

A Functional Cerebral Systems Approach to Depression:
Contributions of the Left and Right Frontal Lobes

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Thesis submitted to the Faculty of
Virginia Polytechnic Institute and State University
in partial fulfillment of the requirements for the degree of

Master of Science

in Psychology

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05/13/2010

Blacksburg, VA

Keywords: laterality, emotion, depression, AAVLT, Neuropsychology

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Abstract

In the majority of the depression literature, there has been little attention paid to the mechanisms underlying the differences that occur among individuals with this label. In a theoretical paper by Shenal, Harrison, and Demaree (2003), they proposed that the differences in depression symptomology may be due to differences in the function (and dysfunction) of the right and left frontal lobes. They go on to explain that each frontal lobe may have a direct influence on patterns of depression symptomology. In the current experiment there was an effort to look at performance differences among depressed and non-depressed males on a tests of affective memory (AAVLT) and functioning for the left (COWAT) and right (RFFT) frontal lobes. Results were non-significant for group based differences but other significant effects were found. Reliable findings included a primacy effect for the recall of words from the negative word list from the AAVLT, whereas a “normal” primacy and recency effects were found for the recall of positive and neutral word lists. There were also significant differences (across trials) for both groups suggesting a “normal” learning curve. It is thought that the non-significant comparisons among the groups are likely due to the qualitatively mild depression scores among participants, which is likely not adequate to capture the level of dysfunction discussed in the original hypothesis.

Acknowledgements

I would like to thank my committee, especially Dr. Harrison, for having as much patience as could be asked for, and my parents for their belief in me when others gave up.

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Introduction

A Functional Cerebral Systems Approach to Depression: Contributions of the Left and Right Frontal Lobes

Depression is described in the DSM-IV as a disorder requiring five or more of the following constellation of symptoms, (1) depressed mood for most of the day, nearly everyday, (2) anhedonia, (3) significant weight loss or weight gain (without dieting), (4) insomnia or hypersomnia nearly everyday, (5) psychomotor agitation or retardation nearly every day (observable by others), (6) fatigue or loss of energy nearly every day, (7) feelings of worthlessness or excessive or inappropriate guilt (which may be delusional), (8) diminished ability to think or concentrate nearly everyday, and (9) recurrent thoughts about death (3). According to World Health Organization statistics Major Depressive disorder is the leading cause of disability in the U.S. for those 15 to 44 years of age (World Health Organization, 2004). It affects approximately 14.8 million American adults or 6.7% of the population in a given year (Kessler, Chiu, Demler, & Walters, 2005). Its prevalence in our society has lead to a vast literature on this topic including that within the field of neuropsychology with an emphasis on what regions of the brain may be contributing to this multi-factor disorder. There is much literature to suggest that dysfunction in the left or right anterior cerebral regions may be responsible. When looked at as a whole, the literature points to a problem that may be better understood when examining the relationship between symptom and associated brain region. In the present paper an experiment will be proposed that will help in describing

manifestations of depression by looking at the difference between depression types based on dysfunctional anterior cerebral regions.

Definition of Neuropsychological Theory

Neuropsychology theory specifies that all behavior is mediated by physical changes in the state of the brain (Heilman & Valenstein, 2003). The reverse is true if the physical state of the brain is changed (i.e., brain lesions). The second major assumption of neuropsychology has to do with the division of labor within the brain or cerebral laterality. According to Kolb and Whishaw, 1990 & Wexler, 1980, the concept of cerebral laterality refers to the functional differences among the left and right cerebral hemispheres. It has been shown that many sensory and motor connections are projected to the contralateral hemisphere (Kolb & Whishaw, 1990; Wexler, 1980). An example of this is that the motor responses of the left extremities are controlled largely by the right cerebral hemisphere.

Right & Left Brain Typical Function

The data on cerebral lateralization of right-handed persons have revealed functional differences among the two hemispheres. The left hemisphere is shown to play a larger role in language and verbal abilities, logical reasoning, verbal memory, arithmetic calculations, and complex voluntary movements (Bryden, 1982; Dimond & Beaumont, 1974; Eccles, 1977). The right hemisphere is implicated in spatial processing including complex geometric patterns, prosody, and musical melody, as well as nonverbal memory (Bryden, 1982; Dimond & Beaumont, 1974). More recently, the neuropsychological literature has demonstrated the functional asymmetry of emotion.

This has been established not only for the perception of emotional content but also for the expression of emotion as well.

Right Hemisphere Model

There are several theories to account for the laterality of emotion. Two of these theories provide the foundation for the current experiment. First, the right hemisphere model states that the right cerebral hemisphere is specialized for the perception and expression of emotion (Borod, Koff, & Caron, 1983; Heilman & Bowers, 1990; Ross, 1985; Tucker, 1981). Much of the literature supports the inference that emotion may be preferentially perceived in the right hemisphere.

Earlier research demonstrates left visual field (right hemisphere) superiority for identifying facial affect (Landis, Assal, & Perret, 1979; Ley & Bryden, 1979; McKeever & Dixon, 1981; Suberi & McKeever, 1977) when studying those without brain damage. It has also been consistently shown in lesion research that those with damage to the right-hemisphere performed significantly worse than patients with left-hemisphere damage when asked to discriminate or to recognize facial affect (Adolphs, Damasio, Tranel, & Damasio, 1996; Borod et al. 1998; Cicone, Wapner, & Gardner, 1980; Etcoff, 1986). It has been theorized by Bowers and Heilman (1984), that the right hemisphere may actually act as a “store of facial emotion icons” or representations. This conclusion is furthered by research that has shown a significant decrease in the ability to perceive affect, either due to right hemisphere injury (Benowitz, et. al. 1983) or intracarotid sodium amytal injection (Ahern, et. al., 1991). The findings from electrophysical recording and neuroimaging studies generally lend support to the idea that the right hemisphere is specialized for the processing of facial emotion. Evoked potential studies

have illustrated greater right- versus left-hemisphere activity during the processing of facial affect (Kestenbaum & Nelson 1992; Laurian, Bader, Lanares, & Oros, 1991; Munte et al., 1998; Vanderploeg, Brown, & Marsh, 1987), and functional magnetic resonance imaging (fMRI) has shown specialization of some right hemisphere structures in the perception of emotion (Narumoto, Okada, Sadato, Fukui, and Yonekura (2001).

Less is known about affective prosody and the right hemisphere's role in its perception. Earlier experiments have implicated that the right hemisphere is relatively specialized for this purpose. Ross (1981), presented communication disorders referred to as the "aprosodias" which reflect disturbance of the ability to perceive (sensory aprosodias) or to express (motor aprosodias) the emotional aspects of speech. This disorder has been observed by Ross, Thompson, & Yenkosky (1997) to occur typically following right hemisphere lesions. A right hemisphere advantage for recognizing speech prosody has also been seen using a dichotic listening paradigm. It was shown that the left ear has an advantage over the right ear in recognizing the emotional components of speech (Carmon & Nachshon, 1973; Haggard & Parkinson, 1971; King & Kimura, 1972; Snyder & Harrison, 1996). In electrophysiological research (Everhart, Carpenter, Carmona, Ethridge, & Demaree, 2003; Jordan, Everhart, & Demaree, 2004; Pihan, Altenmuller, & Ackermann, 1997) and neuroimaging research (Buchanan et al., 2000; George et al., 1996; Imaizumi et al., 1997) the right hemisphere's role in the perception of affective prosody has been shown. It was also shown in research by Borod (1993) that patients with right brain damage experience more problems with the expression of emotional prosody. Those with right hemisphere damage may even speak in a monotone

voice (Ross & Mesulam, 1979; Tucker, Watson, & Heilman, 1977; Williamson, Shenal, Harrison, & Demaree, 2003).

In the right hemisphere model, the experience of emotion through mood and affect is thought to be predominantly handled by the right hemisphere. Gainotti (1972) noticed this when he recorded “indifferent” and “catastrophic” reactions after administering right- and left-intracarotid sodium amytal injections. In experiments involving electrophysiological parameters measured during emotionally charged states there was found to be relative right cerebral activation (decrease in alpha power using electroencephalography [EEG]) during the recollection of past events associated with anger or relaxation (see Foster & Harrison 2002). In other EEG studies, there was evidence of relative right cerebral activation when participants were asked to generate emotional imagery (Karlín, Weinapple, Rochford, & Goldenstein, 1979) and during hypnotically induced depression (Tucker, Stenslie, Roth, & Shearer, 1981). There has also been evidence, using quantitative EEG, that magnitude of cerebral activation is related to the intensity of emotional arousal (Foster & Harrison, 2002).

Many experiments have been done using behavioral and physiological measures to investigate individuals experiencing higher levels of negative affect (e.g. depression, anxiety, or anger). Early research on anxiety found evidence that increased right visual field errors occur on verbal and spatial tasks during periods of increased anxiety (Tucker, Antes, Stenslie, & Barnhardt, 1978). A right ear bias in the perception of tones in subjects with high-trait anxiety (Tucker et al., 1978) and during periods of depressed mood (Tucker et al., 1981) was also seen. The authors of these papers hypothesized that the results were indicative of increased left-hemisphere activity during depression and

anxiety. But it has been argued by Everhart & Harrison (2000) and Silberman & Weingartner (1986) that these results may suggest that the “poorer” left hemisphere performance may come from increased right-hemisphere activation relative to the left. Everhart & Harrison (2000) also saw an association between high trait anxiety and increased accuracy for recognition of negative affective faces presented to the left visual field (right hemisphere) in comparison to low-trait anxiety individuals. Other emotional states and disorders have been linked to increased (or decreased) right-hemisphere activity. Heller (1993) suggested that depressed states were associated with relatively increased right versus left frontal activity and decreased right posterior activity.

Valence Model of Emotion

The second major theory in the neuropsychology of emotion is the valence theory. This theory states that the right hemisphere may be specialized for negative emotion while the left is specialized for positive emotion (Ehrlichman, 1987; Silberman & Weingartner, 1986). Of note, a variant of this theory as proposed by Bryden, 1982 and Davidson, 1984, is that the differential specialization of the two hemispheres for positive (left) and negative (right) emotions only applies to the expression and experience of the valence.

In many of the studies described above, hemispheric differences have been described as a function of whether the emotional valence is positive or negative. It has been consistently shown that patients may have more difficulty perceiving negative rather than positive emotion following injury to the right hemisphere (see Adolphs et al., 1996; Borod et al., 1998). Happy faces are more easily identifiable by normal participants than are other affective faces (Everhart et al., 2003; Everhart & Harrison, 2000). Moreover,

the ability to perceive happy faces is usually preserved after right hemispheric injury (Adolphs et al., 1996). In other studies using amytal injections it was found that an injection at the left carotid artery produced a “catastrophic reaction” which was described as crying, pessimistic statements, guilt, complaints, and worries about the future (Rossi & Rosadini, 1967; Silberman & Weingartner, 1986). A contrasting effect was shown to occur with amytal injection at the right carotid artery, which resulted in a euphoric reaction that consisted of lack of apprehension, smiling, laughing, mimicry, and a sense of well-being. The mood changes that occur with the amytal injection of one hemisphere are thought to represent the release of the opposite hemisphere from contralateral inhibitory influences (Silberman & Weingartner, 1986). These studies among others with patients that have left or right hemispheric brain damage have provided support for the valence hypothesis. More recently Mollet et al. 2007, provided evidence of hallucination based on affective valence with negative hallucinations prevalent in the left hemisphere.

There has also been evidence to support the valence hypothesis through tachistoscopic studies. In an experiment done by Reuter-Lorenz, Givis, and Moscovitch (1983), either happy or sad faces were presented to one visual field while simultaneously presenting a neutral expression in the opposing visual field. The participants were asked to identify the side that the emotional face appeared on. The researchers found shorter reaction times for happy faces shown in the right visual field (left hemisphere) and sad faces in the left visual field (right hemisphere). Natale, Gur, and Gur (1983) presented happy, sad, and chimeric faces to participants and found that the chimeric faces were more likely to be called positive when presented in the right visual field. Subsequent research found that in some cases, negative affective faces are identified more rapidly or

more accurately when presented within the left visual field (Everhart & Harrison, 2000; Harrison & Gorelczenko, 1990). Moreover a left visual field negative affective perceptual bias was demonstrated in the perception of neutral faces by hostile, angry, violent prone individuals (Harrison and Gorelczenko, 1990)

In other studies, there has been mixed support of the theory that the left and right hemispheres are specialized for the perception of positive and negative emotions. In the Adolphs et al. (1996) study, it was reported that brain-damaged patients showed no impairment on facial expression recognition if they had left hemisphere lesions. According to Borod et al. (1998), it appeared that patients with right brain damage were significantly more impaired relative to patients with left brain damage and normal controls across channels and valence.

In regards to emotional expression, early studies of asymmetry in facial expression noted that the tendency for emotion to be expressed to a greater degree on the left side of the face, was more pronounced if the emotion was negative (Sackeim & Gur, 1978; Silberman & Weingartner, 1986). Similarly, using electromyography, Schwartz, Ahern, and Brown (1979) found that right sided contractions were stronger during times of happiness and excitement, whereas emotions like sadness and fear created greater left side contractions. In a review of literature by Borod (1993), which included studies that looked at emotional expression in three modalities (facial, prosodic, and lexical) in both brain damaged patients and normal controls, it was concluded that for facial emotional valence there were no overall differences between right and left brain injured groups. It was noted in the same review that a number of studies demonstrated deficits in positive and negative emotion using patients with right brain damage. The findings were divided

equally between the two valences. Borod also concluded (in regards to expression of prosody) that patients with right brain injuries are more impaired in terms of emotional processing as opposed to those with left brain damage or normals, regardless of valence.

In a second review done by Borod, Haywood, and Koff (1997), they suggests that the left hemiface is more involved than the right during the facial expression of emotion. Additionally, it was seen that left facial asymmetries occurred more often when the emotion was negative. The same review showed no instances of right sided asymmetries for negative emotions, whereas a 15% incidence of right-sided asymmetry for positive emotion was shown. According to these findings it could be assumed that positive emotional expression is associated with the left hemisphere (Borod et al., 1997) but at the very least it may be of bilateral origin (Ehrlichman, 1987).

In observations of brain damaged patients, left-hemisphere lesions were more likely to exhibit “catastrophic” behavior, whereas right hemisphere lesions were more likely to exhibit “indifference reactions” (Heilman et al., 1975). In poststroke patients it was common for left hemisphere lesions (particularly in the left frontal pole) to evidence depression symptoms (Robinson, Starr, & Price, 1984; Starkstein, Boston, & Robinson, 1988). Something to note is that lesions within the right posterior region are also associated with an increased occurrence of depression (Robinson et al., 1984), a finding that is consistent with Heller’s (1993) model of emotion that separates arousal and valence.

The most substantial evidence that supports the valence hypothesis comes from the large literature produced from EEG research. There have been many studies that have associated relatively increased left-hemisphere activity with positive emotional states and

relatively increased right-hemisphere activity with negative emotional states (Davidson & Fox, 1982; Davidson & Henriques, 2000; Davidson et al., 1979; Ekman & Davidson, 1993; Ekman, Davidson, and Friesen, 1990; Everhart, Demaree, and Harrison, in press; Fox & Davidson, 1987, 1988; Koek et al., 1999; Lee et al., 2004; Mandal et al., 1999; Reuter-Lorenz & Davidson, 1981; Schaffer, Davidson, & Saron, 1983; Sutton & Davidson, 2000; Tomarken, Davidson, Wheeler, & Doss, 1992; Tucker, 1981; Waldenstein et al., 2000; Wheeler, Davidson, & Tomarken, 1993). In the Davidson et al. (1979) study, the participants described their emotional responses to television shows that they watched with varying emotional content. The frontal EEG leads displayed relative left-hemisphere activity when the participant indicated a positive emotional response, whereas the opposite pattern was seen when the participant indicated a negative emotional response. Infants displayed similar results when presented with a picture of a person displaying a negative or positive facial expression (Davidson & Fox, 1982). In general, findings in neuroimaging studies have shown the same results as the experiments above (Bench et al., 1992; Passero, Nardini, & Battistini, 1995; Wager, Phan, Liberzon, & Taylor-Tovares et al., 2007). It is important however to point out that there have been EEG studies of emotion that have either had no significant results or had the opposite pattern of activation shown (Cole & Ray, 1985; Schelburg, Besthern, Klos, & Gasser, 1990; Schelburg, Besthern, Pflieger, & Gasser, 1993; Tucker & Dawson, 1984).

Anterior asymmetries have been researched with patients with other diagnoses. In some studies it has been shown that individuals with high trait anxiety have greater right versus left cerebral blood flow (Naveteur, Roy, Ovelac, & Steinling, 1992). There have been some discrepant findings with these results (Everhart and Harrison, 2000; Nitschke

et al., 2000). These inconsistencies may be a result of the high prevalence of comorbid depression, as well as subtypes even within anxiety that include anxious apprehension or anxious arousal (Clark & Watson, 1991; Nitschke et al., 2000).

The Quadrant Model of Depression

There is a substantial literature on the neuropsychology of depression. The best way to conceptualize the current literature including what may seem like inconsistencies, is presented in our model proposed in *Clinical Neurophysiology* (Shenal, Harrison, and Demaree, 2003; see also Foster et al., in press). This model seems to allow for a better understanding of the basis of cerebral activation and arousal theory. The model presented in the Shenal et al. 2003 article accounts for the many diverse cognitive, behavioral, and affective symptoms that are all a part of the diagnosis of depression. It divides the brain into four neuroanatomical divisions (left anterior, left posterior, right anterior, right posterior), three of which fit the current neuropsychological sequelae of depression. Dysfunction occurring in one or more of these areas, may account for the variability found within the broad diagnostic category of “depression”. In the following summary, the function of each quadrant of the brain will be discussed in relation to depression.

Davidson (1995) reported that when healthy controls experienced sadness there was an increase in right-hemisphere activation and a decrease in left-hemisphere activation. He suggested that left-hemisphere activation may cause approach behavior, responses to reward, and increased positive mood. He further hypothesized that a decrease in left-hemisphere activation may cause a decrease in positive affect. It is also suggested in Davidson (1995) that these findings may point to trait like characteristics of depression and may also point to a vulnerability to depression.

Heller et al., 1998, suggested that depression was more severe after left-hemisphere damage than after right-hemisphere damage. One other study that reported more severe depression after left-hemisphere damage was Robinson, Starr, Lipsy, Rao, and Price (1985). In the same study it was noted that the severity of depression was correlated positively with the proximity of the lesion to the left-frontal pole. That being said, left-frontal dysfunction may be accountable for several symptoms associated with a diagnosis of depression.

It is possible that an individual with decreased left-hemisphere arousal may have limited affect and therefore be diagnosed with depression. The anterior left-hemisphere depressive sequelae include limited positive affect (including deficits in processing and executing sequential positive behaviors), sparsity of speech, decreased positive affect initiation, behavioral slowing, and restricted social approach behaviors. Several studies have reported left-anterior dysfunction and noted its relationship to depressed mood (Banich, Stolar, Heller, and Goldman, 1992; Debner et al. 2000; Fleminger, 1991; Henriques and Davidson, 1991). In the Debner et al. (2000) study it was shown that depressed individuals after being assessed at two different times still evidenced decreased left-frontal activation. This stability has also been shown in other experiments (Allen et al., 2004; Tomarken et al., 1992; Vuga et al., 2006).

The right-hemisphere has been implicated in the perception and expression of emotion (Borod, Andelman, Obler, Tweedy, and Welkowitz, 1992; Borod et al. 1998; Montreys and Borod, 1998). According to Davidson (1995), activation of the anterior right-hemisphere is associated with negative emotions. It has also been found to be related to withdrawal from adverse stimuli, negative affect and avoidance (Davidson

1993, 1995, 1998; Davidson, Abercrombie, Nitschke, and Putnum, 1999; Davison and Irwin, 1999; Heller et al. 1998). Activation of this hemisphere may increase negative affect which in turn could lead to a diagnosis of depression.

Although right-frontal activation may lead to negative affect, it is also possible for dysfunction within this area to be associated with depression. A lesion in this area may affect an individual and their ability to withdraw from aversive stimuli or to practice proper avoidance behavior. These behavioral symptoms may be assessed as a form of learned helplessness. It has also been seen in the literature that right-frontal lesions may result in the inability to properly regulate emotion causing one to cry more easily (Heilman et al., 2003; Robinson et al., 1993; Wilson, 1924). Researchers have reported that either right-frontal damage or fronto-temporal damage may interfere with executive functions yielding primary or secondary depressed affect (Liotti and Mayburg, 2001; Mayburg, 2001). It has also been shown that decreased right-frontotemporal rCBF is associated with depression (Lesser et al., 1994). It is possible that either right-hemispheric activation or deactivation may be diagnosed as depression. Considering the broad spectrum of symptoms associated with “depression”, the term may lack good neuropsychological description of all of the symptoms that may be caused by right frontal dysfunction (e.g., flat affect and difficulty in sequencing negative emotional expression). Dysfunction here alters affective speech resulting in an expressive aprosodia with involuntary or intense emotional expression, problems with regulating negative affect, and social impropriety.

There is very little literature on the contributions of the left-posterior cerebrum in depression. There is an emerging literature on its contributions to emotion (Cox, 2007)

which may soon lead to research in the arena of depression. Regardless, the sparsity of research on the functional role of this region in depression is vital and elaboration on this discussion is deferred pending additional research.

Patients with right-posterior dysfunction often develop receptive aprosodia, with impairment in recognizing affective or prosodic components of speech often accompanied by deficits in recognizing facial expressions (Ross, Thompson, & Yenkosky 1997). These symptoms were also noted by other researchers (Dekosky, Heilman, Bowers, and Valenstein, 1980; Heller et al. 1998; Tucker, Watson, Heilman, 1977). Deficits within this area may cause problems in the ability to detect, to comprehend, and to respond appropriately to emotional stimuli. Decreased arousal in the right-posterior region may lead to a depressive disorder. Jorge, Robinson, Starksein, and Arndt (1993) found that anxious-depressed patients exhibit right-hemisphere dysfunction whereas nonanxious-depressed patients displayed left-hemisphere dysfunction. Researchers have also found that patients with right-hemisphere damage evidence impairments in visuo-spatial processing and constructional tasks. Moreover, they may be diagnosed as depressed following a right-hemisphere stroke with behavioral features that often include bland or flattened affect and often emotional emptiness or non-responsiveness (Crews and Harrison, 1995).

The posterior right-hemisphere may contribute to a diagnosis of depression in multiple ways. If the area is damaged it may lead to a dysfunction in the ability to perceive visual, auditory, and somatosensory emotional stimuli. It may also be difficult for an individual with right posterior brain impairment to have insight into emotional problems with diminished emotional arousal as well.

Neurocognitive Differences of Depressed Individuals

It has been common practice to use cognitive measurement tools to assess those with affective disorders (Emerson, Harrison, Everhart, & Williamson, 2001; Everhart, Demaree, Harrison, in press; Mollet and Harrison, 2006). As noted previously, there is a substantial literature describing the neuropsychological deficits associated with depression. There have also been numerous studies that have illustrated neurocognitive performance difficulties in those that are depressed (Austin, Mitchell, & Wilhelm, et al. 1999; Crews & Harrison, 1995; Christensen, Griffiths, Mackinnon, & Jacomb, 1997; Franke et al., 1993; Nathan, Wilkinson, Stammers, & Low, 2001). The majority of this literature has focused on the effect that depression has on executive function.

Executive function has been hypothesized to be responsible for the set of behavioral competencies that integrate basic cognitive processes (Della, Sala, Gray, Spinnler, & Trivelli, 1998) and that permits sensitive and flexible responses. Executive functioning capabilities may include self directed planning and strategy formation, goal directed behavior, cognitive flexibility, and non-perseverative behavior (Crawford & Henry, 2005; Perret, 1974). Consistent with this view, patients that are depressed often perform poorly on tests of executive function such as the nonverbal Wisconsin Card Sorting Test (WCST; Franke et al., 1993; Pendleton, Henderson, Jones, & Welch, 1988), tests of verbal and phonemic fluency (Brown, Scott, Bench, & Dolan, 1994; Emerson, Mollet, & Harrison, 2004; Trichard et al., 1995), and the Stroop Interference Test (Nathan, Wilkinson, Stammers, & Low, 2001). Moreover, performance on the RFFT has

been shown to vary especially with activation over the right frontal lobe using quantitative EEG (Foster & Harrison, 2005) and to vary reliably with a hostile, angry, violent prone disposition (Williamson & Harrison, 2002)

Considering the great deal of evidence that frontal structures are largely responsible for executive processes (see Shallice, 1988; Stuss & Benson, 1986) the presence of frontal abnormalities would suggest that executive deficits within this area of cognition should be evident. Two measures of fluency, which have been utilized as indicators of frontal lobe dysfunction, are the Ruff Figural Fluency Task (RFFT; Ruff, Light, & Evans, 1987) and the Controlled Oral Word Association Test (COWAT; Benton & Hamsher, 1976).

The RFFT has been used as a measure of design fluency (Demakis & Harrison, 1997) and has good test-retest reliability (Ruff et al., 1987) and interrater reliability (Berning, Weed, & Aloia, 1998). The RFFT has also been shown to be sensitive to right frontal lobe dysfunction (Ruff, Allen, Farrow, Niemann, & Wylie, 1994). Patients with severe head injuries perform poorer on the RFFT (Ruff, Evans, & Marshall, 1986) as do patients with right frontal lobe lesions (Baldo, Shimamura, Delis, Kramer, and Kaplan, 2001). The RFFT may be sensitive to the cognitive impairments associated with depression.

The COWAT is also sensitive to frontal lobe dysfunction (Bolter, Long, & Wagner, 1983; Loring Meador, & Lee, 1994; Pendleton, Heaton, Lehman, & Hulihan, 1982; Perret, 1974). But, unlike the RFFT, it is used as an assessment of verbal fluency (Benton & Hamsher, 1976). It was shown that measures of semantic and phonemic (verbal) fluency reveal similar impairments when the frontal lobe is damaged suggesting

a similar frontal system for both (Henry and Crawford, 2004). Considering the left hemispheres influence on verbal/linguistic tasks and the right hemisphere's influence on visuo-spatial (design) tasks it can be hypothesized that both the COWAT and RFFT, respectively may be able to differentiate between left, right, and bi-frontal damage. In relation to the proposed model of depression, either tool may be able to help differentiate types of depression on the basis of fluency or non-fluency in a verbal or design domain. The following proposal begins to examine this possibility.

Rationale

The purpose of the current experiment is to differentiate between design non-fluent and verbally non-fluent depressed individuals, dividing the groups using performance measures from the RFFT and the COWAT. QEEG will be used as a method of recording differences in activation among the two groups primarily focused on relative activation or deactivation within the frontal lobes. Based on the current model of depression proposed by Shenal et al. (2003) and Foster et al. (in press), there should be groups of individuals who will have symptoms of depression, but who will differ in the hemispheric activation involved in their symptomology.

The hypothesized results are as follows: (1) There will be separate groups of depressed individuals (verbally non-fluent/design non-fluent, verbally fluent/design non-fluent, and verbally non-fluent/design fluent); (2) The groups should have relative activation differences over the right and left frontal lobe. Individuals who are depressed and verbally non-fluent should evidence heightened left frontal delta (or less left frontal Beta) magnitude when compared to those who are depressed and design non-fluent and should show a significant decrease in words remembered on the AAVLT compared to

normal controls. They should also show a learning bias for negative words; (3) Individuals who are depressed and design non-fluent should evidence heightened right frontal delta (or less right frontal Beta) magnitude when compared to those who are depressed and verbally non-fluent and should show a significant decrease in words remembered on the AAVLT compare to normal controls. This group should show a learning bias for negative words; (4) The normal control group should out perform the depressed individuals on the AAVLT. They should also evidence less delta magnitude (or greater Beta magnitude) over the frontal lobes when compared to either depressed group. The normal control group should also show normal primacy and recency effects when taking the AAVLT.

It is expected that this research will eventually lead to the identification of subtypes of depression whose symptoms may differ individually, as predicted by the Shenal model which offers an improved understanding of relative cerebral activation. The differences among depression types have been noted to some degree by multiple authors (Bruder, et al., 1995; Heller et al. 1998). The current methodology may help to explain some of the discrepancies found within the literature that have noted differential activation patterns of those with depression (see above). It may be possible once depression is better defined, to be able to inform therapy to reach those who may have been resistant to therapy due to the inability to properly respond to cognitive behavioral (verbal) treatment.

Methods

Participants

A total of 20 men participated in exchange for extra credit in the undergraduate psychology course in which they were enrolled. The Coren, Porac, and Duncan Laterality Questionnaire (CPD; Coren, Porac, Duncan, 1979) was used to assess handedness. To be considered for inclusion participants had to score at least ≥ 7 on the CPD. Scores range from -13 to +13. Positive scores indicate increased right handedness. Inclusion criterion consisted of having no history of significant head injury or neuropsychological disorder and no current health problems as assessed by the Behavioral Neuroscience Medical History Questionnaire (see appendix). All participants were further classified using the Beck Depression Inventory (Beck, 1987) to identify those with depressed mood for participation in the project.

Participants were assigned to a Low Figural Fluency group based on their performance on the RFFT given in a group setting. Specifically, the total number of unique designs produced, were used. Participants were selected who fall one standard deviation below the mean on the RFFT (Ruff, 1996).

Participants were also assigned to a Low Verbal Fluency group based on their performance on the COWAT. Specifically, the total number of unique words produced will be used to measure verbal fluency. The fluency groups were selected from those that are in the top and bottom third of the participant scores, respectively.

Participants also completed the Cook-Medley Hostility Inventory (CMHO; Cook & Medley, 1954) to assess for hostility, which has been shown to influence performance on cognitive tasks (Everhart, Demaree, & Harrison, in press; Mollet & Harrison, 2006)

Classification Measures

Handedness was measured using a valid self-report questionnaire (Coren, Porac, Duncan, 1979; see Appendix A)) consisting of 13 items that assesses lateral preference for the hand, foot, eye, and ear. Items are scored as +1 for “right-handed,” -1 for “left-handed,” or 0 for “both” (right and left responses). This information was assessed during the screening phase.

The Behavioral Neuroscience Medical History Questionnaire (see Appendix B) will be administered during online screening to assess for past and present head injury, stroke, seizures, paralysis, medical illness, psychological or psychiatric problems, sensory impairments, prescription medication use, and problems or pain related to movement. Individuals endorsing relevant problems or histories other than depression will be excluded from this research.

Depression was assessed using the validated self-report Beck Depression Inventory (Beck, 1987). Scores on this 21 question measure range from 0 to 63. Participants’ scores of 0 to 5 were considered non-depressed. 10 non-depressed individuals were asked to participate. Ten depressed individuals (scoring 15 or above on the BDI) were also asked to participate in the second phase of the experiment.

Anxiety was assessed during the initial screening. The State-Trait Anxiety Inventory (Spielberger, 1985), a validated self-report measure was used. It is a 40-item questionnaire which assesses both state and trait anxiety. The inventory was not used as

an exclusionary measure. However, it was used to further identify affective characteristics of the participants.

Participants were asked to complete the Cook-Medley Hostility Inventory (CMHO; Cook & Medley, 1954), a 50- Item true/false questionnaire that has been shown to be “a valid predictor of medical, psychological, and interpersonal outcomes”, as quoted in Everhart, Demaree, and Harrison, 2007. Previous research has also found good convergent and discriminate validity (Barefoot, Dodge, Peterson, Dahlstrom, & Williams, 1989) when the CMHO was compared to the Minnesota Multiphasic Personality Inventory.

Fluency Measures

The Ruff Figural Fluency Test (RFFT; Ruff, 1996; Ruff et al.1987) is a measure of design fluency consisting of five individual parts, with each part consisting of different, self generated stimulus patterns. The participants are instructed to draw as many novel designs as possible by connecting at least two of the dots comprising a 5 dot matrix. Nonverbal fluency is then considered to be the total number of unique designs drawn during the 5 one minute trials. The Low Figural Fluency group will be identified in the initial screening sessions in the laboratory phase. Fifteen design non-fluent individuals will be asked to participate in the experimental phase. The RFFT has been shown to be sensitive to right frontal function/capacity (Demakis and Harrison, 1997; Ruff, Light, & Evans, 1987; Baldo, Shimamura, Delis, Kramer, & Kaplan, 2001). Moreover, performance has been shown to vary reliably with right frontal lobe activation using qEEG (Foster, Williamson, & Harrison, 2005).

The Controlled Oral Word Association Test (COWAT; Benton & Hamsher, 1976) is a measure of verbal fluency consisting of three trials on which participants are instructed to generate and write as many words as possible in 1 min. beginning with a specified letter (F, A, and S). Proper names, numbers and perseverative repetitions of the same word, are not allowed. The final score is the sum of the acceptable words produced across the three trials. The COWAT has been shown to be sensitive to left frontal function/capacity (Stuss, 1986). Fifteen verbally non-fluent individuals will be asked to participate. This measure will be used as a descriptive measure to further identify differences between the groups.

Memory Measure

The Affective Auditory Verbal Learning Test (AAVLT; Snyder & Harrison, 1997; Mollet & Harrison, 2006; Snyder, Harrison, and Shenal, 1998) is a measure of affective memory consisting of five trials of 15 words, of which participants must attempt to recall as many words as possible after all of the words have been read. Each affect condition has either a theme of positive, negative, or neutral valence words. The number of correct reproductions from each trial of each condition will be used in the analysis.

Procedure

Participants were initially screened by administering the BDI, STAI, CMHO, CPD, and the medical history questionnaire. Following the screening, participants were invited to the next phase of the investigation, which was a group administration of the written COWAT and RFFT. The participants were given a brief description of the experimental procedure and an opportunity to answer any questions.

Following the collection of the screening data the participants were then asked to take the AAVLT. At this point the participants read the following instructions:

You will be hearing a list of words. Listen carefully to the words. When the list is finished I want you to write as many words as you can remember. You may write the words in any order. Just try to remember as many as you can.

The experimenter recorded all of the responses on a data sheet. When the participant ceased to recall words the trial was ended. Trial 1 was followed by Trial 2-5. The instructions for those trials was read as follows:

You are going to hear the same list of words. Again, listen carefully to the words. When the list is finished I want you to write as many words as you can remember, including the words you wrote before. You may write the words in any order. Just try to remember as many as you can.

Responses were recorded in the same manner as in Trial 1. The entire format was repeated with the other two lists. The Neutral list was always given first. Order for negative and positive lists was counterbalanced within each group. When the AAVLT has been completed, participants were thanked for their participation, and dismissed.

Statistical Methods

To examine primacy and recency effects, a four factor mixed design ANOVA will be used. The ANOVA will be performed on the AAVLT data with a fixed effect of group (normal and depressed) with repeated measures of valence (neutral, positive, and negative), trial (verbal learning trials 1-5), and word location (beginning, middle, and end).

Results

There were several committee-approved changes to the original proposed methods section due to unforeseen problems with data collection. The first change was the exclusion of all qEEG data collection and analysis due to technical difficulties with the apparatus and software. The second modification was of the ANOVA AAVLT analysis to explore only the depressed vs non-depressed groups. The final analysis excluded the use of the fluency categories (low figural fluency and low verbal fluency). The exclusion of the low figural and low verbal fluency categories was necessary due to time constraints for data collection, the low number of individuals who participated and were depressed, and the inability to get equal or adequate sample numbers per fluency cell with these constraints.

To maximize the size of the depressed group (N= 10) two exclusion criteria were loosened to include two more participants in the depressed group (from N= 8). This included one individual who was left handed (CPD= -5) and using a new depressed group minimum BDI score of 13. To get an adequate sample, and to account for possible problems with heterogeneity, a paired comparison approach was used where equal

numbers of depressed (N=10) and non-depressed (N=10) individuals were matched by laterality questionnaire score, order in which they received the RFFT and COWAT (Order), order in which they received the positive and negative word lists (Valance: 1= positive first 2= negative first), and Age. The only differences found in the matched control sample was a laterality questionnaire score of -2 instead of -5 for the left-handed matched control, and a mean age difference between normal comparison and depressed comparison groups of 1.1 years (range 0-3 years difference) per match.

Self Report Questionnaire Analysis for the Paired Sample

An a priori significance of $p < .05$ was used for all analyses. One-way ANOVAs were used to compare group means from the Coren, Porac, and Duncan Laterality Questionnaire (Coren, Porac, & Duncan, 1979) and the Beck Depression Inventory (Beck, 1987). Results indicated that the groups were statistically equivalent on the laterality questionnaire ($F(1, 19) = .01, p = .90$). The mean score for the depressed group on the laterality questionnaire was 6.30 (SD = 5.81). The mean score for the non-depressed group on the laterality questionnaire was 6.60 (SD = 5.21). Scores for the depressed and non-depressed groups on the Beck Depression Inventory (Beck, 1987) were significantly different ($F(1, 19) = 57.05, p < .01$). The depressed group ($M = 17.00, SD = 4.59$) scored significantly higher on the Beck Depression Inventory (Beck, 1987) relative to the non-depressed group ($M = 3.9, SD = 2.99$). See Figure 1 for the means of this sample comparison. There were non-significant one-way ANOVA group differences for Order $F(1, 19) = 2.86, p = .11$, Valance $F(1, 19) = .82, p = .50$, and Age $F(1, 19) = .20, p = .66$.

Two one-way ANOVAs were used to examine group differences in levels of state-anxiety and trait-anxiety reported. Separate ANOVAs were computed for the state-anxiety scores and the trait-anxiety scores. A main effect for group ($F(1,19) = 7.58, p = .013$) on self-reported level of state-anxiety was found. Post-hoc comparisons indicated that the depressed group ($M = 38.90, SD = 9.53$) scored significantly higher on the STAI-S than the non-depressed group ($M = 28.90, SD = 6.40$). Figure 1 shows the mean comparisons for this ANOVA. A main effect of group ($F(1, 19) = 20.35, p < .01$) on the level of self-reported trait-anxiety during data collection was also found. Post-hoc comparisons using Tukey's HSD, indicated that depressed men ($M = 46.90, SD = 10.14$) reported experiencing significantly more trait-anxiety on the STAI-T than non-depressed men ($M = 30.80, SD = 4.93$). See Figure 1 for a visual display of means compared in this ANOVA.

A one-way ANOVA was used to examine group differences in the level of hostility as measured by the CMHO (Cook & Medley, 1954). A main effect for group was found ($F(1, 19) = 5.97, p < .05$) on the level of hostility reported during data collection. Post-hoc comparisons indicated that depressed men ($M = 28.90, SD = 7.57$) reported significantly more hostility than non depressed men ($M = 20.40, SD = 7.97$). See Figure 1 for a graphical display from this means comparison.

Three one-way ANOVAs were used to assess differences among the depressed groups as a function of the verbal and figural fluency measures. There were no significant mean differences between depressed groups on the verbal fluency measure (COWAT) ($F(1, 19), p = .95$). Non-significant depressed group differences were found on the figural fluency measure (RFFT: M) for both unique designs (RFFTUD) ($F(1, 19) = .03, p = .87$)

and perseverative errors (RFFTP) ($F(1, 19) = .08, p = .77$). See Figure 2 for a visual display of the COWAT & RFFT means.

Affective Auditory Verbal Learning Analysis (AAVLT)

All post hoc comparisons were performed using a Tukey HSD. Data were analyzed using a 4-way mixed design ANOVA with the fixed effect of Group (depressed and non-depressed) with the repeated measures of Valence (neutral, positive, negative), Trial (1-5), and Location (1-3). The analysis revealed a significant interaction of Valence x Location, $F(4, 72) = 5.45; p < .0007$. The graph of this interaction has been displayed in Figure 5. The analysis also illustrated a simple effect for location when the negative word set was read, $F(2,297) = 27.26, p < .0001$ (see Figure 3). Post hoc comparisons (Winer, 1971) indicated that participants learning the negative word list remembered significantly more words at Location 1, than at Location 2 or Location 3.

The analysis also revealed a significant main effect for trial (1-5; see Figure 4) of the AAVLT, $F(4,18) = 12.91; p < .0001$ and a main effect for location (1-3), $F(2,36) = 31.19; p < .0001$. Post hoc comparisons indicated a significant difference between trial one and each of the other trials. A significant difference was found between trial 2 and trial 5. A significant difference was also found between trial 5 and trials 1 & 2. No other main effects or interaction effects were significant. Please see Table 1 for a list of means and standard deviations for each variable.

Discussion

In the current experiment none of the hypothesized group differences were significant. This is thought to have happened for several reasons; the first was the small

number of depressed individuals recruited for participation. In the original proposal an n of 15 individuals per cell was necessary to adequately measure the group and fluency categorical variables. In the sample used in the final analysis, a total of 10 depressed participants and 10 non-depressed matched controls were used. Among those participants that were used, all of the depressed individuals were in the same qualitative depression category of “Mild” depression (BDI range= 13-27) according to the BDI. The non-significant group effects may illustrate the very function of the qualitative rating system, in that the significant difference found in BDI scores between the groups, may not in fact be as diagnostic as the qualitative ratings. It is thought that if there had been more individuals in the “moderate” or “severe” categories (or a mean “depressed” group BDI score closer to the maximum of 63), the sample may have been a better representation of a clinically depressed population, which has been shown to perform significantly worse on multiple measures of learning and cognition when compared to a non-depressed group (Crews, Harrison, and Rhodes, 1999; Emerson, Harrison, Everhart, and Williamson, 2001; Behnken et al., 2010 & Reppermund et al., 2007).

In this experiment, there were group differences on multiple measures of negative affect (please see results section or Figures 2-4). There have been other studies that have shown the relationship of high depression scores and high scores on measures of anxiety (Crews and Harrison, 1994) and hostility (Everhart, Demaree, Harrison, 2008). This experiment corroborates with those findings, in that individuals that had a higher depression score, were also more likely to have high scores on other measures of negative affect.

One interesting interaction in this experiment was the valence by location effect in which it was shown that the negative word list on the AAVLT caused a primacy or perseveration effect (please see Figure 5 for a graphical display). This effect occurred without respect to depressed group, which was counter to the original hypothesis. Additionally, both the neutral and positive word lists showed a normal primacy and recency effect across groups. This may have occurred for a couple of reasons. The first is that the AAVLT may have some utility as a negative affect primer. Hearing and learning negative words over multiple trials may cause, temporary affect changes (relative right hemisphere activation) in individuals, and cause a learning bias for words at the beginning of a set, while distracting further learning of words in the middle or at the end of the negative word set. In an experiment done by Snyder, Harrison, and Shenal (1998), they showed an increase in autonomic nervous system activity (mean diastolic blood pressure increases) when the AAVLT negative word list was read, compared to the positive word list (ANS decreases) or neutral word list (non-significant diastolic differences), which supports a priming or perseveration explanation of the results. In other similar research, Shenal, Rhodes, and Harrison (2000), were able to show a link from the AAVLT negative word list to cardiovascular reactivity among individuals without negative affect disorders. This demonstrates that these effects also carried over to other control groups. In an experiment that directly tested the use of the AAVLT negative word list as a method of mood induction, there were significant results when high and low hostile groups were compared (Demaree & Everhart, 2004).

An alternative explanation of the primacy effect found in this experiment may be that it is more functionally related to the other co-morbid negative affect scores, in that it

may be associated with the relative right hemispheric activation shown to occur during negative affect (Heller 1993; Everhart, Demaree, and Harrison, 2008). It may be that either anxiety or hostility, play a more central role in this phenomenon. The primacy effect has been shown to occur in experiments of negative affect that look at high-hostility groups (Everhart, Demaree, and Wuensch, 2003) or high-anxiety groups (Everhart, Harrison, Shenal, Williamson, and Wuensch, 2002). It may be that the general right hemispheric activation could be driving this effect. Further research will be necessary to discover the utility of the negative word list of the AAVLT as a negative affect manipulation. It should also be noted that the AAVLT positive word list recency effect found in other studies (Snyder, Harrison, Shenal, 1998; Demaree and Everhart, 2004; Demaree et al., 2004; Everhart and Demaree, 2003) was not supported here.

The sample used in this experiment showed a normal learning curve. The total words learned increased over trials regardless of valence or group. This was counter to the hypothesis that the depressed group would learn significantly less total words and words per trial except for acquisition on the negative words list of the AAVLT. This result lends support to the idea that the “depressed” group in this sample may not have been a good representation of a clinically depressed sample. Other studies of learning have shown that clinically depressed individuals evidence major learning deficits when compared to controls on tests of verbal and non-verbal memory (Behnken et al., 2010 & Reppermund et al., 2007), validating the likelihood that the original hypothesis was justifiable, and that our sample may not be a good representation of a clinically depressed one.

Future research could be focused in a few directions, the first being experiments that will focus on the affective lists of the AAVLT as tools to manipulate immediate affective states. The AAVLT positive and negative word lists have already shown some utility in this fashion (Snyder, Harrison, Shenal, 1998; Demaree and Everhart, 2004; Demaree et al., 2004; Everhart and Demaree, 2003) but it has not been looked at while accounting for possible covariates (like depression or anxiety). If it does still maintain its effectiveness as an emotional primer when looking across groups with and without affective dysfunction, it may allow us to examine aspects of short term emotional regulation and its relationship to more stable clinically diagnosed disorders.

Another future research idea may be to actually attempt to re-run the originally proposed experiment. The focus of the second effort should be in partnering with local outpatient care facilities to attain an adequate depressed population that is clinically diagnosed and, therefore, more representative of the population in question. This would once again allow an investigation of the differences within the depressed population. One new aspect that could be added to the original proposition could be the examination of the qualitative ratings of the BDI to examine their utility. It may be that different levels of depression actually have functional differences on tests of learning, memory, and cognition. This could be examined with the added effectiveness of qEEG to get at the neurological basis for these differences that have so far only been noted in theoretical papers (Shenal, Harrison, and Demaree, 2003).

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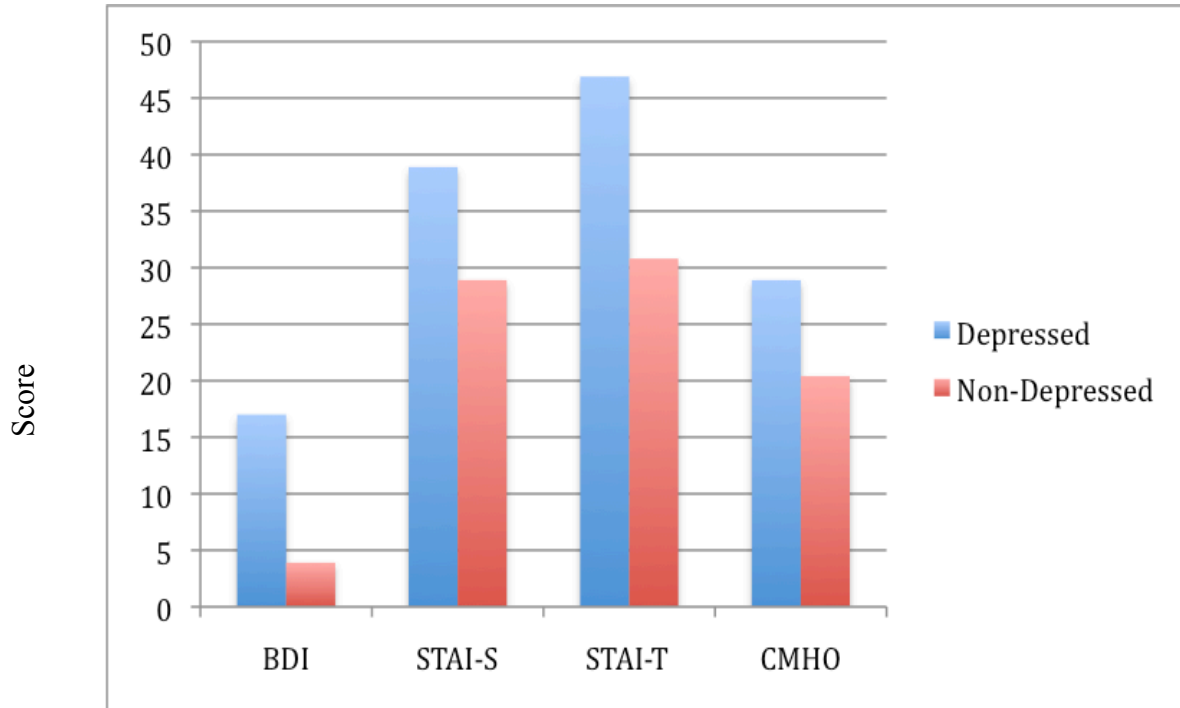
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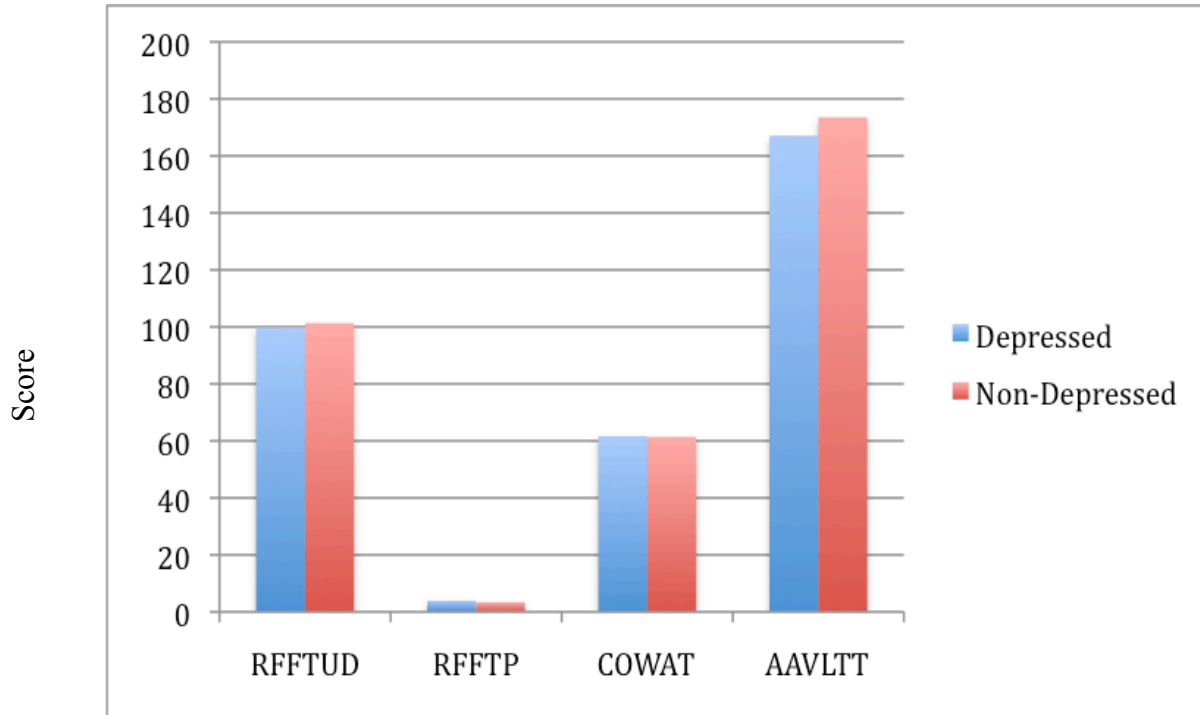
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Self Report Questionnaire Score by Depression Group

Figure 1. Mean BDI, STAI-S, STAI-T, and CMHO scores for the depressed and non-depressed groups.



Measurement Score by Depression Group

Figure 2. Mean RFFT Unique Design, RFFT Perserveration, COWAT, and AAVLT Total score for the depressed and non-depressed groups.

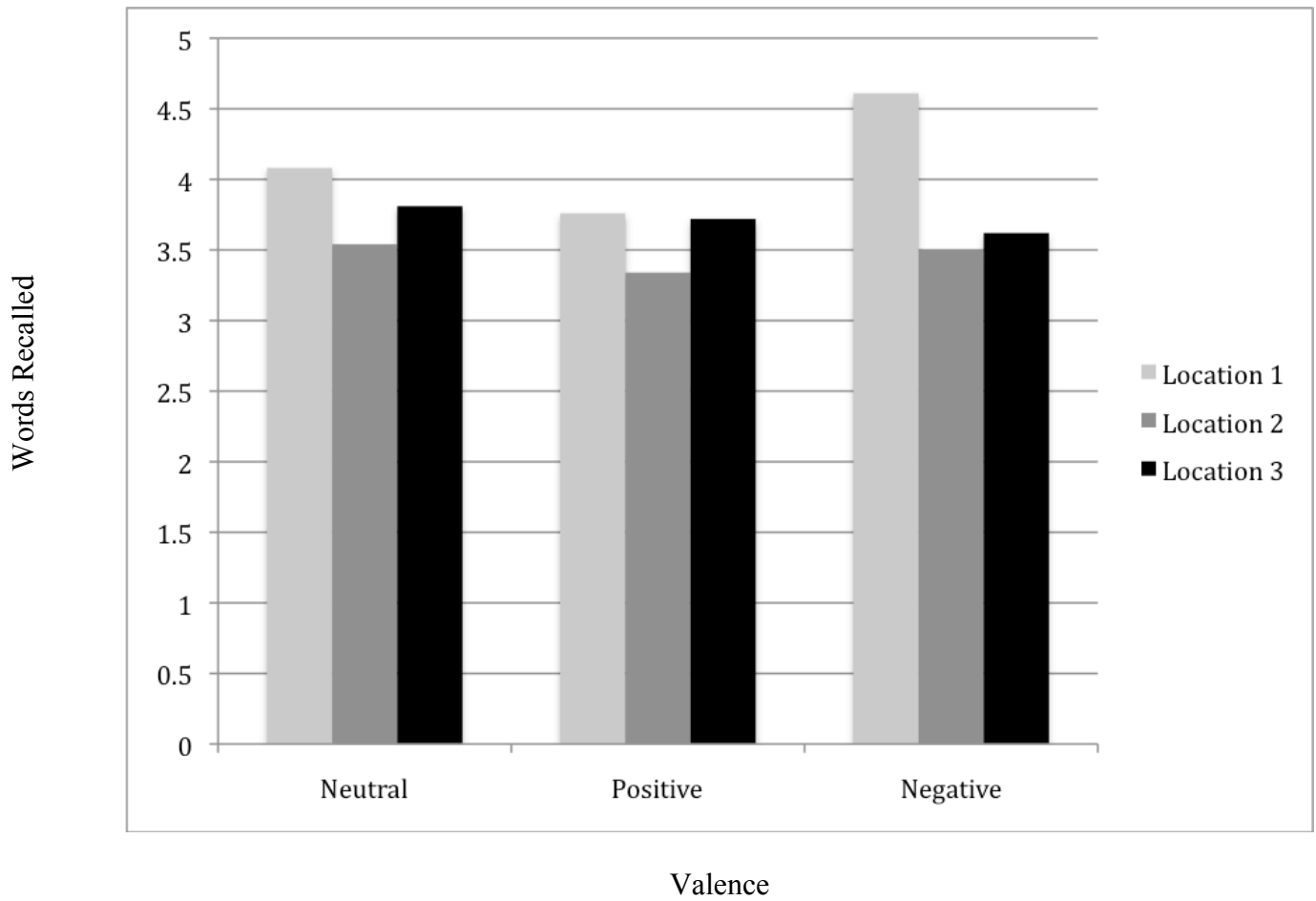


Figure 3. Mean number of words recalled by location (1-3) and valence (neutral, positive, and negative).

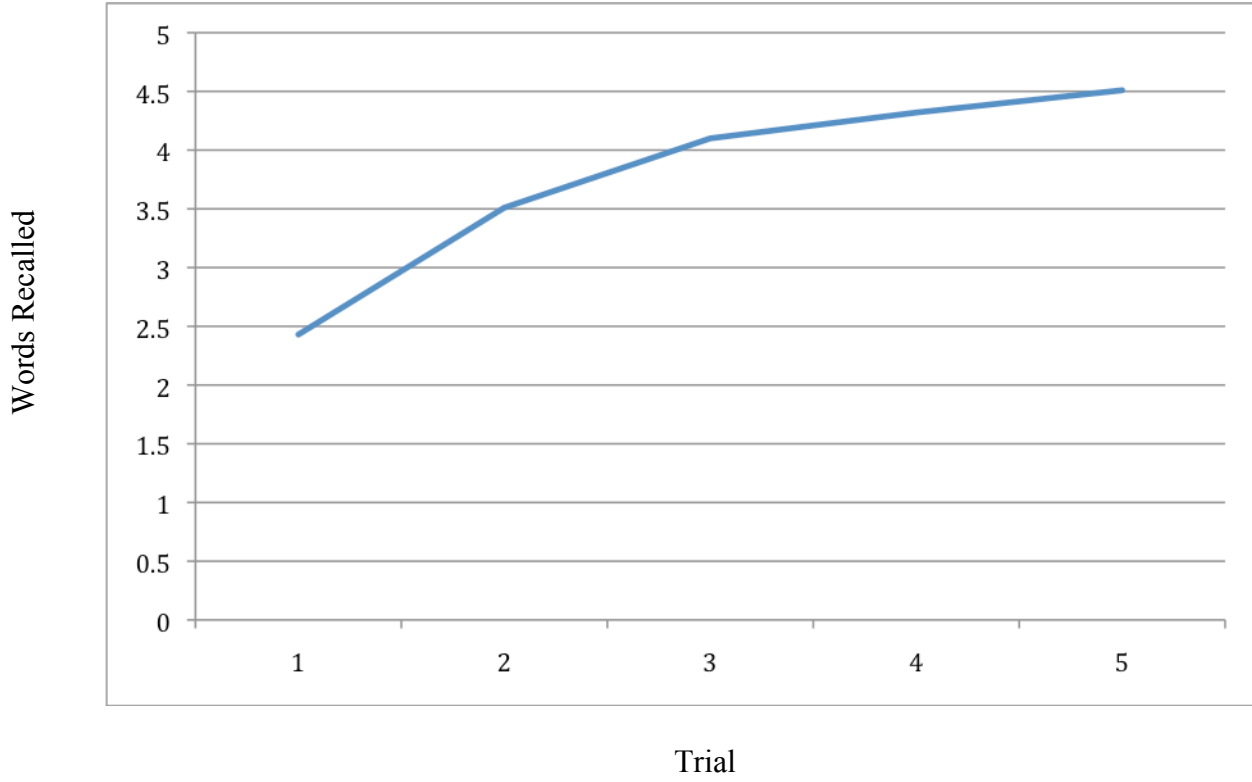


Figure 4. Mean number of words recalled as a function of trial (1-5).

Table 1

Descriptives Table of Means and Standard Deviations of Variables

Variable	Dep. Mean	Non-Dep. Mean	Dep. SD	Non-Dep. SD
BDI*	17	3.9	4.59	2.99
STAI-S*	38.9	28.9	9.53	6.4
STAI-T*	46.9	30.8	10.14	4.95
CMHO*	28.9	20.4	7.59	7.97
RFFTUD	99.7	101.3	21.58	22.1
RFFTP	3.9	3.4	4.78	2.87
COWAT	61.7	61.4	11.05	12.65
AAVTLT	167.1	173.5	23.32	14.83

*Probability Note. Variables noted with * reflect significance of $p < .05$.*

Appendix A

Laterality Questionnaire

Participant #: _____

Circle the appropriate number after each item.

With which hand would you throw a ball to hit a target?	1	-1	0
With which hand do you draw?	1	-1	0
With which hand do you use an eraser on paper?	1	-1	0
With which hand do you remove the top card when dealing?	1	-1	0
With which foot do you kick a ball?	1	-1	0
If you had to pick up a pebble with your toes, which foot would you use?	1	-1	0
If you had to step up on a chair, which foot would you place on the chair first?	1	-1	0
Which eye would you use to peep through a keyhole? 1 -1 0	1	-1	0
If you had to look into a dark bottle to see how full it was which eye would you use?	1	-1	0
Which eye would you use to sight down a rifle? 1 -1 0	1	-1	0
If you wanted to listen to a conversation going on behind a closed door, which ear would you place against the door?	1	-1	0
If you wanted to listen to someone's heartbeat, which ear would you place against his or her chest?	1	-1	0
Into which ear would you place your earphone of a transistor radio? 1 -1 0	1	-1	0

of Right + # of Left = Total Score
 _____ + _____ = _____

Is mother right or left hand dominant? _____

Is father right or left hand dominant? _____

Appendix B

Medical History Questionnaire

1	Do you have any history of congenital or developmental problems ?	Y	N
2	Do you have any history of learning disabilities or special education?	Y	N
3	Have you ever suffered a head injury resulting in a hospital stay longer than 24 hours	Y	N
4	Have you ever been knocked out or rendered unconscious (more than 5 minutes)?	Y	N
5	Have you ever suffered "black-out" or fainting spells?	Y	N
6	Do you have a history of other neurological disorders (e.g. stroke or brain tumor)?	Y	N
7	Have you ever received psychiatric/psychological care or counseling?	Y	N
8	Have you ever been hospitalized in a psychiatric facility/hospital?	Y	N
9	Have you ever been diagnosed with a psychiatric/psychological disorder?	Y	N
10	Have you ever been administered any (neuro)psychological tests or measures?	Y	N
11	Do you have a history of substance abuse or alcohol abuse?	Y	N
12	Do you have a history of high blood pressure?	Y	N
13	Do you have any uncorrected visual or hearing impairments?	Y	N
14	Are you able to read, write, and speak English effectively?	Y	N
15	Do you consume three or more alcoholic more than two nights a week?	Y	N
16	Have you ever experienced a medical or psychiatric condition that could potentially affect cognitive functioning, such as stroke, electroconvulsive treatment, epilepsy, brain surgery, encephalitis, meningitis, multiple sclerosis, Parkinson's Disease,	Y	N
17	Huntington's Chorea, Alzheimer's dementia, Schizophrenia, Bipolar Disorder ?	Y	N
18	Have you ever used smoked or used tobacco products?	Y	N
19	Do you use any unprescribed or "illegal/street" drugs?	Y	N
	Are you taking any of the following medications: antidepressant, antianxiety, antipsychotic?	Y	N
20	Are you taking any allergy or cold medication?	Y	N

MORE ON NEXT PAGE----->

If you answered "yes" to any of the above please explain fully:

Appendix C

Cook Medley Hostility Scale

Direction: If a statement is true or mostly true, as pertaining to you, circle the letter T. If a statement is false, or usually not true about you, circle the letter F.

Try to give a response to every statement.

1. When I take a new job, I like to be tipped off on who should be gotten next to.	T	F
2. When someone does me wrong, I feel I should pay him back if I can, just for the principle of the thing.	T	F
3. I prefer to pass by school friends, or people I know but have not seen for a long time, unless they speak to me first.	T	F
4. I often had to take orders from someone who did not know as much as I did.	T	F
5. I think a great many people exaggerate their misfortunes in order to gain the sympathy and help of others.	T	F
6. It takes a lot of argument to convince most people of the truth.	T	F
7. I think most people lie to get ahead.	T	F
8. Someone has it in for me.	T	F
9. Most people are honest chiefly through the fear of getting caught.	T	F
10. Most people will use somewhat unfair means to gain profit or an advantage, rather than lose it.	T	F
11. I commonly wonder what hidden reason another person may have for doing something nice for me.	T	F
12. It makes me impatient to have people ask my advice or otherwise interrupt me when I am working on something important.	T	F
13. I feel that I have often been punished without cause. T F	T	F
14. I am against giving money to beggars. T F	T	T
15. Some of my family have habits that bother me very much. T F	T	F
16. My relatives are nearly all in sympathy with me. T F	T	F
17. My way of doing things is apt to be misunderstood by others. T F	T	F
18. I don't blame anyone for trying to grab everything they can get in this world.	T	F
19. No one cares what happens to you.	T	F
20. I can be friendly with people who do things I consider wrong.	T	F
21. It is safer to trust nobody.	T	F
22. I do not blame a person for taking advantage of someone who lays himself open to it.	T	F
23. I have often felt that strangers were looking at me critically.	T	F
24. Most people make friends because friends are likely to be useful to them.	T	F
25. I am sure that I am being talked about.	T	F
26. I am likely not to speak to people until they speak to me.	T	F
27. Most people inwardly dislike putting themselves out to help other people.	T	F
28. I tend to be on guard with people who are somewhat more friendly than I had expected.	T	F

29. I have sometimes stayed away from another person because I feared doing or saying something that I might regret afterwards.	T	F
30. People often disappoint me.	T	F
31. I like to keep people guessing what I'm going to do next.	T	F
32. I frequently ask people for advice.	T	F
33. I am not easily angered.	T	F
34. I have often met people who are supposed to be experts who were no better than I.	T	F
35. It makes me think of failure when I hear of the success of someone I know well.	T	F
36. I would certainly enjoy beating a crook at his own game. T F	T	F
37. I have at times had to be rough with people who were rude or annoying.	T	F
38. People generally demand more respect for their own rights than they are willing to allow for others.	T	F
39. There are certain people whom I dislike so much I am inwardly pleased when they are catching it for something they have done.	T	F
40. I am often inclined to go out of my way to win a point with someone who	T	F
has opposed me.	T	F
41. I am quite often not in on the gossip and talk of the group I belong to.	T	F
42. The man who had the most to do with me when I was a child (such as my father, step- father, etc.) was very strict with me.	T	F
43. I have often found people jealous of my good ideas just because they had not thought of them first.	T	F
44. When a man is with a woman, he is usually thinking of things related to her sex.	T	F
45. I do not try to cover up my poor opinion or pity of a person so that he won't know how I feel.	T	F
46. I have frequently worked under people who seem to have things arranged so that they get credit for good work, but are able to pass off mistakes to those under them.	T	F
47. I strongly defend my own opinions as a rule.	T	F
48. People can pretty easily change me even though I thought that my mind was made up on a subject.	T	F
49. Sometimes I am sure that other people can tell what I'm thinking.	T	F
50. A large number of people are guilty of bad sexual conduct.	T	F

