weingartner, H., Kaye, W., Smallberg, S.A., Ebert, M.A., Gillin, J.C., and Sitaram, N. (1981). Memory failures in progressive idiopathic dementia. Journal of Abnormal Psychology, 96, 187-196.
- Weingartner, H., Miller, H., and Murphy, D.L. (1977). Mood-state dependent retrieval of verbal associations. Journal of Abnormal Psychology, 86, 276-284.
- Weingartner, H. and Silberman, E. (1984). Cognitive changes in depression. In R.M. Post and J.C. Ballenger (Eds.), The Neurobiology of Mood Disorders (pp. 121-135). Baltimore: Williams and Wilkins.
- Wells, C.E. (1979). Pseudodementia. American Journal of Psychiatry, 136, 895-900.
- Wragg, R.E. and Jeste, D.V. (1989). Overview of depression and psychosis in Alzheimer's disease. American Journal of Psychiatry, 146, 577-587.
- Zubenko, G.S. and Moossy, J. (1988). Major depression in primary dementia: Clinical and neuropathological correlates. Archives of Neurology, 45, 1182-1186.
- Zuroff, D.C., Colussy, S.A., and Wielgus, M.S. (1983). Selective memory and depression: A cautionary note concerning response bias. Cognitive Therapy and Research, 7, 223-232.

APPENDIX A: EXAMPLES OF GEOMETRIC AND NONSENSE FIGURES FROM
KIMURA'S RECURRING FIGURES TEST

GEOMETRIC FIGURES

NONSENSE FIGURES



APPENDIX B: RAW DATAAbbreviations Used in Raw Data Listing:

ID: Subject Identification Number

SEX: 1 = Male
2 = Female

AGE: Age in Years

ED: Years of Education

MMSE: Mini-Mental State Exam Score

DRSTOT: Dementia Rating Scale Total Score

HAM: Hamilton Depression Rating Scale Score

CGI: Clinical Global Impression for Depression Rating

1 = not present
2 = very mild
3 = mild
4 = moderate
5 = moderately severe
6 = severe
7 = extremely severe

BARFIQ: Estimated Full Scale IQ score based on the Barona demographic equation

THITS: Total hits, or correct "yes" responses on Kimura Recurring Figures (geometric and nonsense figures combined)

TFA: Total false alarms, or incorrect "yes" responses (geometric and nonsense figures combined)

TPR: Pr, or discrimination score for total figures (geometric and nonsense figures combined)

TBR: Br, or response bias score for total figures (geometric and nonsense figures combined)

GHITS: Hits, or correct "yes" responses on geometric figures

GFA: False alarms, or incorrect "yes" responses on geometric figures

GPR: Pr, or discrimination score for geometric figures

GBR: Br, or response bias score for geometric figures

NHITS: Hits, or correct "yes" responses on nonsense figures

NFA: False alarms, or incorrect "yes" responses on nonsense figures

NPR: Pr, or discrimination score for nonsense figures

NBR: Br, or response bias score for nonsense figures

DEMENTED GROUP RAW DATA

ID	SEX	AGE	ED	MMSE	DRSTOT	HAM	CGI	BARFIO
2	2	66	9	19	104	6	1	94.57
11	2	77	14	22	102	8	2	113.75
12	2	76	8	13	95	6	1	97.40
16	1	70	12	22	116	3	1	110.62
18	1	85	12	21	97	4	1	110.14
22	1	76	12	19	92	10	2	103.53
23	2	75	12	18	100	9	2	107.44
24	1	72	12	19	114	6	2	103.06
25	2	81	9	19	106	1	1	97.22
26	1	76	14	22	121	1	1	114.22
31	2	84	10	19	105	5	1	102.89
32	1	74	12	17	93	3	1	103.06
34	1	79	8	19	87	2	1	102.29
43	2	79	14	20	110	9	3	108.68
46	1	80	8	19	115	4	1	97.74
49	2	71	12	19	120	2	1	103.19
50	1	86	8	18	71	6	1	98.21

ID	THITS	TFA	TPR	TBR	GHITS	GFA	GPR	GBR	NHITS	NFA	NPR	NBR
2	56	84	.00	.99	28	42	-.01	.98	28	42	-.01	.98
11	29	25	.30	.60	20	17	.41	.96	9	8	.18	.3
12	41	30	.37	.57	20	9	.49	.43	21	21	.24	.
16	52	26	.61	.80	25	1	.84	.22	27	25	.36	.
18	33	41	.10	.54	17	21	.10	.56	16	20	.09	.
22	37	49	.08	.63	19	23	.13	.63	18	26	.02	.63
23	27	38	.03	.47	22	33	.00	.78	5	5	.06	.14
24	38	46	.13	.63	26	31	.18	.89	12	15	.07	.39
25	33	14	.42	.29	23	11	.54	.59	10	3	.28	.11
26	28	31	.13	.43	2	4	-.02	.10	26	27	.27	.88
31	43	39	.30	.66	28	32	.23	.98	15	7	.36	.27
32	10	15	.00	.18	8	14	-.04	.32	2	1	.05	.04
34	29	44	-.01	.52	11	15	.04	.37	18	29	-.05	.65
43	36	32	.26	.52	9	6	.18	.18	27	26	.33	.92
46	50	62	.15	.87	28	33	.20	.98	22	29	.09	.75
49	39	39	.23	.60	27	29	.26	.93	12	10	.19	.30
50	54	81	.00	.96	28	40	.04	.98	26	41	-.05	.92

DEPRESSED GROUP RAW DATA

ID	SEX	AGE	ED	MMSE	DRSTOT	HAM	CGI	BARFIQ
3	2	69	9	28	131	33	5	93.92
4	2	72	10	29	136	27	5	96.28
7	1	66	18	27	142	21	4	120.19
8	1	74	12	28	128	20	5	104.95
13	1	80	9	26	137	18	4	97.09
15	2	77	16	25	129	16	4	119.37
17	1	71	16	27	125	18	4	118.77
21	2	80	8	29	130	19	4	94.09
29	2	74	11	28	130	30	6	88.75
35	1	80	15	27	126	27	5	114.69
36	1	70	12	29	137	17	4	103.06
37	2	72	14	30	136	21	4	111.99
38	2	74	10	28	126	22	4	98.17
40	2	83	12	27	130	20	4	104.13
42	2	70	12	30	136	18	4	106.97
45	1	75	13	29	139	24	4	114.22
51	1	71	16	30	140	15	4	120.66

ID	THITS	TFA	TPR	TBR	GHITS	GFA	GPR	GBR	NHITS	NFA	NPR	NBR
3	51	44	.38	.84	25	12	.59	.71	26	32	.16	.90
4	31	5	.68	.28	16	0	.77	.07	15	5	.56	.40
7	55	10	.85	.82	28	0	.97	.40	27	10	.70	.83
8	50	39	.42	.80	27	10	.70	.83	23	29	.12	.78
13	25	19	.30	.46	10	2	.42	.14	15	17	.17	.68
15	3	0	.06	.01	2	0	.07	.01	1	0	.04	.01
17	42	28	.41	.57	16	1	.53	.07	26	27	.27	.88
21	48	40	.37	.76	26	23	.37	.86	22	17	.37	.64
29	29	31	.15	.43	14	7	.33	.26	15	24	-.04	.55
35	37	12	.51	.30	21	3	.66	.24	16	9	.35	.34
36	27	3	.44	.07	16	0	.56	.03	11	3	.32	.12
37	43	9	.65	.32	28	3	.90	.83	15	6	.38	.25
38	51	39	.44	.83	23	2	.75	.23	28	37	.11	.98
40	28	22	.24	.35	27	20	.47	.90	1	2	-.01	.06
42	39	20	.45	.44	22	2	.72	.21	17	18	.17	.52
45	46	13	.66	.46	28	7	.81	.91	18	6	.49	.29
51	30	4	.48	.10	27	2	.89	.53	3	2	.06	.06

DEMENTED/DEPRESSED GROUP RAW DATA

ID	SEX	AGE	ED	MMSE	DRSTOT	HAM	CGI	BARFIO
5	2	79	9	8	60	20	5	98.64
9	2	90	14	22	112	23	4	101.94
14	2	70	9	23	121	26	4	94.39
19	2	75	7	22	111	15	4	86.71
27	1	79	16	20	111	16	4	119.24
28	1	81	8	11	61	13	4	93.96
33	1	76	12	24	119	16	4	103.53
39	2	68	13	22	115	20	4	103.96
41	1	67	12	22	105	22	4	108.26
44	2	84	8	22	113	18	4	94.09
47	1	79	19	22	122	11	4	121.13
48	2	85	10	23	98	20	4	97.69

ID	THITS	TFA	TPR	TBR	GHITS	GFA	GPR	GBR	NHITS	NFA	NPR	NBR
5	1	1	.01	.02	0	1	-.02	.03	1	0	.04	.01
9	25	26	.19	.53	13	11	.27	.51	12	15	.10	.55
14	29	31	.15	.43	11	19	-.06	.43	18	12	.35	.45
19	26	35	.05	.44	7	3	.18	.10	19	32	-.08	.70
27	44	46	.23	.71	17	7	.43	.31	27	39	.03	.95
28	34	37	.16	.53	25	23	.33	.82	9	14	-.01	.33
33	33	33	.19	.49	8	2	.23	.08	25	31	.15	.86
39	31	13	.39	.26	14	2	.44	.10	17	11	.34	.40
41	10	14	.01	.17	0	0	.01	.01	10	14	.02	.35
44	3	0	.06	.01	2	0	.07	.01	1	0	.04	.01
47	34	33	.21	.50	9	3	.25	.11	25	30	.17	.85
48	24	19	.20	.29	2	1	.05	.04	22	18	.35	.66