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UNIVERSITY OF OKLAHOMA

GRADUATE COLLEGE

THE CONTRIBUTION OF DEPRESSION AND ANXIETY TO POOR ATTENTION PERFORMANCE ON NEUROPSYCHOLOGICAL ASSESSMENT MEASURES

A Dissertation

SUBMITTED TO THE GRADUATE FACULTY

in partial fulfillment of the requirements for the

degree of

Doctor of Philosophy

By

SID DICKSON
Norman, Oklahoma
2000

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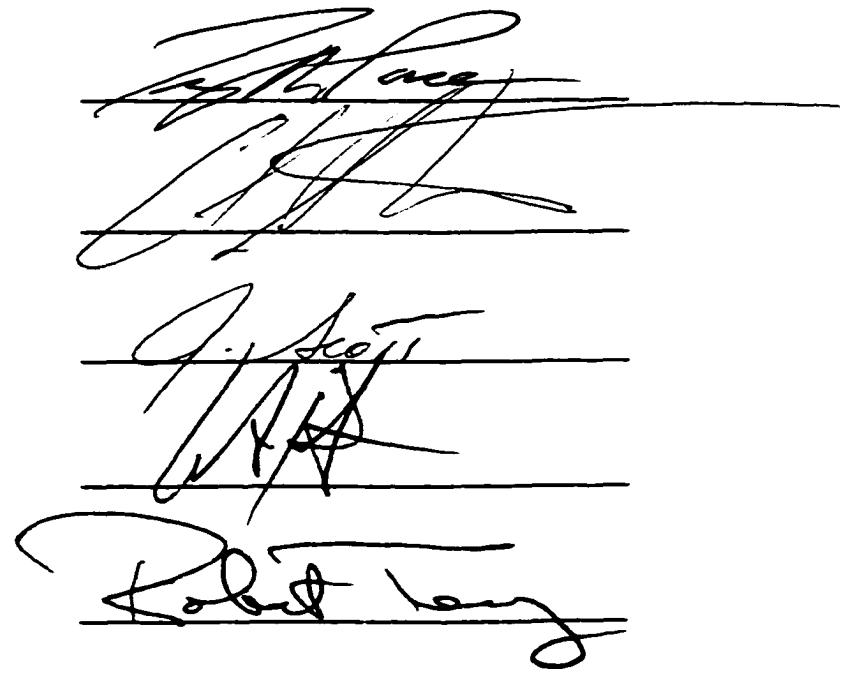
A Dissertation APPROVED FOR THE
DEPARTMENT OF EDUCATIONAL PSYCHOLOGY

BY



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ABSTRACT

Depression and anxiety are encountered on a frequent basis and their effect on cognitive processes can have significant implications for psychotherapy and neuropsychological evaluations.

Empirical studies of their specific effects on neuropsychological test performance are a relatively recent trend and results have been mixed due to various methodological problems (Sweet, Newman, & Bell, 1992) and their significance with respect to cognition has been questioned (Reitan & Wolfson, 1997).

The present study explored the effect of differing levels of anxiety and depression (on MMPI Scales 2 and 7) on attention performance (neuropsychological tests of attention) in 1209 cases gathered retrospectively. Referral sources included neurology, tumor clinic, epilepsy clinic, attorneys, and other allied professions.

Results supported several theoretical models of cognition that predict adverse effects of depression on attention ability, however the amount of variance contributed to attention performance by depression was small (5%). Results also supported predictions of Processing Efficiency Theory (Eysenck & Calvo, 1992) and the Yerkes-Dodson Law that anxiety can improve performance on attention tests.

The present study represented a unique contribution to this area due to its large sample size, use of standardized measures, and diverse patient populations.

The Contribution of Depression and Anxiety to Poor Attention Performance on Neuropsychological Assessment Measures

Depression and anxiety are ubiquitous in the patient populations with which neuropsychologists and clinical and counseling psychologists work (Sweet, Newman, & Bell, 1992). Recognition of the impact of emotional distress on cognitive processes, such as attention, is important for the work in which psychologists engage, particularly for assessment and psychotherapy. Decreased attentional skills due to emotional distress can have implications for psychotherapy by limiting the patient's ability to attend to, and follow, therapist questions and comments. Patient inefficiency in encoding important information relevant to therapeutic change may also hinder compliance and recall of assigned homework and treatment strategies. Within the field of neuropsychology, it is especially important to understand the impact of emotional distress on attentional processes. The ability to distinguish between disordered attention secondary to emotional distress and disordered attention due to organic etiology is of crucial importance. Understanding the extent to which emotional distress contributes to disordered attention can be critical to proper interpretation of neuropsychological assessment results, specifically deficits in attention and recall.

Interest in the effects of emotional distress and psychiatric disturbance on neuropsychological test performance is a relatively recent trend. Sweet et al. (1992) reported that, of 94 studies published on the effects of emotional distress on neuropsychological performance between 1960 and 1975, only 5 examined affective

disorders, and of the 14 published between 1975 and 1978, only 2 were concerned with depressive disorders. However, between 1978 and 1992 over 40 were published, with the majority of those having been published from 1986 to 1992. One reason for the lack of earlier empirical study in this area was a variety of methodological problems observed by Miller (1975). He noted a paucity of standardized measures across the studies at that time that limited efforts to compare findings. He also pointed out the inadequate diagnostic specificity and the use of differing diagnostic criteria prevalent during that time period. Since then, there has been an increased use of standardized neuropsychological tests and batteries and more rigorous diagnostic criteria through the development of the DSM-III-R (American Psychiatric Association, 1987) and subsequent DSM-IV (American Psychiatric Association, 1994; Sweet et al., 1992).

While increased focus on the effects of psychiatric disturbance on neuropsychological test performance is noteworthy, the empirical investigations of those effects have not been without potential confounds. According to Sweet et al. (1992), potential confounding, or moderating, variables in the investigations of neuropsychological performance primarily involve motivation, malingering, and medical factors (Sweet et al., 1992).

Richards and Ruff (1989) tested the hypothesis that reduced motivation accounts for cognitive deficits in depressed patients by randomly assigning two groups of subjects, depressed and nondepressed, to either a motivation or non-motivation condition. Motivation manipulation involved encouragement, a monetary incentive, and performance feedback. Results showed that motivation was indeed lower for depressed subjects, however it did not significantly affect neuropsychological performance. The authors

concluded that, although depressed patients may be less motivated, reduced motivation may not fully account for observed cognitive deficits in depressed patients.

Another potential confounding variable in empirically evaluating the impact of emotional disturbance on neuropsychological performance is malingering, or the "deliberate and conscious feigning of symptoms or the gross exaggeration of symptoms for the purpose of attaining monetary or other external rewards" (Sweet et al., 1992). Thought to be relatively rare, malingering can pose significant diagnostic and assessment difficulties by artificially generating increased mood symptom endorsement and inaccurate and false cognitive profiles. As such, they may artificially skew investigative findings.

Confounding medical factors mentioned by Sweet et al. (1992) include the pharmacological treatment of depression, anxiety, and other psychological conditions. Medications used in the treatment of these conditions have been shown in some studies to impair neuropsychological functioning, however results have generally been mixed. For example, some studies found improved cognitive performance following (primarily) pharmacological treatment of depression (Brumback & Staton, 1980; Fromm & Schopflocher, 1984). Others either observed no changes (Curran, Shine, & Lader, 1986; Telford & Worrall, 1978) or observed only perceptual motor slowing (Squire, Judd, Janowsky, & Huey, 1980). Commonly prescribed medications that were found to exert an adverse neuropsychological effect, at least in certain individuals, included anxiolytics, antidepressants, antipsychotics, anticonvulsants, and even antihypertensives (Dodrill, 1988; Hartman, 1988; Solomon et al., 1983; Trimble, 1987). Particularly troublesome was lithium's adverse affect on memory which, according to Jamison and Asiskal (1983), accounted for the most frequently reported side-effect leading to lithium noncompliance.

Shaw, Stokes, Mann, & Manevitz (1987) reported that over 80% of their subjects receiving lithium complained of neuropsychological side-effects. While some medications may cloud neuropsychological interpretation, many psychiatric and brain-injured patients usually perform better on neuropsychological measures due to a decrease in depression's negative influence on cognitive functioning (Glenn & Joseph, 1987).

Does Emotional Distress Have a Detrimental Effect on Cognition?

While the prevailing opinion in neuropsychology appears to be that emotional disturbances such as depression and anxiety do have an adverse affect on neuropsychological performance (Sweet et al., 1992), there are those who contend otherwise. In a lengthy review of the interaction of emotional disturbance with neuropsychological deficits, Reitan and Wolfson (1997) question this widely held assumption. They acknowledge, as most do, that intellectual and cognitive impairment represents a stressful situation that can cause emotional difficulties and problems of adjustment. They also agree that brain-injured individuals, if not experiencing impaired self-awareness (Prigatano & Schacter, 1991), experience a significant reduction in ability from his or her previous level of functioning which may cause anxiety, feelings of inadequacy in terms of meeting normal responsibilities, and feelings of depression due to failure and inadequate performance (Reitan & Wolfson, 1997). However, they do not readily accept the prevalent belief that emotional distress has a significant effect on neuropsychological functioning, and they point to studies that disagree with that contention. For example, Alvarez (1962) compared Trail Making Test (TMT; Reitan, 1979) performance in depressed versus brain-injured patients. He utilized 32 persons with

unequivocal brain damage (variety of conditions) and 32 patients with severe depression (33% had attempted suicide). He believed that depressed participants might be limited in their performances because of psychomotor slowness and a diminution of the effort needed to perform well. The TMT was selected because it requires "focused attention, selective responses to appropriate stimuli, and a deliberate effort to complete the task as quickly as possible" (Alvarez, 1962). Comparisons of the two groups on the MMPI (Butcher, Dahlstrom, Graham, Tellegen, & Kaemmer, 1989) indicated that the depressed patients had statistically significant elevations over the brain-injured group on Depression, Hysteria, Psychasthenia, Paranoia, Schizophrenia, and the F scale. Results on the TMT showed performance by the brain-injured group to be significantly poorer than the depressed group ($p<.001$) on Parts A and B. Moreover, performance of the depressed group was found to be "similar to that reported by Reitan for his non-brain-injured control groups" (Alvarez, 1962). The implication here by Reitan and Wolfson was that since depressed individuals performed better than brain injured individuals (i.e., similar to controls) they were not impaired by depression as measured by the TMT.

In another study cited by Reitan and Wolfson, Vingerhoets, DeSoete, and Jannes (1995) investigated the relationships between measures of emotional status and cognitive test performances in patients who were awaiting open-heart surgery and in the same patients following surgery. The researchers described an impending open-heart surgery as "one of the most frightening medical procedures", and they felt it provided a "natural stress paradigm" to evaluate the "impact of emotional state on neuropsychological test performance" (Vingerhoets, DeSoete, & Jannes, 1995). Measures of stress included anxiety, using the A-State scale of the State-Trait Anxiety Inventory (Spielberger,

Gorsuch, & Lushene, 1970), and depression, using the Beck Depression Inventory (Beck, 1987). Measures of neuropsychological performance included 11 tests "selected to cover an extensive range of cognitive functions." Both sets of tests were administered to 130 patients before surgery and 109 of the same patients 7-8 days after surgery. Not surprisingly, the results showed significant elevations for anxiety and depression before surgery. Following the surgery, anxiety and depression were lowered but only by a significant degree for anxiety. Correlational analyses were run between measures of emotional status and neuropsychological performance and the results showed no significant relationship for either pre- or post-surgical testing. According to Reitan and Wolfson (1997), results such as these suggest that "neuropsychological abilities are quite robust, even under conditions of rather striking personal stress and anxiety." The implication being that even high levels of stress and anxiety have limited-to-no impact on neuropsychological functioning.

What about clinical depression at levels requiring hospitalization? Kaufman, Grossman, and Kaufman (1994) used the Kaufman Short Neuropsychological Assessment Procedure (KSNAP) to compare cognitive performances of 56 hospitalized inpatients with clinical depression to normal matched controls. They analyzed results according to tests grouped in terms of the three functional units proposed by Luria: (1) attention-orientation representing a low level of cognitive complexity; (2) successive and simultaneous processes, representing an intermediate level of cognitive complexity; and (3) high-level planning ability, representing a high level of cognitive complexity. Results showed no significant differences at any task level, suggesting that clinical depression at levels

requiring hospitalization had no adverse effect on those cognitive functions measured by Kaufman and his colleagues.

Perhaps one of the more enduring assumptions in neuropsychology is the adverse effect of depression on the cognitive functions of the elderly, the so-called "pseudodementia" syndrome. As Reitan and Wolfson (1997) observe, a review of the literature by Bieliauskas (1993), concluded that "depressive-like symptoms have little or no impact on cognitive functions." He further asserted that, "the case for emotional influence on cognitive abilities in the elderly (i.e., pseudodementia) is vastly overrated", and "if elderly patients do present with cognitive difficulties, (they) are more likely disease-based rather than the result of emotional factors such as depression." Bieliauskas and colleagues conducted studies of their own in which nursing home patients and elderly medical outpatients were studied using various measures of cognition (Bieliauskas, Costello, & Terpenning, 1991; Bieliauskas & Lamberty, 1991; Bieliauskas, Lamberty, & Boczar, 1991). In all three studies, they found no significant effects of depression on cognitive abilities for their elderly samples. Although no significant effects of depression were observed in their samples, Bieliauskas allowed that there may have been an adverse influence on cognitive symptoms in patients with a psychiatric history of primary depression coupled with sufficient loss of self-esteem.

Reitan and Wolfson (1997) reviewed findings from studies of MMPI profiles in brain-injured individuals as well as individuals with emotional disorders. The MMPI is considered the instrument "used most frequently to assess the emotional status of patients referred for neuropsychological examinations" (Mittenberg, Tremont, & Rayls, 1996). As Reitan and Wolfson note, it possesses several advantages in the evaluation of personality

and emotional characteristics: it is self-administered and relatively easy to score; has empirically validated scales that have established meanings; is an objectively interpreted instrument; provides clear, valid descriptions of psychological problems, symptoms, and characteristics in a broadly acceptable clinical language; has clinical interpretation strategies that are easily learned; and possesses scales that have high reliability. Reitan and Wolfson reviewed studies of MMPI profiles in an effort to find support for the position that "significant emotional disturbances impact adversely on both neuropsychological test performances and on functional outcome" (Kay, 1993). In one study they reviewed, Gass (1991) analyzed a group of 105 patients referred for neuropsychological evaluation whose neurological examination did not identify any evidence of brain damage. The referrals were from a VA hospital consisting of psychiatric, neurologic, and rehabilitation services. Findings revealed "relatively weak relationships between MMPI indices and Halstead-Reitan Battery (HRB) scores." As a result, Gass concluded that, "as a general rule, these widely-used neuropsychological measures are largely resilient to the effects of emotional and personality factors in patients referred for neuropsychological testing." He further cautioned that a "conservative stance is recommended in attributing poor performance on these tests to psychological factors, particularly as inferred from elevated MMPI scores." Gass argued that HRB tests seem to be generally robust in the presence of psychopathology, and he asserted that "traditional interpretive lore that surrounds the use of various MMPI scores and patterns to make inferences with regard to cognitive functioning may be inaccurate."

In another study, Gass and Daniel (1990) evaluated the effect of emotional factors on Trail-Making Test – Part B performances. They concluded that performance on the

Trail Making Test was resistant to a variety of emotional influences and, though psychiatric symptoms and severe anxiety impairs performances, it is rarely to the extent caused by brain damage.

In a study investigating the relationships between MMPI scores of 59 psychiatric patients and measures of attention, concentration, and memory derived from the Wechsler Memory Scale (WMS), Gass, Burda, Starkey, and Dominguez (1992) found uniformly low correlations between MMPI variables and memory performance. They concluded patients' subjective complaints are unreliable indicators of actual ability. Taken together, it appears that Reitan and Wolfson (1997) have provided compelling evidence that emotional distress does not cause neuropsychological impairment equal to that seen in brain injured individuals. Nonetheless, their evidence also indicates that emotional distress can affect neuropsychological performance, at least at levels somewhere on a continuum between the performance of normal controls and that of brain-injured participants.

Supportive Evidence for an Adverse Effect of Depression on Cognition

Veiel (1997) performed a meta-analysis of "all studies published since 1975 and meeting stringent methodological and sample selection criteria" to assemble a profile of neuropsychological deficits of clinically depressed (major depression) but otherwise unimpaired individuals. Veiel's findings supported a profile "consistent with a global-diffuse impairment of brain functions with particular involvement of the frontal lobes." In fact, the severity of cognitive deficiencies he profiled were observed to be similar to those seen "in moderately severe traumatic brain injury" (Veiel, 1997).

Veiel's (1997) meta-analysis narrowed a large pool of research down to 13 studies using 6 stringent screening criteria, which he noted sacrificed research breadth for methodological stringency. The results of the analysis were grouped in the following nine categories: Attention/Concentration; Verbal Fluency; Scanning and Visuo-Motor Tracking; Verbal Learning-Acquisition; Verbal Learning-Retention/Retrieval; Nonverbal Learning-Acquisition; Nonverbal Learning-Retention/Retrieval; Visuo-Spatial Functions; and Mental Flexibility-Control.

Results for Attention/Concentration from the resulting 13 studies generally included only Digit Span Forward (only a few included Digit Span Backward of the Wechsler scales; Wechsler, 1981, 1987). Results showed only a 0.18 standard deviation between depressed and non-depressed controls. In the category of Verbal Fluency, tests included only the Controlled Oral Word Association Test (FAS test; Benton & Hamsher, 1989) and results showed a 0.55 standard deviation. Scanning and Visuo-Motor Tracking included Trail Making Test Part A and the WAIS-R Digit Symbol subtest (Wechsler, 1981) and results revealed almost a full standard deviation between groups at 0.93. Visuo-Spatial Functions included the following tests: Rey Complex Figure Test (Rey, 1964) and the Block Design and Object Assembly subtests of the WAIS-R (Wechlser, 1981). Results indicated a 0.81 standard deviation between groups in this category. Verbal Learning-Acquisition included immediate recall from many of the standard memory tests and results indicated a 0.90 standard deviation. Verbal Learning-Retention/Retrieval included delayed recall of at least several minutes, and findings showed a 0.91 standard deviation. Nonverbal Learning-Acquisition and Nonverbal Learning-Retention/Retrieval had standard deviations of 0.93 and 0.83, respectively, and included visually presented

material from recurring figures (Williams, Iacono, Remick, & Greenwood, 1990) and Rey's Complex Figure (Rey, 1964). The final category revealed the most striking distinction. Mental Flexibility and Control included measures commonly regarded as very sensitive to most kinds of brain dysfunction, and especially to frontal lobe dysfunction (Veiel, 1997): Trail Making Test Part B (time) and the Color-Word score of the Stroop Test. Results for this category was 2 full standard deviations between groups. To summarize, the cognitive functions observed to be most affected by depression (i.e., above 0.50 standard deviation) were, in order: Mental Flexibility and Control, Scanning and Visuo-Motor Tracking, Nonverbal Learning-Acquisition, Verbal Learning-Retention/Retrieval, Verbal Learning-Acquisition, Nonverbal Learning-Retention/Retrieval, Visuo-Spatial Functions, and Verbal Fluency. As Veiel (1997) observed, the obtained profile of cognitive deficiencies appeared "at first glance" to match that which would raise the question of impaired frontal lobe functions.

Sweet, Newman and Bell (1992) observed a similar profile in their review. They noted a pattern of "decreased cognitive efficiency or mild attentional or mild memory problems", typically evidenced by the following patterns: slowed information processing (e.g., slowness on all Stroop Color-Word pages), impaired word recall with normal recognition, impaired incidental learning with normal intentional learning, and impaired recall of easy word pairs (often with normal recall of difficult word pairs).

A number of studies have demonstrated a negative effect of depression on motor tasks. In a study by Raskin, Friedman, and DiMascio (1982; in Grant & Adams, 1986), 277 depressed patients were matched on age, sex, and education with 112 normal controls in a multicenter research project. Findings showed that depressed subjects performed

poorly on a number of motor performance tasks, including tapping, aiming, and circle tracing. Impairments were also found on nonsense syllable learning, Stroop scores, and the Clock Reversal Test. In another study, Cohen et al. (1982) examined motor performance (i.e., grip strength) and cognitive function (i.e., various mental tasks such as working with "trigrams") in depressed patients by severity level (severely depressed, moderately depressed, euthymic and normal mood). Results demonstrated deficits in motor and cognitive performance of depressed patients that appeared to be proportionate to depression severity.

Taken together, findings tend to consistently show that "cortically mediated intellectual functions are spared" (Grant & Adams, 1986), such as repetition, reading, naming, mathematics, and motor praxis. However, deficits that are more prominent tend to be those dependent on arousal, attention, and concentration. Grant and Adams (1986) observed that depressed patients suffer deficits in attention on tasks requiring "effort." Although some investigators conclude that depressed patients have "motivational disorders", Grant and Adams argued there is more to it than "motivation." For example, they ask how can one conclude that poor vigilance or grip strength can be simply due to lack of "motivation"? Ultimately, they concluded that depressed patients are simply "less with it" than unimpaired controls, suggesting a deficit in information processing. In profiling memory deficits of depressed individuals, they argued that the depressive state of the individual impedes the reception of new information as well as its initial processing. This ineffective initial acquisition appears to be central to later failures in recall. However, once information is encoded it appears that depressed patients tend to retain it. Retrieval deficits are common as well, especially for spontaneous recall, due in part to the poor

initial processing, however performance tends to improve on less stringent recall testing (e.g., recognition memory; Grant & Adams, 1986).

In summary, depressive states tend to exert adverse effects on cognition in the form of global and diffuse impairment of brain functions with particular involvement of the frontal lobes, noted in at least one study to reach levels seen in moderately severe traumatic brain injury. Adverse effects that have been noted include impairment in mental flexibility; scanning and visuomotor tracking; and both verbal and nonverbal acquisition, retention, and retrieval. Deficits have also been noted on measures of cognitive efficiency, attentional performance, information processing, and incidental learning. Cognitive functions that are typically spared tend to be those cortically-mediated intellectual functions such as repetition, reading, naming, and mathematics.

Supportive Evidence for an Adverse Effect of Anxiety on Cognition

There is evidence to suggest that anxiety, as a form of emotional distress, exerts an adverse effect on cognition. Anxiety, as with many psychological states, exists on a continuum of severity from simple worry and rumination to chronic and severe levels such as that observed in Post-traumatic Stress Disorder (PTSD; APA, 1994). The research presented below highlights studies that have focused on varying levels of anxiety and stress.

Pruzinsky and Borkovec (1990) recruited 56 college students comprising two groups, worriers and nonworriers. Subjects engaged in either brief relaxing imagery or stressful imagery. Before and after the imagery tasks, measures of focused attention and anagram measures were obtained. The results revealed that worriers reported more

negative daydreaming, greater difficulty with attentional control and greater obsessional symptoms. They also evidenced significantly more negatively affect-laden cognitive intrusions during relaxed wakefulness and focused attention.

In another study utilizing college students, Gillis (1993) investigated the hypothesis that stress impairs judgment (among other hypotheses which were not supported). He had 98 undergraduates complete a complex multiple-cue judgment task. Subjects were then assessed for (1) their exposure to two potential sources of stress, life events and irrational thinking and (2) the amount of personal dysphoria they were experiencing. Measures included the Life Experiences Survey, Dysfunctional Attitude Scale, State-Trait Anxiety Inventory, and Beck Depression Inventory. Results indicated that subjective distress, depression and state anxiety were significantly related to poor judgmental performance. In addition, results suggested that potential external sources of stress do not negatively affect judgment unless they generate subjective distress at the time those judgments are made.

High and low state anxiety was studied in a sample of community-dwelling elderly volunteers by Rankin, Gilner, Gfeller, and Katz (1994). Participants were administered the State-Trait Anxiety Inventory for Children, Mini-Mental Status Examination, and subtests of the Wechsler Memory Scale—Revised. Results indicated that anxiety (low and high) adversely affected sustained attention, but the findings were not significant for verbal and figural memory tasks.

If cognitive processes are affected by anxiety states at the lower end of the continuum, what about more intense pathological conditions of anxiety? One would reasonably infer that there is an inverse relationship where anxiety severity increases as cognitive performance decreases. However, "cognitive performance" covers a lot of

neuropsychological ground. It is possible that an inverse relationship exists but only for certain cognitive domains as was observed in a number of the studies on depressive effects on cognition. For example, in attentional processes, information processing, and encoding. Vasterling, Brailey, Constans, and Sutker (1998) investigated attention and memory performances in Persian Gulf War veterans with and without PTSD diagnoses. Veterans who were diagnosed with PTSD exhibited relative deficiencies in performance on tasks of sustained attention, mental manipulation (mental arithmetic), initial information acquisition, and retroactive memory interference. They also committed more errors of commission and intrusion. Veterans' tendencies toward response disinhibition and intrusion on cognitive tasks was positively correlated with re-experiencing symptoms and negatively correlated with avoidance-numbing symptoms. Vasterling and colleagues observed that the veterans' pattern of cognitive deficits were consistent with models of PTSD that emphasize the role of hyperarousal and involvement of frontal-subcortical systems. Moreover, their data suggested that intrusion of traumatic memories in PTSD might not be limited to trauma-related cognitions but instead reflect a more generalized pattern of disinhibition.

Individuals suffering from Obsessive Compulsive Disorder (OCD; APA, 1994) are also at risk for adverse cognitive effects due to anxiety. In one recent study by Clayton, Richards, and Edwards (1999) individuals diagnosed with OCD were studied along with a panic disordered and control group. Results showed significantly poorer performance on a series of psychometric tasks of selective attention. The researchers concluded that the data supported the hypothesis that OCD individuals have a diminished ability to selectively

ignore competing external (sensory) and internal (cognitive) stimuli, especially intrusive thoughts.

Further support was found by Schmidtke, Schorb, Winkelmann and Hohagen (1998). They investigated "frontal lobe performance" in 29 unmedicated OCD patients who were matched on age, gender, and intelligence with a double-size control group of normals. Participants were administered 12 neuropsychological tests, most of which are thought to be sensitive to different aspects of frontal lobe functioning. Results indicated that OCD patients were *unimpaired* on tests of abstraction, problem-solving, set-shifting, response inhibition, and reaction speed, however they evidenced deficits of approximately one standard deviation on timed tests of verbal and nonverbal fluency and attentional processing. Schmidtke and colleagues theorized that the obtained neuropsychological profile is related to "dysfunctioning within the anterior cingulate, but not the dorsolateral prefrontal circuit." Similar results were obtained by Veale, Sahakian, Owen, and Marks (1996) who found impairment on an attentional shifting task in 40 matched OCD patients.

Finally in another study of anxiety and cognition, Channon, Flynn, and Robertson (1992) compared 18 adults with Tourette syndrome with 22 controls. Participants were assessed on a number of clinical and experimental measures of attention and self-report measures of mood, anxiety, and obsessionality. Results revealed that the Tourette group was more depressed, anxious, and obsessional, and they performed worse on complex measures of attention, including serial addition, block sequence span (forward), trail-making, and a letter cancellation task.

In summary, anxiety states tend to exert adverse effects on cognition in the form of impairment in attentional control, sustained and selective attention performance, poor

judgment, attentional shifting, and initial information acquisition. Deficits have also been noted in mental arithmetic and verbal and nonverbal fluency. Evidence of overall disinhibition is characteristic as well, in the form of cognitive intrusions, retroactive interference and errors of commission and intrusions. Cognitive functions that were noted to be spared (in OCD patients) were abstraction, problem-solving, set-shifting, response inhibition, and reaction speed.

Cognitive Processes in Depression

To attempt to better understand obtained neuropsychological profiles, cognitive theorists have developed models of cognitive processing. One of the more compelling approaches is that articulated by Ingram (1984), the information processing model (or information processing "approach"). In the information processing model, network theory is utilized to conceptualize a process called "spreading activation." According to this theory, memory is composed of cognitive networks of associated concepts and descriptive propositions. Previous information and events that have been encoded into memory are represented by these propositions, and each memory unit is composed of a cluster of components (concepts and propositions) making up the memory. These clusters are referred to as memory "nodes." Network theory proposes that, in order for a memory or cognition to reach an individual's conscious awareness, its corresponding node must be activated above some minimum threshold. Once activation reaches a sufficient level, the person consciously experiences the memory.

Theoretically, there are at least two ways that a memory may be activated sufficiently to reach consciousness. One is through the presentation of an environmental stimulus array that corresponds to an active memory node. Ingram presents the example of an individual who has been wanting to buy a Porsche. Although the individual might not be consciously experiencing thoughts about the Porsche, if one of them happens to pass by on the street the person will be "reminded" of the desire to own one. Network theory would argue that this happens because the energy from the Porsche stimulus pattern activates the corresponding "Porsche memory node" to a sufficient level to reach

conscious awareness so the individual is reminded of, and begins to think about, the Porsche.

A second way in which memory nodes may be activated is through the spread of activation. Network theory assumes that memories are connected with each other through associative linkages. Theoretically, memories that are similar conceptually, or have somehow become associated for the individual, are linked through associative pathways. The strength of these pathways is seen as a function of how strongly the memories are associated. Strongly associated memories will have strong and more closely associated linkages, whereas weakly associated memories will have weaker or perhaps no associative pathways. Presumably, when a memory is activated, activation is presumed to spread along its associative pathways causing other memory nodes to become more likely to be activated. Memory nodes which stand the highest chance of being activated in this manner are those that are connected through the strongest associative pathways. It is theorized that this spreading activation of memories may be analogous to the person's stream of consciousness. To return to the Porsche example, not only is the person reminded of the desire to buy one, but a flood of associated cognitions may become conscious as well, such as the inability to buy one on his present salary, the need for a raise or promotion, or a project under way that could lead to a promotion, and so on (Ingram, 1984).

With regard to affect, Ingram proposes that affect can be conceptualized in terms of affective structures called "primitive emotion nodes." Each specific emotion such as depression, anger, joy, or fear is theoretically represented by a particular node or unit in memory. Connected to each emotion node is a set of features associated with the emotion, such as its subjective experience, its unique autonomic response pattern, verbal

labels used to describe the emotion, and cognitions containing descriptions of events that evoke that emotion. When a particular node is activated, the emotion is experienced and activation is channeled through its interconnections to evoke the emotion's other manifestations. Additionally, each emotion node is thought to be associated with a particular cognitive network consisting of emotion-related memories and cognitions. Although some connections to the emotion node are believed to be innate, such as the connection to nodes that trigger autonomic responses, cognitive linkages are largely learned and are generally established through "contiguity" during life events (Ingram, 1984). Ingram presented the example of attending the funeral of a friend. A link is established when the sadness felt at the funeral (depression emotion node activation) becomes associated with a cognitive node representing descriptions of funerals. Thus, through acculturation, learning, and innate programming, emotion nodes are viewed as being linked with particular cognitive networks containing emotion-congruent content.

Components of the information-processing framework that are particularly relevant to the present study of attention and general cognition are the "depth of processing model" and "cognitive capacity." As network theory seeks to describe the structure of memory, depth of processing seeks to describe the process by which information is encoded into memory. At its basic level, the depth of processing model proposes that information is more likely to be fully perceived and encoded when it is processed "deeper," where depth refers to the degree and extent of cognitive analysis the information receives. In other words, the more cognitive analysis a piece of information receives, the more likely it is to be comprehended and understood. Ingram (1984) refers to this cognitive analysis as "cognitive elaboration." That is, information that receives

analysis is elaborated upon cognitively. When received, information is processed at different cognitive depths. These different depths may be viewed as corresponding roughly to different cognitive networks, with larger and more intricate networks being seen as representing deeper and deeper cognitive depths.

The concept of cognitive capacity has sometimes been referred to as the same as attention or consciousness (Lachman, Lachman & Butterfield, 1979). According to the model, individuals have processing limits. They can only process a finite amount of information at any given time, or, stated another way, the amount of attention they can pay is limited. An individual's processing capacity is limited, and when this capacity is exceeded, no more information can be attended to or processed. Not all information utilizes the same proportion of capacity, however. For example, the information required to drive a car may engage very little cognitive capacity if the person has driven a car for a long time (i.e., an overlearned skill), on the other hand it may engage large portions of cognitive capacity if the skill is just being learned. To tie this concept back in with the depth of processing model discussed above, in order for an individual to process information at a deeper level (i.e., increased cognitive elaboration) a relatively larger proportion of cognitive capacity must be utilized. That is, it will take nearly all of their attention.

So, how is depression presumed to affect these cognitive processes? The information-processing model of depression acknowledges that any psychological state is a complex process involving the interaction of a variety of factors, however it views those factors as converging upon a basic mechanism called the "depression emotion node" (Bower, 1981 as cited in Ingram, 1984). It proposes that the phenomenological

experience of depression, along with the onset of depressive symptoms, results from the activation of an individual's depression node (although other emotion nodes may be activated concurrently). This activation of the depression node is determined by the appraisal of life events, referring to the manner in which life events are linked to the contents of existing cognitive structures (e.g., attitudes, beliefs). Appraisal is viewed as the process that gives subjective meaning to external events and is generally thought to be determined by an individual's (1) beliefs about the parameters of a particular life event, and (2) beliefs as to the effects of the event. For example, suppose a woman has been left by her husband for another woman. If she believes that she will never see him again (a parameter of the event) and that she will not be able to function without him (a perceived effect of the event), then it is likely that the event will be cognitively appraised in such a way as to activate her depression node.

A variety of factors are assumed to either shorten or lengthen the duration of depressive episodes, however in the absence of those factors it is believed that the underlying mechanism that determines depression duration is level of activation. Once activated, a depression node is thought to experience a period of decay until the activation level falls below a threshold, at which time the individual no longer experiences depressive affect. The higher the initial activation level, the longer it will take to decay to subthreshold levels. The initial activation level is, in turn, determined by the value that the individual places on the negative event (i.e., how it is appraised).

The activation of a depression node is presumed to be necessary and sufficient to cause depressive affect (Ingram, 1984), however the information processing model postulates a somewhat different set of cognitive processes and mechanisms which

act to maintain the depressive affect. The depression node is viewed as a central part of certain cognitive networks with associated linkages to various other units in the network. Due to the associative nature of the networks, it is thought that other units will be the representations of past events associated with depression. In addition, cognitions that were related to past depressive feelings would also be linked to the network. These particular memory units will in turn be linked to other units with which they have become associated through past experiences, however the strongest associative links will be with the memory units representing the present depressing situation.

The concept of spreading activation maintains that, when the depression node is activated above the threshold, activation spreads through the depression-associated network, causing its various contents to become more likely to be brought to conscious awareness. This presumably may set up a "cognitive loop" process where thoughts, memories, and associations consistent with an individual's mood become more accessible to the individual (Clark & Isen, 1982). Importantly, due to the depression node being relatively central to this particular network, it is thought that as activation cycles through the network, it is eventually fed back (though at a slightly lower level due to signal decay), causing the depression node to remain activated. For the person experiencing this, it is as if negative memories and cognitions keep coming back again and again to consciousness, thus maintaining the depressive feelings.

Important to the present study is the notion of "available cognitive capacity." As activation spreads it is viewed as occupying a proportional amount of the person's available cognitive capacity. With greater magnitudes of activation, there are greater levels of spreading activation and more associations in the network are activated above

consciousness. As those associations are activated above threshold and the person begins to actively think about them, a larger proportion of the limited capacity is engaged. Consequently, persons undergoing a depressive episode will have a high degree of attention focused upon themselves and their cognitions as available capacity becomes increasingly occupied by spreading activation (Ingram, 1984).

Ingram illustrates this process with the following example. Suppose an individual has just lost an important job. It is assumed that this loss will activate the person's depression node, which in turn sends activation spreading throughout its associated network. In other words, in addition to the initial depression that the individual feels, he will think about losing the job and will be more likely to think about past depressive experiences (as depression-associated memory nodes are activated). As more and more associations become activated, related past cognitions that are related to depression may be experienced (e.g., guilt, self-degradation, low self-esteem). Thus, as the individual has depressive cognitions, the depression is maintained as activation is recycled back to the depression node.

When presented with a task or new information, not only is adequate available cognitive capacity necessary to attend to it but the incoming information is addressed according to its similarity to the person's current cognitive contents. The implication of this is that, unless the new task or information is unusually strong (to exceed activation threshold), information that is not particularly relevant to current cognitive content will not be processed (or not fully processed) because it is not associated with the presently active network (i.e., it is not related to the depression-associated network and is more related to networks that are presently inactive).

Evidence for this limited cognitive capacity or "limited resource" hypothesis has found support from studies of memory and aging and mixed groups of bipolar and unipolar depressives (see King, Caine, & Cox, 1993) and other studies of self-focused attention (Ellis, 1991; Ingram, Lumry, Cruet, & Seiber, 1987; Lemelin, Baruch, Vincent, Laplante, Everett, & Vincent, 1996).

Other related and empirically-supported hypotheses posited to explain depressives' poor attentional (and memory) performance are: automatic versus effortful processing and the reduced initiative hypothesis. First articulated by Hasher and Zacks in 1979 (see King, Caine, & Cox, 1993), effortful and automatic processing are distinguished by processes that are either intentional and conceptually driven (effortful) or automatic and "data driven" (automatic). These processes are usually apparent in explicit versus implicit memory tasks, where explicit memory tasks are effortful and implicit memory tasks are automatic. Explicit memory tasks are those in which recall and recognition are assessed directly with the person being conscious of the task and requiring conceptual processing (e.g., recalling a word within the context of a list). In contrast, implicit memory tasks do not necessarily involve the person's awareness and are thought to be an unconscious activation of an item (e.g., a word) in the person's lexicon that subsequently makes the item more accessible to consciousness.

Studies of implicit versus explicit memory indicate that depressed individuals are generally more impaired versus controls on explicit (i.e., effortful) memory tasks. For example, in an extensive review of such studies Hartlage, Alloy, Vazquez, and Dykman (1993) concluded the following: (1) Depression interferes with effortful processing, and the degree of interference is determined by the degree of effortfulness of the task, the

severity of depression, and the valence of the stimulus material to be processed; and (2) depression interferes only minimally with automatic processes.

The reduced initiative hypothesis stipulates that depressives' poor attentional performance is due to a tendency to be self-focused and to fail to direct sufficient attentional resources to an external task. For example, Channon, Baker, and Robertson (1993) found that deficits in short-term memory of depressed patients was the result of difficulties in attentional regulation, rather than a "simple defect in storage capacity." In a dramatic demonstration of this process Hertel and Rude (1991) showed that, by experimentally eliciting increased "focused attention", they were able to improve memory scores of depressed patients such that it matched the performance of formerly depressed (i.e., recovered) patients and non-psychiatric controls. Thus, these studies would suggest that attentional resources of depressed patients, given the right conditions, might be shifted "outward" to focus on external tasks.

Another hypothesis investigating the effects of depression on cognition is that of selective attentional, or negative bias. It is hypothesized that depressives have enhanced memory (and possibly attention) for negatively toned material (King, Caine, & Cox, 1993). For example, on memory tasks depressed patients remembered anxiety-provoking information more vividly and "agreeable" information more poorly than did controls (Mialet, Pope, & Yurgelun-Todd, 1996). A study by Pace and Dixon (1993) supports this hypothesis. In a study examining the effects of 6-8 sessions of Beck's cognitive therapy on mildly and moderately depressed college students' depressive symptoms and depressive self-schemata, results demonstrated not only that depressives show a clear preference in

recall for negative self-referent judgments, but that brief cognitive therapy can significantly decrease that negative bias.

In 1989, Schwartz and Garamoni described an information-processing model of positive and negative cognition, the States of Mind (SOM) model. Based on the "golden section proportion", an extensively studied phenomena in personal construct psychology whereby individuals differentiate dichotomous judgments in a ratio of approximately 61.8% to 38.2% (e.g., in a balance of positive (P) and negative (N) adjectives, P/(P+N)=.618), the States of Mind model proposes five distinct states of mind that are conceptualized in terms of cognitive balance and quantitatively defined by homeostatic set point ratios of positive cognitions to total positive plus negative cognitions.

The five states of mind consist of three SOMs (positive dialogue, internal dialogue of conflict, and negative dialogue) that retain a dialectical interaction between positive and negative thoughts and two SOMs (positive monologue and negative monologue) that are imbalanced positively or negatively to the degree that they virtually abandon a dialectical process. Each SOM is defined its ratio of positive to negative cognitions. The most optimal state of mind, positive dialogue, is characterized by a ratio of .618 positive cognitions to .382 negative cognitions. It allows a general positivity in cognition and mood while preserving maximal attentiveness to negative, threatening events. The individual with this SOM will readily recognize negative cognitions and likely engage in sufficient facilitory self-talk and positive coping strategies to alleviate distress.

Negative dialogue is a SOM characterized by a ratio of .382 positive cognitions to .618 negative cognitions. Individuals with this SOM maintain a background of continual, moderate negativity such that, when negative events occur, they are less shocking and

more easily assimilated into existing structures. Such persons are usually observed to be moderately anxious or depressed. The system strives to maintain a "preferred" (or homeostatic) level of dysphoria or fear.

The internal dialogue of conflict SOM is characterized by symmetrically balanced structures such that the ratio of positive to negative cognitions is .50. This results in equal salience of positive and negative information as well as maximal uncertainty, a state that is not optimal as it is associated with indecision and doubt. Clinically, it may manifest as mild levels of anxiety, depression, and obsessionality.

Positive monologue is one of the SOMs characterized by a ratio that is asymmetrical to the degree that a dialectical process is virtually abandoned. It consists of positive cognitions with a ratio of .69 or more. In this state, positive thoughts and feelings exceed the optimal balance. Ratios in these ranges (i.e., that are monologic versus dialogic) are inherently unstable, and there is a tendency to strive toward a more balanced and less extreme dialogic SOM. Clinically, this excess of positive cognitions may manifest as mania or hypomania. With rising positivity, there is reduction in uncertainty at the expense of a loss of salience of negative events. Consequently, important threatening events may go unnoticed to the detriment of the individual.

At the other end of the spectrum is the negative dialogue SOM characterized by positive cognitions at a ratio of .31 or less (and negative cognitions at .69 or more). According to Schwartz and Garamoni (1989), this SOM is not as enduring as the other states of mind (especially the dialogic SOMs) and is more transitory, which may explain the tendency for some severe unipolar depressives to experience spontaneous remission. The negative monologue is usually associated with extremely severe psychopathological

states and is characterized clinically by profound depression or acute panic. It is qualitatively distinct and exhibits "distinct structural and information processing properties. In addition to its hypothesized instability, there are "internal and external pressures" exerted on the individual to move towards more positive thinking - to reestablish a more balanced, dialogic SOM (Schwartz & Garamoni, 1989).

Garamoni et al. (1991) found support for the States of Mind model in a study of 39 outpatients with major depression. Correlational analysis confirmed that the balance of positive and negative affective symptoms (P/(P+N)) in this sample closely approximated the golden section (i.e., 0.37). More recently, Nasby and Russell (1997) investigated whether the States of Mind model could successfully differentiate between Vietnam combat veterans who suffered from posttraumatic stress disorder (PTSD) and Vietnam combat veterans who did not. After matching the groups by combat exposure and controlling for general psychopathology, their results demonstrated more maladaptive SOM in PTSD veterans than combat veterans without PTSD.

Finally, a theoretical model well known to the field of psychology is the cognitive model of depression posited by Aaron Beck (Beck, Rush, Shaw & Emery, 1979). In its simplest form, it consists of three specific concepts which seek to explain the psychological underpinnings of depression: (1) the cognitive triad, (2) schemas, and (3) cognitive errors or faulty information processing.

The cognitive triad is composed of three major patterns of cognition: a negative view of self, world, and future. With regard to the self, the individual develops a view of himself as defective, inadequate, or worthless. His view of the world is distorted, and he may envisage an environment that places exorbitant demands on him, presents unfair

obstacles, and sets him up for failure. The future may be equally bleak. As the depressed individual makes long-range projections, he expects his situation to continue as it has or become worse. Frustration and hardship are expected.

Schemas are theoretical cognitive structures that organize environmental stimuli along unique patterns (unique to the individual). Although different persons may conceptualize the same situation in different ways, a particular individual tends to be consistent in his responses to similar types of events. Relatively stable cognitive patterns form the basis for the regularity of interpretations of a particular set of situations (Beck et al., 1979). Depressed individuals will tend to activate idiosyncratic negative schemas that may not logically relate to the situation at hand. As these idiosyncratic schemas become more active, they are evoked by a wider range of stimuli which are less logically related to them, and they lose increasingly more voluntary control over their thinking processes. With increasing levels of severity, they are unable to invoke other more appropriate schemas. As the depression deepens, cognitive organization may become so independent of external stimulation that the individual is unresponsive to changes in his immediate environment (Beck et al., 1979).

Faulty information processing, or cognitive errors, refer to systematic errors in the thinking of the depressed person. These systematic errors serve to maintain the individual's belief in the validity of his negative concepts despite the presence of contradictory evidence. According to Beck and colleagues (1979), depressed individuals' thinking tends to be "primitive" in nature so that global judgments are made regarding events that impinge on their lives. The meanings that they attribute to external events are

likely to be extreme, negative, absolute, and judgmental. In turn, the emotional response tends to be negative and extreme.

Consistent with previously mentioned models of depressive cognition, the cognitive model of depression proposed by Beck and colleagues (1979) conceptualizes depressive cognition as significantly self-, or internally, focused. In addition, external stimuli tend to be distorted or significantly self-, or internally, focused. In addition, external stimuli tend to be distorted or unattended to, which may lead to attentional deficits, decreased concentration, and inefficient memory encoding/retrieval.

Psychophysiological Measures

In addition to neuropsychological measures of cognitive performance to ascertain deviations in depressives, psychophysiological mechanisms can also be assessed and compared to controls. As a component of their study on focused attention, Hertel and Rude (1991) incorporated a reaction time component to assess participants' mental demands. Reaction time to an auditory signal was assessed and found to be significantly slower in the depressed group. Moreover, reaction time was not affected by increases in attentional focus, though it was in the control group (i.e., increased attentional focus improved reaction time in controls).

Psychophysiological studies which are particularly relevant to attention are those of evoked potentials and contingent negative variation (CNV). Evoked potentials are waves of cerebral electrical activity arising in response to a sensory stimulus (auditory, visual, or somesthetic). These potentials can only be observed by repeating the stimulus, averaging several repetitions, and subtracting the background noise of the

electroencephalogram (EEG). The early components of the evoked potential (i.e., before 100 milliseconds) are usually linked to characteristics of the stimulus, however components which arrive later are related to the attention which the stimulus has aroused. For example, the evoked potential commonly used is the "P300" (positive deflection, 300 ms) component. At the level of the P300, studies of depressed patients unanimously show a reduction in amplitude (Mialet, Pope & Yurgelun-Todd, 1996). Yee and Miller (1994) compared dysthymics to anhedonic and normal controls on P300 evoked potentials and found that dysthymic individuals are hyporesponsive at various stages of information processing. Yet it was unclear whether they were deficient in the amount of available attentional resources for information processing or deficient in the allocation of those resources.

In addition to P300 potentials, recent studies have attempted to utilize changes in the attenuation of the "N2 Vertex wave" in participants during a selective attention task (Mialet et al., 1996). Changes observed are suggestive of a deficit of the "attentional trace" at the initial stage of information processing (El Massioui, 1988). Mialet et al. (1996) interpret these changes as evidence of limited availability of attentional resources in depressed patients, thus constraining them to "mobilize controlled attention for operations that would normally be easily handled by automatic processing."

Contingent negative variation (CNV) consists of a slow negative potential which develops in the frontal regions of the brain during the interval between an alerting stimulus and a response and represents the physiological correlate of anticipation in a motor or mental performance (Mialet et al., 1996). In summarizing the findings of CNV in depressives, Mialet and colleagues report a clear decrease in the amplitude of the CNV

and an association of CNV with relative insensibility of depressives to contextual (environmental) cues.

Additional studies investigating speech (larynghographic recordings), eye movements, and visual tracking in depressives have also revealed impairment, lending additional support for the hypothesis of an overall reduction in depressed patients' attentional performance (Mialet et al., 1996). In fact, Mialet and colleagues compare attention in depressives to that of schizophrenic patients, stating that depressives display an "impoverished intensity" of attention rather than an impaired ability to orient their attention.

Cognitive Processes in Anxiety

As reported earlier, there is ample support for an adverse effect of anxiety states on cognitive performance, however there are studies which have actually reported equal or better performance of anxious individuals over controls on cognitive performance (Dibartolo, Brown, & Barlow, 1997; Eysenck & Calvo, 1992). To attempt to account for the discrepancy in findings, Eysenck and Calvo (1992) developed the processing efficiency theory of anxiety. Processing efficiency theory conceptualizes *state* anxiety as the crucial factor determining individual differences in internal processing and performance. Eysenck and Calvo (1992) assert that state anxiety is determined through an interaction of trait anxiety and situational threat or stress, however they also note that it is often difficult to distinguish between trait and state anxiety empirically because they both correlate very highly (often 0.70 or higher). Thus, many studies do not attempt to disentangle the two forms.

Two components of anxiety, worry and emotionality, are identified in processing efficiency theory. Emotionality refers to an individual's awareness of the physical aspects of anxiety (e.g., increased heart rate, or sweating), whereas the worry component consists of self-preoccupation, concern over evaluation, and concern over level of performance (Eysenck & Calvo, 1992). The worry component is viewed as occupying the cognitive component of state anxiety, and it is hypothesized to play an integral role in the articulatory loop of the working-memory system devised by Baddeley (1986; see Eysenck & Calvo, 1992). Baddeley's working-memory system consists of three components, all having limited capacity: a modality-free central executive resembling attention, which is involved in active processing; an articulatory loop specializing in rote verbal rehearsal used

for temporary storage of verbal information (often involving subvocalizations); and a visual-spatial sketch pad specializing in visual and/or spatial information. The central executive is considered the most important component of the working-memory system, and it is used on tasks that require planning or decision making and as a problem-solver when lower processing systems seem inadequate.

According to Eysenck and Calvo (1992), the worry component of anxiety is not only thought to consume some of an individual's valuable processing and storage resources, but is also thought to serve a motivational function via a "control system". Importantly, the functioning of this control system leads to an allocation of additional processing resources (i.e., effort) and to the initiation of processing activities (e.g., strategies) designed to improve performance, especially when the person perceives their performance as poor (or is given feedback to that effect). If successful, such attempts at allocating additional resources increase available working memory capacity and may, in fact, *improve* performance provided there are not undue demands placed on their overall resources (elaborated further below).

A fundamental theoretical distinction set forth in processing efficiency theory, one which seeks to explain the inconsistent findings in other studies of anxiety and performance, is that between performance effectiveness and processing efficiency. Performance effectiveness simply refers to the quality of *task performance* (as in test scores), whereas processing efficiency refers to the *relationship between* the effectiveness of performance and the effort, or amount of processing resources, invested. This relationship is defined as performance effectiveness divided by effort, though "not in a strict mathematical sense" (Eysenck & Calvo, 1992).

Processing efficiency theory differs from other theories of anxiety and performance in a number of ways. First, worry has motivational as well as attentional interference effects. Second, the motivational effect is generally positive in that it leads to increased effort or compensatory strategies. Third, the notion that the effects of anxiety on performance differ from the effects on performance *efficiency* is unique to this theory. Fourth, anxiety affects both the storage and the processing capacity of the working-memory system which are available for task performance, rather than simply the storage capacity of short-term memory or attentional resources as hypothesized in other theories. Finally, at a conceptual level it is assumed within other theories (see Eysenck & Calvo, 1992) that stressful situations or threats cause anxious individuals to respond relatively passively with self-preoccupation, which either interferes with attention to task-relevant processing or motivates them to avoid the task, thus reducing on-task effort. Performance efficiency theory, on the other hand, assumes that anxious individuals respond to stressful situations *dynamically*. They worry about possible aversive consequences but try to avoid them by allocating further resources to the task, however that strategy can only be useful to a point given the finite resources available at any given time.

To elaborate, when presented with a simple task, anxious and non-anxious individuals are roughly equal in the amount of attentional resources required for the task. However, when task demands increase, the amount of attentional resources required by each begins to diverge. The central executive (of the anxious individual) begins to direct additional resources to the task, and if the individual is aware of poor task performance, he will begin to worry (in the articulatory loop) about adverse consequences of his poor performance and will be motivated to improve that performance. This motivational

function entails the allocation of further resources (i.e., effort) and processing activities (i.e., strategies) in an attempt to improve performance. This compensatory response may lead to improved performance but not always. Objectively, task performance, as measured by test scores, may not reveal any significant differences between anxious and non-anxious controls unless secondary (concurrent) tasks are employed to further tax the capacity of the available resources. It is then that differences are often detected, highlighting the anxious individual's re-distribution of resources in an effort to compensate for poor task performance. Numerous confirmatory studies have been conducted on performance efficiency, utilizing ever-inventive research strategies to uncover this underlying process (Eysenck & Calvo, 1992; Elliman, Green, Rogers & Finch, 1997; Dibartolo et. al, 1997).

Eysenck and Calvo (1992) outline the following predictions of the processing efficiency theory, all of which are supported by empirical studies cited in their monograph: (1) Anxiety typically impairs processing efficiency more than performance effectiveness, and (2) adverse effects of anxiety on task performance generally become stronger as task demands on working memory capacity increase. The implications of the first prediction, also supported empirically, are as follows:

(a) highly anxious participants should report higher levels of subjective effort than low-anxious participants on comparable tasks
(b) anxiety will typically have an adverse effect on a secondary task performed concurrently with a primary task
(c) anxiety will reduce spare processing capacity (assessed by responding to probes) during the performance of a central task
(d) motivational factors enhancing effort typically benefit the performance of low-anxious individuals more than high-anxious individuals
(e) the performance of a central task will be adversely affected by an additional load to a greater extent in anxious than in non-anxious groups

(f) impaired processing efficiency produced by anxiety can be detected by lengthened processing time (e.g., response or reaction time)
(g) the greater impairment effect of anxiety on efficiency than on effectiveness can be detected by psychophysiological measures

The second prediction by Eysenck and Calvo, that adverse effects of anxiety on task performance generally become stronger as task demands on working memory capacity increase, includes the following empirically supported implications:

(a) the effects of anxiety on task performance depend on the amount of resources required by the task (measurable by the susceptibility of that task to interference by a concurrent load)
(b) anxiety reduces temporary storage capacity
(c) anxiety has powerful adverse effects on tasks with high storage and processing demands
(d) anxiety does not generally impair performance on tasks not involving the central executive and/or articulatory loop components of the working-memory system (e.g., undemanding or automatic tasks)

Processing efficiency theory has held up well under empirical scrutiny. As recently as 1997, Elliman and colleagues examined the effect of anxiety on three groups of anxious individuals consisting of low, medium, and high anxiety. Participants performed a high-processing load measure of sustained attention, including accuracy and response scores, and basic measures of psychomotor performance. Results revealed that high levels of anxiety were associated with longer response times and a decline in performance accuracy. Since all participants performed equally well on basic psychomotor speed, the researchers were able to demonstrate that the increase in response times was unique to the high-anxious group. In other words, high-anxious individuals used more processing capacity (and thus more response time) than their lower anxious counterparts. Thus, they were performing less efficiently as they attempted to maintain similar levels of performance.

Finally, in a study by Dibartolo and colleagues (1997), participants with generalized anxiety disorder (GAD) were compared to normal controls on two information processing signal detection tasks. The first task consisted of neutral distractors, and the second consisted of administering negative feedback cues. GAD participants evidenced impaired performance on the first task, however they unexpectedly improved on the second task to match normal controls despite reporting significantly higher levels of worry and negative affectivity. Dibartolo and colleagues interpreted this finding as demonstrating Eysenck and Calvo's (1992) concept of motivational functioning within the articulatory loop and central executive. In other words, they believed GAD participants experienced increased worry about their performance and consequently reallocated additional resources to the second task condition. Thus, improving their scores.

Presumed Anatomical Basis for Attentional Processes

Drawing on earlier work conducted with schizophrenia and other disorders affecting attention, Mirsky (1987) postulated that attention could be subdivided into a number of different elements, including the capacity to focus on or select some aspect of the environment, the ability to sustain or maintain focus for a period of time, the ability to encode or manipulate information held in memory, and the ability to shift adaptively from one aspect of the environment to another. These four elements of attention (focus-execute, sustain, encode, and shift) can be measured by a variety of neuropsychological tests of attention. In a study conducted through the Laboratory of Psychology and Psychopathology of the National Institute of Mental health (see Culbertson & Krull, 1996) a factor analysis was used to analyze a battery of neuropsychological tests thought to be sensitive to various aspects of attention. The analysis yielded four factors which supported the aspects of attention proposed by Mirksy (1987). As reported by Culbertson and Krull (1996), the factor associated with the "focus-execute" component was related to perceptual motor speed and loaded on such tests as Trail Making (Reitan, 1979), a letter cancellation procedure, a measure similar to Coding from the Wechsler scales (Wechsler, 1990), and the Stroop test (C.J. Golden, 1978). The factor associated with the "sustain" element of attention was vigilance and loaded on scores from a Continuous Performance Test (omissions, commission errors, and reaction time; Rosvold, Mirsky, Sarason, Bransome, & Beck, 1956). The "encode" element was best captured by a factor loading on numerical or mnemonic tasks, as on Digit Span and Arithmetic from the Wechsler scales. Finally, the "shift" element of attention was measured best by a factor loading on

flexibility, as measured by scores from the Wisconsin Card Sorting Test (WCST; Heaton, 1981).

Mirsky (1987) integrated the neuropsychological testing data and findings from previous neuroanatomic studies to arrive at descriptions of the neuroanatomic localization of the elements of attention. The following functional specialization was suggested: The "focus-execute" function is likely shared by superior temporal and inferior parietal cortices, as well as by structures that make up the corpus striatum (including the caudate, putamen, and globus pallidus). The motor component of the execute function is felt to be mediated by the inferior parietal and corpus striatal regions of the brain. The "sustain" function of attention is thought to be mediated by rostral structures, including the tectum, mesopontine reticular formation, and reticular and midline thalamic nuclei. The "encoding" function is believed to be accomplished by the hippocampus, which provides an essential mnemonic function required for some aspects of attention. Finally, the ability to "shift" attention from one aspect of the environment to the other is supported by the prefrontal cortex. As Mirsky observed, this attentional system within the brain is very widespread and, therefore, quite vulnerable so that damage or dysfunction in any one of these brain regions can lead to specific deficits in attentional function.

Purpose of Study

Depression and anxiety are encountered on a frequent basis in psychological and neuropsychological patient populations (Sweet, Newman, & Bell, 1992), therefore it is paramount to have an adequate understanding of the impact of these conditions on cognitive processes. As mentioned previously, decreased attention can have significant implications for psychotherapy by limiting the patient's ability to attend to, incorporate, and apply therapeutic principles and insight. For the neuropsychologist, it is crucial to appreciate the role of emotional distress in impaired attention to help clarify its contribution versus CNS injury.

Empirical studies exploring the relationship of emotional distress and neuropsychological test performance are a relatively recent trend and results have been mixed due to various methodological problems (e.g., not using standardized measures, inadequate diagnostic specificity, small sample sizes; Sweet et al., 1992). Furthermore, the very notion of emotional distress exerting a significant impact on attentional processes has been called into question (Reitan & Wolfson, 1997).

The present study seeks to determine the impact of differing levels of anxiety and depression on attentional performance in both patients with neurologic insult and those without. It represents a unique contribution to this area of research due to its large sample size, use of standardized measures, and diverse patient population characterizing a rich array of presenting etiologies within the context of a large academic medical center. Based upon the existing literature reviewed, the following research questions and hypotheses were generated.

Research Questions and Hypotheses

Patients were routinely administered the MMPI as part of a flexible neuropsychological battery, thus MMPI t-scores on scales 2 (Depression) and 7 (Psychasthenia) were used to assess severity levels of anxiety and depression. Analyses were then conducted on the group as a whole, followed by analyses on neurologic (i.e., some evidence of neurologic insult) and non-neurologic (i.e., no evidence of neurologic insult) patients. The following research questions and hypotheses were posed:

1. *Which neuropsychological instruments seemed to best measure attention in this sample?*

What is the factor structure of the purported neuropsychological measures of attention in this sample? Which measures appear to best assess the construct known as attention? What is the relationship, if any, to Mirsky's (1987) attentional model? Which instruments appear to be the most useful to include in subsequent analyses with this sample? It was expected that the factor structure of test scores in this sample would closely match that of Mirsky's factor structure (i.e., the four factors: focus-execute, sustain, encode, and shift). It was also expected that the tests found to load most highly on attention components would be those identified by Mirsky or tests roughly equivalent within the test battery used with this sample.

2. *Do legal cases differ significantly on attention performance from the sample as a whole?*

Does having a legal case pending cause a patient to perform significantly different on neuropsychological measures of attention compared to the rest of the outpatient sample? If indeed this is the case, patients with a legal case pending will be selected from the

sample and analyzed separately. Sweet and colleagues (1992) identify motivational variables and malingering as potential confounding factors in investigations of neuropsychological performance. Of the studies reviewed for this study, it was unclear as to how significant legal status affected attention performance on neuropsychological tests. It is expected that, of those presenting for neuropsychological evaluation due to a legal case pending, motivation levels and blatant attempts at malingering will be sufficiently varied as to not represent a significant confound in this outpatient sample. Thus, it is expected that no significant difference will be found from the overall sample, and it will be possible to include their scores in subsequent analyses.

3. *What is the relationship between depression and attention in the outpatient sample?*

What is the relationship between scale 2 MMPI t-scores and the neuropsychological measures of attention administered to this outpatient sample? The information processing approach (Ingram, 1984) predicts that depression significantly impairs attentional resources. Therefore, it is expected that scale 2 MMPI t-scores will be significantly correlated with attention measures, such that increasing depression results in poorer attention performance.

3b. *What is the relationship between depression severity level (Low and High) and attention in this outpatient sample?*

When scale 2 MMPI t-scores are divided into Low (D > 45 and D < 55) and High (D > 70) Depression, while controlling for anxiety (Pt < 60), what relationship exists with attention measures? It is expected that higher depression levels will result in fewer attentional resources being available for attention-demanding tasks, therefore higher levels should result in significantly poorer attention performance than lower depression levels.

4. *What is the relationship between anxiety and attention in this outpatient sample?*

What is the relationship between scale 7 MMPI t-scores and the neuropsychological measures of attention administered to this outpatient sample? Processing efficiency theory (Eysenck & Calvo, 1992) predicts that anxiety places increased demands on attentional processes such that more effort is required for attention tasks than is normally required. However, it is sometimes difficult to detect this increased demand on attentional resources empirically. Consequently, studies have been mixed in finding attentional deficits due to anxiety. Nonetheless, with this large sample it is expected that scale 7 MMPI t-scores will be significantly correlated with attention measures, such that increasing anxiety results in poorer attention performance.

4b. *What is the relationship between anxiety severity level and attention in this outpatient sample?*

When scale 7 MMPI t-scores are divided into Low (Pt > 45 and D < 55) and High (Pt > 70) Anxiey, while controlling for depression (D < 60), what relationship exists with attention measures? Processing efficiency theory predicts that increasing levels of anxiety place more and more demands on attentional processes. Therefore, it is expected that higher anxiety levels will result in significantly poorer attention performance than lower anxiety levels.

The Yerkes-Dodson Law (Revelle & Loftus, 1992) predicts optimal performance at Moderate levels of anxiety, and poorer performance at low and high anxiety states. The Yerkes-Dodson Law has been supported in numerous empirical studies (Revelle & Loftus, 1992) and is hypothesized to be a function of the central nervous system (CNS). At very low levels of anxiety, the CNS is not sufficiently attentive to environmental cues, thus less

encoding takes place. At high levels of anxiety, the CNS is overly preoccupied with the stressor responsible for the anxiety and internal body functioning to be sufficiently attentive to environmental cues, which also results in less encoding. To assess for the effects of Yerkes-Dodson, Scale 7 will be divided into four groups: No Anxiety, Low Anxiety, Moderate Anxiety, and High Anxiety and attention measures will be compared along those dimensions.

5. *What relationship exists with attention when depression and anxiety severity levels are both High, and both Low?*

When scale 2 and scale 7 MMPI t-scores are both High (D and Pt > 70), what relationship exists with attention measures? Similarly, when scale 2 and scale 7 MMPI t-scores are both Low (D and Pt > 45 and < 55), what relationship exists with attention measures? Vanderploeg, Kizilbash, Curtiss, and Schinka (1998) reported significant (detrimental) effects on memory by depression compounded with anxiety. Moreover, effects were stronger with high levels versus low. Therefore, it is expected that similar effects will be obtained with attention performance.

6. *When the sample is divided into two groups according to neurologic status, is there a significant difference on neuropsychological measures of attention?*

It is expected that, by virtue of their neurological insult, neurologic patients will evidence significantly more impairment on neuropsychological measures of attention.

6b. *Are there significant differences in depression levels for neurologics versus non-neurologics?*

Are there significant differences on scale 2 MMPI t-scores between neurologic and non-neurologic groups? As Reitan and Wolfson (1997) observe, intellectual and cognitive

impairment represents a stressful situation that can cause emotional difficulties with awareness of one's reduction in ability from previous levels of functioning. It is expected that this sample will be no different. Neurologic patients should report higher levels of depression than non-neurologic patients.

6c. *Are there significant differences in anxiety levels for neurologics versus non-neurologics?*

Are there significant differences on scale 7 MMPI t-scores between neurologic and non-neurologic groups? Anxiety and depression are frequently comorbid conditions (Vanderploeg et al., 1998), therefore it is expected that a number of depressed neurologics will likely evidence concomitant anxiety. Consequently, it is expected that neurologic patients will report higher anxiety levels than non-neurologic patients.

6d. *Are depressed neurologics more affected on attention measures than non-depressed neurologics?*

When neurologics are separated by depression level (D > 70), is there a significant difference in performance on measures of attention? It is expected that neurologics will, as a group, evidence impaired attention due their neurologic insult. According to the information processing approach, depression will also impair attention performance. Therefore, it is expected that depression will impair attention over and above that existing due to the underlying neurologic condition.

6e. *Are anxious neurologics more affected on attention measures than non-anxious neurologics?*

When neurologics are separated by anxiety level (Pt > 70), is there a significant difference in performance on measures of attention? In keeping with processing efficiency

theory, it is expected that anxiety will impair attention over and above that existing due to the underlying neurologic condition. However, it is recognized that processing efficiency has stated that attention deficits will not always be revealed due to increased effort by anxious individuals to perform better.

6f. *Are depressed non-neurologics more affected on attention measures than non-depressed non-neurologics?*

When non-neurologics are separated by depression level (D > 70), is there a significant difference in performance on measures of attention? It is expected that depression will impair attention performance as predicted by the information processing approach.

6g. *Are anxious non-neurologics more affected on attention measures than non-anxious non-neurologics?*

When non-neurologics are separated by anxiety level (D > 70), is there a significant difference in performance on measures of attention? It is expected that anxiety will impair performance in non-neurologics, however it is also recognized that anxiety deficits are not always revealed according to processing efficiency theory.

Method

Setting and Participants

The data for the study were gathered retrospectively from the records of patients who had been referred to the Neuropsychological Assessment Laboratory at the Department of Psychiatry and Behavioral Sciences, University of Oklahoma Health Sciences Center (OUHSC) between 1955 and 1997. The Neuropsychological Laboratory at OUHSC receives approximately 300 referrals per year for neuropsychological assessment from neurology, tumor clinic, epilepsy clinic, attorneys, and other allied professions. Assessments usually consisted of a flexible battery of tests (i.e., a core battery of tests given to all patients plus additional tests added by the neuropsychologist based on the patient's presenting problem or etiology). Tests were administered by a Master's level psychometrist, pre-doctoral intern, post-doctoral fellow, or neuropsychologist. Testing time varied according to patient but was generally between 3-8 hours with appropriate breaks to prevent fatigue effects. Participants were individuals referred for neuropsychological evaluation that completed valid MMPI's (Minnesota Multiphasic Personality Inventory; Butcher, Dahlstrom, Graham, Tellegen, & Kaemmer, 1989) and were at least 18 years old. For the years 1963 through 1997, 1,654 completed MMPI's were available, with the majority encompassing the years 1977 through 1997. Use of the MMPI was discontinued in 1992, in favor of the MMPI-2. In the total sample, there were 701 (58%) MMPI's and 508 (42%) MMPI-2's administered.

Of the 2,942 cases in the database, 1,209 (41.1%) participants were selected for analysis. Demographic information for this sample is presented in Table 1 below.

Table 1

Demographic Information for Participants

Variable	Mean	SD
Age	42.01	16.61
Education	13.20	2.81

	Frequencies	Percent
Gender		
Male	673	55.7
Female	536	44.3
Ethnicity		
Caucasian	1079	89.2
African American	76	6.3
Native American	21	1.7
Hispanic	6	0.5
Asian American	5	0.4
Other	3	0.2
Unknown	4	0.3

Measures

The Minnesota Multiphasic Personality Inventory - 2nd Edition (MMPI-2; Butcher, Dahlstrom, Graham, Tellegen, & Kaemmer, 1989), a widely used tool in the assessment of emotional/personality functioning, was utilized for the present study. Basic scales 2 (D; Depression) and 7 (Pt; Psychasthenia) were used as a measure of depression and anxiety, respectively. Participants were eliminated if they had MMPI T-score elevations above 70 on L or K scales, or above 80 on F. Similar procedures were utilized by Vanderploeg et al. (1998).

Measures of Attention

Digit Span

WAIS-R Digit Span (Wechsler, 1987) scaled score Forward and Backward raw scores. Digit Span consists of two parts: Digits Forward requires the subject to repeat sequences of three to nine digits; Digit Backward sequences are two to eight numbers long, and the subject must say them in reverse order (Spreen & Strauss, 1998). Moderate correlations have been achieved with the PASAT, RBMT (Rivermead Behavioural Memory Test), WMS scores, CVLT, TPT (Tactual Performance Test) Memory, and numerous other tests utilizing attention and memory.

Visual Memory Span

WMS-R Visual Memory Span (Wechsler, 1987) forward and backward raw scores. The Visual Memory Span consists of two cards on each of which eight squares are printed in a nonlinear pattern, red squares for the forward span and green for reversed span.

Administration procedures are the same as for Digit Span, requiring two trials at each level regardless of whether the first was passed (Lezak, 1995).

Mental Control

WMS-R Mental Control (Wechsler, 1987) consists of (1) counting backwards from 20 in 30 seconds; (2) reciting the alphabet in 30 seconds; and (3) counting from 1 to 40 by 3's in 45 seconds. Its attentional component is "consistently attested to by factor analytic studies" (Lezak, 1995).

Digit Symbol

WAIS-R Digit Symbol (Wechsler, 1987) scaled score. Digit Symbol is a symbol substitution task consisting of four rows containing, in all, 100 small blank squares, each paired with a randomly assigned number from one to nine. Following a practice trial on the first seven, the task is to fill in the blank spaces with the symbol that is paired to the number above the blank space as quickly as possible for 90 seconds (Lezak, 1995). Among other functions such as psychomotor speed and visuomotor coordination, it is a measure of focused attention, requiring the individual to reject irrelevant information while attending to relevant input (Spreen & Strauss, 1998).

Symbol Digit Modalities Test

Symbol Digit Modalities Test (Smith, 1991) oral administration raw score and written administration raw score. This test preserves the substitution format of Digit Symbol but reverses the presentation of material so that the symbols are printed and the numbers are written, enabling the individual to respond with the more familiar act of number writing. It also allows for a spoken response trial. As with Digit Symbol, 90 seconds are allowed for each trial, however there are 110 items instead of 100 (Lezak, 1995). Although Lezak

(1995) maintains that this test primarily measures complex scanning and visual tracking, Spreen and Strauss (1998) include it as a measure of focused attention.

Trail Making Test

Trail Making Test (Reitan & Wolfson, 1985) Part A time in seconds and Part B time in seconds. The individual must first draw lines to connect consecutively numbered circles on one work sheet (Part A) and then connect the same number of consecutively numbered and lettered circles on another worksheet by alternating between the two sequences (Part B; Lezak, 1995). Trail Making is a test involving focused attention, motor speed, and complex visual scanning (Lezak, 1995; Spreen & Strauss, 1998).

Finger Tapping

Tapping (Reitan & Wolfson, 1985) dominant hand mean number of taps and nondominant hand mean number of taps. Finger Tapping consists of a tapping key with a device for recording the number of taps. Each hand makes five 10-second trials with brief rest periods between trials. The score for each hand is the average for a set of five trials that do not vary more than 5 taps (Lezak, 1995). Though not a formal test of attention, it taps motor speed and manual dexterity and is sensitive to the presence and laterality of brain lesion (Lezak, 1995; Spreen & Strauss, 1998).

Grooved Pegboard

Grooved Pegboard (Reitan & Wolfson, 1985) dominant hand time in seconds and nondominant hand time in seconds. Grooved Pegboard consists of a small board containing a 5 X 5 set of slotted holes angled in different directions. Each peg has a ridge along one side requiring it to be rotated into position for correct insertion. The score is time to completion, and its complexity makes it sensitive for measuring general slowing

and progression of disease processes, and by assisting in identifying lateralized impairment (Lezak, 1995). It is not recognized as a formal test of attention.

Conners' Continuous Performance Test (CPT)

Conners' Continuous Performance Test (CPT; Conners, 1995) number of hits, omission errors, commission errors, and hit reaction time. The Standard CPT in Conners' program requires the individual to press the appropriate key (e.g., space bar) for any letter *except* the letter X. There are six blocks, each with three 20-trial sub-blocks. For each block, the sub-blocks have different interstimulus intervals: 1, 2, or 4 seconds. Each letter is displayed for 250 milliseconds. The CPT assesses lapses in attention or vigilance and impulsivity (Spreen & Strauss, 1998).

Seashore Rhythm Test

Seashore Rhythm Test (Reitan & Wolfson, 1985) total errors. The Seashore Rhythm Test is the most widely used test for nonverbal auditory perception. It also consists of concentration and tracking components. It requires the individual to discriminate between like and unlike pairs of musical beats.

Speech Perception Test

Speech Sounds Perception Test (Reitan & Wolfson, 1985) total errors. This test consists of 60 sets of nonsense syllables each beginning and ending with different consonants but based on the vowel sound "ee." It is administered by tape recording, and individuals must note what they think they heard on a four-choice form laid out in six 10-item sections labeled A to F. Since it is a rapidly paced test, it is thought to be sensitive to attentional deficits (Lezak, 1995).

WMS-R Attention/Concentration Index

WMS-R (Wechsler, 1987) Attention/Concentration Index score. This score is derived from the WMS-R Digit Span, Visual Memory Span, and Mental Control subtest scores.

Wisconsin Card Sorting Test (WCST)

Wisconsin Card Sorting Test (WCST; Heaton, et al., 1993) number correct, number of errors, number of perseverative responses, number of categories completed, and failure to maintain set. This test is designed to assess the ability to form abstract concepts, to shift and maintain set, and utilize feedback (Spreen & Strauss, 1998). It consists of four stimulus cards (or representations of cards on a computer screen), red triangle, two green stars, three yellow crosses, and four blue circles. The individual is asked to match each of 64 cards, which have designs similar to those on the stimulus cards, varying in color, geometric form, and number, to one of the four key cards and is given feedback each time whether he or she is right or wrong. There is no time limit.

Paced Auditory Serial Addition Test (PASAT)

Paced Auditory Serial Addition Test (PASAT; Gronwall, 1977) total errors on each of trials 1-4. The PASAT is a serial-addition task used to assess capacity and rate of information processing and sustained and divided attention. The individual is required to comprehend the auditory input, respond verbally, inhibit encoding of his or her own response while attending to the next stimulus in a series, and perform at an externally determined pace. A prerecorded tape delivers a random series of 61 numbers from 1 to 9. The individual is instructed to add pairs of numbers such that each number is added to the one that immediately precedes it: the second is added to the first, the third to the second, the fourth to the third, and so on. The same 61 numbers, given in the same sequence, are

presented in four different trials, each trial differing in its rate of digit presentation (2.4, 2.0, 1.6, 1.2 seconds).

Stroop Color and Word Test

Stroop (Golden, 1978) Word score, Color score, and Color-Word score. This test is a measure of selective attention and cognitive flexibility was originally developed by Stroop (1935). A number of versions have been developed, but the one utilized here is the version developed by Golden (1978). In Golden's version, three cards are used consisting of 100 items on each. On the first card, the individual is asked to read the color words (red, green, or blue) printed in black ink as quickly as possible for 45 seconds. On the second, the individual reads the color of ink, each item consisting of "XXX" as quickly as possible for 45 seconds. On the final card, the individual must name the color in which the color words are printed and disregard their verbal content (i.e., inhibit the natural urge to read the word).

Word Fluency

Benton Word Fluency (Benton, Hamsher, & Sivan, 1994) total number of words generated. The purpose of this test is to evaluate the spontaneous production of words beginning with a given letter (C, F, and L; or F, A, and S). The individual is asked to produce orally as many words as possible, for each letter, given a 60 second time limit for each trial. The score is the combined total for the three trials added to an education correction factor.

Category Fluency

Animal naming (Goodglass & Kaplan, 1987) total number of words. This test serves the same purpose as Word Fluency, however on this task the individual is confined to a category and asked to produce as many animal names as possible within 60 seconds.

Results

Analyses were conducted using SPSS 9.0 (Statistical Package for the Social Sciences - Version 9.0). Participants completing valid MMPI's were selected from the overall sample. Valid MMPI's were defined as t-score elevations less than 70 on scales L and K and less than 80 on scale F (Vanderploeg, et al., 1998).

Preliminary Analyses

A selection of only cases in which the MMPI-2 was administered, allowing for potentially less-confounding interpretations, resulted in an undesirable sample size (N=390). Consequently, fewer analyses would be possible and with less statistical power. With a goal of significantly increasing the total sample size, the decision was made to utilize the total MMPI dataset, which includes individuals administered the original MMPI (N=641). Understandably, there are normative issues with regard to combining the two datasets. Namely, scaling differences between the two instruments. On the original MMPI, each clinical scale is comprised of t-scores that are linear combinations of the underlying raw score distribution for that scale. As such, they retain the original raw score distribution (e.g., in skewness), and the linear t-score transformation does not change these distributions (aside from rounding errors). As a result, the same linear t-score value can have different meanings (i.e., different relative standings or percentiles) for different scales (Tellegen & Ben-Porath, 1992). The MMPI-2 was developed utilizing uniform t-scores to provide direct comparisons across scales. For example, a t-score of 80 (98.6^{th} percentile) on Scale 8 corresponds to a t-score of 80 (98.6^{th} percentile) on Scale 9 (for

that same individual or another individual). Procedures to derive uniform t-scores are described in detail by Tellegen and Ben-Porath (1992).

Because raw scores tend to be higher for the MMPI-2 than for the MMPI normative sample, t-scores tend to be lower on the MMPI-2 than on the MMPI. These findings led to a recommendation in the MMPI-2 manual (Butcher et al., 1989) that less-restrictive criteria that recognize potential significance of elevation in the 65-69 t-score range be used in interpreting MMPI-2 profiles.

To determine the degree to which the two MMPI datasets differed in this particular sample, a one-way ANOVA was performed. As expected, the two groups were significantly different ($p<0.01$; see Table 2 below) on all five scales (L, F, K, D, and Pt). T-scores for the MMPI-2 were lower than the MMPI T-scores for all scales except Scale L. This was expected. Harrell, Honaker, and Parnell (1992) found significantly lower t-scores on several of the clinical scales but found a higher Scale L for the MMPI-2. They administered the MMPI, MMPI-2, or both, in a counterbalanced repeated-measures design (N=131). Subject rank order T scores and dispersion of the basic clinical scales did NOT differ between the tests, and measures of profile similarity indicated congruence between the two instruments. Among subjects who completed both instruments, code-type concordance was not significantly lower than stability rates of the tests. The results supported the assignment of 65T as the lower boundary of clinical elevation on the MMPI-2 and the psychometric equivalence of the MMPI-2 and MMPI with respect to T scores, score rankings, and measures of score distribution. In a clinical review, Clavelle (1992) asked clinical psychologists (N=35) to review pairs of MMPI and MMPI-2 profiles derived from the same test responses and estimated that 92-96% of their diagnoses and

89-93% of their narrative interpretations would be essentially the same from one version of the MMPI to the next.

Table 2

ANOVA of MMPI and MMPI-2 Scales L, F, K, D, and Pt

	N	Mean (S.D.)	F	Sig.
MMPI L			19.232	.0001
MMPI	641	51.20 (7.3)		
MMPI-2	390	53.01 (8.24)		
MMPI F			44.762	.0001
MMPI	641	60.67 (9.02)		
MMPI-2	390	56.63 (10.62)		
MMPI K			44.351	.0001
MMPI	641	51.90 (8.5)		
MMPI-2	390	48.13 (10.24)		
MMPI D			11.465	.001
MMPI	641	71.06 (15.76)		
MMPI-2	390	66.65 (14.45)		
MMPI Pt			21.753	.0001
MMPI	641	66.55 (13.82)		
MMPI-2	390	61.57 (13.52)		

Note: MMPIL=Scale L; MMPIF=Scale F; MMPIK=Scale K; MMPID=Scale D; MMPIPt=Scale Pt

To determine the effect size of the differences between the two groups (i.e., the degree to which their distributions do not overlap), a MANOVA was performed to derive the Eta Squared for each comparison. The effect sizes are summarized in Table 3 below. Cohen (1977) suggests the following effect size conventions: "0.2 = small, 0.5 = medium, and 0.8 = large effect size." The resulting Eta Squared values suggested effect sizes less than 0.04, indicating very small differences between the two MMPI datasets. It is likely that the significant differences found in the MANOVA were due to the large overall sample size. With t-score differences less than T=4.98, the original MMPI dataset was considered appropriate for inclusion in subsequent analyses, however separate analyses were performed using "MMPI version administered" as a covariate. This was done as another safeguard against this potentially confounding issue

Table 3

Effect Size Differences Between MMPI and MMPI-2 Groups

Variable	Sum of Squares	df	Mean Square	F	Sig.	Eta Squared
MMPI L	1107.534	1	1107.534	19.04	.000	.016
MMPI F	4481.732	1	4481.732	47.34	.000	.038
MMPI K	3870.380	1	3870.380	46.355	.000	.037
MMPI D	2973.480	1	2973.480	12.139	.001	.010
MMPI Pt	4338.107	1	4338.107	22.530	.000	.018

Note: MMPIL=Scale L; MMPIF=Scale F; MMPIK=Scale K; MMPID=Scale D; MMPI Pt=Scale Pt

Descriptive statistics were performed to examine variables for errors, number of valid cases, and measures of central tendency, variability, and shape of distribution. Data were recoded as necessary and incorrectly coded variables were removed. Descriptive and Frequency statistics are presented in Appendix A. Variables for the Stroop Color Word Naming Test, PASAT, Symbol Digit Modalities Test and CPT were removed from subsequent analyses due to low sample size (N=19, N=30, N=34, and N=51, respectively).

Next, a correlation matrix was constructed to explore the relationships between emotional distress (MMPI Scales 2 and 7) and attentional measures. Significant correlations are reported in Table 4 below (only those with >0.35 absolute value). Significant correlations (* $p < .05$; ** $p < .01$) were obtained between Scales 2 and 7 and attention measures, though they were very modest. MMPID (Scale 2) was significantly correlated with LPID (-.070*), LPTND (-.079*), ROCOPY (.069*), SPTE (.073*), TAPD (-.069*), and WDSY (-.099**). MMPI PT (Scale 7) was significantly correlated with LPID (-.121**), LPTND (-.119**), and ROCOPY (.130**).

Table 4

Correlation Matrix of Significant Correlations

ANIM & LPID	.365	WCSTC & WCSTE	-.438
ANIM & ROCOPY	-.537	WCSTC & WCSTFM	.381
DYND & DYNND	.835	WCSTC & WCSTPR	-.432
DYND & TAPD	.448	WCSTCT & WCSTE	-.830
DYND & TAPND	.365	WCSTCT & WCSTPE	-.538
LPID & LPTND	.858	WCSTCT & WCSTPR	-.707
LPID & ROCOPY	-.667	WCSTE & WCSTPE	.718
LPID & WRAC	-.410	WCSTE & WCSTPR	.809
LPTND & ROCOPY	-.643	WCSTPE & WCSTPR	.499
LPTND & WRAC	-.351	MMPI D & MMPI F	.474
ROCOPY & WRAC	.528	MMPI D & MMPI Pt	.740
TAPND & TAPD	.739	MMPI F & MMPI K	-.368
TRLA & TRLB	.545	MMPI F & MMPI Pt	.517
TRLA & WDSY	-.422	WDSP & WDSY	.382
TRLB & WDSY	-.460	WDSP & WFSIQ	.557
WDSY & WFSIQ	.462	WFSIQ & EDUC	.442
WDSY & AGE	-.413		

Note. All correlations significant at $p<0.01$. Only correlations greater than 0.35 absolute value listed. MMPIL=Scale L; MMPIF=Scale F; MMPIK=Scale K; MMPID=Scale D; MMPIPt=Scale Pt; ANIM=Category Fluency; DYND=Dominant Grip Strength;

(table continued)

DYNND=Nondominant Grip Strength; LPID=Dominant Grooved Pegboard; LPTND=Nondominant Grooved Pegboard; TAPD=Dominant Finger Tapping; TAPND=Nondominant Finger Tapping; ROCOPY=Rey-O Copy; TRLA=Trails A; TRLB=Trails B; WCSTC=Wisconsin Card Sorting Test Correct; WCSTCT=WCST Number of Categories; WCSTE=WCST Errors; WCSTFM=WCST Failure to Maintain Set; WCSTPE=WCST Perseverative Errors; WCSTPR=WCST Perseverative Responses; WDSP=WAIS-R Digit Span; WDSY=WAIS-R Digit Symbol; WRAC=WMS-R Attention/Concentration Index; WFSIQ=WAIS-R Full Scale IQ; AGE=Age; EDUC=Education.

Next, a factor analysis was conducted to explore the underlying factor structure of attention test scores in this sample and to compare to the factor structure obtained by Mirsky (1987). Results of the factor analysis are presented in Table 5 below. Utilizing a Principal Component Analysis extracton method and Varimax (with Kaiser Normalization) rotation method, six factors were identified. Loadings for Factor 1 consisted of Category Fluency, Grooved Pegboard (dominant and nondominant), Rey-O Copy, and WMS-R Attention/Concentration Index. Factor 2 included the following Wisconsin Card Sort Scores (WCST): Number Correct, Number of Categories, Number of Errors, Number of Perseverative Errors, and Number of Perseverative Responses. Factor 3 was comprised of: Grip Strength (dominant and nondominant), and Finger Tapping (dominant and nondominant). Factor 4 consisted of both Finger Tapping scores, Verbal Fluency, Trails A and B, and WAIS-R Digit Symbol. Factor 5 included WCST scores for Number Correct and Failure to Maintain Set. Finally, Factor 6 consisted of Seashore Rhythm, Speech Sounds Perception Test, and WAIS-R Digit Span.

Table 5

Exploratory Factor Analysis of Attention Test Scores

Variable	Factor 1	Factor 2	Factor 3	Factor 4	Factor 5	Factor 6
LPID	.873					
LPTND	.834					
WRAC	-.631					
ROCOPY	-.856					
ANIM	.700					
WCSTC		-.484			.664	
WCSTCT		-.906				
WCSTE		-.960				
WCSTPE		.729				
WCSTPR		.842				
DYND			.867			
DYNND			.881			
TAPD			.727	-.346		
TAPND			.736	-.332		
BWFTW				-.420		
TRLA				.790		
TRLB				.742		
WDSY				-.681		
WCSTFM					.901	
RHYE						.732
SPTE						.584
WDSP						-.643
ARITH						-.584

Note. Extraction Method: Principal Component Analysis. Rotation Method: Varimax with Kaiser Normalization. ANIM=Category Fluency; ARITH= WAIS-R Arithmetic; BWFTW=Verbal Fluency; DYND=Dominant Grip Strength; DYNND=Nondominant Grip Strength; LPID=Dominant Grooved Pegboard; LPTND=Nondominant Grooved Pegboard; RHYE=Seashore Rhythm; SPTE=Speech Sounds Perception Test; TAPD=Dominant Finger Tapping; TAPND=Nondominant Finger Tapping; ROCOPY=Rey-O Copy; TRLA=Trails A; TRLB=Trails B; WCSTC=Wisconsin Card Sorting Test Correct; WCSTCT=WCST Number of Categories; WCSTE=WCST Errors; WCSTFM=WCST Failure to Maintain Set; WCSTPE=WCST Perseverative Errors; WCSTPR=WCST Perseverative Responses; WDSP=WAIS-R Digit Span; WDSY=WAIS-R Digit Symbol; WRAC=WMS-R Attention/Concentration Index.

An Analysis of Variance (ANOVA) was conducted to determine if participants with a legal case pending (N=178 or 14.7%) differed significantly from the overall sample. As discussed earlier, a confounding factor in other studies exploring attention has been that of symptom exaggeration, and individuals with legal cases pending are at increased risk for exaggeration of symptoms due to potential financial gain (Sweet et al., 1992). Results are presented in Table 6 below.

Table 6

ANOVA of Groups by Legal Status

Variable	N	Mean (S.D.)	F	Sig.
MMPI D			17.78	.0001
Legal	178	69.93 (15.65)		
Not Legal	791	67.61 (14.51)		
MMPI Pt			18.74	.0001
Legal	178	65.47 (14.09)		
Not Legal	791	62.03 (12.97)		

Note: MMPID=Scale D; MMPIPt=Scale Pt.

Results of the ANOVA revealed significant differences ($p < 0.01$) between groups on MMPI scales D and Pt by legal status, therefore individuals referred for testing due to a legal case pending were removed from subsequent analyses. Results were not significant for MMPI scales L, F, or K.

Next, a canonical correlation was performed between the set of 22 attention variables (test scores) and the set of two emotional distress variables (Scales D and Pt).

Canonical analysis is the most general of the multivariate techniques (Tabachnick & Fidell, 1989). In fact, other procedures such as multiple regression, discriminant function analysis, and MANOVA are special cases of it. However, canonical correlation is also one of the least used. According to Tabachnick and Fidell, the most critical limitation is difficulty interpreting the solution due to computation of canonical pairs that are independent of all others and sensitivity to variables added to each set on both sides of the equation. Sample size is also an issue. A ratio of at least 10 cases for every variable is desirable. For the present study, sample size was adequate, and interpretability was facilitated by maintaining the same set of variables for each analysis (except for the one addition of a covariate).

The first canonical correlation was .23 (5% of variance). The second canonical correlation was not interpreted due to nonsignificance. With both canonical correlations included, Wilks Lambda = .92, $p < .001$, and with the first canonical correlation removed, Wilks Lambda = .97, $p = .084$. The first pair of canonical variates therefore accounted for the significant relationships between the two sets of variables. Data on the first pair of canonical variates appear in Table 7 below, along with correlations between the variables and the canonical variates, standardized canonical variate coefficients, within-set variance accounted for by the canonical variates (percent of variance), redundancies, and the canonical correlation.

With a cutoff correlation of .30 as suggested by Tabachnick and Fidell (1989), the variables in the attention set that were correlated with the first canonical variate were Rey-O copy, WCST errors, WCST perseverative errors, Finger Tapping, Trails B time, WAIS-R Digit Symbol scaled score, and WAIS-R Digit Span scaled score. Among emotional

distress variables, only MMPI Scale D (depression) was significantly correlated. The first pair of canonical variates indicate that higher depression scores (.45) are associated with lower scores on Rey-O copy (-.34), more WCST errors (.30) and perseverative errors (.35), fewer dominant (-.52) and nondominant (-.44) taps on Finger Tapping, longer total time on Trails B (.46), and lower scaled scores on WAIS-R Digit Symbol (-.75) and Digit Span (-.39).

Table 7

Canonical Correlation

	Correlation	Coefficient
Attention Test set		
ANIM	.16	.07
LPID	.26	.47
LPTND	.18	-.49
ROCOPY	-.34	-.38
WRAC	-.07	.33
WCSTC	-.04	.23
WCSTCT	-.12	.16
WCSTE	.30	.31
WCSTPE	.35	.17
WCSTPR	.19	.06
WCSTFM	-.01	-.21
DYND	-.21	.20
DYNND	-.22	-.22
TAPD	-.52	-.38
TAPND	-.44	.03
BWFTW	-.26	.03
TRLA	.25	-.27
TRLB	.46	.19
WDSY	-.75	-.61
WDSP	-.39	-.08
RHYE	.19	.09
SPTE	.17	.05

(table continued)

Table 7 (continued)

Canonical Correlation

	Correlation
Percent of variance	9.90
Redundancy	0.50
Emotional Distress set	
MMPI D	.45
MMPI Pt	-.27
Percent of variance	0.70
Redundancy	13.60
Canonical correlation	0.23

Note. Second canonical variate was not significant ($p = 0.084$); ANIM=Category Fluency; DYND=Dominant Grip Strength; DYNND=Nondominant Grip Strength; LPID=Dominant Grooved Pegboard; LPTND=Nondominant Grooved Pegboard; TAPD=Dominant Finger Tapping; TAPND=Nondominant Finger Tapping; RHYE=Seashore Rhythm; ROCOPY=Rey-O Copy; SPTE=Speech Sounds Perception Test; TRLA=Trails A; TRLB=Trails B; WCSTC=Wisconsin Card Sorting Test Correct; WCSTCT=WCST Number of Categories; WCSTE=WCST Errors; WCSTFM=WCST Failure to Maintain Set; WCSTPE=WCST Perseverative Errors; WCSTPR=WCST Perseverative Responses; WDSP=WAIS-R Digit Span; WDSY=WAIS-R Digit Symbol; WRAC=WMS-R Attention/Concentration Index; MMPID=Scale D; MMPIPt=Scale Pt.

Next, an identical canonical correlation was performed with the addition of MMPI_VER (version of MMPI administered) as a covariate (Table 8 below). The first canonical correlation was .28 (8% of variance). The second canonical correlation was .22 (5% of variance). With both canonical correlations included, Wilks Lambda = .88, $p < .001$, and with the first canonical correlation removed, Wilks Lambda = .95, $p < .05$. The first pair of canonical variates therefore accounted for the significant relationships between the two sets of variables. Data on the two pairs of canonical variates appear in Table 8, along with correlations between the variables and the canonical variates, standardized

canonical variate coefficients, within-set variance accounted for by the canonical variates (percent of variance), redundancies, and canonical correlations.

Total percent of variance and total redundancy indicate that the first pair of canonical variates was marginally related with moderate redundancy (39%), however the second pair was circumspect due to very large redundancy (60%).

With a cutoff correlation of .30, the variables in the attention set that were correlated with the first canonical variate were WCST errors, WCST perseverative errors, WCST perseverative responses, dominant and nondominant Finger Tapping, Trails B time, WAIS-R Digit Symbol scaled score, and Speech Perception Test errors. Among emotional distress variables, only MMPI Scale D (depression) was significantly correlated. The first pair of canonical variates indicate that higher depression scores (.85) are associated with more WCST errors (.39), perseverative errors (.33), perseverative responses (.31), fewer dominant (-.47) and nondominant (-.41) taps on Finger Tapping, longer total time on Trails B (.40), lower scaled scores on WAIS-R Digit Symbol (-.71) and more errors on the Speech Perception Test (.34).

Table 8

Canonical Correlation with Covariate

	First canonical variate**		Second canonical variate*	
	Correlation	Coefficient	Correlation	Coefficient
Attention Test set				
ANIM	-.04	.14	.29	-.07
LPID	-.11	-.03	.46	.36
LPTND	-.07	-.10	.42	-.30
ROCOPY	.08	-.29	-.59	-.34
WRAC	.11	.31	-.21	.22
WCSTC	-.11	-.01	.24	.45
WCSTCT	-.27	.40	.14	.13
WCSTE	.39	.43	-.04	-.11
WCSTPE	.33	.20	.21	.11
WCSTPR	.31	.04	-.04	.36
WCSTFM	-.05	-.13	.19	-.08
DYND	.04	.20	-.07	.02
DYNND	.02	-.05	-.07	-.03
TAPD	-.47	-.40	-.1	.12
TAPND	-.41	-.05	-.13	-.15
BWFTW	-.18	.04	-.33	-.13
TRLA	.23	-.13	.25	.11
TRLB	.40	.09	.09	-.06
WDSY	-.71	-.64	-.35	-.28
WDSP	-.25	.03	-.11	.09
RHYE	.04	-.07	.08	.11
SPTE	.34	.19	-.15	-.10
MMPI_VER	-.28	-.41	.78	.66
Percent of variance	8.17		8.79	Total = 16.96
Redundancy	0.58		0.42	Total = 1.00
Emotional Distress set				
MMPI D	.85	1.45	-.53	.43
MMPI Pt	.29	-.81	-.96	-1.28
Percent of variance	2.85		2.88	Total = 5.73
Redundancy	39.87		60.13	Total = 100.00
Canonical correlation	0.28		0.22	

(table continued)

Note. *p < 0.05 **p < 0.01; ANIM=Category Fluency; DYND=Dominant Grip Strength; DYNND=Nondominant Grip Strength; LPID=Dominant Grooved Pegboard; LPTND=Nondominant Grooved Pegboard; TAPD=Dominant Finger Tapping; TAPND=Nondominant Finger Tapping; RHYE=Seashore Rhythm; ROCOPY=Rey-O Copy; SPTE=Speech Sounds Perception Test; TRLA=Trails A; TRLB=Trails B; WCSTC=Wisconsin Card Sorting Test Correct; WCSTCT=WCST Number of Categories; WCSTE=WCST Errors; WCSTFM=WCST Failure to Maintain Set; WCSTPE=WCST Perseverative Errors; WCSTPR=WCST Perseverative Responses; WDSP=WAIS-R Digit Span; WDSY=WAIS-R Digit Symbol; WRAC=WMS-R Attention/Concentration Index; MMPID=Scale D; MMPIPt=Scale Pt.

The variables in the attention set that were correlated with the second canonical variate were dominant and nondominant Grooved Pegboard, Rey-O copy, Verbal Fluency, and WAIS-R Digit Symbol. In the emotional distress set, variables with significant correlations included both Scale D (depression) and Scale Pt (anxiety). The second pair of canonical variates indicated that lower depression scores (-.53) and anxiety scores (-.96) were associated with longer time to complete Grooved Pegboard for dominant (.46) and nondominant (.42) upper extremities, lower scores on Rey-O copy (-.59), fewer words on Verbal Fluency (-.33), and lower WAIS-R Digit Symbol scaled scores (-.35).). However, as mentioned above total redundancy for this canonical pair was 60% indicating a large portion of the variance shared between the two sets of variables. Therefore, interpretation of this canonical pair would be specious.

In conclusion, results of the first canonical correlation revealed that depression was significantly correlated with the first canonical variate (Emotional Distress) but only contributed 0.7% of the variance. Variables significantly correlated with the second canonical variate (Attention Tests) were Rey-O Copy, WCST Errors and Perseverative Errors, Finger Tapping (both hands), Trails B, and WAIS-R Digit Symbol and Digit Span, and they contributed approximately 10% of the variance. Overall, the pair of canonical

variates (Emotional Distress and Attention Tests) was modestly correlated (0.23) with only 5% of the variance explained.

The second canonical correlation, which included the version of MMPI administered to the participants, revealed minor differences. Only the first pair of canonical variates was interpretable due to large redundancy in the second pair (60%) of canonical variates. In the second canonical correlation, depression was significantly correlated with the first canonical variate (Emotional Distress) and contributed slightly more variance (2.85%). Variables significantly correlated with the second canonical variate (Attention Tests) differed somewhat. Rey-O Copy and WAIS-R Digit Span were dropped in favor of WCST Perseverative Responses and the Speech Sounds Perception Test. The percent of variance contributed was 8.17%. Overall, the pair of canonical variates (Emotional Distress and Attention Tests) was modestly correlated (0.28) with only 5% of the variance explained. Furthermore, the covariate MMPI Version Administered (MMPI_VER) was not significantly correlated with the second canonical variate (Attention Tests).

Next, a series of Multiple Analyses of Covariance (MANCOVA) were performed to determine the relationship between severity levels of emotional distress and attention measures while controlling for either depression or anxiety scores. Severity levels for all analyses were determined using the same procedure utilized by Vanderploeg et al. (1998):

"High Depression" = T-score on D > 70, T-score on Pt < 60;

"Low Depression" = T-score on D > 45 and < 55, T-score on Pt < 60;

"High Anxiety" = T-score on Pt > 70, T-score on D < 60;

"Low Anxiety" = T-score on Pt > 45 and < 55, T-score on D < 60;

"High Depression and Anxiety" = T-score on D and Pt > 70; and

"Low Depression and Anxiety" = T-score on D and Pt > 45 and < 55.

A MANCOVA was performed comparing attention scores between groups by level of anxiety (i.e., No, Low, Moderate, and High Anxiety) while controlling for depression scores. Table 9 (below) presents significant interactions ($p < 0.05$) and significant post-hoc comparisons utilizing Games-Howell's (1976) procedure for unequal n's/variances to determine significant pairwise comparisons. Mean differences (I minus J) are presented in parentheses. Significant interactions were found only for the three variables LPID ($p = .012$), ROCOPY ($p = .003$), and WRAC ($p = .028$).

Table 9

MANOVA by Anxiety Severity Levels Controlling for Depression with Post-Hoc Comparisons

Variable I v. J levels (Means)	N		N	F	Sig.
LPID				3.66	.012*
Moderate (72.05)	283	High (65.42)	245		.035*
ROCOPY				4.67	.003**
No (34.95)	56	High (36.77)	245		.021*
Low (35.40)	125	High (36.77)	245		.027*
Moderate (35.70)	283	High (36.77)	245		.027*
WRAC				3.05	.028*
Moderate (99.76)	83	High (103.05)	45		.013*

Note: Only significant interactions are shown (* $p < 0.05$ or ** $p < 0.01$); LPID=Dominant Grooved Pegboard; ROCOPY=Rey-O Copy; WRAC=WMS-R Attention/Concentration Index.

A similar MANCOVA was performed comparing attention scores between groups by level of depression (No, Low, Moderate, and High Depression) while controlling for anxiety. Table 10 (below) presents significant interactions ($p < 0.05$) and significant post-hoc comparisons utilizing Games-Howell's procedure for unequal n's/variances to determine significant pairwise comparisons. Mean differences (I minus J) are presented in parentheses.

Table 10

MANOVA by Depression Severity Levels Controlling for Anxiety with Post-Hoc Comparisons

Variable I v. J levels (Means)	N		N	F	Sig.
LPID				3.28	.021*
Moderate (74.40)	251	High (66.63)	318		.013*
LPTND				3.15	.024*
Moderate (83.08)	251	High (66.63)	318		.028*
TAPD				3.50	.015*
Low (46.64)	106	Moderate (44.10)	251		.002**
Low (46.64)	106	High (43.94)	318		.000**
WCSTCT				3.23	.022*
No (4.68)	34	High (3.95)	251		.035*
WCSTE				4.70	.003**
No (32.86)	34	Moderate (4.01)	251		.015*
No (32.86)	34	High (41.95)	318		.012*

(table continued)

Table 10

MANOVA by Depression Severity Levels Controlling for Anxiety with Post-Hoc Comparisons

Variable I v. J levels (Means)	N		N	F	Sig.
WCSTPR				2.97	.031*
No (21.15)	34	Moderate (28.26)	251		.007**
No (21.15)	34	High (28.77)	318		.002**
WDSP				3.68	.012*
No (9.97)	34	Moderate (8.48)	251		.026*
WDSY				3.28	.021*
No (8.33)	34	Moderate (7.11)	251		.048*
WRAC				4.65	.003**
Moderate (99.03)	251	High (102.90)	318		.002**

Note: Only significant interactions are shown (* $p < 0.05$ or ** $p < 0.01$); ANIM=Category Fluency; DYND=Dominant Grip Strength; DYNND=Nondominant Grip Strength; LPID=Dominant Grooved Pegboard; LPTND=Nondominant Grooved Pegboard; TAPD=Dominant Finger Tapping; TAPND=Nondominant Finger Tapping; RHYE=Seashore Rhythm; ROCOPY=Rey-O Copy; SPTE=Speech Sounds Perception Test; TRLA=Trails A; TRLB=Trails B; WCSTC=Wisconsin Card Sorting Test Correct; WCSTCT=WCST Number of Categories; WCSTE=WCST Errors; WCSTFM=WCST Failure to Maintain Set; WCSTPE=WCST Perseverative Errors; WCSTPR=WCST Perseverative Responses; WDSP=WAIS-R Digit Span; WDSY=WAIS-R Digit Symbol; WRAC=WMS-R Attention/Concentration Index; MMPID=Scale D; MMPI PT=Scale Pt.

Next, a series of MANOVA procedures were performed to assess the effect of both depression and anxiety. Attention performance of participants with both High scores ($T > 70$) on Scales 2 and 7 was compared to those without High scores. Results were significant for several attention variables (see Table 11 below), however a comparison of

the means revealed surprising results. Except for SPTE, all other variable means indicated improvement in performance of participants. Attention performance of participants with both Low scores (T >45 and <55) on Scales 2 and 7 was compared to those without Low scores, however results were not significant.

The sample was subsequently divided into two groups by presence (neurologic, N=256) or absence (non-neurologic, N=287) of documented neurologic injury. The neurologic group consisted of mixed etiologies (see Table 12 below). Neurologic injury due to trauma comprised 49.2% of the sample, followed by degenerative processes (27.7%), vascular disorders (19.9%), and neoplasms (11.7%). The remaining etiologies (2.6%) comprised less than 1% each. A MANOVA was performed to compare the two groups on performance on attention measures. Results are presented in Table 13 below and show significant interactions for the following variables: BWFTW ($p < .001$), LPID ($p < .007$), LPTND ($p = .012$), ROCOPY ($p = .005$), TAPD ($p = .019$), TAPND ($p = .015$), TRLA ($p < .001$), TRLB($p < .001$), WDSP ($p < .001$), WDSY ($p < .001$), and WRAC ($p = .024$). All mean differences were in the expected direction of performance for each test.

Table 11

MANOVA of Attention Measures for Both High Depression and Anxiety

Variable	High D/A (N=204)	Low D/A (N=505)	F	Sig.
BWFTW	33.97 (8.33)	32.60 (8.32)	.3.91	048*
LPID	65.46 (25.69)	72.13 (30.89)	7.42	.007**
LPTND	76.30 (21.38)	80.77 (26.23)	4.66	.031*
ROCOPY	36.78 (4.31)	35.62 (4.51)	9.85	.002**
SPTE	8.30 (4.39)	7.63 (3.17)	5.14	.024*
WRAC	103.07 (11.54)	100.79 (13.43)	4.53	.034*

Note: * $p < .05$; ** $p < .01$; BWFTW=Verbal Fluency; LPID=Dominant Grooved Pegboard; LPTND=Nondominant Grooved Pegboard; ROCOPY=Rey-O Copy; SPTE=Speech Perception Test; WRAC=WMS-R Attention/Concentration Index.

Table 12

Frequencies by Neurologic Diagnosis

Diagnostic Category	N
Trauma	126
Degenerative Process	71
Vascular Disorders	51
Neoplasm	30

(table continued)

Table 12 (continued)

Frequencies by Neurologic Diagnosis

Diagnostic Category	N
Seizure Disorders	24
Infectious Disease	19
Metabolic or Toxic Disorder	15
Congenital	4
Miscellaneous (Cranial/Peripheral nerve disorder, spinal disorder, migraines, etc.)	4

Table 13

MANOVA by Neurologic Status

Variable	Neurologic (N=256)	Non-neurologic (N=287)	F	Sig.
BWFTW	31.85 (9.6)	34.89 (6.88)	18.29	.000**
LPID	70.81 (29.52)	64.62 (23.38)	7.41	.007**
LPTND	79.94 (25.54)	75.06 (19.21)	6.42	.012*
ROCOPY	35.95 (3.98)	36.89 (3.74)	8.01	.005**
TAPD	43.45 (8.74)	45.07 (7.28)	5.58	.019*
TAPND	38.89 (7.85)	40.35 (6.21)	5.90	.015*

(table continued)

Table 13 (continued)

MANOVA by Neurologic Status

Variable	Neurologic (N=256)	Non-neurologic (N=287)	F	Sig.
TRLA	53.13 (46.06)	39.18 (13.64)	23.96	.000**
TRLB	132.69 (90.43)	105.02 (54.44)	19.11	.000**
WDSP	8.09 (2.67)	9.22 (2.72)	23.70	.000**
WDSY	6.15 (3.02)	8.29 (2.75)	74.80	.000**
WRAC	101.44 (14.19)	103.87 (10.70)	5.14	.024*

Note: Only significant interactions are shown (* $p < 0.05$ or ** $p < 0.01$);
LPID=Dominant Grooved Pegboard; LPTND=Nondominant Grooved Pegboard;
TAPD=Dominant Finger Tapping; TAPND=Nondominant Finger Tapping;
ROCOPY=Rey-O Copy; TRLA=Trails A; TRLB=Trails B; WDSP=WAIS-R Digit Span;
WDSY=WAIS-R Digit Symbol; WRAC=WMS-R Attention/Concentration Index

Next, a series of MANOVA's were utilized for comparisons between neurologic and non-neurologic groups on levels of anxiety and depression. Results are presented in Table 14 below. Depression and anxiety levels were significantly lower in the neurologic group as compared to the non-neurologic group.

Table 14

MANOVA of Depression and Anxiety Level by Neurologic Status

Variable	Neurologic (N=354)	Non-neurologic (N=409)	F	Sig.
Neurologic Status				
MMPI D	68.31 (14.85)	70.81 (15.8)	5.024	.025*
MMPI Pt	62.25 (13.59)	67.33 (14.14)	25.458	.000**

Note: * $p < 0.05$; ** $p < 0.01$; MMPID=Scale D; MMPIPt=Scale Pt.

The neurologic group was divided into two groups by depression level (i.e., depressed or non-depressed) and compared on attention measures using MANOVA (see Table 15 below). Significant differences were found on ROCOPY ($p = .005$), ANIM ($p = .034$), and LPID ($p = .021$). Interestingly, performance was better for the depressed group on ROCOPY and LPID. Performance was worse for the depressed group on ANIM.

Next, the neurologic group was divided into two groups by anxiety level (i.e., anxious or non-anxious) and compared on the attention measures, again utilizing the MANOVA procedure. Significant differences were found for the following variables: ROCOPY ($p < .001$), WCSTFM ($p = .011$), BWFTW ($p = .025$), LPID ($p = .001$), and LPTND ($p = .01$). For all variables performance was significantly better in the anxious group.

Next, the non-neurologic group was divided into two groups by depression level (i.e., depressed or non-depressed) and compared on attention measures. Significant differences were found on the following variables: TAPD ($p = .016$), TAPND ($p = .022$), TRLA ($p = .015$), WCSTCT ($p = .023$), WCSTE ($p = .013$), WCSTPR ($p = .001$), WDSY ($p < .001$), LPID ($p = .024$), and LPTND ($p = .036$). For all of these variables except LPID and LPTND, performance was significantly worse in the depressed group. Performance was improved for the depressed group on LPID and LPTND.

Finally, the non-neurologic group was divided into two groups by anxiety level (i.e., anxious or non-anxious) and compared on attention measures. No significant interactions were found.

Table 15

MANOVA of Attention Measures for Depression and Anxiety Level by Neurologic Status

Variable	High	Low	F	Sig.
Neurologic by Depression	(N=98)	(N=158)		
ROCOPY	36.83 (3.78)	35.40 (4.01)	8.044	.005**
ANIM	11.4 (2.28)	12.04 (2.33)	4.56	.034*
LPID	65.43 (23.98)	74.14 (32.11)	5.355	.021*
Neurologic by Anxiety	(N=74)	(N=184)		
ROCOPY	37.31 (3.38)	35.39 (4.08)	12.828	.000**
WCSTFM	0.77 (0.48)	1.04 (0.81)	6.642	.011*
BWFTW	33.95 (8.69)	30.99 (9.84)	5.100	.025*
LPID	61.27 (16.74)	74.68 (32.60)	11.301	.001**
LPTND	73.50 (17.69)	82.57 (27.73)	6.787	.010*

(table continued)

Table 15

MANOVA of Attention Measures for Depression and Anxiety Level by Neurologic Status

Variable	High	Low	F	Sig.
Non-neurologic by Dep	(N=147)	(N=140)		
TAPD	44.06 (8.79)	46.13 (5.08)	5.854	.016*
TAPND	39.54 (7.23)	41.21 (4.79)	5.309	.022*
TRLA	41.10 (15.59)	37.17 (10.93)	6.047	.015*
WCSTCT	3.86 (1.22)	4.21 (1.34)	5.251	.023*
WCSTE	42.07 (14.69)	37.78 (14.43)	6.232	.013*
WCSTPR	29.65 (15.14)	24.32 (10.84)	11.652	.001**
WDSY	7.70 (2.79)	8.91 (2.59)	14.332	.000**
LPID	61.58 (19.16)	67.81 (26.81)	5.159	.024*
LPTND	72.75 (15.71)	77.49 (22.11)	4.427	.036*
Non-neurologic by Anxiety				
(no significant interactions)				

Note: * $p < 0.05$; ** $p < 0.01$; ANIM=Category Fluency; DYND=Dominant Grip Strength; DYNND=Nondominant Grip Strength; LPID=Dominant Grooved Pegboard; LPTND=Nondominant Grooved Pegboard; TAPD=Dominant Finger Tapping; TAPND=Nondominant Finger Tapping; RHYE=Seashore Rhythm; ROCOPY=Rey-O Copy; SPTE=Speech Sounds Perception Test; TRLA=Trails A; TRLB=Trails B; WCSTC=Wisconsin Card Sorting Test Correct; WCSTCT=WCST Number of Categories; WCSTE=WCST Errors; WCSTFM=WCST Failure to Maintain Set; WCSTPE=WCST Perseverative Errors; WCSTPR=WCST Perseverative Responses; WDSP=WAIS-R Digit Span; WDSY=WAIS-R Digit Symbol; WRAC=WMS-R Attention/Concentration Index

Discussion

The purpose of this study was to explore the relationship between emotional distress and neuropsychological test performance given the frequency with which psychologists and neuropsychologists encounter depression and anxiety. Empirical studies exploring this relationship have been a relatively recent trend, and results have been mixed due to various methodological problems (Sweet et al., 1992). The present study aimed to address this problem by determining the impact of differing levels of anxiety and depression on attention performance in both patients with neurologic insult and those without.

Relationships Among the Variables

Initial correlational analyses revealed relationships in the expected directions with respect to various scaling characteristics. For example, significant inverse correlations were found between tests that consist of total time to complete (e.g., Grooved Pegboard) and number correct (e.g., Rey-O Copy), reflecting expected directions of performance. Tests scored in the same direction according to performance were positively correlated, and as expected, tests tended to correlate with each other by area (e.g., motor) and with themselves (e.g., Dominant and Nondominant Finger Tapping). In addition, MMPI Scale 2 (Depression) and Scale 7 (Anxiety) were highly correlated, as expected.

Unfortunately, it was necessary to remove a number of key measures of attention from all analyses due to low sample sizes: Conners' Continuous Performance Test (CPT) scores (N=51), Paced Auditory Serial Addition Test (PASAT) scores (N=30), Symbol Digit Modalities Test - Oral and Written scores (N=34) and Stroop Color and Word Test

scores (N=19). As discussed previously, these tests are good measures of attention ability and have been shown to be sensitive to the effects of depression and anxiety (Lezak, 1995; Spreen & Strauss, 1998; Sweet, Newman & Bell, 1992; and Veiel, 1997).

A factor analysis was performed in an attempt to derive similar factor loadings as that derived by Mirsky (1987) and to explore the factor structure underlying the data utilized for this study. Attempts to replicate Mirsky's analysis were significantly limited by the removal of the Stroop and CPT variables, representing almost half (4) of Mirsky's original 10 variables. Removal of variables associated with the Stroop Color and Word Test and Conners' Continuous Performance Test (CPT) was necessitated by low sample sizes for those tests. Comparisons with Mirsky's original factor analysis was further limited by the lack of a suitable analogue to the Talland Letter Cancellation Test, used by Mirsky in his original study. However, when the remaining variables were submitted to factor analysis, nearly analogous factor loadings were derived as originally found by Mirsky (see Table 5).

Mirsky's original factor analysis identified four factors: "focus-execute", "sustain", "encode", and "shift", however in the current study six factors were derived. In the current analysis, Trail Making (Parts A and B), Digit Symbol, Finger Tapping and Verbal Fluency, loaded together on Factor 4, which may reflect Mirsky's first factor, "focus-execute." Mirsky's second factor ("sustain") consisted of CPT scores, which were removed from this analysis, thus no comparison was available. His third factor ("encode") consisted of Arithmetic and Digit Span scale scores, however factor loadings for Arithmetic and Digit Span loaded on Factor 6 in the current analysis with the Speech Perception Test and Seashore Rhythm. Loadings for this factor may have represented an

"Auditory Attention" or "Auditory Processing" component. Mirsky's fourth factor ("shift") consisted of WCST errors, which appeared to correspond with Factor 2 in the current study. This factor consisted of 5 out of the 6 WCST scores (i.e., all but Failure to Maintain Set).

The remaining factors that were derived in the current analysis did not correspond to any of Mirsky's original factors. Factor 3 appeared to represent a straightforward motor component, consisting of Grip Strength (both hands) and Finger Tapping (both hands). Factor 1 was baffling and consisted of Category Fluency, Grooved Pegboard, Rey-O Copy, and the WMS-R Attention/Concentration Index. It is possible that tests with large variability [e.g., Grooved Pegboard Dominant: 71.51 (30.91); Grooved Pegboard Nondominant: 81.34 (28.32); and WMS-R Attention/Concentration Index: 101.69 (13.69)] loaded on the first factor, which is a potential artifact of Principal Component Analysis. Equally puzzling was Factor 5, which consisted of WCST Number Correct and WCST Failure to Maintain Set, both of which loaded separately from the other WCST scores.

It is difficult to derive rich interpretative findings from the factor analysis performed in the current study due to the loss of key attention test scores (e.g., CPT and Stroop) and the lack of direct comparison to Mirsky's original analysis. Nevertheless, a few conclusions may be drawn. First, it appeared that the underlying structure of the dataset is represented by six factors or components of attention, as contrasted with Mirsky's four components. The first factor is enigmatic, and a meaningful label or theoretical construct could not be derived. The second factor seemed to represent a cognitive "shifting" component, probably mediated by prefrontal cortex. A third factor

was derived, and appeared to represent a straightforward "motor" component. This activating or driving function likely incorporates lower midbrain structures such as the caudate, putamen, and globus pallidus. The fourth factor appeared to represent a "focusing" or "execute" function, probably mediated by superior temporal and inferior parietal cortices. The fifth factor incorporated two of the Wisconsin Card Sorting Test scores apart from those represented in a separate derived factor. It is possible that they are tapping into another aspect of cognitive flexibility, problem solving, or reasoning, however it is unusual for WCST scores to load separately. Thus, it is unclear what this finding means. The final factor appeared to represent an auditory processing component of attention, which is represented primarily in the temporal cortices, although the spatial aspects of auditory processing probably takes place in the nondominant parietal cortex (usually right hemisphere).

The Significance of Medico-Legal Issues

It was hypothesized that, of those cases presenting for neuropsychological evaluation due to a legal case pending, motivation levels and attempts at malingering would be sufficiently varied as to not represent a significant confound in this large sample. However, this was not the case. Participants who presented for a neuropsychological evaluation that were involved in seeking financial compensation differed significantly from those without financial incentive. Results strongly supported findings that individuals with potential financial gain are at risk for exaggeration of symptoms and invalid response sets, thus presenting a potential confound in empirical studies (Sweet et al., 1992).

Consequently, participants with a legal case pending were removed from subsequent analyses.

Depression and Attention

Clinical lore and increasing numbers of empirical studies inform us that depression can significantly limit attentional resources (Beck et al., 1979; Ingram, 1984; and Sweet et al., 1992). One goal of the current study was to explore the degree to which depression contributes to the variance in attention scores as measured by neuropsychological measures of attention. A second goal was to explore the degree to which individual neuropsychological measures of attention are affected by depression scores.

Two canonical correlations were performed (Tables 7 and 8). The first canonical correlation included measures of emotional distress (MMPI Scales 2 and 7) and neuropsychological measures of attention, while the second correlation included those same measures but also included the version of MMPI administered as a covariate, to ensure that this was not a potential confound. Moderate-to-high correlations were obtained in each canonical correlation between depression and attention measures (relationships with anxiety to be discussed in the next section), and though significant, the percent of variance contributed in each solution was surprisingly minute. The first canonical correlation accounted for 5% ($r = 0.23$) of the variance, and the second canonical correlation (with the covariate of MMPI version) accounted for slightly more, 8% ($r = 0.28$).

In the first canonical correlation, moderate-to-high correlations (>0.30 absolute value) indicated that as depression ($r = .45$) increased performance was adversely affected

on (in descending order) WAIS-R Digit Symbol, Dominant Finger Tapping, Trails B, Nondominant Finger Tapping, WAIS-R Digit Span, WCST Perseverative Errors, Rey-O Copy, and WCST Errors. When version of MMPI administered was added as a covariate, Rey-O Copy and WAIS-R Digit Span dropped out and WCST Perseverative Responses and Speech Perception Test were added (given a conservative >0.30 absolute value criterion). In descending order, as depression (r = .85) increased performance was adversely affected on WAIS-R Digit Symbol, Dominant Finger Tapping, Nondominant Finger Tapping, Trails B, WCST Errors, WCST, Perseverative Errors, Speech Perception Test, and WCST Perseverative Responses. Thus, it appeared that WAIS-R Digit Symbol, Finger Tapping, and the Wisconsin Card Sorting Test were most sensitive to the affects of depression on attention, followed closely by WAIS-R Digit Span, Rey-O Copy, and the Speech Perception Test. Consideration of these patterns with respect to the factor loadings discussed previously (in the context of Mirsky's findings), seemed to suggest that depression adversely affected the "Shift" and "Focus/Execute" components of attention and possibly some aspects of the "Encode" component.

Next, attention measures were analyzed by depression severity levels, while controlling for the effect of anxiety. Post-hoc procedures were performed to explore pairwise comparisons (Table 10). Results showed that performance on attention measures was significantly affected by depression severity. Increasing levels of depression resulted in poorer performance on Dominant Finger Tapping; WCST Categories, Errors, and Perseverative Responses; WAIS-R Digit Span and Digit Symbol; and the WMS-R Attention/Concentration Index. A curious finding was improved performance on Grooved Pegboard (Dominant and Nondominant) as depression increases from Moderate to High

Depression. In fact, for both variables mean scores were lower than the No Depression group (though this was not significant). Data were re-checked and confirmed on this result, and no reason could be determinded for such a finding.

Anxiety and Attention

Another goal of the current study was to explore the degree to which anxiety contributes to the variance in attention scores as measured by neuropsychological measures of attention. A second goal was to explore the degree to which individual neuropsychological measures of attention are affected by anxiety scores. As reported earlier, ample support exists for an adverse effect of anxiety states on cognitive performance, however some studies report equal or better performance of anxious individuals over controls (Dibartolo, et al., 1997; Eysenck & Calvo, 1992). Moreover, the Yerkes-Dodson Law (Revelle & Loftus, 1992) predicts optimal performance at moderate levels of anxiety.

The two canonical correlations discussed previously (Tables 7 and 8) included anxiety (MMPI Scale 7) as part of the emotional distress variable set. The first canonical correlation included measures of emotional distress and neuropsychological measures of attention, while the second correlation included those same measures but also included the version of MMPI administered as a covariate. Moderate-to-high correlations were obtained in each canonical correlation between anxiety and attention measures, and though significant, the percent of variance contributed in each solution was surprisingly minute. Furthermore, anxiety was much less correlated with attention measures than depression in both canonical correlations (<.30 absolute value). In fact, anxiety was inversely correlated

in the first canonical correlation (r = -.27) and positively correlated in the second canonical correlation (r = .29). Overall, the first canonical correlation accounted for 5% (r = 0.23) of the variance, and the second canonical correlation (with the covariate of MMPI version) accounted for slightly more, 8% (r = 0.28). However, given the small correlations for anxiety and reversed directional relationships, interpretations are extremely guarded. It is likely that depression contributed most of what little variance in attention was contributed to the solutions by emotional distress.

Next, attention measures were analyzed by anxiety severity levels, while controlling for the effect of depression. Post-hoc procedures were performed to explore pairwise comparisons (Table 9). Results showed that performance on attention measures was significantly affected by anxiety severity, but in a positive sense. Increasing levels of anxiety resulted in improved performance on Dominant Grooved Pegboard, Rey-O Copy, and the WMS-R Attention/Concentration Index. No significant adverse effects were observed on attention, even for participants in the High Anxiety group.

Improved performance with increasing anxiety supported the Yerkes-Dodson Law, however better performance at the High Anxiety level was not expected. In examining mean trends for all attention measures, performance was observed to be significantly improved for most variables at the High Anxiety level. This was possibly due to the assignment of all MMPI Scale 7 scores greater than T=70 to the High Anxiety group. If the floor were raised for admission to the High Anxiety group (e.g., T=80), it is possible that more high-anxious individuals would be reflected in the means and evidence worse performance. Put another way, perhaps individuals "captured" in the low end of the High Anxiety group are reflective of the "moderate anxiety" level described in the Yerkes-

Dodson Law, thus they perform optimally - which is reflected in the means of the High Anxiety group of the current study.

Depression and Anxiety Combined

The combination of both Low Depression and Low Anxiety (T > 45 and <55) did not exert adverse effects on attention performance. However, when the effects of both High Depression and High Anxiety were analyzed a surprising finding emerged. Performance improved on Verbal Fluency, Grooved Pegboard (dominant and nondominant), Rey-O Copy, and WMS-R Attention/Concentration Index. The only variable in which performance decreased was on the Speech Perception Test. It is not clear why such a finding would have emerged. One could speculate that the performance improvements noted previously in the separate analysis of High Anxiety (i.e., in the context of Yerkes-Dodson) overwhelms the effects of High Depression when they are experienced together. Another related possibility might be that participants reporting both high anxiety and high depressive symptoms are experiencing a more "agitated" depression versus one consisting of "psychomotor retardation."

Neurologics versus Non-neurologics

Two groups were identified: those participants with documented neurologic injury and those clearly absent neurologic injury. The neurologic group was comprised of almost half (49.2%) neurologic injury due to trauma. Nonetheless, it was a mixed neurologic group (i.e., multiple etiologies represented).

When groups were compared by performance on attention measures, the non-neurologic group outperformed the neurologic group as expected. The non-neurologic group performed better on Verbal Fluency, Grooved Pegboard, Finger Tapping, Trails A and B, Rey-O Copy, WAIS-R Digit Span and Digit Symbol, and the WMS-R Attention/Concentration Index.

When the two groups were compared by level of depression and anxiety, the neurologic group scored lower on depression and anxiety, which was unexpected. This finding was despite the fact that neurologic groups will often elevate on Scales 2 and 7 simply due to the endorsement of somatic complaints arising from their illness or injury (Gass & Wald, 1997). A likely explanation for decreased emotional distress in this group was impaired self-awareness (Prigatano & Schacter, 1991). Since nearly half of the neurologic group was composed of individuals with documented brain trauma, it is possible that those individuals had decreased awareness of their cognitive and physical deficits. With reduced insight into their deficits such individuals are less likely to develop a reactive depression, because they simply fail to appreciate how different they are compared to pre-injury functioning.

Next, the neurologic group was itself divided into depressed and non-depressed groups to assess the effect of depression on attention measures in this population. Significant differences were found on the Rey-O Copy, Category Fluency, and Grooved Pegboard. Performance was worse for the depressed group on Category Fluency, however the depressed group performed better than the non-depressed group on Grooved Pegboard (Dominant only) and Rey-O Copy. Anxiety level was not controlled in this

analysis due to low cell sizes, thus anxiety may have contributed to better performance by the depressed group.

The neurologic group was also divided into anxious and non-anxious groups to assess the effect of anxiety on measures of attention. Significant differences were found on Rey-O Copy, Verbal Fluency, Grooved Pegboard, and WCST Failure to Maintain Set. Performance on all variables was better in the anxious group.

Next, the non-neurologic group was divided according to depression level and compared on attention measures. Significant differences were found on Finger Tapping; Grooved Pegboard; Trails A; WAIS-R Digit Symbol; and WCST Categories, Errors, and Perseverative Responses. Performance was worse in the depressed group for all variables except Grooved Pegboard. Performance on Grooved Pegboard was better for the depressed group. Again, this may be due to the influence of anxiety level, which was not controlled for due to low cell sizes.

When the non-neurologic group was divided into two groups according to anxiety level and compared on attention measures, no significant results were obtained. However, results for Grooved Pegboard (Dominant) approached significance ($p = .067$) with cell means indicating improved performance by the anxious group. Non-significant results were likely due to low cell sizes.

While depression adversely affected attention performance in both neurologic and non-neurologic populations alike, it is likely that the depression experienced by both are indeed different. It is conceivable that depressive symptoms experienced by non-neurologic individuals are more chronic in nature, whereas depressive symptoms experienced by neurologic individuals are more reactive or situational in nature. If this is

indeed the case, not only would depressive symptoms in neurologic individuals be expected to be of shorter duration, but they may actually be a good prognostic sign. A sign that the neurologic individual is perhaps gaining better insight or awareness of their situation.

Relationship of Current Findings to Past Research: Contributions and Limitations

Findings from the current study are consistent with past research (Cohen et al., 1982, and Grant & Adams, 1986, Raskin et al., 1982, Sweet et al., 1992) and the stringent meta-analysis of Veiel (1997), which demonstrate an adverse effect of depression on cognition, especially attentional processes. The results of the current study do not readily support the position of researchers such as Reitan and Wolfson (1997) that cognition is unaffected by depression when compared to controls. However, the current study also revealed that depression actually contributed little (5%) to the overall variance in attention scores.

Increases in the severity levels of depression were commensurate with worsening performance on certain attention measures. This finding was consistent with the theoretical models proposed regarding cognitive processes in depressed individuals (Beck et al., 1979; Hasher & Zacks, 1979; Ingram, 1984; King et al., 1993; Pace & Dixon, 1993; and Schwartz & Garamoni, 1989). While specific findings with respect to poor performance on individual tests of attention cannot support one particular theory of cognitive processing over another, the findings do support the general premise germane to each of the models reviewed: over-emphasis on, or pre-occupation with, internal events or psychological processes at the expense of directing attentional resources out toward the

environment. The outcome of which is poor attention to external demands or tasks and poor encoding for later retrieval, which has implications for memory processes.

Empirical studies of anxiety and attention have been mixed. Some studies find improved attention in anxious individuals. Eysenck and Calvo (1992) described the Processing Efficiency Theory of anxiety, which predicts that under some circumstances performance will improve as anxiety increases. The present findings supported Eysenck and Calvo's theory. Attention, as measured by certain assessment measures, improved as anxiety increased. This was true even in the presence of high levels of depression. Specific to Processing Efficiency Theory is the notion that anxious individuals must expend more internal effort compared to controls to accomplish the same tasks. This process was not explored in the current study, therefore it is not known if anxious individuals in this sample found it necessary to expend more mental effort to achieve the improved attention scores. This is an empirical question open for future study.

In addition, findings from the current study demonstrated partial support for the Yerkes-Dodson Law (Revelle & Loftus, 1992). Attention scores improved for the High Anxiety group (as measured by Scale 7). Yerkes-Dodson posits optimal performance at Moderate levels of anxiety and decreasing performance at higher levels of anxiety. However, as mentioned previously the High Anxiety group in the current study was defined as scores on Scale 7 greater than T=70. Therefore, it is possible that the High Anxiety group in the current study "captured" some of the individuals at the low end of this group (e.g., T > 70 but less than 80) who could have been more appropriately labeled as a "Moderate Anxiety" group in another study focusing on the Yerkes-Dodson relationship. Furthermore, it is possible that further support for Yerkes-Dodson could

have been found if the High Anxiety group been further refined to represent individuals scoring at the upper bounds of Scale 7 (e.g., T > 80 or higher). It is possible that attention performance may have dropped off dramatically at the upper ends of the scale.

A limitation of the current study was the unfortunate exclusion of three important, and sensitive, neuropsychological measures of attention: the Paced Auditory Serial Attention Test (PASAT), Conners' Continuous Peformance Test (CPT), and Stroop Color and Word Test. These sensitive measures may have significantly added to the current findings. It is hoped that future studies exploring the issue of emotional distress and attention ability will include these important tests in their analyses. Another limitation of this study was the necessary combining of original MMPI and MMPI-2 data. Although it was statistically determined that the two groups had virtually non-overlapping distributions (i.e., very small effect size differences), it is possible that the fundamental scaling differences in the two instruments may have introduced one or more unknown confounds. Also, the use of a mixed neurologic group due to small cell sizes instead of using one category of brain injury (e.g., trauma or degenerative diseases) may have introduced a confound. Patients presenting with differing mechanisms of brain injury may perform differently on attention ability in the presence of depression and/or anxiety. Future studies could explore these issues with more homogenous neurologic populations.

The current study represents a unique contribution to the literature due to its large sample size, use of standardized measures, and diverse patient population representing a wide array of presenting etiologies within the context of a large academic medical center. The findings from this study provide important information regarding the relationship of emotional distress, specifically depression and anxiety, and its affect on

neuropsychological measures of attention. While one should be cautious in generalizing the findings to other populations not referred for neuropsychological evaluation (e.g., outpatient psychotherapy patients), the current findings represent an important addition to psychological and neuropsychological research.

Conclusions

It is widely held among psychologists that depressive and anxiety symptoms exert significant and deleterious effects on attention resources. In particular, it is believed by many neuropsychologists that depression and anxiety can adversely influence performance on neuropsychological measures of attention and memory. Although findings from the current study do not dispute those positions, the statistical results were not as robust as expected.

The findings suggest that depression exerts a more adverse effect on attention than anxiety. That being said, however, the contribution of depression in this study to the overall variance in attention scores was much less than expected (less than 1% of the variance, in fact) and may not be particularly meaningful clinically. Nonetheless, the modest findings obtained in this sample may be atypical. Adverse effects may occur, and clinicians should always be mindful of the potential deleterious influence of depressive or anxiety symptoms.

The Processing Efficiency Theory of anxiety (Eysenck & Calvo, 1992) predicts that anxious individuals may perform worse, the same, or better than non-anxious controls depending on the nature of the task. Depending on the variables and type of analysis performed, the current findings found either little-or-no effect of anxiety on attention or modest, inverse correlations (i.e., slightly worse performance).

When individuals presenting with a medico-legal case were analyzed separately, findings supported empirical studies show those groups are significantly different and, may in fact, present a confounding influence on research findings.

Finally, mixed neurologic cases were compared to cases without neurologic insult. Results demonstrated less depression and anxiety among neurologic individuals, however performance among non-neurologic individuals was superior to that of individuals with documented neurologic injury.

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APPENDIX A

Descriptive Statistics

Variable	N	Mean	Standard Deviation
Age	1209	42.01	16.61
Gender			
Male	673		
Female	536		
Education	1209	13.20	2.81
Race			
Caucasian	1079		
African American	76		
Native American	21		
Hispanic	6		
Asian American	5		
Other	3		
Unknown	4		
MMPI-2 L Scale	1209	52.10	7.68
MMPI-2 F Scale	1209	58.95	9.92
MMPI-2 K Scale	1209	50.42	9.31
MMPI-2 Scale D	1209	70.08	15.72
MMPI-2 Scale Pt	1209	65.13	14.00
WAIS-R FSIQ	1143	95.71	15.19
WAIS-R Digit Span	1140	8.78	2.69
WAIS-R Digit Symbol	1140	7.22	3.06
Oral Symbol Digit Modalities	34	45.68	12.04
PASAT Trial 1	30	10.57	8.54
PASAT Trial 2	30	14.93	7.97
PASAT Trial 3	30	20.73	8.92
PASAT Trial 4	30	26.00	8.83
WMS-R VMS Forward	138	7.83	2.05
WMS-R VMS Backward	138	6.92	2.26
WMS-R Mental Control	138	5.10	1.23
WMS-R Att/Conc Index Score	281	94.49	16.48

Seashore Rhythm	507	5.82	4.93
Speech Sounds Percep Test	509	7.99	5.34
Grip Strength - Dom	511	35.88	13.04
Grip Strength - Non-Dom	504	32.75	12.33
Grooved Pegboard - Dom	584	89.64	41.37
Grooved Peg - Non-Dom	583	99.30	44.16
Finger Tapping - Dom	539	44.04	9.72
Finger Tapping - Non-Dom	523	39.55	8.46
Trails A	921	44.33	33.87
Trails B	912	115.83	84.73
Word Fluency	409	32.15	11.71
Category Fluency	335	15.41	5.52
Stroop Word Score	19	93.11	15.08
Stroop Color Score	19	66.95	13.26
Stroop Color-Word Score	19	37.95	8.82
CPT Hits	51	318.24	6.77
CPT Omissions	51	5.76	6.77
CPT Commissions	51	12.53	8.47
CPT Hit Reaction Time	51	442.35	76.19
WCST Correct	232	69.17	15.09
WCST Errors	232	40.05	23.98
WCST Perseverative Resp	228	26.87	23.96
WCST Perseverative Errors	231	19.86	15.59
WCST Number of Categories	233	4.39	2.00
WCST Failure to Maintain Set	216	0.95	1.29

APPENDIX B

UNIVERSITY OF OKLAHOMA

GRADUATE COLLEGE

THE CONTRIBUTION OF DEPRESSION AND ANXIETY TO POOR ATTENTION PERFORMANCE ON NEUROPSYCHOLOGICAL ASSESSMENT MEASURES

A Dissertation Prospectus

SUBMITTED TO THE GRADUATE FACULTY

in partial fulfillment of the requirements for the

degree of

Doctor of Philosophy

By

SID DICKSON
Norman, Oklahoma
1999

THE CONTRIBUTION OF DEPRESSION AND ANXIETY TO POOR ATTENTION PERFORMANCE ON NEUROPSYCHOLOGICAL ASSESSMENT MEASURES

A Dissertation APPROVED FOR THE
DEPARTMENT OF EDUCATIONAL PSYCHOLOGY

BY

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The Contribution of Depression and Anxiety to Poor Attention Performance on Neuropsychological Assessment Measures

Depression and anxiety are ubiquitous in the patient populations with which neuropsychologists and clinical and counseling psychologists work (Sweet, Newman, & Bell, 1992). Recognition of the impact of emotional distress on cognitive processes, such as attention, is important for the work in which psychologists engage, particularly for assessment and psychotherapy. Decreased attentional skills due to emotional distress can have implications for psychotherapy by limiting the patient's ability to attend to, and follow, therapist questions and comments. Patient inefficiency in encoding important information relevant to therapeutic change may also hinder compliance and recall of assigned homework and treatment strategies. Within the field of neuropsychology, it is especially important to understand the impact of emotional distress on attentional processes. The ability to distinguish between disordered attention secondary to emotional distress and disordered attention due to organic etiology is of crucial importance. Understanding the extent to which emotional distress contributes to disordered attention can be critical to proper interpretation of neuropsychological assessment results, specifically deficits in attention and recall.

Interest in the effects of emotional distress and psychiatric disturbance on neuropsychological test performance is a relatively recent trend. Sweet et al. (1992) reported that, of 94 studies published on the effects of emotional distress on neuropsychological performance between 1960 and 1975, only 5 examined affective disorders, and of the 14 published between 1975 and 1978, only 2 were concerned with

depressive disorders. However, between 1978 and 1992 over 40 were published, with the majority of those having been published from 1986 to 1992. One reason for the lack of earlier empirical study in this area was a variety of methodological problems observed by Miller (1975). He noted a paucity of standardized measures across the studies at that time that limited efforts to compare findings. He also pointed out the inadequate diagnostic specificity and the use of differing diagnostic criteria prevalent during that time period. Since then, there has been an increased use of standardized neuropsychological tests and batteries and more rigorous diagnostic criteria through the development of the DSM-III-R (American Psychiatric Association, 1987) and subsequent DSM-IV (American Psychiatric Association, 1994; Sweet et al., 1992).

While increased focus on the effects of psychiatric disturbance on neuropsychological test performance is noteworthy, the empirical investigations of those effects have not been without potential confounds. According to Sweet et al. (1992), potential confounding, or moderating, variables in the investigations of neuropsychological performance primarily involve motivation, malingering, and medical factors (Sweet et al., 1992).

Richards and Ruff (1989) tested the hypothesis that reduced motivation accounts for cognitive deficits in depressed patients by randomly assigning two groups of subjects, depressed and nondepressed, to either a motivation or non-motivation condition. Motivation manipulation involved encouragement, a monetary incentive, and performance feedback. Results showed that motivation was indeed lower for depressed subjects, however it did not significantly affect neuropsychological performance. The authors

concluded that, although depressed patients may be less motivated, reduced motivation may not fully account for observed cognitive deficits in depressed patients.

Another potential confounding variable in empirically evaluating the impact of emotional disturbance on neuropsychological performance is malingering, or the "deliberate and conscious feigning of symptoms or the gross exaggeration of symptoms for the purpose of attaining monetary or other external rewards" (Sweet et al., 1992). Thought to be relatively rare, malingering can pose significant diagnostic and assessment difficulties by artificially generating increased mood symptom endorsement and inaccurate and false cognitive profiles. As such, they may artificially skew investigative findings.

Confounding medical factors mentioned by Sweet et al. (1992) include the pharmacological treatment of depression, anxiety, and other psychological conditions. Medications used in the treatment of these conditions have been shown in some studies to impair neuropsychological functioning, however results have generally been mixed. For example, some studies found improved cognitive performance following (primarily) pharmacological treatment of depression (Brumback & Staton, 1980; Fromm & Schopflocher, 1984). Others either observed no changes (Curran, Shine, & Lader, 1986; Telford & Worrall, 1978) or observed only perceptual motor slowing (Squire, Judd, Janowsky, & Huey, 1980). Commonly prescribed medications that were found to exert an adverse neuropsychological effect, at least in certain individuals, included anxiolytics, antidepressants, antipsychotics, anticonvulsants, and even antihypertensives (Dodrill, 1988; Hartman, 1988; Solomon et al., 1983; Trimble, 1987). Particularly troublesome was lithium's adverse affect on memory which, according to Jamison and Asiskal (1983), accounted for the most frequently reported side-effect leading to lithium noncompliance.

Shaw, Stokes, Mann, & Manevitz (1987) reported that over 80% of their subjects receiving lithium complained of neuropsychological side-effects. While some medications may cloud neuropsychological interpretation, many psychiatric and brain-injured patients usually perform better on neuropsychological measures due to a decrease in depression's negative influence on cognitive functioning (Glenn & Joseph, 1987).

Does Emotional Distress Have a Detrimental Effect on Cognition?

While the prevailing opinion in neuropsychology appears to be that emotional disturbances such as depression and anxiety do have an adverse affect on neuropsychological performance, there are those who contend otherwise (Sweet et al., 1992). In a lengthy review of the interaction of emotional disturbance with neuropsychological deficits, Reitan and Wolfson (1997) question this widely held assumption. They acknowledge, as most do, that intellectual and cognitive impairment represents a stressful situation that can cause emotional difficulties and problems of adjustment. They also agree that brain-injured individuals, if not experiencing impaired self-awareness (Prigatano & Schacter, 1991), experience a significant reduction in ability from his or her previous level of functioning which may cause anxiety, feelings of inadequacy in terms of meeting normal responsibilities, and feelings of depression due to failure and inadequate performance (Reitan & Wolfson, 1997). However, they do not readily accept the prevalent belief that emotional distress has a significant effect on neuropsychological functioning, and they point to studies that disagree with that contention. For example, Alvarez (1962) compared Trail Making Test (TMT; Reitan, 1979) performance in depressed versus brain-injured patients. He utilized 32 persons with

unequivocal brain damage (variety of conditions) and 32 patients with severe depression (33% had attempted suicide). He believed that depressed participants might be limited in their performances because of psychomotor slowness and a diminution of the effort needed to perform well. The TMT was selected because it requires "focused attention, selective responses to appropriate stimuli, and a deliberate effort to complete the task as quickly as possible" (Alvarez, 1962). Comparisons of the two groups on the MMPI (Butcher, Dahlstrom, Graham, Tellegen, & Kaemmer, 1989) indicated that the depressed patients had statistically significant elevations over the brain-injured group on Depression, Hysteria, Psychasthenia, Paranoia, Schizophrenia, and the F scale. Results on the TMT showed performance by the brain-injured group to be significantly poorer than the depressed group ($p<.001$) on Parts A and B. Moreover, performance of the depressed group was found to be "similar to that reported by Reitan for his non-brain-injured control groups" (Alvarez, 1962). The implication here by Reitan and Wolfson was that since depressed individuals performed better than brain injured individuals (i.e., similar to controls) they were not impaired by depression as measured by the TMT.

In another study cited by Reitan and Wolfson, Vingerhoets, DeSoete, and Jannes (1995) investigated the relationships between measures of emotional status and cognitive test performances in patients who were awaiting open-heart surgery and in the same patients following surgery. The researchers described an impending open-heart surgery as "one of the most frightening medical procedures", and they felt it provided a "natural stress paradigm" to evaluate the "impact of emotional state on neuropsychological test performance" (Vingerhoets, DeSoete, & Jannes, 1995). Measures of stress included anxiety, using the A-State scale of the State-Trait Anxiety Inventory (Spielberger,

Gorsuch, & Lushene, 1970), and depression, using the Beck Depression Inventory (Beck, 1987). Measures of neuropsychological performance included 11 tests "selected to cover an extensive range of cognitive functions." Both sets of tests were administered to 130 patients before surgery and 109 of the same patients 7-8 days after surgery. Not surprisingly, the results showed significant elevations for anxiety and depression before surgery. Following the surgery, anxiety and depression were lowered but only by a significant degree for anxiety. Correlational analyses were run between measures of emotional status and neuropsychological performance and the results showed no significant relationship for either pre- or post-surgical testing. According to Reitan and Wolfson (1997), results such as these suggest that "neuropsychological abilities are quite robust, even under conditions of rather striking personal stress and anxiety." The implication being that even high levels of stress and anxiety have limited-to-no impact on neuropsychological functioning.

What about clinical depression at levels requiring hospitalization? Kaufman, Grossman, and Kaufman (1994) used the Kaufman Short Neuropsychological Assessment Procedure (KSNAP) to compare cognitive performances of 56 hospitalized inpatients with clinical depression to normal matched controls. They analyzed results according to tests grouped in terms of the three functional units proposed by Luria: (1) attention-orientation representing a low level of cognitive complexity; (2) successive and simultaneous processes, representing an intermediate level of cognitive complexity; and (3) high-level planning ability, representing a high level of cognitive complexity. Results showed no significant differences at any task level, suggesting that clinical depression at levels

requiring hospitalization had no adverse effect on those cognitive functions measured by Kaufman and his colleagues.

Perhaps one of the more enduring assumptions in neuropsychology is the adverse effect of depression on the cognitive functions of the elderly, the so-called "pseudodementia" syndrome. As Reitan and Wolfson (1997) observe, a review of the literature by Bieliauskas (1993), concluded that "depressive-like symptoms have little or no impact on cognitive functions." He further asserted that, "the case for emotional influence on cognitive abilities in the elderly (i.e., pseudodementia) is vastly overrated", and "if elderly patients do present with cognitive difficulties, (they) are more likely disease-based rather than the result of emotional factors such as depression." Bieliauskas and colleagues conducted studies of their own in which nursing home patients and elderly medical outpatients were studied using various measures of cognition (Bieliauskas, Costello, & Terpenning, 1991; Bieliauskas & Lamberty, 1991; Bieliauskas, Lamberty, & Boczar, 1991). In all three studies, they found no significant effects of depression on cognitive abilities for their elderly samples. Although no significant effects of depression were observed in their samples, Bieliauskas allowed that there may have been an adverse influence on cognitive symptoms in patients with a psychiatric history of primary depression coupled with sufficient loss of self-esteem.

Reitan and Wolfson (1997) reviewed findings from studies of MMPI profiles in brain-injured individuals as well as individuals with emotional disorders. The MMPI is considered the instrument "used most frequently to assess the emotional status of patients referred for neuropsychological examinations" (Mittenberg, Tremont, & Rayls, 1996). As Reitan and Wolfson note, it possesses several advantages in the evaluation of personality

and emotional characteristics: it is self-administered and relatively easy to score; has empirically validated scales that have established meanings; is an objectively interpreted instrument; provides clear, valid descriptions of psychological problems, symptoms, and characteristics in a broadly acceptable clinical language; has clinical interpretation strategies that are easily learned; and possesses scales that have high reliability. Reitan and Wolfson reviewed studies of MMPI profiles in an effort to find support for the position that "significant emotional disturbances impact adversely on both neuropsychological test performances and on functional outcome" (Kay, 1993). In one study they reviewed, Gass (1991) analyzed a group of 105 patients referred for neuropsychological evaluation whose neurological examination did not identify any evidence of brain damage. The referrals were from a VA hospital consisting of psychiatric, neurologic, and rehabilitation services. Findings revealed "relatively weak relationships between MMPI indices and Halstead-Reitan Battery (HRB) scores." As a result, Gass concluded that, "as a general rule, these widely-used neuropsychological measures are largely resilient to the effects of emotional and personality factors in patients referred for neuropsychological testing." He further cautioned that a "conservative stance is recommended in attributing poor performance on these tests to psychological factors, particularly as inferred from elevated MMPI scores." Gass argued that HRB tests seem to be generally robust in the presence of psychopathology, and he asserted that "traditional interpretive lore that surrounds the use of various MMPI scores and patterns to make inferences with regard to cognitive functioning may be inaccurate."

In another study, Gass and Daniel (1990) evaluated the effect of emotional factors on Trail-Making Test – Part B performances. They concluded that performance on the

Trail Making Test was resistant to a variety of emotional influences and, though psychiatric symptoms and severe anxiety impairs performances, it is rarely to the extent caused by brain damage.

In a study investigating the relationships between MMPI scores of 59 psychiatric patients and measures of attention, concentration, and memory derived from the Wechsler Memory Scale (WMS), Gass, Burda, Starkey, and Dominguez (1992) found uniformly low correlations between MMPI variables and memory performance. They concluded patients' subjective complaints are unreliable indicators of actual ability. Taken together, it appears that Reitan and Wolfson (1997) have provided compelling evidence that emotional distress does not cause neuropsychological impairment equal to that seen in brain injured individuals. Nonetheless, their evidence also indicates that emotional distress can affect neuropsychological performance, at least at levels somewhere on a continuum between the performance of normal controls and that of brain-injured participants.

Supportive Evidence for an Adverse Effect of Depression on Cognition

Veiel (1997) performed a meta-analysis of "all studies published since 1975 and meeting stringent methodological and sample selection criteria" to assemble a profile of neuropsychological deficits of clinically depressed (major depression) but otherwise unimpaired individuals. Veiel's findings supported a profile "consistent with a global-diffuse impairment of brain functions with particular involvement of the frontal lobes." In fact, the severity of cognitive deficiencies he profiled were observed to be similar to those seen "in moderately severe traumatic brain injury" (Veiel, 1997).

Veiel's (1997) meta-analysis narrowed a large pool of research down to 13 studies using 6 stringent screening criteria, which he noted sacrificed research breadth for methodological stringency. The results of the analysis were grouped in the following nine categories: Attention/Concentration; Verbal Fluency; Scanning and Visuo-Motor Tracking; Verbal Learning-Acquisition; Verbal Learning-Retention/Retrieval; Nonverbal Learning-Acquisition; Nonverbal Learning-Retention/Retrieval; Visuo-Spatial Functions; and Mental Flexibility-Control.

Results for Attention/Concentration from the resulting 13 studies generally included only Digit Span Forward (only a few included Digit Span Backward of the Wechsler scales; Wechsler, 1981, 1987). Results showed only a 0.18 standard deviation between depressed and non-depressed controls. In the category of Verbal Fluency, tests included only the Controlled Oral Word Association Test (FAS test; Benton & Hamsher, 1989) and results showed a 0.55 standard difference. Scanning and Visuo-Motor Tracking included Trail Making Test Part A and the WAIS-R Digit Symbol subtest (Wechsler, 1981) and results revealed almost a full standard difference between groups at 0.93. Visuo-Spatial Functions included the following tests: Rey Complex Figure Test (Rey, 1964) and the Block Design and Object Assembly subtests of the WAIS-R (Wechlser, 1981). Results indicated a 0.81 standard difference between groups in this category. Verbal Learning-Acquisition included immediate recall from many of the standard memory tests and results indicated a 0.90 standard difference. Verbal Learning-Retention/Retrieval included delayed recall of at least several minutes, and findings showed a 0.91 standard difference. Nonverbal Learning-Acquisition and Nonverbal Learning-Retention/Retrieval had standard differences of 0.93 and 0.83, respectively, and

included visually presented material from recurring figures (Williams, Iacono, Remick, & Greenwood, 1990) and Rey's Complex Figure (Rey, 1964). The final category revealed the most striking distinction. Mental Flexibility and Control included measures commonly regarded as very sensitive to most kinds of brain dysfunction, and especially to frontal lobe dysfunction (Veiel, 1997): Trail Making Test Part B (time) and the Color-Word score of the Stroop Test. Results for this category was 2 full standard differences between groups. To summarize, the cognitive functions observed to be most affected by depression (i.e., above 0.50 standard difference) were, in order: Mental Flexibility and Control, Scanning and Visuo-Motor Tracking, Nonverbal Learning-Acquisition, Verbal Learning-Retention/Retrieval, Verbal Learning-Acquisition, Nonverbal Learning-Retention/Retrieval, Visuo-Spatial Functions, and Verbal Fluency. As Veiel (1997) observed, the obtained profile of cognitive deficiencies appeared "at first glance" to match that which would raise the question of impaired frontal lobe functions.

Sweet, Newman and Bell (1992) observed a similar profile in their review. They noted a pattern of "decreased cognitive efficiency or mild attentional or mild memory problems", typically evidenced by the following patterns: slowed information processing (e.g., slowness on all Stroop Color-Word pages), impaired word recall with normal recognition, impaired incidental learning with normal intentional learning, and impaired recall of easy word pairs (often with normal recall of difficult word pairs).

A number of studies have demonstrated a negative effect of depression on motor tasks. In a study by Raskin, Friedman, and DiMascio (1982; in Grant & Adams, 1986), 277 depressed patients were matched on age, sex, and education with 112 normal controls in a multicenter research project. Findings showed that depressed subjects performed

poorly on a number of motor performance tasks, including tapping, aiming, and circle tracing. Impairments were also found on nonsense syllable learning, Stroop scores, and the Clock Reversal Test. In another study, Cohen et al. (1982) examined motor performance (i.e., grip strength) and cognitive function (i.e., various mental tasks such as working with "trigrams") in depressed patients by severity level (severely depressed, moderately depressed, euthymic and normal mood). Results demonstrated deficits in motor and cognitive performance of depressed patients that appeared to be proportionate to depression severity.

Taken together, findings tend to consistently show that "cortically mediated intellectual functions are spared" (Grant & Adams, 1986), such as repetition, reading, naming, mathematics, and motor praxis. However, deficits that are more prominent tend to be those dependent on arousal, attention, and concentration. Grant and Adams (1986) observed that depressed patients suffer deficits in attention on tasks requiring "effort." Although some investigators conclude that depressed patients have "motivational disorders", Grant and Adams argued there is more to it than "motivation." For example, they ask how can one conclude that poor vigilance or grip strength can be simply due to lack of "motivation"? Ultimately, they concluded that depressed patients are simply "less with it" than unimpaired controls, suggesting a deficit in information processing. In profiling memory deficits of depressed individuals, they argued that the depressive state of the individual impedes the reception of new information as well as its initial processing. This ineffective initial acquisition appears to be central to later failures in recall. However, once information is encoded it appears that depressed patients tend to retain it. Retrieval deficits are common as well, especially for spontaneous recall, due in part to the poor

initial processing, however performance tends to improve on less stringent recall testing (e.g., recognition memory; Grant & Adams, 1986).

In summary, depressive states tend to exert adverse effects on cognition in the form of global and diffuse impairment of brain functions with particular involvement of the frontal lobes, noted in at least one study to reach levels seen in moderately severe traumatic brain injury. Adverse effects that have been noted include impairment in mental flexibility; scanning and visuomotor tracking; and both verbal and nonverbal acquisition, retention, and retrieval. Deficits have also been noted on measures of cognitive efficiency, attentional performance, information processing, and incidental learning. Cognitive functions that are typically spared tend to be those cortically-mediated intellectual functions such as repetition, reading, naming, and mathematics.

Supportive Evidence for an Adverse Effect of Anxiety on Cognition

There is evidence to suggest that anxiety, as a form of emotional distress, exerts an adverse effect on cognition. Anxiety, as with many psychological states, exists on a continuum of severity from simple worry and rumination to chronic and severe levels such as that observed in Post-traumatic Stress Disorder (PTSD; APA, 1994). The research presented below highlights studies that have focused on varying levels of anxiety and stress.

Pruzinsky and Borkovec (1990) recruited 56 college students comprising two groups, worriers and nonworriers. Subjects engaged in either brief relaxing imagery or stressful imagery. Before and after the imagery tasks, measures of focused attention and anagram measures were obtained. The results revealed that worriers reported more

negative daydreaming, greater difficulty with attentional control and greater obsessional symptoms. They also evidenced significantly more negatively affect-laden cognitive intrusions during relaxed wakefulness and focused attention.

In another study utilizing college students, Gillis (1993) investigated the hypothesis that stress impairs judgment (among other hypotheses which were not supported). He had 98 undergraduates complete a complex multiple-cue judgment task. Subjects were then assessed for (1) their exposure to two potential sources of stress, life events and irrational thinking and (2) the amount of personal dysphoria they were experiencing. Measures included the Life Experiences Survey, Dysfunctional Attitude Scale, State-Trait Anxiety Inventory, and Beck Depression Inventory. Results indicated that subjective distress, depression and state anxiety were significantly related to poor judgmental performance. In addition, results suggested that potential external sources of stress do not negatively affect judgment unless they generate subjective distress at the time those judgments are made.

High and low state anxiety was studied in a sample of community-dwelling elderly volunteers by Rankin, Gilner, Gfeller, and Katz (1994). Participants were administered the State-Trait Anxiety Inventory for Children, Mini-Mental Status Examination, and subtests of the Wechsler Memory Scale—Revised. Results indicated that anxiety (low and high) adversely affected sustained attention, but the findings were not significant for verbal and figural memory tasks.

If cognitive processes are affected by anxiety states at the lower end of the continuum, what about more intense pathological conditions of anxiety? One would reasonably infer that there is an inverse relationship where anxiety severity increases as cognitive performance decreases. However, "cognitive performance" covers a lot of

neuropsychological ground. It is possible that an inverse relationship exists but only for certain cognitive domains as was observed in a number of the studies on depressive effects on cognition. For example, in attentional processes, information processing, and encoding. Vasterling, Brailey, Constans, and Sutker (1998) investigated attention and memory performances in Persian Gulf War veterans with and without PTSD diagnoses. Veterans who were diagnosed with PTSD exhibited relative deficiencies in performance on tasks of sustained attention, mental manipulation (mental arithmetic), initial information acquisition, and retroactive memory interference. They also committed more errors of commission and intrusion. Veterans' tendencies toward response disinhibition and intrusion on cognitive tasks was positively correlated with re-experiencing symptoms and negatively correlated with avoidance-numbing symptoms. Vasterling and colleagues observed that the veterans' pattern of cognitive deficits were consistent with models of PTSD that emphasize the role of hyperarousal and involvement of frontal-subcortical systems. Moreover, their data suggested that intrusion of traumatic memories in PTSD might not be limited to trauma-related cognitions but instead reflect a more generalized pattern of disinhibition.

Individuals suffering from Obsessive Compulsive Disorder (OCD; APA, 1994) are also at risk for adverse cognitive effects due to anxiety. In one recent study by Clayton, Richards, and Edwards (1999) individuals diagnosed with OCD were studied along with a panic disordered and control group. Results showed significantly poorer performance on a series of psychometric tasks of selective attention. The researchers concluded that the data supported the hypothesis that OCD individuals have a diminished ability to selectively

ignore competing external (sensory) and internal (cognitive) stimuli, especially intrusive thoughts.

Further support was found by Schmidtke, Schorb, Winkelmann and Hohagen (1998). They investigated "frontal lobe performance" in 29 unmedicated OCD patients who were matched on age, gender, and intelligence with a double-size control group of normals. Participants were administered 12 neuropsychological tests, most of which are thought to be sensitive to different aspects of frontal lobe functioning. Results indicated that OCD patients were *unimpaired* on tests of abstraction, problem-solving, set-shifting, response inhibition, and reaction speed, however they evidenced deficits of approximately one standard deviation on timed tests of verbal and nonverbal fluency and attentional processing. Schmidtke and colleagues theorized that the obtained neuropsychological profile is related to "dysfunctioning within the anterior cingulate, but not the dorsolateral prefrontal circuit." Similar results were obtained by Veale, Sahakian, Owen, and Marks (1996) who found impairment on an attentional shifting task in 40 matched OCD patients.

Finally in another study of anxiety and cognition, Channon, Flynn, and Robertson (1992) compared 18 adults with Tourette syndrome with 22 controls. Participants were assessed on a number of clinical and experimental measures of attention and self-report measures of mood, anxiety, and obsessionality. Results revealed that the Tourette group was more depressed, anxious, and obsessional, and they performed worse on complex measures of attention, including serial addition, block sequence span (forward), trail-making, and a letter cancellation task.

In summary, anxiety states tend to exert adverse effects on cognition in the form of impairment in attentional control, sustained and selective attention performance, poor

judgment, attentional shifting, and initial information acquisition. Deficits have also been noted in mental arithmetic and verbal and nonverbal fluency. Evidence of overall disinhibition is characteristic as well, in the form of cognitive intrusions, retroactive interference and errors of commission and intrusions. Cognitive functions that were noted to be spared (in OCD patients) were abstraction, problem-solving, set-shifting, response inhibition, and reaction speed.

Cognitive Processes in Depression

To attempt to better understand obtained neuropsychological profiles, cognitive theorists have developed models of cognitive processing. One of the more compelling approaches is that articulated by Ingram (1984), the information processing model (or information processing "approach"). In the information processing model, network theory is utilized to conceptualize a process called "spreading activation." According to this theory, memory is composed of cognitive networks of associated concepts and descriptive propositions. Previous information and events that have been encoded into memory are represented by these propositions, and each memory unit is composed of a cluster of components (concepts and propositions) making up the memory. These clusters are referred to as memory "nodes." Network theory proposes that, in order for a memory or cognition to reach an individual's conscious awareness, its corresponding node must be activated above some minimum threshold. Once activation reaches a sufficient level, the person consciously experiences the memory.

Theoretically, there are at least two ways that a memory may be activated sufficiently to reach consciousness. One is through the presentation of an environmental

stimulus array that corresponds to an active memory node. Ingram presents the example of an individual who has been wanting to buy a Porsche. Although the individual might not be consciously experiencing thoughts about the Porsche, if one of them happens to pass by on the street the person will be "reminded" of the desire to own one Network theory would argue that this happens because the energy from the Porsche stimulus pattern activates the corresponding "Porsche memory node" to a sufficient level to reach conscious awareness so the individual is reminded of, and begins to think about, the Porsche.

A second way in which memory nodes may be activated is through the spread of activation. Network theory assumes that memories are connected with each other through associative linkages. Theoretically, memories that are similar conceptually, or have somehow become associated for the individual, are linked through associative pathways. The strength of these pathways is seen as a function of how strongly the memories are associated. Strongly associated memories will have strong and more closely associated linkages, whereas weakly associated memories will have weaker or perhaps no associative pathways. Presumably, when a memory is activated, activation is presumed to spread along its associative pathways causing other memory nodes to become more likely to be activated. Memory nodes which stand the highest chance of being activated in this manner are those that are connected through the strongest associative pathways. It is theorized that this spreading activation of memories may be analogous to the person's stream of consciousness. To return to the Porsche example, not only is the person reminded of the desire to buy one, but a flood of associated cognitions may become conscious as well,

such as the inability to buy one on his present salary, the need for a raise or promotion, or a project under way that could lead to a promotion, and so on (Ingram, 1984).

With regard to affect, Ingram proposes that affect can be conceptualized in terms of affective structures called "primitive emotion nodes." Each specific emotion such as depression, anger, joy, or fear is theoretically represented by a particular node or unit in memory. Connected to each emotion node is a set of features associated with the emotion, such as its subjective experience, its unique autonomic response pattern, verbal labels used to describe the emotion, and cognitions containing descriptions of events that evoke that emotion. When a particular node is activated, the emotion is experienced and activation is channeled through its interconnections to evoke the emotion's other manifestations. Additionally, each emotion node is thought to be associated with a particular cognitive network consisting of emotion-related memories and cognitions. Although some connections to the emotion node are believed to be innate, such as the connection to nodes that trigger autonomic responses, cognitive linkages are largely learned and are generally established through "contiguity" during life events (Ingram, 1984). Ingram presented the example of attending the funeral of a friend. A link is established when the sadness felt at the funeral (depression emotion node activation) becomes associated with a cognitive node representing descriptions of funerals. Thus, through acculturation, learning, and innate programming, emotion nodes are viewed as being linked with particular cognitive networks containing emotion-congruent content.

Components of the information-processing framework that are particularly relevant to the present study of attention and general cognition are the "depth of processing model" and "cognitive capacity." As network theory seeks to describe the

structure of memory, depth of processing seeks to describe the process by which information is encoded into memory. At its basic level, the depth of processing model proposes that information is more likely to be fully perceived and encoded when it is processed "deeper," where depth refers to the degree and extent of cognitive analysis the information receives. In other words, the more cognitive analysis a piece of information receives, the more likely it is to be comprehended and understood. Ingram (1984) refers to this cognitive analysis as "cognitive elaboration." That is, information that receives analysis is elaborated upon cognitively. When received, information is processed at different cognitive depths. These different depths may be viewed as corresponding roughly to different cognitive networks, with larger and more intricate networks being seen as representing deeper and deeper cognitive depths.

The concept of cognitive capacity has sometimes been referred to as the same as attention or consciousness (Lachman, Lachman & Butterfield, 1979). According to the model, individuals have processing limits. They can only process a finite amount of information at any given time, or, stated another way, the amount of attention they can pay is limited. An individual's processing capacity is limited, and when this capacity is exceeded, no more information can be attended to or processed. Not all information utilizes the same proportion of capacity, however. For example, the information required to drive a car may engage very little cognitive capacity if the person has driven a car for a long time (i.e., an overlearned skill), on the other hand it may engage large portions of cognitive capacity if the skill is just being learned. To tie this concept back in with the depth of processing model discussed above, in order for an individual to process information at a deeper level (i.e., increased cognitive elaboration) a relatively larger

proportion of cognitive capacity must be utilized. That is, it will take nearly all of their attention.

So, how is depression presumed to affect these cognitive processes? The information-processing model of depression acknowledges that any psychological state is a complex process involving the interaction of a variety of factors, however it views those factors as converging upon a basic mechanism called the "depression emotion node" (Bower, 1981 as cited in Ingram, 1984). It proposes that the phenomenological experience of depression, along with the onset of depressive symptoms, results from the activation of an individual's depression node (although other emotion nodes may be activated concurrently). This activation of the depression node is determined by the appraisal of life events, referring to the manner in which life events are linked to the contents of existing cognitive structures (e.g., attitudes, beliefs). Appraisal is viewed as the process that gives subjective meaning to external events and is generally thought to be determined by an individual's (1) beliefs about the parameters of a particular life event, and (2) beliefs as to the effects of the event. For example, suppose a woman has been left by her husband for another woman. If she believes that she will never see him again (a parameter of the event) and that she will not be able to function without him (a perceived effect of the event), then it is likely that the event will be cognitively appraised in such a way as to activate her depression node.

A variety of factors are assumed to either shorten or lengthen the duration of depressive episodes, however in the absence of those factors it is believed that the underlying mechanism that determines depression duration is level of activation. Once activated, a depression node is thought to experience a period of decay until the activation

level falls below a threshold, at which time the individual no longer experiences depressive affect. The higher the initial activation level, the longer it will take to decay to subthreshold levels. The initial activation level is, in turn, determined by the value that the individual places on the negative event (i.e., how it is appraised).

The activation of a depression node is presumed to be necessary and sufficient to cause depressive affect (Ingram, 1984), however the information processing model postulates a somewhat different set of cognitive processes and mechanisms which act to maintain the depressive affect. The depression node is viewed as a central part of certain cognitive networks with associated linkages to various other units in the network. Due to the associative nature of the networks, it is thought that other units will be the representations of past events associated with depression. In addition, cognitions that were related to past depressive feelings would also be linked to the network. These particular memory units will in turn be linked to other units with which they have become associated through past experiences, however the strongest associative links will be with the memory units representing the present depressing situation.

The concept of spreading activation maintains that, when the depression node is activated above the threshold, activation spreads through the depression-associated network, causing its various contents to become more likely to be brought to conscious awareness. This presumably may set up a "cognitive loop" process where thoughts, memories, and associations consistent with an individual's mood become more accessible to the individual (Clark & Isen, 1982). Importantly, due to the depression node being relatively central to this particular network, it is thought that as activation cycles through the network, it is eventually fed back (though at a slightly lower level due to signal decay),

causing the depression node to remain activated. For the person experiencing this, it is as if negative memories and cognitions keep coming back again and again to consciousness, thus maintaining the depressive feelings.

Important to the present study is the notion of "available cognitive capacity." As activation spreads it is viewed as occupying a proportional amount of the person's available cognitive capacity. With greater magnitudes of activation, there are greater levels of spreading activation and more associations in the network are activated above consciousness. As those associations are activated above threshold and the person begins to actively think about them, a larger proportion of the limited capacity is engaged. Consequently, persons undergoing a depressive episode will have a high degree of attention focused upon themselves and their cognitions as available capacity becomes increasingly occupied by spreading activation (Ingram, 1984).

Ingram illustrates this process with the following example. Suppose an individual has just lost an important job. It is assumed that this loss will activate the person's depression node, which in turn sends activation spreading throughout its associated network. In other words, in addition to the initial depression that the individual feels, he will think about losing the job and will be more likely to think about past depressive experiences (as depression-associated memory nodes are activated). As more and more associations become activated, related past cognitions that are related to depression may be experienced (e.g., guilt, self-degradation, low self-esteem). Thus, as the individual has depressive cognitions, the depression is maintained as activation is recycled back to the depression node.

When presented with a task or new information, not only is adequate available cognitive capacity necessary to attend to it but the incoming information is addressed according to its similarity to the person's current cognitive contents. The implication of this is that, unless the new task or information is unusually strong (to exceed activation threshold), information that is not particularly relevant to current cognitive content will not be processed (or not fully processed) because it is not associated with the presently active network (i.e., it is not related to the depression-associated network and is more related to networks that are presently inactive).

Evidence for this limited cognitive capacity or "limited resource" hypothesis has found support from studies of memory and aging and mixed groups of bipolar and unipolar depressives (see King, Caine, & Cox, 1993) and other studies of self-focused attention (Ellis, 1991; Ingram, Lumry, Cruet, & Seiber, 1987; Lemelin, Baruch, Vincent, Laplante, Everett, & Vincent, 1996).

Other related and empirically-supported hypotheses posited to explain depressives' poor attentional (and memory) performance are: automatic versus effortful processing and the reduced initiative hypothesis. First articulated by Hasher and Zacks in 1979 (see King, Caine, & Cox, 1993), effortful and automatic processing are distinguished by processes that are either intentional and conceptually driven (effortful) or automatic and "data driven" (automatic). These processes are usually apparent in explicit versus implicit memory tasks, where explicit memory tasks are effortful and implicit memory tasks are automatic. Explicit memory tasks are those in which recall and recognition are assessed directly with the person being conscious of the task and requiring conceptual processing (e.g., recalling a word within the context of a list). In contrast, implicit memory tasks do

not necessarily involve the person's awareness and are thought to be an unconscious activation of an item (e.g., a word) in the person's lexicon that subsequently makes the item more accessible to consciousness.

Studies of implicit versus explicit memory indicate that depressed individuals are generally more impaired versus controls on explicit (i.e., effortful) memory tasks. For example, in an extensive review of such studies Hartlage, Alloy, Vazquez, and Dykman (1993) concluded the following: (1) Depression interferes with effortful processing, and the degree of interference is determined by the degree of effortfulness of the task, the severity of depression, and the valence of the stimulus material to be processed; and (2) depression interferes only minimally with automatic processes.

The reduced initiative hypothesis stipulates that depressives' poor attentional performance is due to a tendency to be self-focused and to fail to direct sufficient attentional resources to an external task. For example, Channon, Baker, and Robertson (1993) found that deficits in short-term memory of depressed patients was the result of difficulties in attentional regulation, rather than a "simple defect in storage capacity." In a dramatic demonstration of this process Hertel and Rude (1991) showed that, by experimentally eliciting increased "focused attention", they were able to improve memory scores of depressed patients such that it matched the performance of formerly depressed (i.e., recovered) patients and non-psychiatric controls. Thus, these studies would suggest that attentional resources of depressed patients, given the right conditions, might be shifted "outward" to focus on external tasks.

Another hypothesis investigating the effects of depression on cognition is that of selective attentional, or negative bias. It is hypothesized that depressives have enhanced

memory (and possibly attention) for negatively toned material (King, Caine, & Cox, 1993). For example, on memory tasks depressed patients remembered anxiety-provoking information more vividly and "agreeable" information more poorly than did controls (Mialet, Pope, & Yurgelun-Todd, 1996). A study by Pace and Dixon (1993) supports this hypothesis. In a study examining the effects of 6-8 sessions of Beck's cognitive therapy on mildly and moderately depressed college students' depressive symptoms and depressive self-schemata, results demonstrated not only that depressives show a clear preference in recall for negative self-referent judgments, but that brief cognitive therapy can significantly decrease that negative bias.

In 1989, Schwartz and Garamoni described an information-processing model of positive and negative cognition, the States of Mind (SOM) model. Based on the "golden section proportion", an extensively studied phenomena in personal construct psychology whereby individuals differentiate dichotomous judgments in a ratio of approximately 61.8% to 38.2% (e.g., in a balance of positive (P) and negative (N) adjectives, P/(P+N)=.618), the States of Mind model proposes five distinct states of mind that are conceptualized in terms of cognitive balance and quantitatively defined by homeostatic set point ratios of positive cognitions to total positive plus negative cognitions.

The five states of mind consist of three SOMs (positive dialogue, internal dialogue of conflict, and negative dialogue) that retain a dialectical interaction between positive and negative thoughts and two SOMs (positive monologue and negative monologue) that are imbalanced positively or negatively to the degree that they virtually abandon a dialectical process. Each SOM is defined its ratio of positive to negative cognitions. The most optimal state of mind, positive dialogue, is characterized by a ratio of .618 positive

cognitions to .382 negative cognitions. It allows a general positivity in cognition and mood while preserving maximal attentiveness to negative, threatening events. The individual with this SOM will readily recognize negative cognitions and likely engage in sufficient facilitory self-talk and positive coping strategies to alleviate distress.

Negative dialogue is a SOM characterized by a ratio of .382 positive cognitions to .618 negative cognitions. Individuals with this SOM maintain a background of continual, moderate negativity such that, when negative events occur, they are less shocking and more easily assimilated into existing structures. Such persons are usually observed to be moderately anxious or depressed. The system strives to maintain a "preferred" (or homeostatic) level of dysphoria or fear.

The internal dialogue of conflict SOM is characterized by symmetrically balanced structures such that the ratio of positive to negative cognitions is .50. This results in equal salience of positive and negative information as well as maximal uncertainty, a state that is not optimal as it is associated with indecision and doubt. Clinically, it may manifest as mild levels of anxiety, depression, and obsessionality.

Positive monologue is one of the SOMs characterized by a ratio that is asymmetrical to the degree that a dialectical process is virtually abandoned. It consists of positive cognitions with a ratio of .69 or more. In this state, positive thoughts and feelings exceed the optimal balance. Ratios in these ranges (i.e., that are monologic versus dialogic) are inherently unstable, and there is a tendency to strive toward a more balanced and less extreme dialogic SOM. Clinically, this excess of positive cognitions may manifest as mania or hypomania. With rising positivity, there is reduction in uncertainty at the

expense of a loss of salience of negative events. Consequently, important threatening events may go unnoticed to the detriment of the individual.

At the other end of the spectrum is the negative dialogue SOM characterized by positive cognitions at a ratio of .31 or less (and negative cognitions at .69 or more). According to Schwartz and Garamoni (1989), this SOM is not as enduring as the other states of mind (especially the dialogic SOMs) and is more transitory, which may explain the tendency for some severe unipolar depressives to experience spontaneous remission. The negative monologue is usually associated with extremely severe psychopathological states and is characterized clinically by profound depression or acute panic. It is qualitatively distinct and exhibits "distinct structural and information processing properties. In addition to its hypothesized instability, there are "internal and external pressures" exerted on the individual to move towards more positive thinking - to reestablish a more balanced, dialogic SOM (Schwartz & Garamoni, 1989).

Garamoni et al. (1991) found support for the States of Mind model in a study of 39 outpatients with major depression. Correlational analysis confirmed that the balance of positive and negative affective symptoms (P/(P+N)) in this sample closely approximated the golden section (i.e., 0.37). More recently, Nasby and Russell (1997) investigated whether the States of Mind model could successfully differentiate between Vietnam combat veterans who suffered from posttraumatic stress disorder (PTSD) and Vietnam combat veterans who did not. After matching the groups by combat exposure and controlling for general psychopathology, their results demonstrated more maladaptive SOM in PTSD veterans than combat veterans without PTSD.

Psychophysiological Measures

In addition to neuropsychological measures of cognitive performance to ascertain differences in depressives, psychophysiological mechanisms can also be assessed and compared to controls. As a component of their study on focused attention, Hertel and Rude (1991) incorporated a reaction time component to assess participants' mental demands. Reaction time to an auditory signal was assessed and found to be significantly slower in the depressed group. Moreover, reaction time was not affected by increases in attentional focus, though it was in the control group (i.e., increased attentional focus improved reaction time in controls).

Psychophysiological studies which are particularly relevant to attention are those of evoked potentials and contingent negative variation (CNV). Evoked potentials are waves of cerebral electrical activity arising in response to a sensory stimulus (auditory, visual, or somesthetic). These potentials can only be observed by repeating the stimulus, averaging several repetitions, and subtracting the background noise of the electroencephalogram (EEG). The early components of the evoked potential (i.e., before 100 milliseconds) are usually linked to characteristics of the stimulus, however components which arrive later are related to the attention which the stimulus has aroused. For example, the evoked potential commonly used is the "P300" (positive deflection, 300 ms) component. At the level of the P300, studies of depressed patients unanimously show a reduction in amplitude (Mialet, Pope & Yurgelun-Todd, 1996). Yee and Miller (1994) compared dysthymics to anhedonic and normal controls on P300 evoked potentials and found that dysthymic individuals are hyporesponsive at various stages of information processing. Yet it was unclear whether they were deficient in the amount of available

attentional resources for information processing or deficient in the allocation of those resources.

In addition to P300 potentials, recent studies have attempted to utilize changes in the attenuation of the "N2 Vertex wave" in participants during a selective attention task (Mialet et al., 1996). Changes observed are suggestive of a deficit of the "attentional trace" at the initial stage of information processing (El Massioui, 1988). Mialet et al. (1996) interpret these changes as evidence of limited availability of attentional resources in depressed patients, thus constraining them to "mobilize controlled attention for operations that would normally be easily handled by automatic processing."

Contingent negative variation (CNV) consists of a slow negative potential which develops in the frontal regions of the brain during the interval between an alerting stimulus and a response and represents the physiological correlate of anticipation in a motor or mental performance (Mialet et al., 1996). In summarizing the findings of CNV in depressives, Mialet and colleagues report a clear decrease in the amplitude of the CNV and an association of CNV with relative insensibility of depressives to contextual (environmental) cues.

Additional studies investigating speech (larynghographic recordings), eye movements, and visual tracking in depressives have also revealed impairment, lending additional support for the hypothesis of an overall reduction in depressed patients' attentional performance (Mialet et al., 1996). In fact, Mialet and colleagues compare attention in depressives to that of schizophrenic patients, stating that depressives display an "impoverished intensity" of attention rather than an impaired ability to orient their attention.

Cognitive Processes in Anxiety

As reported earlier, there is ample support for an adverse effect of anxiety states on cognitive performance, however there are studies which have actually reported equal or better performance of anxious individuals over controls on cognitive performance (Dibartolo, Brown, & Barlow, 1997; Eysenck & Calvo, 1992). To attempt to account for the discrepancy in findings, Eysenck and Calvo (1992) developed the processing efficiency theory of anxiety. Processing efficiency theory conceptualizes *state* anxiety as the crucial factor determining individual differences in internal processing and performance. Eysenck and Calvo (1992) assert that state anxiety is determined through an interaction of trait anxiety and situational threat or stress, however they also note that it is often difficult to distinguish between trait and state anxiety empirically because they both correlate very highly (often 0.70 or higher). Thus, many studies do not attempt to disentangle the two forms.

Two components of anxiety, worry and emotionality, are identified in processing efficiency theory. Emotionality refers to an individual's awareness of the physical aspects of anxiety (e.g., increased heart rate, or sweating), whereas the worry component consists of self-preoccupation, concern over evaluation, and concern over level of performance (Eysenck & Calvo, 1992). The worry component is viewed as occupying the cognitive component of state anxiety, and it is hypothesized to play an integral role in the articulatory loop of the working-memory system devised by Baddeley (1986; see Eysenck & Calvo, 1992). Baddeley's working-memory system consists of three components, all having limited capacity: a modality-free central executive resembling attention, which is

involved in active processing; an articulatory loop specializing in rote verbal rehearsal used for temporary storage of verbal information (often involving subvocalizations); and a visual-spatial sketch pad specializing in visual and/or spatial information. The central executive is considered the most important component of the working-memory system, and it is used on tasks that require planning or decision making and as a problem-solver when lower processing systems seem inadequate.

According to Eysenck and Calvo (1992), the worry component of anxiety is not only thought to consume some of an individual's valuable processing and storage resources, but is also thought to serve a motivational function via a "control system". Importantly, the functioning of this control system leads to an allocation of additional processing resources (i.e., effort) and to the initiation of processing activities (e.g., strategies) designed to improve performance, especially when the person perceives their performance as poor (or is given feedback to that effect). If successful, such attempts at allocating additional resources increase available working memory capacity and may, in fact, *improve* performance provided there are not undue demands placed on their overall resources (elaborated further below).

A fundamental theoretical distinction set forth in processing efficiency theory, one which seeks to explain the inconsistent findings in other studies of anxiety and performance, is that between performance effectiveness and processing efficiency. Performance effectiveness simply refers to the quality of *task performance* (as in test scores), whereas processing efficiency refers to the *relationship between* the effectiveness of performance and the effort, or amount of processing resources, invested. This

relationship is defined as performance effectiveness divided by effort, though "not in a strict mathematical sense" (Eysenck & Calvo, 1992).

Processing efficiency theory differs from other theories of anxiety and performance in a number of ways. First, worry has motivational as well as attentional interference effects. Second, the motivational effect is generally positive in that it leads to increased effort or compensatory strategies. Third, the notion that the effects of anxiety on performance differ from the effects on performance *efficiency* is unique to this theory. Fourth, anxiety affects both the storage and the processing capacity of the working-memory system which are available for task performance, rather than simply the storage capacity of short-term memory or attentional resources as hypothesized in other theories. Finally, at a conceptual level it is assumed within other theories (see Eysenck & Calvo, 1992) that stressful situations or threats cause anxious individuals to respond relatively passively with self-preoccupation, which either interferes with attention to task-relevant processing or motivates them to avoid the task, thus reducing on-task effort. Performance efficiency theory, on the other hand, assumes that anxious individuals respond to stressful situations *dynamically*. They worry about possible aversive consequences but try to avoid them by allocating further resources to the task, however that strategy can only be useful to a point given the finite resources available at any given time.

To elaborate, when presented with a simple task, anxious and non-anxious individuals are roughly equal in the amount of attentional resources required for the task. However, when task demands increase, the amount of attentional resources required by each begins to diverge. The central executive (of the anxious individual) begins to direct additional resources to the task, and if the individual is aware of poor task performance,

he will begin to worry (in the articulatory loop) about adverse consequences of his poor performance and will be motivated to improve that performance. This motivational function entails the allocation of further resources (i.e., effort) and processing activities (i.e., strategies) in an attempt to improve performance. This compensatory response may lead to improved performance but not always. Objectively, task performance, as measured by test scores, may not reveal any significant differences between anxious and non-anxious controls unless secondary (concurrent) tasks are employed to further tax the capacity of the available resources. It is then that differences are often detected, highlighting the anxious individual's re-distribution of resources in an effort to compensate for poor task performance. Numerous confirmatory studies have been conducted on performance efficiency, utilizing ever-inventive research strategies to uncover this underlying process (Eysenck & Calvo, 1992; Elliman, Green, Rogers & Finch, 1997; Dibartolo et. al, 1997).

Eysenck and Calvo (1992) outline the following predictions of the processing efficiency theory, all of which are supported by empirical studies cited in their monograph: (1) Anxiety typically impairs processing efficiency more than performance effectiveness, and (2) adverse effects of anxiety on task performance generally become stronger as task demands on working memory capacity increase. The implications of the first prediction, also supported empirically, are as follows:

(a) highly anxious participants should report higher levels of subjective effort than low-anxious participants on comparable tasks
(b) anxiety will typically have an adverse effect on a secondary task performed concurrently with a primary task
(c) anxiety will reduce spare processing capacity (assessed by responding to probes) during the performance of a central task

(d) motivational factors enhancing effort typically benefit the performance of low-anxious individuals more than high-anxious individuals
(e) the performance of a central task will be adversely affected by an additional load to a greater extent in anxious than in non-anxious groups
(f) impaired processing efficiency produced by anxiety can be detected by lengthened processing time (e.g., response or reaction time)
(g) the greater impairment effect of anxiety on efficiency than on effectiveness can be detected by psychophysiological measures

The second prediction by Eysenck and Calvo, that adverse effects of anxiety on task performance generally become stronger as task demands on working memory capacity increase, includes the following empirically supported implications:

(a) the effects of anxiety on task performance depend on the amount of resources required by the task (measurable by the susceptibility of that task to interference by a concurrent load)
(b) anxiety reduces temporary storage capacity
(c) anxiety has powerful adverse effects on tasks with high storage and processing demands
(d) anxiety does not generally impair performance on tasks not involving the central executive and/or articulatory loop components of the working-memory system (e.g., undemanding or automatic tasks)

Processing efficiency theory has held up well under empirical scrutiny. As recently as 1997, Elliman and colleagues examined the effect of anxiety on three groups of anxious individuals consisting of low, medium, and high anxiety. Participants performed a high-processing load measure of sustained attention, including accuracy and response scores, and basic measures of psychomotor performance. Results revealed that high levels of anxiety were associated with longer response times and a decline in performance accuracy. Since all participants performed equally well on basic psychomotor speed, the researchers were able to demonstrate that the increase in response times was unique to the high-anxious group. In other words, high-anxious individuals used more processing capacity

(and thus more response time) than their lower anxious counterparts. Thus, they were performing less efficiently as they attempted to maintain similar levels of performance.

Finally, in a study by Dibartolo and colleagues (1997), participants with generalized anxiety disorder (GAD) were compared to normal controls on two information processing signal detection tasks. The first task consisted of neutral distractors, and the second consisted of administering negative feedback cues. GAD participants evidenced impaired performance on the first task, however they unexpectedly improved on the second task to match normal controls despite reporting significantly higher levels of worry and negative affectivity. Dibartolo and colleagues interpreted this finding as demonstrating Eysenck and Calvo's (1992) concept of motivational functioning within the articulatory loop and central executive. In other words, they believed GAD participants experienced increased worry about their performance and consequently reallocated additional resources to the second task condition. Thus, improving their scores.

Presumed Anatomical Basis for Attentional Processes

Drawing on earlier work conducted with schizophrenia and other disorders affecting attention, Mirsky (1987) postulated that attention could be subdivided into a number of different elements, including the capacity to focus on or select some aspect of the environment, the ability to sustain or maintain focus for a period of time, the ability to encode or manipulate information held in memory, and the ability to shift adaptively from one aspect of the environment to another. These four elements of attention (focus-execute, sustain, encode, and shift) can be measured by a variety of neuropsychological

tests of attention. In a study conducted through the Laboratory of Psychology and Psychopathology of the National Institute of Mental health (see Culbertson & Krull, 1996) a factor analysis was used to analyze a battery of neuropsychological tests thought to be sensitive to various aspects of attention. The analysis yielded four factors which supported the aspects of attention proposed by Mirksy (1987). As reported by Culbertson and Krull (1996), the factor associated with the "focus-execute" component was related to perceptual motor speed and loaded on such tests as Trail Making (Reitan, 1979), a letter cancellation procedure, a measure similar to Coding from the Wechsler scales (Wechsler, 1990), and the Stroop test (C.J. Golden, 1978). The factor associated with the "sustain" element of attention was vigilance and loaded on scores from a Continuous Performance Test (omissions, commission errors, and reaction time; Rosvold, Mirsky, Sarason, Bransome, & Beck, 1956). The "encode" element was best captured by a factor loading on numerical or mnemonic tasks, as on Digit Span and Arithmetic from the Wechsler scales. Finally, the "shift" element of attention was measured best by a factor loading on flexibility, as measured by scores from the Wisconsin Card Sorting Test (WCST; Heaton, 1981).

Mirsky (1987) integrated the neuropsychological testing data and findings from previous neuroanatomic studies to arrive at descriptions of the neuroanatomic localization of the elements of attention. The following functional specialization was suggested: The "focus-execute" function is likely shared by superior temporal and inferior parietal cortices, as well as by structures that make up the corpus striatum (including the caudate, putamen, and globus pallidus). The motor component of the execute function is felt to be mediated by the inferior parietal and corpus striatal regions of the brain. The "sustain"

function of attention is thought to be mediated by rostral structures, including the tectum, mesopontine reticular formation, and reticular and midline thalamic nuclei. The "encoding" function is believed to be accomplished by the hippocampus, which provides an essential mnemonic function required for some aspects of attention. Finally, the ability to "shift" attention from one aspect of the environment to the other is supported by the prefrontal cortex. As Mirsky observed, this attentional system within the brain is very widespread and, therefore, quite vulnerable so that damage or dysfunction in any one of these brain regions can lead to specific deficits in attentional function.

Purpose of Study

Depression and anxiety are encountered on a frequent basis in psychological and neuropsychological patient populations (Sweet, Newman, & Bell, 1992), therefore it is paramount to have an adequate understanding of the impact of these conditions on cognitive processes. As mentioned previously, decreased attention can have significant

implications for psychotherapy by limiting the patient's ability to attend to, incorporate, and apply therapeutic principles and insight. For the neuropsychologist, it is crucial to appreciate the role of emotional distress in impaired attention to help clarify its contribution versus CNS injury.

Empirical studies exploring the relationship of emotional distress and neuropsychological test performance are a relatively recent trend and results have been mixed due to various methodological problems (e.g., not using standardized measures, inadequate diagnostic specificity, small sample sizes; Sweet et al., 1992). Furthermore, the very notion of emotional distress exerting a significant impact on attentional processes has been called into question (Reitan & Wolfson, 1997).

The present study seeks to determine the impact of differing levels of anxiety and depression on attentional performance in both patients with neurologic insult and those without. It represents a unique contribution to this area of research due to its large sample size, use of standardized measures, and diverse patient population characterizing a rich array of presenting etiologies within the context of a large academic medical center. Based upon the existing literature reviewed, the following research questions and hypotheses were generated.

Research Questions and Hypotheses

Since patients were routinely administered the MMPI as part of a flexible neuropsychological battery, MMPI t-scores on scales 2 (Depression) and 7 (Psychasthenia) will be used to assess severity levels of anxiety and depression. Analyses will then be conducted on the group as a whole, followed by analyses on neurologic (i.e.,

some evidence of neurologic insult) and non-neurologic (i.e., no evidence of neurologic insult) patients. The following research questions and hypotheses are posed:

1. *Which neuropsychological instruments seemed to best measure attention in this sample?*

What is the factor structure of the purported neuropsychological measures of attention in this sample? Which measures appear to best assess the construct known as attention? What is the relationship, if any, to Mirsky's (1987) attentional model? Which instruments appear to be the most useful to include in subsequent analyses with this sample? It is expected that the factor structure of test scores in this sample will closely match that of Mirsky's factor structure (i.e., the four factors: focus-execute, sustain, encode, and shift). It is also expected that the tests found to load most highly on attention components will be those identified by Mirsky or tests roughly equivalent within the test battery used with this sample.

2. *Do legal cases differ significantly on attention performance from the sample as a whole?*

Does having a legal case pending cause a patient to perform significantly different on neuropsychological measures of attention compared to the rest of the outpatient sample? If indeed this is the case, patients with a legal case pending will be selected from the sample and analyzed separately. Sweet and colleagues (1992) identify motivational variables and malingering as potential confounding factors in investigations of neuropsychological performance. Of the studies reviewed for this study, it was unclear as to how significant legal status affected attention performance on neuropsychological tests. It is expected that, of those presenting for neuropsychological evaluation due to a legal

case pending, motivation levels and blatant attempts at malingering will be sufficiently varied as to not represent a significant confound in this outpatient sample. Thus, it is expected that no significant difference will be found from the overall sample, and it will be possible to include their scores in subsequent analyses.

3. *What is the relationship between depression and attention in the outpatient sample?*

What is the relationship between scale 2 MMPI t-scores and the neuropsychological measures of attention administered to this outpatient sample? The information processing approach (Ingram, 1984) predicts that depression significantly impairs attentional resources. Therefore, it is expected that scale 2 MMPI t-scores will be significantly correlated with attention measures, such that increasing depression results in poorer attention performance.

3b. *What is the relationship between depression severity level (Low and High) and attention in this outpatient sample?*

When scale 2 MMPI t-scores are divided into Low (D > 45 and D < 55) and High (D > 70) Depression, while controlling for anxiety (Pt < 60), what relationship exists with attention measures? It is expected that higher depression levels will result in fewer attentional resources being available for attention-demanding tasks, therefore higher levels should result in significantly poorer attention performance than lower depression levels.

4. *What is the relationship between anxiety and attention in this outpatient sample?*

What is the relationship between scale 7 MMPI t-scores and the neuropsychological measures of attention administered to this outpatient sample? Processing efficiency theory (Eysenck & Calvo, 1992) predicts that anxiety places increased demands on attentional processes such that more effort is required for attention tasks than is normally required.

However, it is sometimes difficult to detect this increased demand on attentional resources empirically. Consequently, studies have been mixed in finding attentional deficits due to anxiety. Nonetheless, with this large sample it is expected that scale 7 MMPI t-scores will be significantly correlated with attention measures, such that increasing anxiety results in poorer attention performance.

4b. *What is the relationship between anxiety severity level (Low and High) and attention in this outpatient sample?*

When scale 7 MMPI t-scores are divided into Low (Pt > 45 and D < 55) and High (Pt > 70) Anxiey, while controlling for depression (D < 60), what relationship exists with attention measures? Processing efficiency theory predicts that increasing levels of anxiety place more and more demands on attentional processes. Therefore, it is expected that higher anxiety levels will result in significantly poorer attention performance than lower anxiety levels.

5. *What relationship exists with attention when depression and anxiety severity levels are both High, and both Low?*

When scale 2 and scale 7 MMPI t-scores are both High (D and Pt > 70), what relationship exists with attention measures? Similarly, when scale 2 and scale 7 MMPI t-scores are both Low (D and Pt > 45 and < 55), what relationship exists with attention measures? Vanderploeg, Kizilbash, Curtiss, and Schinka (1998) reported significant (detrimental) effects on memory by depression compounded with anxiety. Moreover, effects were stronger with high levels versus low. Therefore, it is expected that similar effects will be obtained with attention performance.

6. *When the sample is divided into two groups according to neurologic status, is there a significant difference on neuropsychological measures of attention?*

It is expected that, by virtue of their neurological insult, neurologic patients will evidence significantly more impairment on neuropsychological measures of attention.

6b. *Are there significant differences in depression levels for neurologics versus non-neurologics?*

Are there significant differences on scale 2 MMPI t-scores between neurologic and non-neurologic groups? As Reitan and Wolfson (1997) observe, intellectual and cognitive impairment represents a stressful situation that can cause emotional difficulties with awareness of one's reduction in ability from previous levels of functioning. It is expected that this sample will be no different. Neurologic patients should report higher levels of depression than non-neurologic patients.

6c. *Are there significant differences in anxiety levels for neurologics versus non-neurologics?*

Are there significant differences on scale 7 MMPI t-scores between neurologic and non-neurologic groups? Anxiety and depression are frequently comorbid conditions (Vanderploeg et al., 1998), therefore it is expected that a number of depressed neurologics will likely evidence concomitant anxiety. Consequently, it is expected that neurologic patients will report higher anxiety levels than non-neurologic patients.

6d. *Are depressed neurologics more affected on attention measures than non-depressed neurologics?*

When neurologics are separated by depression level (D > 70), is there a significant difference in performance on measures of attention? It is expected that neurologics will,

as a group, evidence impaired attention due their neurologic insult. According to the information processing approach, depression will also impair attention performance. Therefore, it is expected that depression will impair attention over and above that existing due to the underlying neurologic condition.

6e. *Are anxious neurologics more affected on attention measures than non-anxious neurologics?*

When neurologics are separated by anxiety level (Pt > 70), is there a significant difference in performance on measures of attention? In keeping with processing efficiency theory, it is expected that anxiety will impair attention over and above that existing due to the underlying neurologic condition. However, it is recognized that processing efficiency has stated that attention deficits will not always be revealed due to increased effort by anxious individuals to perform better.

6f. *Are depressed non-neurologics more affected on attention measures than non-depressed non-neurologics?*

When non-neurologics are separated by depression level (D > 70), is there a significant difference in performance on measures of attention? It is expected that depression will impair attention performance as predicted by the information processing approach.

6g. *Are anxious non-neurologics more affected on attention measures than non-anxious non-neurologics?*

When non-neurologics are separated by anxiety level (D > 70), is there a significant difference in performance on measures of attention? It is expected that anxiety

will impair performance in non-neurologics, however it is also recognized that anxiety deficits are not always revealed according to processing efficiency theory.

Method

Setting and Participants

The data for the study will be gathered retrospectively from the records of patients who had been referred to the Neuropsychological Assessment Laboratory at the Department of Psychiatry and Behavioral Sciences, University of Oklahoma Health Sciences Center (OUHSC) between 1955 and 1997. The Neuropsychological Laboratory at OUHSC receives approximately 300 referrals per year for neuropsychological assessment from neurology, tumor clinic, epilepsy clinic, attorneys, and other allied professions. Assessments usually consisted of a flexible battery of tests (i.e., a core battery of tests given to all patients plus additional tests added by the neuropsychologist based on the patient's presenting problem or etiology). Tests were administered by a Master's level psychometrist, pre-doctoral intern, post-doctoral fellow, or neuropsychologist. Testing time varied according to patient but was generally between 3-8 hours with appropriate breaks to prevent fatigue effects. Participants will be individuals referred for neuropsychological evaluation that completed valid MMPI's (Minnesota Multiphasic Personality Inventory; Butcher, Dahlstrom, Graham, Tellegen, & Kaemmer, 1989) and were at least 18 years old. For the years 1963 through 1997, 1,654 completed MMPI's were available, with the majority encompassing the years 1977 through 1997. Use of the MMPI was discontinued in 1992, in favor of the MMPI-2. In the total sample, there were 701 (58%) MMPI's and 508 (42%) MMPI-2's administered.

Of the 2,942 cases in the database, 1,209 (41.1%) participants were selected for analysis. Demographic information for this sample is presented in Table 1 below.

Table 1

Demographic Information for Participants

Variable	Mean	SD
Age	42.01	16.61
Education	13.20	2.81

	Frequencies	Percent
Gender		
Male	673	55.7
Female	536	44.3
Ethnicity		
Caucasian	1079	89.2
African American	76	6.3
Native American	21	1.7
Hispanic	6	0.5
Asian American	5	0.4
Other	3	0.2
Unknown	4	0.3

Measures

The Minnesota Multiphasic Personality Inventory - 2nd Edition (MMPI-2; Butcher, Dahlstrom, Graham, Tellegen, & Kaemmer, 1989), a widely used tool in the assessment of emotional/personality functioning, will be utilized for the present study. Basic scales 2 (D; Depression) and 7 (Pt; Psychasthenia) will be used as a measure of depression and anxiety, respectively. Participants will be eliminated if they had MMPI T-score elevations above 70 on L or K scales, or above 80 on F. Similar procedures were utilized by Vanderploeg et al. (1998).

Measures of Attention

Digit Span

WAIS-R Digit Span (Wechsler, 1987) scaled score Forward and Backward raw scores. Digit Span consists of two parts: Digits Forward requires the subject to repeat sequences of three to nine digits; Digit Backward sequences are two to eight numbers long, and the subject must say them in reverse order (Spreen & Strauss, 1998). Moderate correlations have been achieved with the PASAT, RBMT (Rivermead Behavioural Memory Test), WMS scores, CVLT, TPT (Tactual Performance Test) Memory, and numerous other tests utilizing attention and memory.

Visual Memory Span

WMS-R Visual Memory Span (Wechsler, 1987) forward and backward raw scores. The Visual Memory Span provides consists of two cards on each of which eight squares are printed in a nonlinear pattern, red squares for the forward span and green for reversed

span. Administration procedures are the same as for Digit Span, requiring two trials at each level regardless of whether the first was passed (Lezak, 1995).

Mental Control

WMS-R Mental Control (Wechsler, 1987) consists of (1) counting backwards from 20 in 30 seconds; (2) reciting the alphabet in 30 seconds; and (3) counting from 1 to 40 by 3's in 45 seconds. Its attentional component is "consistently attested to by factor analytic studies" (Lezak, 1995).

Digit Symbol

WAIS-R Digit Symbol (Wechsler, 1987) scaled score. Digit Symbol is a symbol substitution task consisting of four rows containing, in all, 100 small blank squares, each paired with a randomly assigned number from one to nine. Following a practice trial on the first seven, the task is to fill in the blank spaces with the symbol that is paired to the number above the blank space as quickly as possible for 90 seconds (Lezak, 1995). Among other functions such as psychomotor speed and visuomotor coordination, it is a measure of focused attention, requiring the individual to reject irrelevant information while attending to relevant input (Spreen & Strauss, 1998).

Symbol Digit Modalities Test

Symbol Digit Modalities Test (Smith, 1991) oral administration raw score and written administration raw score. This test preserves the substitution format of Digit Symbol but reverses the presentation of material so that the symbols are printed and the numbers are written, enabling the individual to respond with the more familiar act of number writing. It also allows for a spoken response trial. As with Digit Symbol, 90 seconds are allowed for each trial, however there are 110 items instead of 100 (Lezak, 1995). Although Lezak

(1995) maintains that this test primarily measures complex scanning and visual tracking, Spreen and Strauss (1998) include it as a measure of focused attention.

Trail Making Test

Trail Making Test (Reitan & Wolfson, 1985) Part A time in seconds and Part B time in seconds. The individual must first draw lines to connect consecutively numbered circles on one work sheet (Part A) and then connect the same number of consecutively numbered and lettered circles on another worksheet by alternating between the two sequences (Part B; Lezak, 1995). Trail Making is a test involving focused attention, motor speed, and complex visual scanning (Lezak, 1995; Spreen & Strauss, 1998).

Finger Tapping

Tapping (Reitan & Wolfson, 1985) dominant hand mean number of taps and nondominant hand mean number of taps. Finger Tapping consists of a tapping key with a device for recording the number of taps. Each hand makes five 10-second trials with brief rest periods between trials. The score for each hand is the average for a set of five trials that do not vary more than 5 taps (Lezak, 1995). The test taps motor speed and manual dexterity and is sensitive to the presence and laterality of brain lesion (Lezak, 1995; Spreen & Strauss, 1998).

Grooved Pegboard

Grooved Pegboard (Reitan & Wolfson, 1985) dominant hand time in seconds and nondominant hand time in seconds. Grooved Pegboard consists of a small board containing a 5 X 5 set of slotted holes angled in different directions. Each peg has a ridge along one side requiring it to be rotated into position for correct insertion. The score is time to completion, and its complexity makes it sensitive for measuring general slowing

and progression of disease processes, and by assisting in identifying lateralized impairment (Lezak, 1995).

Conners' Continuous Performance Test (CPT)

Conners' Continuous Performance Test (CPT; Conners, 1995) number of hits, omission errors, commission errors, and hit reaction time. The Standard CPT in Conners' program requires the individual to press the appropriate key (e.g., space bar) for any letter *except* the letter X. There are six blocks, each with three 20-trial sub-blocks. For each block, the sub-blocks have different interstimulus intervals: 1, 2, or 4 seconds. Each letter is displayed for 250 milliseconds. The CPT assesses lapses in attention or vigilance and impulsivity (Spreen & Strauss, 1998).

Seashore Rhythm Test

Seashore Rhythm Test (Reitan & Wolfson, 1985) total errors. The Seashore Rhythm Test is the most widely used test for nonverbal auditory perception. It also consists of concentration and tracking components. It requires the individual to discriminate between like and unlike pairs of musical beats.

Speech Perception Test

Speech Sounds Perception Test (Reitan & Wolfson, 1985) total errors. This test consists of 60 sets of nonsense syllables each beginning and ending with different consonants but based on the vowel sound "ee." It is administered by tape recording, and individuals must note what they think they heard on a four-choice form laid out in six 10-item sections labeled A to F. Since it is a rapidly paced test, it is thought to be sensitive to attentional deficits (Lezak, 1995).

WMS-R Attention/Concentration Index

WMS-R (Wechsler, 1987) Attention/Concentration Index score. This score is derived from the WMS-R Digit Span, Visual Memory Span, and Mental Control subtest scores.

Wisconsin Card Sorting Test (WCST)

Wisconsin Card Sorting Test (WCST; Heaton, et al., 1993) number correct, number of errors, number of perseverative responses, number of categories completed, and failure to maintain set. This test is designed to assess the ability to form abstract concepts, to shift and maintain set, and utilize feedback (Spreen & Strauss, 1998). It consists of four stimulus cards (or representations of cards on a computer screen), red triangle, two green stars, three yellow crosses, and four blue circles. The individual is asked to match each of 64 cards, which have designs similar to those on the stimulus cards, varying in color, geometric form, and number, to one of the four key cards and is given feedback each time whether he or she is right or wrong. There is no time limit.

Paced Auditory Serial Addition Test (PASAT)

Paced Auditory Serial Addition Test (PASAT; Gronwall, 1977) total errors on each of trials 1-4. The PASAT is a serial-addition task used to assess capacity and rate of information processing and sustained and divided attention. The individual is required to comprehend the auditory input, respond verbally, inhibit encoding of his or her own response while attending to the next stimulus in a series, and perform at an externally determined pace. A prerecorded tape delivers a random series of 61 numbers from 1 to 9. The individual is instructed to add pairs of numbers such that each number is added to the one that immediately precedes it: the second is added to the first, the third to the second, the fourth to the third, and so on. The same 61 numbers, given in the same sequence, are

presented in four different trials, each trial differing in its rate of digit presentation (2.4, 2.0, 1.6, 1.2 seconds).

Stroop Color and Word Test

Stroop (Golden, 1978) Word score, Color score, and Color-Word score. This test is a measure of selective attention and cognitive flexibility was originally developed by Stroop (1935). A number of versions have been developed, but the one utilized here is the version developed by Golden (1978). In Golden's version, three cards are used consisting of 100 items on each. On the first card, the individual is asked to read the color words (red, green, or blue) printed in black ink as quickly as possible for 45 seconds. On the second, the individual reads the color of ink, each item consisting of "XXX" as quickly as possible for 45 seconds. On the final card, the individual must name the color in which the color words are printed and disregard their verbal content (i.e., inhibit the natural urge to read the word).

Word Fluency

Benton Word Fluency (Benton, Hamsher, & Sivan, 1994) total number of words generated. The purpose of this test is to evaluate the spontaneous production of words beginning with a given letter (C, F, and L; or F, A, and S). The individual is asked to produce orally as many words as possible, for each letter, given a 60 second time limit for each trial. The score is the combined total for the three trials added to an education correction factor.

Category Fluency

Animal naming (Goodglass & Kaplan, 1987) total number of words. This test serves the same purpose as Word Fluency, however on this task the individual is confined to a category and asked to produce as many animal names as possible within 60 seconds.

Results

Analyses will be conducted using SPSS 9.0 (Statistical Package for the Social Sciences - Version 9.0). Participants will be selected from the overall sample if they completed valid MMPI's, defined as t-score elevations below 70 on scales L or K, or below 80 on scale F (Vanderploeg, et al., 1998).

Descriptive statistics will be performed to examine variables for errors, number of cases, measures of central tendency, variability, and shape of distribution. Results will be described in Table form. Data will be recoded as necessary.

Next, a correlation matrix will be constructed to explore the relationships between emotional distress (MMPI Scales 2 and 7) and attentional measures. This will be followed by a factor analysis to identify the neuropsychological tests that best assess attention in this sample and which attentional construct or factor they appear to measure.

A Multiple Analysis of Variance (MANOVA) will be conducted to determine if the participants with a legal case pending (N=178 or 14.7%) differ significantly from the overall sample. If it is determined that they may skew the data, they will be removed from subsequent analyses.

A series of canonical correlation analyses will be conducted to determine the extent to which attention measures contribute to prediction of depression scores and to prediction of attention scores in the overall sample.

Next, canonical correlation analyses will be conducted to determine which attention measures contribute to prediction of severity level of depression and severity level of anxiety for the overall sample. Severity levels will be determined using the same procedure used by Vanderploeg et al. (1998; see below).

The sample will be subsequently divided into two groups according to neurologic status (presence or absence of documented neurologic injury). In each of these MANOVA analyses, the attention scores will be the dependent variables and MMPI t-scores on D and Pt will be the grouping variables. Next, a series of two MANOVAs will be conducted by dividing the entire sample (both neurologic and non-neurologic) into groups that scored high versus low on the MMPI D and Pt scales in the following manner:

"High Depression" = T-score on D > 70, T-score on Pt < 60, (N = ?),

"Low Depression" = T-score on D > 45 and < 55, T-score on Pt < 60, (N = ?);

"High Anxiety" = T-score on Pt > 70, T-score on D < 60, (N = ?), "Low Anxiety" = T-score on Pt > 45 and < 55, T-score on D < 60, (N = ?); and

"High Depression and Anxiety" = T-score on D and Pt > 70, (N = ?), "Low Depression and Anxiety" = T-score on D and Pt > 45 and < 55, (N = ?).

Next, a series of MANOVA's will be utilized for additional group comparisons to answer the remaining research questions. This will be followed by separate analyses utilizing "version of MMPI administered" as a covariate to explore potential confounds of using both versions in the same dataset.

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