Depression and Neuropsychological Impairment

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Depression and Neuropsychological Impairment

by

Diane Goulet Fisher

A Thesis Submitted to the Faculty of the Graduate School of Loyola University of Chicago in Partial Fulfillment of the Requirements for the Degree of Master of Arts

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VITA

The author, Diane Goulet Fisher, is the daughter of Bernard Thomas Goulet, Sr., and Barbara Geary Goulet. She was born November 26, 1958, in Springfield, Illinois.

Her elementary education was at St. Joseph's Grade School in Springfield, Illinois. Her secondary education was at Ursuline Academy of Springfield, Illinois. The author received her Bachelor of Science degree with High Honors in Psychology at the University of Illinois-Champaign in 1980. While attending the University of Illinois, she was a member of Kappa Kappa Gamma Sorority.

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The author is married to Mr. Westby G. Fisher and currently resides in Oak Park, Illinois.
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CHAPTER I

INTRODUCTION

Neuropsychological Deficits and Psychiatric Disorder

Within recent years, the field of clinical neuropsychology has received increased attention in the literature and has undergone extensive refinement and review of diagnostic practices. Although neuropsychological testing has many practical applications, perhaps its most acknowledged and frequent usefulness has been in the area of diagnosis (Lezak, 1976). Traditionally, neuropsychological assessment has been used to measure cerebral dysfunction resulting from cerebral vascular accidents, tumors, trauma, and other central pathophysiologic disorders. The utility of neuropsychological tests for behaviorally assessing the presence of brain damage has been consistently and repeatedly demonstrated (e.g. Filskov, & Goldstein, 1974; Golden, 1977; Golden, Hammeke, & Purisch, 1981; Golden et al., 1981; Lewis, Golden, Moses, Osmon, Purisch, & Hammeke, 1979; Malec, 1978; Purisch, Golden, & Hammeke, 1978; Russell, 1970).

Clinical application of these neuropsychological tests has increasingly demanded subtle and complex discriminations to be made between psychiatric and brain-based impairment. There are multiple clinical problems posed by the interrelationship between emotional and neuropathologic symptomatology. Patients with both neurologic and func-
tional disorders commonly present with similar psychological symptoms including anxiety, depression or psychosis, or any combination of these, (Heaton, Baade, & Johnson, 1978). Often psychiatric patients exhibit detectable cognitive deficits on the Mental Status Exam and psychological testing in the absence of significant structural intracranial pathology, or in the presence of equivocal EEG or CT scan findings. In a study of psychiatric patients referred for computerized tomography (CT) scans to rule out brain disorder, Tsai and Tsuang (1981) found that 25% were unnecessarily tested, and suggest that the findings highlight the need for refinement of available neuropsychological tests to facilitate more efficient screening of psychiatric patients.

To the extent that emotional variables affect performance on neuropsychological tests, the validity of the use of these tests with populations that may have psychiatric disorders requires special consideration. Lewis, Allen, and Frieswky (1983) suggest that the assessment of the interplay between organic, intrapsychic and situational factors is a clinically fruitful focus of neuropsychological evaluation. They conclude that this approach replaces the previous conceptualization of dysfunction as exclusively psychiatric or neuropathologic in nature, suggesting that both factors are active in influencing a patient's cognitive functioning. Diagnostically, accurate delineation of the presentation of functionally related deficits from those associated with an underlying organic substrate are needed, and clearly carry great prognostic significance for the individual patient in planning future treatment and predicting recovery of functioning.
In sum, the issue of diagnostic accuracy in the use of neuropsychological testing with psychiatric patients has generated a good deal of controversy in the literature. While various theoretical schools argue that psychopathology is either exclusively brain-based or psychological in nature, others consider the interplay of these factors to be of primary importance.

There have been general findings in the literature that suggest a variety of deficits in cognitive functioning which may be related to psychiatric variables. Numerous authors have noted that the majority of research in this area is based on studies of schizophrenics, or those included under the global category of affective disorder (Heaton, & Crowley, 1981; Malec, 1978; Miller, 1975). Several studies observed differing patterns of neuropsychological impairment across diagnostic subtypes of schizophrenia (Hirsch, & DeWolfe, 1977; Malec, 1978). In general, there is evidence that schizophrenic patients frequently receive neuropsychological test scores in the ranges typically characteristic of brain-damaged patients (Klonoff, Fibiger, & Hutton, 1970; Malec, 1978; Matarazzo et al., 1976).

Carpenter (1983) noted that a generalized deficit is less apparent in the test performance of affectives compared to schizophrenics. He concluded that schizophrenics appear to be more impaired than affectives on many neuropsychological measures, except for equal impairment on measures of nondominant, anterior brain functioning. Perkins (1974) noted that both psychotic non organic patients as well as non psychotic psychiatric patients received false positive scores on the Category
test, which is commonly regarded as a general measure of brain-based impairment. In a study comparing electroencephalograph (EEG) patterns, neuropsychological test performance, and intellectual abilities of schizophrenic and affective disorder patients, Flor-Henry (1976) found support for differing patterns of deficit between groups. There is consistent documentation of neuropsychological test performance differences between various psychiatric groups, however, the level and qualitative nature of neuropsychological test performance of various psychiatric groups appears to vary. There is little agreement on specific patterns of performance characteristic of each group.

A number of authors point out that false diagnosis of brain-damage in psychiatric populations is a common concern. Goldstein (1978) and Matarazzo et al. (1976) note the frequency of false-positive classifications of brain-damage in schizophrenic populations. The lack of specific diagnostic criteria, difficulty interpreting the patient's test performance, and need for concurrent personality and psychological assessment are emphasized. Albert (1981) and Colbert and Harrow (1966) agree that the issue of accurate diagnosis becomes particularly difficult in differentiating manifestations of a dementia versus depressive illness. Perkins (1974) observed a number of false positive ratings within a mixed psychiatric population on the Category test. Studies such as these suggest that a significant percentage of psychiatric patients referred for neuropsychological assessment are falsely classified as brain-damaged due to the influence of psychological illness.

The complexity of the issue of the accuracy of neuropsychological
tests with psychiatric populations is underscored by the presence of contrasting views within the literature. Heaton, Baade and Johnson (1978) summarized studies of psychiatric patient's neuropsychological performance and concluded that (with the exception of chronic and process schizophrenics) these tests were able to discriminate between organic and psychiatric populations with sufficient clinical accuracy. The authors observed that all psychiatric diagnostic groups, excluding chronic and process schizophrenics, consistently performed better than organics on a variety of neuropsychological tests. In a study of schizophrenic subtypes, Heaton (1975) concluded that the majority of chronic schizophrenics look organic because many of them are organic. In their review of the literature, Heaton et al. (1978) observed that one could assume at least a 30% organic base rate in most psychiatric patients sent for neuropsychological testing. Thus, these studies suggest that neuropsychological tests are valid in psychiatric populations, and that when deficits are noted they reflect an underlying brain-based impairment. Given this possibility, the significant proportion of depressed patients who are not brain-damaged and receive impaired neuropsychological test scores must be accounted for. The attribution of impaired test scores in psychiatric patients to an underlying organic base rate does not explain the changes observed in performance across repeated neuropsychological testing.

The presence of an underlying organic base rate is discussed by Lewis, Allen and Frieswyk (1983) in terms of "cortical vulnerability". The authors suggest that repeated neuropsychological evaluation docu-
ments the enduring substrate of cortical dysfunction which is apparent even following remission of the clinical psychiatric syndrome. Lewis et al.'s (1983) view of the organic versus functional deficits considers a number of possible factors:

A growing body of research suggest that the dysfunction is in the realms of hemispheric differentiation and activation, hormonal and metabolic variables and cerebrovascular functioning and that these forms of organic impairment may play a contributing role in the development of psychopathology... In these cases, the neurological substrate of dysfunction is frequently mild and may be viewed as "cortical vulnerability." That is, in the absence of significant environmental or intrapsychic stress, the impairment has no obvious effect on everyday functioning....Under (certain) conditions, the cortical vulnerability becomes sharply manifest.(p.67)

Logically, this argument could also be proposed for a significant proportion of depressed patients who are classified as organic on neuropsychological tests. However, this consideration does not allow adequate delineation of the amount of underlying organic-brain pathology from that attributable to emotional variables, nor does it describe the observed transient nature of emotionally-related cognitive deficits.

Several studies provide evidence of psychiatric patient's improvement over time on neuropsychological test performance after various types of psychological treatment, (Malloy et al., 1982; Kronfol, des Hamsher, Digre, & Waziri, 1978; Small, Small, Milstein, & Moore, 1972; Sternberg, & Jarvik, 1976). These findings strongly suggest an effect of transient emotional factors which either exaggerate or mimic actual brain-based impairment. Lewis et al. (1983) note that the fluctuating expression of cortical dysfunction and the exacerbating impact of acute psychiatric syndromes necessitates repeated testing over time to diagnose the nature and severity of cerebral impairment. It appears that
the ability of neuropsychological tests to discriminate between psychiatric and neurologic disorders may be enhanced by establishing norms and patterns descriptive of certain psychiatric subgroups, as well as observing patterns of change over time.

**Depression and Neuropsychological Impairment**

The overlap of behavioral symptoms presented by depressed and organic patients is an unresolved diagnostic issue. Heaton and Crowley (1981) reviewed studies relating emotional disturbance to neuropsychological impairment and noted that few studies included depressed patients. They concluded that the majority of these studies evidenced little if any relationship between level of depression and neuropsychological test performance in psychiatric patients. Others find clear evidence that some patients' depressive states exaggerate or introduce impairment of the type measured by neuropsychological tests (Kronfol et al., 1978; Goldstein, Filskov, Weaver, & Ives, 1977; Miller, 1975; Sweet, 1983). Gainotti (1972) observed that depression is often a part of the symptomatology that exists with demonstrable brain damage. Colbert and Harrow (1966) noted that much of the diagnostic confusion is caused by ambiguous, global criteria for defining the interrelationships and differences between depression and organicity.

Several investigators (e.g. Albert, 1981; Morstyn, Hochnadel, Kaplan, & Gutheil, 1982) have noted that depression in particular produced dysfunction that could be mistaken for irreversible cognitive change, and they discuss the need to discriminate dementia masquerading as depression and vice versa. In elderly patients depression often
results in cognitive impairment similar to dementia. Dementia is a general term that refers to a permanent impairment of intellectual function due to brain dysfunction.

Research sought to identify specific patterns of deficit associated with depressive syndromes. McAllister (1983) observed that depression is frequently associated with diffuse, global cognitive deficits, and that a clear pattern of deficits distinct from dementia related deficits is not apparent. Spar (1982) listed a number of qualitative distinctions between demented and pseudodemented patients. Pseudodementia is a functional disorder, often depression, which masquerades as dementia. For example, pseudodemented patients' test performances were characterized by a greater frequency of "near miss" answers, "don't know"s, patchy memory loss, variable level of performance on similar tasks, and greater self-awareness of the patient's deficits than seen in demented patients. LaRue (1982) observed distinctive signs in memory performance of patients with pseudodementia, including mild to moderate deficits in recall of new information, but not complete disorientation to space or time. LaRue added that these patients give a pattern of "don't know" errors without significant confabulation of responses or intrusion of irrelevant information.

LaRue (1982) and Spar (1982) concluded that qualitative distinctions facilitate the differentiation of demented from pseudodemented patients, but added that the continued risk of diagnostic error demands more information. McAllister (1983) stated that accurately diagnosed pseudodemented patients tended to have a very good prognosis. He noted
that the patients' deficits often resolve completely when the associated psychopathology is treated. Hence, the observed deficits are not indicative of an underlying dementing illness. In general, this perspective contrasts with Lewis et al.'s (1983) view of an underlying cortical vulnerability in psychopathology associated with cognitive impairment. McAllister (1983) concluded that further definition, diagnostic criteria, and classification of the pseudodemented syndrome will enhance clinical, theoretical and etiological understanding of these patients, with the ultimate goal of more accurate diagnosis, prognosis, and treatment planning. McAllister (1983) and LaRue (1982) stress the need for neuropsychological data and longitudinal assessment in clarifying these diagnostic issues. Morstyn et al. (1982), Carpenter (1983), and Lewis et al. (1983) concurred that identification of an organic disorder co-existing with depression is best discerned through repeated evaluation of neuropsychological test performance. If a good deal of a patient's impairment is functional in nature, then the patient will improve in neuropsychological performance as the depression improves. If an underlying neurological substrate is present in depressed patients, this should also become evident over repeated testing as the affective illness becomes less severe.

The influence of depression on neuropsychological test performance is problematic in all ages of depressed patients. Thus, the difficulty is not confined to differentiating demented from pseudodemented patients (Sweet, 1983). The qualitative distinctions that authors in this area have proposed may facilitate differentiation of neuropathology versus
depression in a broader clinical range of patients. Additionally, in quantitative analyses with a range of patients, the consensus appears to be that depression consistently mimics brain-damage, as observed on neuropsychological tests.

While it is not surprising to find deficits in test performance consistent with the expected syndrome of depression (i.e., psychomotor retardation and impaired concentration), the documentation of unexpected deficits and evidence of a lateralized deficit in clinically depressed patients need to be explained. Glass, Uhlenhuth, Hartel, Matuzas, & Fischman, (1981) have found evidence of cognitive dysfunction in depression even in ambulatory out-patients without substantial impairment in attention or motor functioning. Similarly, Small et al. (1972) claimed that neuropsychological test performance of psychiatric patients did not correspond with clinical signs and symptoms. Using a non-clinical populations, Harris, Gross, and Van Nieuwkirk (1981) and Tucker, Stenske, Roth, and Shearer (1981) studied depression in normal adults and noted the modulating effects of the students' emotional state on cognitive tasks. Tucker et al. (1981) observed evidence of impaired imagery, auditory attentional biases, and impaired information-processing. The authors' EEG analyses yielded asymmetries in the frontal lobe, with greater activity in the right-frontal area of the brain while the students were in a depressed state. They inferred from these observations that the modulating effects of the right-anterior region were implicated in emotional arousal and information-processing efficiency. Harris et al. (1981) found impaired Tactual Performance Test scores when normal
adults were in a depressed state. As Weingartner and Silberman (1982) suggest, caution is necessary in generalizing the results of studies such as these to populations of psychiatric patients. These studies do, however, provide convincing evidence of a relationship between a depressed emotional state and impairment in cognitive functioning.

Among the studies investigating the specific pattern of cognitive impairment evidenced by depressed patients, there appears to be consistent evidence suggesting nondominant hemisphere dysfunction (Carpenter, 1983; Goldstein et al., 1977; Kronfol et al., 1978). Carpenter (1983) summarizes recent literature in neuropsychology which has found consistent right-hemisphere performance deficits, with some evidence of milder dominant hemisphere deficits. Carpenter notes that performance on tasks requiring speed, higher spatial functioning and sensorimotor abilities of the nondominant side of the body are frequently impaired. Flor-Henry's (1976) findings support the presence of nondominant, fronto-temporal dysfunction indicated on neuropsychological, intellectual, and EEG measures. Yeudall (1977) found that 88% of depressed patients exhibited impairment on neuropsychological tests involved with temporal-frontal nondominant hemisphere functioning. Weingartner and Silberman (1982) cited a number of studies which found relatively greater deficits in performance on right-hemisphere associated spatial and holistic tasks among depressed patients. They summarized a number of studies which also suggest a reduced ability to perform tasks requiring sustained motor effort.

In their extensive reviews of depressed patient's psychological
testing performance, Weingartner and Silberman (1982) and Miller (1975) concluded that the extent of cognitive impairment was consistently related to the intensity and severity of the depression. D'Elia and Perris' (1973) EEG study also noted a laterialized difference in activity which was proportional to the degree of depression. From a study of neuropsychological and EEG measures of depressed patients, Flor-Henry (1976) concluded that laterality of dysfunction was related to the severity of depression.

While studies such as these appear very promising, a great deal more data need to be collected to document the presence of lateraled dysfunction and its relation to cognitive impairment. Miller (1975) provided a comprehensive review of depression and concurrent deficits which described impaired performance on intelligence tests, motoric slowness, impaired verbal learning and free recall task performance, and lower intellectual speed in depressed patients. Miller (1975) and McAllister (1983) concurred that observations of depression-related deficits provided little overall evidence of a pattern of deficit unique to depression.

It is important to realize that the observed depression-related deficits may not be consistent across time and may not be clearly delineated from possible underlying brain-based impairment. Studies demonstrating improvement in neuropsychological test performance following treatment for depression provide further support for the idea of a significant relationship between depression and cognitive impairment. Boyar (1981) and Sweet (1983) suggested that comparisons across time
would allow a clearer picture of the inconsistencies of psychiatric deficits. Boyar (1981) noted that interpretive error is more likely with one testing, and recommended repeated testing whenever there is a possibility of psychiatric factors affecting neuropsychological performance. Matarazzo et al.'s (1976) findings with schizophrenic, normal and brain-damaged patients provided evidence of improvement in test performance over time, and raised the issue of stability and instability of cognitive deficits in schizophrenic populations. The authors suggested the practice of repeated neuropsychological testing over time in order to gain diagnostic information when questioning organicity versus psychiatric involvement. Small et al. (1972) and Malloy et al. (1982) supported this finding by showing similar post-treatment improvement in mixed psychiatric groups on neuropsychological and intellectual measures. Sternberg and Jarvik (1976) observed that depressed patients' short-term memory functioning improved as their depression was alleviated. Small et al. (1972) suggested a need to examine which tests remain stable over time despite clinical improvement, and how test performance varies with diagnostic and treatment variables.

Several studies have documented neuropsychological test improvement over time in psychiatric patients treated with electroconvulsive therapy (ECT). The majority of these studies were designed to assess the effects of ECT treatment on cognitive functioning, and unexpectedly found deficits in the pre-test neuropsychological performance of depressed patients. Kronfol et al. (1978) observed that depressed subject's were impaired on right-hemisphere function psychological tests
prior to ECT treatment, and that these cognitive deficits improved as
the depression improved. Goldstein et al. (1977) found similar right­
hemisphere impairment in depressed patient's performance on Halstead-
Reitan Neuropsychological measures prior to ECT treatment. Often, it is
not clear in the literature what proportion of these patients had a spe­
cific diagnosis of depression. Malloy et al. (1982) reported that psy­
chiatric patients, who scored within the brain-damaged range on Hal­
stead-Reitan and WAIS tests, improved their test performance after ECT
therapy on the majority of measures. Following a review of a number of
studies reporting improvement in neuropsychological test performance
after ECT treatment, Sweet (1983) noted that it is difficult to explain
such dramatic post-treatment improvement on any other basis than
decreased depression. Sweet examined several case studies of depressed
patients, and observed that repeated neuropsychological testing shows
improvement corresponding to mood change and greater than expected for a
practice effect. In these case studies, improvement over time was
observed in numerous neuropsychological measures including: the Trail
Making, Stroop Color-Word, Finger Tapping, Wechsler Memory Scale and
Category tests.

In general, the literature has not isolated patterns of deficit
specific to depression or patterns of recovery relative to diagnostic or
treatment variables. Although a number of studies have attempted to
measure the effects of depression on neuropsychological test perform­
ance, few have been conducted with a standardized and validated battery
of neuropsychological tests. The diversity of neuropsychological tests
available is evident in the depression-related literature, and makes it difficult to integrate the quantitative findings. Perhaps because of this limitation, there is a lack of specification of observed deficits in functioning in the majority of the literature (with some exceptions, i.e. Miller 1975). Similarly, most studies have not attempted to observe patterns of deficit across groups and reported on characteristic profiles of dysfunction which might enable future studies to obtain normative data for clinical use. While there appears to be qualitative clinical signs characteristic of depressed patient's test performance, it is not clear whether a consistent pattern will emerge in quantitative assessments of deficit.

In addition, many past studies including pre- and post-test measures of depression have focused on the neurological effects of ECT therapy and have not provided control groups to compensate for practice effect improvement. Goldstein et al. (1977) noted that depressed patients referred for ECT therapy often have not responded to traditional psychotherapy and pharmacologic interventions and may have some form of neurologic deficit which is different from that of a more typical population of depressed patients. Thus, neuropsychological observations of ECT patients may not be adequately representative of depressed patients in general, and may confound the discrimination of emotional versus neurologically based disorder.

Specific diagnostic criteria for the depression diagnosis and observation of type and severity of depression in the experimental group are needed, with further consideration of variables such as history of
substance abuse, medication levels, and treatment modalities other than ECT which are not presently covered adequately in the literature.

**Statement of the Problem and Hypotheses**

The present study is designed to assess the consistency of patterns of deficit exhibited by a group of depressed psychiatric patients before and after treatment. The design includes consideration of variables such as age, education, history of substance abuse, and current mode of treatment. The generalizability of the hypothesized deficits evidenced in ECT patients and normal adults can be examined with the present group of depressed inpatients receiving other treatment modalities.

Furthermore, the study is designed to clarify the relationship between depression and neuropsychological test performance, including a wide range of functions, and a number of commonly used neuropsychological measures. Several specific hypotheses will be tested, based on the evidence in past literature. It is hypothesized that: 1) depressed patients will initially appear more impaired on neuropsychological tests than those in the nondepressed group. Therefore, depressed patient's impairment on specific subtests or across a general level of test performance will falsely classify depressed patients in ranges characteristic of brain-damaged patients. 2) Depressed patients' impairment on neuropsychological measures will lessen as the depression improves, and the improvement observed in neuropsychological test performance after treatment will be greater than the practice effect observed in the control group's pre- to post-test improvement, supporting the hypothesis that
many depression-related deficits are transient. Finally, 3) the measure of degree of depression will be correlated with degree of impairment on the neuropsychological subtest scores. A relationship between change in degree of depression and change in degree of impairment from the pre to the post-testing is expected, although the nature of this relationship is not clear from past studies. It is hoped that further information on a pattern of deficit associated with depression will be obtained. The possibility of an underlying "cortical vulnerability", or brain-based substrate of impairment which is exacerbated during episodes of depression will be examined by comparing post-test scores of depressed and nondepressed subjects.
CHAPTER II

METHOD

Subjects

Depressed subjects consisted of fifteen patients admitted to the inpatient psychiatric ward of Illinois Masonic Medical center with a diagnosis of depression. Appropriate patients were selected from a review of the psychiatric records over an eight month period, with patients who satisfied the criteria and consented to participate in the study included in the final group. Patients whose diagnosis changed over the course of treatment after initial testing were eliminated from the study. The diagnosis of depression was preliminarily made on the basis of the initial psychiatric evaluation and treatment plan filled out by the admitting psychiatrist within 24 to 36 hours after admission. All psychiatric diagnoses were confirmed using DSM-III criteria reviewed in a structured diagnostic interview given by the examiners, including the following diagnostic categories: Major Depression, Major Depressive Episode, Dysthymic Disorder and Atypical Depression. The depressive disorder was also assessed by the initial Beck Depression Inventory score given before subjects were chosen to be included in the final group. Nondepressed comparison group subjects consisted of fifteen nondepressed volunteers, including out-patients at the Chronic Pain and Headache Treatment Center of Illinois Masonic Medical Center, and volun-
teer hospital employees. The subjects included in the final comparison group did not have a diagnosis of depression in their medical charts, and were checked for depression on their initial Beck Depression Inventory scale measure. The groups were equated for age, sex, and education. The age range included adults up to sixty years of age. The mean age of the inpatient group ($M=28.5$, $SD=10.6$) did not significantly differ from the mean age of the nondepressed comparison group ($M=33.9$, $SD=8.4$), $t(28)=-1.52$, $p=.14$, and the average educational level for the inpatient group ($M=13.7$, $SD=2.2$), did not significantly differ from the comparison group average ($M=14.0$, $SD=1.5$), $t(28)=-.48$, $p=.63$. The groups also did not significantly differ in handedness (73% were right-handed in the inpatient group, 80% in the comparison group) or sex (60% were females within the inpatient group, and 66% were females in the comparison group). All subjects with a history or suspicion of neurological illness, significant drug abuse, acute medication side effects, or change of diagnosis during the course of treatment were excluded from the study.

**Materials**

**Beck Depression Inventory**

The subjects then completed the Beck Depression Inventory (BDI) checklist (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961). The BDI is a self-report measure consisting of twenty-one items related to various aspects of depression. Each item is defined by four statements of increasing severity and the subject is instructed to choose the statement that is most applicable to him/herself. The scale asks the subject
to answer in the context of how he/she has been feeling "in the past week, including today," (Beck et al., 1961). This phrase is valuable for the purposes of this study because it focuses the depressed subject on the current episode of depression and does not include past history. This scale is one of the most frequently used in clinical research on depression and is a reliable and valid measure shown to be adequate as a measure of depression in populations similar to those included in this study (Beck, Gram & Dein, 1976; Hughes, O'Hara & Rehm, 1982; Prusoff, Klerman & Paykel, 1972). The BDI measures the degree of the depressive symptomatology, and as Prusoff et al. (1972) pointed out rates the presence or absence of symptoms and thus is valuable in rating recovery over time.

Neuropsychological Screening Battery

Typically, the primary function of a neuropsychological screening is to enable clinicians to differentiate between psychiatric and organic patients. The neuropsychological screening battery used in this study is comprised of twelve popular standardized instruments. The twelve independent subtests assess a wide range of functions, including receptive and expressive language, fine-motor functioning, visual-spatial ability, psychomotor speed, immediate and short-term semantic and figural memory, and general cognitive efficiency. Wysocki & Sweet (1981) demonstrated the usefulness of this battery in differentiating psychiatric, normal, and brain-damaged patients. The screening battery consists of: (1) the Spatial Relations Test (Halstead & Wepman, 1949), (2) the Stroop Color-Word test (Golden, 1978; Stroop, 1935), (3) the Luria-Ne-
The screening battery subtests include a wide range of tasks which allow assessment of a diversity of neuropsychological functions. 1) The Spatial Relations test is a part of the Aphasia Screening test and asks the subject to copy a Greek cross. The scoring is based on the quality of the design the subject is able to produce. 2) The Stroop Color-Word test is composed of three sheets of paper with columns of words which the subject must read as quickly as possible. The Word sheet involves simply reading the words; red, blue and green. The Color sheet contains columns of x's in the above colors. The subject names the colors as quickly as possible. The third sheet combines the words on the first sheet with the colors on the second, i.e. the word "red" would be printed in the color green. The subject must ignore the word and name the colors as quickly as possible. The scores on the three tasks involve the number of items the subject completes in 45 seconds. 3) The Luria-Nebraska Pathognomonic items include a wide range of tactual, verbal, writing, drawing, and calculational skills drawn from the Screening Battery subtests. In describing the Luria-Nebraska Pathognomonic Items, Lewis et al. (1979) stated that, "poor performance on these items is highly correlated with brain dysfunction, but is rarely found in patients with other problems such as psychiatric disorders." 4) The Wechsler Memory Scale subtests include the Visual Reproduction (Figural
memory) and Logical Memory for Stories subtest measures. The subject recalls three designs and two short stories, immediately after presentation of each, and then after a period of approximately 30 minutes delay.

5) The Trail-Making test involves measures A and B. Trails A requires connecting numbers on a sheet sequentially from one to twenty-five, while Trails B requires alternating between sequential numbers and letters. 6) The Finger Tapping Test measures the average number of taps the subject completes in ten seconds for each hand. 7) The Digit Symbol subtest is taken from the Wechsler Adult Intelligence Scale (Wechsler, 1955), and involves coding the appropriate symbol under each number as quickly as possible. The code is displayed in a key at the top of the page, and the score is based on the number completed in ninety seconds.

Procedure

Subjects for the treatment group were selected as soon as possible after admission to the inpatient psychiatric service of Illinois Masonic. All treatment group subjects had a clinical diagnosis of depression. Comparison group subjects were selected from the pain treatment clinic outpatient population, and from volunteer hospital employees. All comparison group subjects were screened out if significant depression was evident upon initial testing. Prior to any subject's participation in the study, he/she was given a consent form which stated that he/she voluntarily agreed to participate in the testing sessions, and agreed to release relevant medical history information to the examiner. The testing was administered by two advanced graduate students in clinical psychology who have received thorough training in the neu-
ropsychological and depression measures. All phases of the testing and scoring of these measures were supervised by Dr. Jerry Sweet, Illinois Masonic staff psychologist. For all subtests (except Spatial Relations) the inter-rater correlation of test scores ranged from .97 to 1.00, while the percentage of agreement for Spatial Relations scores (with a range from 1 to 3) was 71%. This reliability coefficient was derived from observation and scoring of test performance of a subsample of subjects, from the depressed and nondepressed groups, simultaneously by both testers.

Hughes et al. (1982) commented that the inclusion of two or more classes of depression assessment allows the most accurate evaluation of the patient's condition. The inclusion criteria for treatment group depressed subjects was based upon three assessments of the patient's psychiatric disorder in the present study. These included the admitting psychiatrist's evaluation and diagnosis made upon admission, the DSM-III criteria reviewed in a structured clinical interview, and an objective self-report measure (the Beck Depression Inventory; Beck, et al., 1961). Patients admitted with an appropriate psychiatric diagnosis of depression by the admitting psychiatrist were asked to participate in the study. They received a structured clinical interview, based upon the criteria cited in the Diagnostic and Statistical Manual (3rd ed., American Psychiatric Association, 1980). On the basis of the interview data and clinical records, those patients who satisfied the requirements for a diagnosis of: Major Depression (296.00), Dysthymic Disorder (300.40) or Atypical Depression (296.82) were included in the study after their
medical charts were reviewed to check factors cited as exclusion criteria for this study.

All volunteers participated in two testing sessions of approximately one hour each. A brief staff feedback sheet summarizing the testing results was made available to the attending psychiatrist of the participating patient. Subjects who were unwilling or unable to participate fully in the initial testing were eliminated from the final experimental groups. Each session consisted of administration of the neuropsychological screening battery and depression inventory (BDI). Inpatients also participated in a brief structured diagnostic interview. The first testing session was administered as soon as possible after admission to the inpatient ward, or for comparison group subjects, upon agreement to participate in the study. The second testing, for psychiatric inpatients, was immediately prior to, or as soon as possible after discharge. For the depressed group, the average period of time from pre- to post-test was 15.5 days (range=6 to 41). Similarly, comparison group subjects were retested at an average of 19.7 days after the pre-test (range=11 to 42 days).
CHAPTER III

RESULTS

Pretest Differences: Depressed and Nondepressed

It was predicted that depressed subjects would be significantly more impaired than the nondepressed group on neuropsychological test measures prior to treatment of the depression. One-way ANOVAs comparing depressed and nondepressed performance on each pre-test subtest yielded significant differences between the groups on ten of the thirteen measures. The means for these data are presented in Table 1. As can be seen, the depressed group was significantly more impaired than the nondepressed on the following ten subtests: the Wechsler Memory Scale Immediate, $F(1,28)=6.85$, $p=.01$, and half-hour delayed Figural memory, $F(1,28)=8.04$, $p=.008$, the Trail-Making A, $F(1,28)=18.35$, $p=.0002$, and Trails B tests, $F(1,28)=6.3$, $p=.02$, Digit Symbol, $F(1,28)=17.56$, $p=.0003$, the Stroop Color page, $F(1,28)=21.72$, $p=.0001$, Word page, $F(1,28)=12.14$, $p=.002$, and Color-Word page, $F(1,28)=13.69$, $p=.001$, the Luria Pathognomonic, $F(1,28)=17.18$, $p=.0003$, and the Beck Depression Inventory, $F(1,28)=116.18$, $p<.0001$.

Additionally, it was predicted that depressed subjects would be more frequently classified as impaired on pre-test neuropsychological measures compared to the nondepressed group.
### TABLE 1

Mean and Standard Deviations of Depressed and Nondepressed Groups: Pre- and Post-Subtest Scores

<table>
<thead>
<tr>
<th>Test</th>
<th>Depressed Pre</th>
<th>Depressed Post</th>
<th>Nondepressed Pre</th>
<th>Nondepressed Post</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean (S.D.)</td>
<td>Mean (S.D.)</td>
<td>Mean (S.D.)</td>
<td>Mean (S.D.)</td>
</tr>
<tr>
<td>Tapping</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dom.</td>
<td>44.2 (12.1)</td>
<td>47.3 (10.3)</td>
<td>49.4 (8.6)</td>
<td>49.6 (6.0)</td>
</tr>
<tr>
<td>Non-Dom.</td>
<td>40.1 (11.6)</td>
<td>42.8 (8.7)</td>
<td>41.2 (7.3)</td>
<td>44.0 (6.5)</td>
</tr>
<tr>
<td>WMS</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Imm.Sem.</td>
<td>16.3 (6.6)</td>
<td>23.4 (5.8)</td>
<td>18.0 (6.1)</td>
<td>23.6 (7.0)</td>
</tr>
<tr>
<td>Del.Sem.</td>
<td>12.4 (5.7)</td>
<td>19.8 (5.9)</td>
<td>15.1 (6.2)</td>
<td>22.4 (7.6)</td>
</tr>
<tr>
<td>Imm.Fig.</td>
<td>7.1 (2.9)</td>
<td>8.9 (2.6)</td>
<td>9.8 (2.7)</td>
<td>10.4 (2.3)</td>
</tr>
<tr>
<td>Del.Fig.</td>
<td>5.7 (2.9)</td>
<td>8.4 (3.3)</td>
<td>8.6 (2.6)</td>
<td>10.4 (2.7)</td>
</tr>
<tr>
<td>Trails A *</td>
<td>36.9 (8.9)</td>
<td>28.9 (8.2)</td>
<td>22.7 (9.2)</td>
<td>21.3 (6.2)</td>
</tr>
<tr>
<td>B *</td>
<td>90.2 (46.9)</td>
<td>66.9 (25.9)</td>
<td>55.3 (26.4)</td>
<td>56.6 (25.4)</td>
</tr>
<tr>
<td>Dig.Sym.</td>
<td>50.3 (11.5)</td>
<td>55.9 (9.0)</td>
<td>70.4 (14.6)</td>
<td>73.0 (15.2)</td>
</tr>
<tr>
<td>Stroop W</td>
<td>93.6 (20.4)</td>
<td>97.9 (22.6)</td>
<td>116.7 (14.7)</td>
<td>117.8 (18.6)</td>
</tr>
<tr>
<td>C</td>
<td>64.1 (9.2)</td>
<td>68.8 (13.6)</td>
<td>84.2 (8.5)</td>
<td>87.4 (8.0)</td>
</tr>
<tr>
<td>CW</td>
<td>35.5 (9.8)</td>
<td>40.9 (10.3)</td>
<td>48.4 (8.9)</td>
<td>52.2 (8.2)</td>
</tr>
<tr>
<td>Pathog. *</td>
<td>12.9 (4.5)</td>
<td>9.3 (3.5)</td>
<td>6.7 (3.7)</td>
<td>5.6 (3.8)</td>
</tr>
<tr>
<td>Sp.Rel. *</td>
<td>1.2 (0.4)</td>
<td>1.1 (0.5)</td>
<td>1.3 (0.5)</td>
<td>1.1 (0.3)</td>
</tr>
<tr>
<td>BDI *</td>
<td>26.2 (8.2)</td>
<td>9.9 (2.1)</td>
<td>2.3 (2.6)</td>
<td>1.7 (2.5)</td>
</tr>
</tbody>
</table>

*Dom. = Dominant Hand
*Non-Dom. = Non-dominant Hand
*WMS = Wechsler Memory Scale
*Imm.Sem. = Immediate Memory for Stories
*Del.Sem. = Delayed Memory for Stories
*Imm.Fig. = Immediate Figural Memory
*Del.Fig. = Delayed Figural Memory
*Dig.Sym. = Digit Symbol
*Stroop W = Word Page
*C = Color Page
*CW = Color-Word Page
*Pathog. = Luria Pathognomonic Scale
*Sp.Rel. = Spatial Relations Test
*BDI = Beck Depression Inventory

*Higher Score = Impairment

NOTE: Due to color blindness, n for control Stroop data = 14.
Tables 2 and 3 show the pre- and post-test Chi-square analyses of the percentage of impaired and non-impaired subjects within each group. The cut-offs used to classify subject's test performance as impaired or non-impaired are commonly used clinically as brain-damage indicators. Specifically, the cut-off criteria for impairment of subtest performance were: Trails A, 40 and above, Trails B, 92 and above (Halstead, 1947); Digit Symbol, scale scores of 6 or below (Wechsler, 1981); Pathognomonic items, the critical cut-offs corrected for age and education were used (Golden, Moses, Graber & Berg, 1981); Tapping-Dominant, females 45 and below, males 49 and below, and Tapping-Nondominant, females 39 and below, males 43 and below (Russell, Neuringer & Goldstein, 1970); Wechsler Memory Scale- all subtests with a Russell rating of 2 or lower (Russell, 1975); Stroop- all pages with a t-score of 35 or lower (Golden, 1978); Spatial Relations, a rating of 2 or below (Russell et al., 1970).

For all subtests, a greater frequency of depressed subjects scored within the impaired range. An average of 48% of the depressed group scored within the impaired range on the pre-test measures, compared to 25% of the nondepressed group. On the post-test, an average of 25% of the depressed group's performance tested within the impaired range compared to 16% of the nondepressed group. However, the corrected Chi-Square yielded a significant difference between groups on the Wechsler Memory scale half-hour Figural Memory pre-test scores only, \( \chi^2 = 9.2, p = .01 \).
### TABLE 2

Pre-Test - Percentage and Frequencies of Subjects Impaired by Group
(Frequencies in Parentheses)

<table>
<thead>
<tr>
<th></th>
<th>Depressed</th>
<th>Nondepressed</th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>%Impaired</td>
<td>%Non-impaired</td>
<td>%Impaired</td>
<td>%Non-impaired</td>
<td>X²</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tapping</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dom.</td>
<td>47 (7)</td>
<td>53 (8)</td>
<td>20 (3)</td>
<td>80 (12)</td>
<td>1.35</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non-Dom.</td>
<td>53 (8)</td>
<td>47 (7)</td>
<td>40 (6)</td>
<td>60 (9)</td>
<td>0.13</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>WMS</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Imm.Sem.</td>
<td>87 (13)</td>
<td>13 (2)</td>
<td>80 (12)</td>
<td>20 (3)</td>
<td>F</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Del.Sem.</td>
<td>87 (13)</td>
<td>13 (2)</td>
<td>80 (12)</td>
<td>20 (3)</td>
<td>F</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Imm.Fig.</td>
<td>80 (12)</td>
<td>20 (3)</td>
<td>40 (6)</td>
<td>60 (9)</td>
<td>3.47</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Del.Fig.</td>
<td>93 (14)</td>
<td>7 (1)</td>
<td>33 (5)</td>
<td>67 (10)</td>
<td>9.19*</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Trails A</td>
<td>40 (6)</td>
<td>60 (9)</td>
<td>7 (1)</td>
<td>93 (14)</td>
<td>F</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>B</td>
<td>33 (5)</td>
<td>67 (10)</td>
<td>13 (2)</td>
<td>87 (13)</td>
<td>F</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dig.Sym.</td>
<td>33 (5)</td>
<td>67 (10)</td>
<td>7 (1)</td>
<td>93 (14)</td>
<td>F</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stroop W</td>
<td>27 (4)</td>
<td>73 (11)</td>
<td>0 (0)</td>
<td>100 (14)</td>
<td>F</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stroop C</td>
<td>27 (4)</td>
<td>73 (11)</td>
<td>0 (0)</td>
<td>100 (14)</td>
<td>F</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CW</td>
<td>33 (5)</td>
<td>67 (10)</td>
<td>7 (1)</td>
<td>93 (13)</td>
<td>F</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pathog.</td>
<td>13 (2)</td>
<td>87 (13)</td>
<td>0 (0)</td>
<td>100 (15)</td>
<td>0.53</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sp.Rel.</td>
<td>20 (3)</td>
<td>80 (12)</td>
<td>27 (4)</td>
<td>73 (11)</td>
<td>F</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Dom. = Dominant Hand
Non-Dom. = Non-dominant Hand
WMS = Wechsler Memory Scale
Imm.Sem. = Immediate Memory for Stories
Del.Sem. = Delayed Memory for Stories
Imm.Fig. = Immediate Figural Memory
Del.Fig. = Delayed Figural Memory
Dig.Sym. = Digit Symbol
Stroop W = Word Page
Stroop C = Color Page
CW = Color-Word Page
Pathog. = Luria Pathognomonic Scale
Sp.Rel. = Spatial Relations Test

* p = 0.01
* F = Fisher's exact test
df = 1
NOTE: Due to color blindness, n for control Stroop data = 14.
TABLE 3

Post-Test - Percentage and Frequencies of Subjects Impaired by Group
(Frequencies in Parentheses)

<table>
<thead>
<tr>
<th></th>
<th>Depressed</th>
<th>Nondepressed</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>%Impaired</td>
<td>%Non-impaired</td>
<td>%Impaired</td>
</tr>
<tr>
<td><strong>Tapping</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dom.</td>
<td>47 (7)</td>
<td>53 (8)</td>
<td>20 (3)</td>
</tr>
<tr>
<td>Non-Dom.</td>
<td>33 (5)</td>
<td>67 (10)</td>
<td>33 (5)</td>
</tr>
<tr>
<td><strong>WMS</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Imm.Sem.</td>
<td>47 (7)</td>
<td>53 (8)</td>
<td>47 (7)</td>
</tr>
<tr>
<td>Del.Sem.</td>
<td>53 (8)</td>
<td>47 (7)</td>
<td>47 (7)</td>
</tr>
<tr>
<td>Imm.Fig.</td>
<td>40 (6)</td>
<td>60 (9)</td>
<td>20 (3)</td>
</tr>
<tr>
<td>Del.Fig.</td>
<td>40 (6)</td>
<td>60 (9)</td>
<td>20 (3)</td>
</tr>
<tr>
<td>Trails A</td>
<td>13 (2)</td>
<td>87 (13)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>B</td>
<td>20 (3)</td>
<td>80 (12)</td>
<td>20 (3)</td>
</tr>
<tr>
<td>Dig.Sym.</td>
<td>7 (1)</td>
<td>93 (14)</td>
<td>7 (1)</td>
</tr>
<tr>
<td>Stroop W</td>
<td>7 (1)</td>
<td>93 (14)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Stroop C</td>
<td>13 (2)</td>
<td>87 (13)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>CW</td>
<td>20 (3)</td>
<td>80 (12)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Pathog.</td>
<td>0 (0)</td>
<td>100 (15)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>Sp.Rel.</td>
<td>13 (2)</td>
<td>87 (13)</td>
<td>7 (1)</td>
</tr>
</tbody>
</table>

Dom. = Dominant Hand  
Non-Dom. = Non-dominant Hand  
WMS = Wechsler Memory Scale  
F = Fisher's exact test  
Imm.Sem. = Immediate Memory for Stories  
Del.Sem. = Delayed Memory for Stories  
Imm.Fig. = Immediate Figural Memory  
Del.Fig. = Delayed Figural Memory  
NOTE: Due to color blindness, n for control Stroop data = 14.
In sum, both the analyses of variance and the clinical impairment cut-offs provide support for the hypothesis that depression is associated with impaired performance on neuropsychological tests compared to nondepressed subjects.

Pre- to Post-test Improvement: Depressed vs. Nondepressed

It was hypothesized that the neuropsychological test performance of the depressed group would improve as improvement in depression occurred. Before examining neuropsychological test performance changes, it was first necessary to document that level of depression improved over time. Thus, a 2(group) by 2(pre-post) analysis of variance was conducted on the BDI scores. The main Group effect revealed that the depressed group, ($M=18.1, SD=8.2$) was significantly more depressed than the comparison group, ($M=2.0, SD=2.5$), $F(1,28)=63.30, p<.0001$, while the pre- to post main effect indicated that overall depression significantly improved, ($M(pre)=14.23, SD=13.5$, $M(post)=5.8, SD=7.3$), $F(1,28)=81.79, p<.0001$. Further, the significant repeated-measures interaction indicates that the depressed group showed significantly more improvement over time ($M(pre)=26.2$, $M(post)=9.9$), compared to the nondepressed group ($M(pre)=2.3$, $M(post)=1.7$), $F(1,28)=71.73, p<.0001$, (see Table 1).

To examine changes in neuropsychological test performance, 2(group) by 2(pre-post testing) analyses of variance were conducted on all subtest scores. The means from these analyses are presented in Table 1. The repeated-measures ANOVA revealed significant main effects
between groups for several measures: the Stroop-Color page, 
\(M(Depr)=67.6, \ SD=9.8, \ M(N-Depr)=86.3, \ SD=6.4\), \(F(1,28)=20.5, \ p=.0001, \)
Stroop-Word page, \(M(Depr)=95.7, \ SD=15.6, \ M(N-Depr)=117.3, \ SD=7.7\), \(F(1,28)=9.95, \ p=.004, \)
and Stroop Color-Word page, \(M(Depr)=38.2, \ SD=6.9, \ M(N-Depr)=50.3, \ SD=6.8\), \(F(1,28)=13.92, \ p=.001, \)
the Immediate Figural Memory, \(M(Depr)=8.0, \ SD=2.6, \ M(N-Depr)=10.4, \ SD=2.5\), \(F(1,28)=7.78, \ p=.009, \)
and Delayed Figural Memory, \(M(Depr)=7.1, \ SD=2.9, \ M(N-Depr)=9.5, \ SD=3.3\), \(F(1,28)=6.44, \ p=.02, \)
the Digit Symbol scale, \(M(Depr)=53.1, \ SD=7.9, \ M(N-Depr)=71.7, \ SD=4.9\), \(F(1,28)=16.92, \ p=.0003, \)
the Trail-Making A, \(M(Depr)=32.9, \ SD=9.1, \ M(N-Depr)=22.0, \ SD=10.0\), \(F(1,28)=20.08, \ p=.0001, \)
Trail-Making B, \(M(Depr)=78.6, \ SD=40.6, \ M(N-Depr)=56.0, \ SD=26.2\), \(F(1,28)=5.05, \ p=.03, \)
and the Luria Pathognomonic scale, \(M(Depr)=11.1, \ SD=3.3, \ M(N-Depr)=6.2, \ SD=4.1\), \(F(1,28)=15.89, \ p=.0004. \)
These results again reflected the fact that depressed patients were significantly more impaired than nondepressed patients on neuropsychological test measures.

The analyses of variance also revealed main effects for pre- to post-testing on numerous subtests (collapsed across groups): the immediate Figural, \(M(Pre)=8.5, \ M(Post)=9.9, \ SD=2.5\), \(F(1,28)=10.03, p=.004, \)
and half-hour delayed Figural memory, \(M(Pre)=7.2, \ M(Post)=9.4, \ SD=2.4\), \(F(1,28)=25.59, \ p=.0001, \)
the Immediate Semantic, \(M(Pre)=17.1, \ M(Post)=23.6, \ SD=3.1\), \(F(1,28)=124.42, p<.0001, \)
and half-hour delayed Semantic Memory for Stories, \(M(Pre)=13.7, \ M(Post)=21.1, \ SD=3.7\), \(F(1,28)=115.09, \ p=.0001, \)
Digit Symbol, \(M(Pre)=60.3, \ M(Post)=64.4,
Non-dominant Finger Tapping, (M(Pre)=40.6, M(Post)=43.4, SD=5.5), F(1,28)=7.55, p=.01, Trails A, (M(Pre)=29.8, M(Post)=25.1, SD=10.1), F(1,28)=7.33, p=.01, the Stroop Color-Word subtest, (M(Pre)=41.7, M(Post)=46.4, SD=6.8), F(1,28)=13.39, p=.001, and the Luria Pathognomonic, (M(Pre)=9.8, M(Post)=7.5, SD=3.8), F(1,28)=12.34, p=.001. These main effects revealed a general tendency for all subjects to improve in test performance from the pre- to the post-test.

Of critical importance to the hypotheses of the present study were the interaction effects. It was hypothesized that depressed subject's test performance would improve over time with improvement of their depression, while the nondepressed group's improvement over time would be less significant and attributable to a practice effect. An examination of the group means (see Table 1) reveals that the depressed group improves in pre- to post-test performance consistently over all the subtests, while the nondepressed group's performance remains relatively constant on some subtests, improving on a smaller proportion of subtests from pre- to post. Several subtests show a notable degree of improvement from pre-to post- for the depressed groups, with a concomitant smaller improvement for the nondepressed group. However this interaction approached significance for only three of the subtests: the Trails B, F(1,28)=3.88, p=.058, the Luria Pathognomonic scale, F(1,28)=3.35, p=.08, and Trails A, F(1,28)=3.49, p=.07. In all cases, t-tests were used to investigate these statistical trends, and results showed that
the depressed group's test performance significantly improved from the pre- to the post-tests on: Trails A, \( M(\text{Pre})=36.9, M(\text{Post})=28.9, SD=9.1 \), \( t(14)=3.42, p=.004 \), Trails B, \( M(\text{Pre})=90.2, M(\text{Post})=66.9, SD=40.6 \), \( t(14)=2.22, p=.04 \), and Luria Pathognomonic, \( M(\text{Pre})=12.9, M(\text{Post})=9.3, SD=3.2 \), \( t(14)=4.2, p=.001 \). The nondepressed group did not show significant improvement over time on the t-tests for these measures.

Correlations of Depression and Test Performance

As a further test of the relationship between depression and neuropsychological test performance, three sets of correlational analyses were conducted: BDI pre-test with neuropsychological pre-test performance, BDI post-test with neuropsychological post-test performance, and changes in BDI score from the pre- to the post-test with change in neuropsychological subtest performance scores from the pre- to the post-test.

The Beck Depression Inventory (BDI) was significantly correlated with twelve subtests (see Table 4), suggesting a relationship between degree of depression and impairment on neuropsychological testing. The post-test BDI significantly correlated with three post-test measures, compared to the correlation of the pre-test BDI with nine pre-test measures. The pre-test BDI was significantly correlated (df=29) with: pre-test measures of the Wechsler Memory immediate Figural, \( r=-.34, p=.03 \), and half-hour delayed Figural Memory, \( r=-.40, p=.01 \), the Trails A, \( r=.53, p=.001 \), and Trails B, \( r=.38, p=.02 \), the Luria Pathogno-
monic, $r=.47$, $p=.004$, Digit Symbol, $r=-.56$, $p=.001$, and Stroop Word page, $r=-.53$, $p=.001$, Color page, $r=-.63$, $p=.0001$, and Color-Word page, $r=-.49$, $p=.004$, measures. The post-test BDI was significantly correlated with: post-test measures of the Wechsler Memory delayed Memory for Stories, $r=-.36$, $p=.02$, Stroop-Color page, $r=-.42$, $p=.02$, and Luria Pathognomonic scale, $r=.44$, $p=.006$.

This suggests that the greater degree of depression present in the pre-test measurements was more highly related to test performance than the milder depression remaining for overall post-test measures. The possibility that a mild degree of depression continues to be related to test performance is suggested by the post-test significant correlations with the BDI.

To investigate the relationship between change in depression rating and change in level of neuropsychological test performance, the amount of change in BDI score from the pre- to the post-test was correlated with the amount of change from pre- to post-test for all subtests (df=29). The amount of change in the BDI measure over time was correlated with with change in Trails A performance, $r=.36$, $p=.02$. The correlation of the changes approached significance for the Wechsler delayed Figural Memory, $r=-.27$, $p=.07$, Trails B, $r=.28$, $p=.06$, Digit Symbol, $r=-.29$, $p=.06$, and Stroop Color-Word page, $r=-.24$, $p=.10$. The correlational analyses support the evidence throughout the results that depression is related to neuropsychological test performance, although the nature of this relationship requires further investigation.
TABLE 4
Correlation of Depression and Test Performance

<table>
<thead>
<tr>
<th>Test</th>
<th>Pre-test BDI</th>
<th>Post-Test BDI</th>
<th>Change in BDI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tapping</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dom.</td>
<td>-0.14</td>
<td>-0.13</td>
<td>-0.16</td>
</tr>
<tr>
<td>Non-Dom.</td>
<td>-0.0009</td>
<td>-0.16</td>
<td>-0.04</td>
</tr>
<tr>
<td>WMS</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Imm.Sem.</td>
<td>-0.08</td>
<td>-0.22</td>
<td>-0.07</td>
</tr>
<tr>
<td>Del.Sem.</td>
<td>-0.14</td>
<td>-0.36*</td>
<td>-0.05</td>
</tr>
<tr>
<td>Imm.Fig.</td>
<td>-0.35*</td>
<td>-0.10</td>
<td>-0.02</td>
</tr>
<tr>
<td>Del.Fig.</td>
<td>-0.41**</td>
<td>-0.16</td>
<td>-0.27</td>
</tr>
<tr>
<td>Trails A</td>
<td>-0.53***</td>
<td>0.04</td>
<td>0.37*</td>
</tr>
<tr>
<td>B</td>
<td>0.38**</td>
<td>0.02</td>
<td>0.28</td>
</tr>
<tr>
<td>Dig.Sym.</td>
<td>-0.56</td>
<td>-0.29</td>
<td>-0.29*</td>
</tr>
<tr>
<td>Stroop W</td>
<td>-0.53</td>
<td>-0.23</td>
<td>-0.19</td>
</tr>
<tr>
<td>Stroop C</td>
<td>-0.63***</td>
<td>-0.42**</td>
<td>-0.06</td>
</tr>
<tr>
<td>CW</td>
<td>-0.49**</td>
<td>-0.18</td>
<td>-0.24</td>
</tr>
<tr>
<td>Pathog.</td>
<td>0.48**</td>
<td>0.45**</td>
<td>0.15</td>
</tr>
<tr>
<td>Sp.Rel.</td>
<td>0.002</td>
<td>0.12</td>
<td>-0.02</td>
</tr>
</tbody>
</table>

Dom. = Dominant Hand
Non-Dom. = Non-dominant Hand
WMS = Wechsler Memory Scale
Imm.Sem. = Immediate Memory for Stories
Del.Sem. = Delayed Memory for Stories
Imm.Fig. = Immediate Figural Memory
Del.Fig. = Delayed Figural Memory
Dig.Sym. = Digit Symbol
Stroop W = Word Page
C = Color Page
CW = Color-Word Page
Pathog. = Luria Pathognomonic Scale
Sp.Rel. = Spatial Relations Test
BDI = Beck Depression Inventory

NOTE: Due to color blindness, n for control Stroop data = 14.
CHAPTER IV

DISCUSSION

In order to assess the relationship of depression to neuropsychological impairment, a depressed and nondepressed group of subjects were given two administrations of a neuropsychological battery and measure of depression. Differences between groups in test performance were examined at each testing session and across testings.

Pre-test Differences between Groups

It was expected that depressed patients would be significantly more impaired than the comparison group on neuropsychological measures. This was supported by the number of significant pre-test differences between the two groups for ten out of fourteen measures. When the pre-test means are examined, it is clear that the depressed group performs at a consistently more impaired level than the comparison group. This finding supports the hypothesis that depression influences test performance as observed in past literature (e.g. Carpenter, 1983; Kronfol et al., 1978; Miller, 1975; Sweet, 1983; Weingartner & Silberman, 1982.) If one considers the experimental groups to differ primarily by depression (with age, education, sex, and handedness being equivalent) then the relationship of depression to deficits on neuropsychological tests appears quite significant. Specific areas of impairment or clearly lateralized deficits are not suggested by the observed results, although an
Overall impairment in level of neuropsychological test performance is evident. The depressed patient's exhibited significant deficits in figural memory, suggesting non-dominant hemisphere impairment. However, the majority of neuropsychological measures which were impaired for the depressed group are considered to be general measures of intellectual efficiency and overall level of neuropsychological functioning involving both hemispheres.

Several authors suggested (e.g. Albert, 1981; Perkins, 1974; Malloy et al., 1982) that a critical clinical issue involves accurate differential diagnosis of brain-based impairment in psychiatric patients. From the observations in the literature, a significant percentage of depressed patients' neuropsychological test scores were expected to fall in the brain-damaged range. While this observation received support in the present study with the observation that an average of 48% depressed patients tested in the brain-damaged range in the pre-test, this percentage did not significantly differ from the nondepressed group, with the exception of one pre-test measure. Similarly, the percentage of depressed subjects within the impaired range decreased on the post-test measures. However, it is difficult to explain the fact that an average of 25% of the nondepressed subjects also tested in the impaired range on pre-test measures, and 15% on the post-test. Although there were no comparison group subjects included who appeared to be depressed or had received depressed BDI scores, the volunteers from the Chronic Pain and Headache Treatment Center may have been susceptible to mediating variables such as distraction and self attention in much the same way as the
depressed patients. This is one alternative to consider in interpreting impaired neuropsychological scores within both groups.

It was hypothesized that depressed patients would fall past common clinical cut-offs for brain damage during the time they are quite depressed, but that this occurrence would be less common when the same patients were less depressed. In these analyses, fewer depressed subjects tested within the impaired range on the post-test on thirteen out of fourteen measures. As several authors have suggested (e.g. Boyar, 1981; Sweet, 1983), the clinical utility of repeated testing in facilitating differential diagnosis of emotional versus brain-based impairment is supported by these observations of improvement.

**Differential Change in Performance Across Groups**

The overall performance of the depressed group (pre- and post-) in comparison with the nondepressed group is also congruent with the hypothesis that depression influences neuropsychological test performance. The depressed group was consistently more impaired than the nondepressed on the majority of neuropsychological measures, and on several, did not reach the level of the nondepressed group even on the post-test measure. The failure of the depressed group to evidence greater improvement can be attributed to several factors. Several patients tested at discharge showed significant improvement in their depression, but remained mildly depressed. Several patients had a history of dysthymic disorder, presenting with a fluctuating, rather chronic pattern of depression with a range of severity. The design of this study dictated that patient's were to be tested as close to the date of discharge as possible, a
restriction that precluded testing the patients when they appeared more fully recovered from their psychological disorder. Additionally, the presence of an underlying substrate of "cortical vulnerability" has been suggested by Lewis et al. (1983), and appears to be a possible explanation for the overall mild impairment in test performance evident in several depressed group post-test scores. Similarly, Heaton et al.'s (1978) suggestion of an underlying base rate of brain-based impairment in psychiatric populations receives some degree of support from the findings in this study. The depressed group may differ from the nondepressed group on personality, motivational and emotional factors (e.g. anxiety) which may also affect test performance. This presents a number of possible alternatives to explain the evidence of impairment on post-test measures for the depressed group compared to the nondepressed group.

The relative lack of significant interactions between groups across pre- to post-test measures can be attributed to several factors. The mild impairment evidenced in the depressed group's post-test scores may have weakened the relationship expected in the hypotheses. Further, on several measures both groups exhibited some degree of improvement due most probably to a practice effect or to generally decreased test anxiety in the second testing session. The interactions that were close to significance were in the direction expected, which was confirmed by the post-hoc t-tests investigating these interactions. Since this testing was voluntarily participated in by all subjects, the range of depression represented in the depressed group may not be adequately representative of the type of depressed patient referred for neuropsycholo-
gical assessment. A selection factor may be present due to the fact that several patients who appeared severely depressed would not participate. However, this underscores the observations of those patients who did agree to participate and appeared consistently impaired throughout the testing.

Correlations of Depression Rating and Test Performance

While a number of studies have used depression rating scales to assess or confirm depression as associated with neuropsychological test performance (e.g. Kronfol et al., 1978; Sternberg, & Jarvik, 1976; Miller, 1975), the degree of direct relationship between these measures is seldom commented on. However, Miller (1975) concluded that the Beck Depression Inventory was not significantly correlated with depressive symptomatology. In contrast to Miller's (1975) conclusion, the BDI rating did correlate significantly with level of performance on several neuropsychological subtests. The fact that the strongest correlation between depression score and test performance can be noted on the pre-test suggests that the neuropsychological tasks are most affected by a severe degree of depression (which in most cases was not present upon post-testing). It was expected that the neuropsychological scores would improve as the depression improved. The correlations found at the pre-test support the suggestion in the literature that the degree of impairment is directly related to the severity of impairment (e.g. Miller, 1975; Weingartner, & Silberman, 1982; D'Elia, & Perris, 1973). Although a number of studies have observed cognitive deficits present with a mild degree of depression, there may be a certain point in which the depres-
sion becomes severe enough to significantly affect test performance. On the other hand, as suggested by Sweet (1983), there may be other moderating variables that correlate and co-vary with depression (e.g. self-focus, self-awareness and attention). The tenuous support offered by the correlation of change in depression with change in subtest score is surprising, and challenges the assumption that a direct linear relationship can be inferred.

**Summary and Conclusions**

The nature of the impairment exhibited by the depressed group appears to be broad and diverse in nature, with little evidence of a particular lateralization of dysfunction. The memory impairment noted in several studies (Sternberg, 1976; Miller, 1975) received some support, with impaired figural memory suggesting non-dominant hemisphere involvement, while the significant impairment noted on measures such as the Stroop Color-Word pages, Digit Symbol, Trail-Making A & B, and the Pathognomonic items indicate a general loss of cognitive efficiency in both right and left hemisphere functions. Clinically, the data suggest that depressed patients might be expected to exhibit the greatest degree of improvement upon repeated testing for measures such as: the Stroop Color-Word pages, the Trail-Making tests, Wechsler Memory scale measures, and overall score on the Pathognomonic items. The Beck Depression Inventory (and similar self-report measures of depression) appear to provide useful assessment of degree of depression as associated with neuropsychological impairment.

There do not appear to be indicators of a specific pattern of def-
icit, nor of clear clinical signs characteristic of depression-related cognitive impairment. The results consistently support the need to seriously assess and consider the influence of depression in using neuropsychological data for differential diagnosis. Perhaps future directions for research will include more specific qualitative delineators of characteristically depressed test performance. Similarly, longitudinal assessment of depression and neuropsychological performance which provides data across a number of testings with varying degrees of depression may be helpful. Additional analyses of the possibility of physical and psychological moderator variables which co-vary with depression may more clearly define and account for neuropsychological test data.

Finally, the recommendation of repeated testing for psychiatric groups appears to be a necessity for accurate clinical diagnosis, in light of the observed inconsistencies of emotionally-based deficits. Heaton and Heaton's (1981) suggestions for obtaining optimal results from difficult or impaired patients is particularly useful for depressed patients. The qualitative nature of their performance, as well as the influence of certain personality variables needs to be considered in interpreting quantitative neuropsychological data. The intermingling of emotional and cognitive functioning observed within this issue remind the clinician who relies on quantitative assessment that the individual must be considered in all his complexity, and that we must remain mindful of the holistic nature of all aspects of human behavior.
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November 22, 1983
Date

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