A Functional Cerebral Systems Approach to Hostility: Changes in Frontal Lobe Delta Activation and Fluency Performance as a Function of Stress

by

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ABSTRACT

Executive functions, potentially including the regulatory control of emotions and expressive fluency (verbal or design), have historically been associated with the frontal lobes. Moreover, research has demonstrated the importance of cerebral laterality with a prominent role of the right frontal regions in the regulation of negative affect (anger, hostility) and in the generation or fluent production of designs rather than verbal fluency (left frontal). In the present research, participants identified with high and with low levels of hostility were evaluated on a design fluency test twice in one experimental session. Before the second administration of the fluency test, each participant underwent cold pressor stress. It was hypothesized that diminished right frontal capacity in high hostiles would be evident through lowered performance on this cognitive stressor. Convergent validity of the "capacity model" was partially supported wherein high hostile men evidenced reduced delta magnitude over the right frontal region after exposure to a physiological stressor but failed to maintain consistent levels of right cerebral activation across conditions. The results suggest an inability for high hostile men to maintain stable levels of cerebral activation after exposure to physiological and cognitive stress. Moreover, low hostiles showed enhanced cognitive performance on the design task with lower levels of arousal (heightened delta magnitude). In contrast, reduced arousal (heightened delta magnitude) yielded increased executive deficits in high hostiles as evidenced through increased perseverative errors on the design fluency task.

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Table of Contents

List of Tables

List of Figures

A Functional Cerebral Systems Approach to Hostility: Changes in Frontal Lobe Delta Activation and Fluency Performance as a Function of Stress

INTRODUCTION

Hostility is a multidimensional construct that may be most aptly defined through neuropsychological models using a functional cerebral systems approach. Typically defined as a trait (Strong, Kahler, Greene, & Schinka, 2005), hostility has been characterized by the experience of negative emotions and a proneness to anger (Bunde & Suls, 2006; Smith, 1994) as well as increased reactivity to stress (Shenal & Harrison, 2003). Further, there is overwhelming evidence that hostility is associated with a number of health problems and risk behaviors (Williams & Barefoot, 1988) indicating an increased cost for health care and counseling. Despite the apparent relationship between hostility and health risk behaviors, there is surprisingly little evidence linking the components of this trait with functional cerebral systems regulating sympathetic nervous system activation and negative emotional perception. The proposed research will attempt to provide some insight into how functional cerebral systems may be altered with hostility by associating changes in patterns of regional cerebral activation with exposure to stress.

Hostility will be conceptualized from a functional cerebral systems approach. Heilman's model of arousal (Heilman, Watson, & Valenstein, 2003) will be introduced to support the position that changes in right cerebral function are related to hostility level. Heller's (1990; 1993) model of emotional asymmetry will be introduced to support the position that changes in right cerebral function are related to changes in emotional processing with hostility. Heller's model will be integrated with Heilman's model of

arousal to account for hostility differences in design fluency after exposure to a physiological stressor. Associations between activation of the right frontal region of the brain and performance on a task that has been demonstrated to require activation in this region will be examined using a dual-task paradigm. Evidence will be presented that hostility is associated with changes in task performance in low and high arousal conditions. Cognitive arousal will be operationally defined by focusing primarily on nonverbal task performance before and after exposure to a physiological stressor. Differences in lateralization of function with respect to hostility will be discussed.

Definition of Hostility

Hostility can be defined using physiological, behavioral, and cognitive terms. From a physiological perspective, hostility has been strongly associated with the development of cardiovascular disease (CVD) (Smith, 1994; Naiura et al., 2002; Smith & Ruiz, 2002; Miller, Smith, Turner, Guijarro, & Hallet, 1996). Hostility is considered to be derived from Type A behavior patterns (Siegman, 1994), which were originally thought to be the primary contributors to CVD. Recent research has shown that high levels of hostility are related to greater cardiovascular reactivity to stressful situations (Rhodes, Harrison, & Demaree, 2002; Smith, Glazer, Ruiz, & Gallo, 2004), higher resting levels of blood pressure (BP) and heart rate (HR) (Demaree & Harrison, 1997; Keefe, Castell, & Blumenthal, 1986), and premature death (Everson et al., 1997). Increased risk for hypertension and CVD among hostile individuals has been well-documented (Zhu et al., 2005; Nelson et al., 2005; Olson et al., 2005), indicating a need for further research examining the effects of hostility level as a function of exposure to stress.

Behaviorally, hostility can be defined as an increased expression of anger (Spicer & Chamberlain, 1996; Anderson, Linden, & Habra, 2006) and aggressive behavior. Heightened levels of hostility have also been related to increased incidents of health risk behaviors, such as increased cigarette (Barefoot, Dodge, Peterson, Dahlstrom, & Williams, 1992) and alcohol (Houston & Vavak, 1991) consumption. Cognitively, hostility may be defined as an increased incidence of cynicism and negative attribution bias (Buss & Perry, 1992), where hostile individuals are more likely to perceive neutral stimuli as negative or offensive in nature. When requested to identify emotional faces tachistoscopically presented to the left visual field (LVF) and right visual field (RVF), individuals were more accurate at identifying emotional faces presented to the LVF (right hemisphere) (Harrison & Gorelczenko, 1990). Further, high hostile individuals were more likely to identify neutral faces as negatively valenced, indicating a bias for facial affect at the LVF.

The multidimensional definition of hostility, along with its strong association with CVD and prevalence in the population (Williams & Williams, 1993; p183) has prompted the development of a neuropsychological model of the functional cerebral systems that involve these dimensions of hostility. Briefly, this capacity model states that physiological arousal is associated with negative emotional processing that is characteristic of high hostile individuals. Changes in physiological arousal and emotional processing with hostility are related to right hemispheric perceptual processing and right frontal regulation of stress (Demaree & Harrison, 1997; Shenal & Harrison, 2003; Rhodes, Harrison, & Demaree, 2002).

Functional Cerebral Systems Approach

The underlying assumption of the proposed research is that there are differences with hostility level in the functional cerebral systems that underlie task completion and changes in arousal states. Functional cerebral systems are defined by Luria (1973) as being composed of interconnected regions that are active to varying degrees during the completion of a task. Depending on the requirements of the task, different cerebral regions that are required for the successful completion of a task may be recruited through the interconnected networks that facilitate them. Understanding the nature of this recruitment may be instrumental in determining the underpinnings of behavioral and physiological differences in hostility.

Essentially, it would be inaccurate to view successful completion of a complex task as resulting from activation of one region of the brain. In contrast, it would also be inaccurate to view all regions of the brain as being equally involved in the completion of a complex task. The most accurate way to conceptualize the involvement of brain regions that contribute to the completion of a task is to view it as a functional system (Luria, 1973) in which cerebral regions and the pathways that interconnect them are active to varying degrees depending on the task requirements.

Functional Cerebral Space Model

Luria's work may be extended to include specific ways in which two or more tasks that recruit similar neural systems may present conflict with respect to function. In other words, carrying out two tasks at the same time will result in poor performance if both tasks require activation from cerebral regions that are anatomically close in proximity. Kinsbourne and Hicks (1978) first proposed the idea of functional cerebral

space, indicating that functional capacity (or optimal ability to complete a task) becomes limited when the same functional cerebral system is used for more than one task. When a continuous activity is carried out, it involves a locus or point at which activation starts. A neural network that involves the spread of activation to corresponding cerebral regions is also integral to completion of continuous tasks. These two units comprise the functional cerebral space involved for carrying out a given task.

Active regions of the brain that are closer in location will more likely experience interference, and this is especially the case for loci in the same hemisphere. Kinsbourne and Cook (1971) found that performing dual tasks such as speaking and tapping the left index finger was carried out more efficiently than speaking and tapping the right index finger. According to this model, the dual task was completed with greater ease when carried out using the left index finger because it employed more anatomically distant regions of the brain. The same effect can be found in dual concurrent tasks that employ manual and cognitive abilities such as typing and counting backwards (Hiscock, Caroselli, & Wood, 2006; Caroselli, Hiscock, & Roebuck, 1997). In this case, optimal typing performance seems to limit cognitive performance for right-handed individuals.

The main assumption of the dual task approach is that capacity to process information is limited (Kinsbourne & Hicks, 1978; Hiscock et al., 2006; Huang & Mercer, 2001; Kahneman, 1973) and must be divided between two tasks. The more demanding a primary task is, the more capacity is required to complete that task at the expense of the secondary task. The reciprocal nature of cerebral regions in a network (Dennet & Kinsbourne, 1992; Kinsbourne, 2000) is directly related to performance on neuropsychological tasks (Kinsbourne, 1993). Further, communication of cerebral

regions in a network may be related to arousal in dual task paradigms (Kinsbourne, 1994). A functional cerebral system can change as a function of arousal state and this change in arousal can affect the way a dual task is carried out.

Heilman's Model of Arousal

Arousal is a physiological preparedness to process sensory and motor stimuli (Heilman et al., 2003; Coull, 1998). The two primary neuropsychological parameters of arousal are laterality (right or left hemisphere processing) and caudality (anterior and posterior processing). Heilman's model presents an outline of the functional system associated with changes in arousal state, and reiterates the importance of frontal lobe connections to posterior regions of the brain in the regulation of arousal and attention. With respect to the functional cerebral systems involved in arousal, the frontocortical-thalamic-mesencephalic reticular formation (mRF) pathway is largely responsible for increased arousal (Heilman et al., 2003). As demonstrated by Moruzzi and Magoun (1949), cortical regions are influenced by changes in function of the mRF, and stimulation of this structure results in an increase in behavioral measures of arousal (i.e.: cage activity in cats).

The frontal lobes have a largely inhibitory role over activation in the mRF (Damasio & Anderson, 2003; Heller, 1993). As follows, any disruption resulting in reduced frontal lobe input to the mRF may result in over arousal. As a result of increased activation of the posterior regions or decreased activation in the frontal regions, changes in arousal are likely to occur. The right frontal region has been implicated in the inhibitory regulation of arousal. Reaction times for right-hand responses were shorter when stimuli were presented to the right hemisphere, whereas reaction times for left-hand

responses were longer when stimuli were presented to the left hemisphere (Heilman & Van Den Abell, 1980). This finding is consistent with the hypothesis that the right hemisphere processes visual stimuli more readily, and implicates the enhanced role of the right hemisphere in arousal.

According to Heilman's model of arousal, the right hemisphere plays a greater role than does the left hemisphere in the arousal-attention domain. Perhaps the most convincing evidence of right hemisphere dominance for arousal comes from Heilman and Van Den Abell (1980). Heilman and Van Den Abell found EEG desynchronization of the right parietal region during presentations of visual stimuli to either hemifield whereas EEG desynchronization of the left parietal region occurred only during presentation to the right hemifield in normal participants.

Additional evidence of right hemisphere involvement in the arousal and attention domain comes from examination of patients that demonstrate hemispatial neglect (Heilman et al., 2003; Heilman, Schwartz, & Watson, 1978), or a commonly reported unawareness of stimuli within the left hemispace. These patients show a variety of behavioral manifestations of inattention, such as copying only the right side of a picture (Heilman, 1995), dressing only the right side of their body (Heilman et al., 2003), and deviation to the right side on a line bisection task (Heilman & Gilmore, 1998). As previously mentioned, these patients often fail to attend to the left side of extrapersonal or intrapersonal space despite absence of damage to sensory and motor areas. Hemispatial neglect is more commonly seen in patients with right hemisphere damage than left, due to the enhanced role of the right hemisphere in arousal and attention (Heilman & Gilmore, 1998; Heilman et al., 2003, Mesulam, 2000).

Physiological Indices

There is evidence that the right hemisphere regulates physiological levels of arousal. Increases in HR and systolic blood pressure (SBP) are associated with increased right cerebral activation. Sodium amobarbitol injections to the left intracarotid artery (Wadda testing) in epileptic patients was related to an increase in HR, whereas the opposite effect was found with injections to the right intracarotid artery (Zamrini et al., 1990). When temporal regions of the right hemisphere are stimulated an increase in HR and SBP are seen (Oppenheimer, Gelb, Girvin, & Hachinski, 1992). The right brain is thought to differentially control sympathetic regulation of the heart (Wittling, 1995; Wittling, Block Schweiger, & Genzel, 1998). Essentially, the above mentioned research shows that incremental changes in SBP and HR are strongly related to right hemisphere function.

Using Galvanic skin response (GSR) as a measure of arousal during exposure to sensory stimuli on the ipsilesional finger, Heilman et al. (1978) found that patients with right hemisphere lesions showed reduced GSR when stimulated compared to normal controls and aphasic patients with left hemisphere lesions. Conversely, patients with left hemisphere lesions showed heightened GSR activity compared to normal controls. Given that right hemispheric lesions are strongly associated with changes in physiological (Heilman et al., 1978), and behavioral (Heilman, 2003; Mesulam, 2000) indices of cerebral activation, relating right lateralization of arousal mechanisms to emotional processing may provide further support for investigating changes in arousal as it relates to hostility.

The Right Hemisphere Model of Emotion

The basic premise of the right hemisphere model of emotion is that the right hemisphere is specialized for processing emotions regardless of valence (Tucker, 1981; Borod, Koff, & Caron, 1983). Similar to those of arousal, neuropsychological parameters of emotional processing include laterality (right or left hemisphere), caudality (anterior or posterior), and processing mode (perception, and expression).

Arousal has been demonstrated to differentially mediate the perception and expression of emotion in right-hemisphere damaged (RHD) and left-hemisphere damaged (LHD) patients (Borod, 1992; Borod et al., 2000; Borod, Bloom, Brickman, Nakhutina, & Curke, 2002). Patients with right hemisphere lesions that demonstrate hemispatial neglect tend to have flat emotional reactions, whereas patients with left hemisphere damage tend to have catastrophic reactions (Heilman et al., 1978; Dekosky, Heilman, Bowers, & Valenstein, 1980) that are not in proportion with the event that induced them. Additionally, RHD patients were less able to detect emotional faces or scenes compared to patients with LHD and normal controls (Dekosky et al., 1980). Walters, Harrison, Williamson, & Foster (2006) reported that patients experiencing visual formaethesias (hallucinations) in the left hemispace (right hemisphere) reported a negative affective valence associated with these formaethesias.

One possible interpretation of these findings is that the right hemisphere mediates emotional expression, as demonstrated by flat affect seen in these individuals compared to LHD individuals and normal controls. Along the same lines, patients with RHD are less able to perceive and to interpret emotional stimuli compared to patients with LHD, and normal controls. Borod (1992) suggests that the right hemisphere advantage for

emotional processing is related to the fact that emotions have spatial and nonverbal properties that are congruent with those mediated by the right hemisphere.

Similar findings have been demonstrated in research examining right hemisphere processing in normal healthy adults. In a dichotic listening paradigm, healthy participants were better able to perceive phonemes conveying emotional prosody (Erhan, Borod, Tenke, & Brider, 1998) when presented to the left ear demonstrating a right hemisphere, left ear advantage (RH-LEA) for emotional phonemes with both positively and negatively valenced emotional tones. Conversely, a left hemisphere, right ear advantage (LH-REA) was found when nonprosodic verbal phonemes were used. For research done in our lab, participants were less able to identify phonemes presented to the left ear after exposure to an affective stressor (Holland, Mollet, Carmona, Addison, & Harrison, 2007), indicating a reduction in the capacity of the right hemisphere to process affective information and direct intention to the left hemibody concurrently. In essence, the addition of emotional prosody may affect the well-documented LH-REA commonly seen in dichotic listening paradigms.

Other Models of Emotion

While an in-depth review of all proposed models of emotion is beyond the scope of the current review, two other models take into account important aspects of the lateralization of emotion, and are worth mentioning here. To reiterate, the right hemisphere model of emotion states that the right hemisphere is dominant for regulating positive and negative valences of emotion with respect to perception and expression (Heilman & Bowers, 1990). The valence model is a modification of the right hemisphere model in that it incorporates relative right and left hemispheric activation for negative

and positive emotions, respectively (Davidson, 1984; Everhart & Demaree, 2003). Using this approach, the left hemisphere is thought to be more dominant in positively valenced emotional reactions in that they are more communicative in nature (Ross et al., 1994; Borod, Koff, & Buck, 1986). In contrast, the right hemisphere is thought to be more dominant in negatively valenced emotions in that they are thought to be more reactive in nature.

Another model outlines the behavioral inhibition system (BIS) and behavioral activation (BAS) system in the regulation of emotional expression and experience. Briefly, this model states that the left frontal region regulates approach emotions, including happiness and anger (Harmon-Jones, 2003). Conversely, the right frontal lobe regulates withdrawal emotions, including those of sadness and fear (Harmon-Jones, 2003). EEG indices of central arousal during the experience of these emotions include alpha desynchronization of the left and right frontal lobes, which indicate relative activation during happy or sad emotional states, respectively. It is apparent that models of emotion take into account different aspects of expression and perception, and incorporate motivational factors into their paradigms.

Demaree, Everhart, Youngstrom, and Harrison (2005) integrate the above models of emotion into a framework of cerebral dominance. By taking into account cerebral dominance as a critical factor in the experience of emotion, Demaree et al. (2005) propose that physiological indices of left-frontal arousal may be associated with feelings of dominance, whereas indices of right-frontal activation may be associated with feelings of submission. While there is disagreement as to the nature of emotional processing of positively valenced emotions, there is much less controversy regarding the notion that

negatively-valenced emotions are processed within the right hemisphere (Demaree et al., 2005; Borod et al., 2002; Davidson, 1984; Ross et al., 1994).

By recognizing the influence of arousal in the expression and perception of emotion, it may be possible to determine a link between changes in arousal and corresponding reports of negative feelings as they relate to right frontal and temporo-parietal activation. Additionally, this theoretical framework may also be extended to explore differences in emotional expression and perception as an indication of changes in arousal. Clearly, it is constructive to take into account interactions with posterior regions of the brain when examining emotional processing.

Heller's Model of Emotional Asymmetry

Integration of the principles of the arousal and right hemisphere models indicates that the right anterior and posterior cerebral regions are differentially activated in emotion. Different cerebral systems may act upon each other in an excitatory or inhibitory manner. The model of emotional asymmetry (Heller, 1990; 1993) has been influential in guiding research concerning selective changes in right frontal activation in mood disorders, such as depression (Nitschke, Heller, Etienne, & Miller, 2004) and anxiety without depression (Nitschke, Heller, Palmieri, & Miller, 1999; Keller et al., 2000). Briefly, this research has consistently found greater right posterior activation in individuals classified as high on anxiety scales.

In this model, Heller proposes that there is reciprocal communication between frontal and parietal regions of the brain that correspond directly with mood state. Arousal and valence are the two dimensions incorporated in this model. Consistent with Heilman's model of arousal (Heilman et al., 2003), the right posterior region plays a

critical role in contributing to changes in mood states through increasing arousal. In contrast, the right frontal region regulates mood states through inhibition of the right parietal region. With respect to caudality, any reduction in activation of the right frontal region leads to an overall increase in arousal (Heller 1990; 1993), which is consistent with Heilman's model of arousal (Heilman et al., 2003).

As previously mentioned, there is a strong association between right hemisphere activation and the experience of negatively valenced moods (Ross et al., 1994; Davidson, 1984). In depression, right frontal activation is demonstrated to be higher than left frontal activation (Nitschke et al., 2004). There is also a corresponding decrease in activation in the right parietal region. An increase in activation in the right frontal region indicates regulation of emotional states and a decrease in sympathetic arousal (Heller, 1990; 1993). There is no known empirical evidence demonstrating the relationship between positive mood valence and cerebral activation (Davidson, 1984). Left frontal activation should be higher than right frontal activation when experiencing a positively valenced mood such as happiness. In this case, right parietal activation should be higher to indicate a heightened state of arousal.

Emotional valence and autonomic arousal are intimately connected (Thayer, 1989), and play a critical role in the development of personality traits (Heller, 1993). Everhart and Harrison (2000) found that anxious men were more accurate in identifying angry faces presented to the left visual field (LVF) than were nonanxious men. Pathological changes in cerebral activation and regional communication have been evidenced to exist as indicated in Heller's capacity model and can be extended to include differences in hostility. Through applying Heller's model to hostility, hostile men are

proposed to evidence reduced right anterior activation with a corresponding increase in right posterior activation. In the following sections, differences in cerebral functioning with respect to hostility will be discussed.

Neuropsychology of Hostility

The neuropsychological effects of hostility have been extensively researched in our laboratory. Evidence of processing differences in motor (Demaree, Higgins, Williamson, & Harrison, 2002), somatosensory (Herridge, Harrison & Demaree, 1997), auditory (Demaree & Harrison, 1997), and visual (Herridge, Harrison, Mollet, & Shenal, 2004) modalities have been found in high hostile individuals. The prevalence of data demonstrating differences in multiple modalities indicates that functional cerebral systems in high hostile individuals may be altered.

Hostility leads to the experience of negative emotion and stress. Mollet and Harrison (2007) found that hostile men demonstrated enhanced ability to recall negatively valenced words on an affective verbal learning task. This finding corresponded with higher scores on an anger scale (Spielberger et al., 1985) relative to low hostile individuals. Previously mentioned neuropsychological models of emotion indicate that the right hemisphere plays a critical role in the processing of negative emotions (Borod, 1992; Demaree et al., 2005) and that arousal systems may be altered as well as emotional systems in hostility. It has been shown that right hemisphere lesions may result in decreased levels of arousal (Heilman et al., 2003) and perceptual inaccuracies in the processing of emotional stimuli (Herridge et al., 2004). This correspondence between changes in arousal and emotional processing may be further exemplified in high levels of hostility.

Physiological Indices of Hostility

Differences in the sympathetic measures of SBP and HR have been seen in low and high hostile individuals. Rhodes, Harrison, and Demaree (2002) exposed low and high hostile individuals to a cold pressor stressor. The researchers found that HR in high hostiles increased significantly from pre stress levels upon being exposed to the stressor. Along the same lines, high hostile men exposed to an affective stressor (angry cries) in a dichotic listening paradigm demonstrated increased HR after exposure to affective stress (Holland, Mollet, Carmona, & Harrison, 2007b). Using the Auditory Affective Verbal Learning Test as a measure of cognitive emotional stress, Shenal and Harrison (2003) found increased SBP after learning a negative word list.

Using a vestibular activation paradigm, Carmona, Holland, Stratton, & Harrison (2008) found differences in sympathetic tone with hostility before and after exposure to vestibular stress. Using skin conductance as a measure of sympathetic tone, Carmona and colleagues found that high hostile men evidenced heightened sympathetic arousal after undergoing full body rotation. Given that sympathetic control of the heart is associated with right hemisphere activation (Wittling et al., 1998; Wittling, 1995), these findings indicate altered right hemispheric arousal and emotional processing in high hostiles.

Hemispheric Activation

Changes in right frontal function may be especially pronounced in high hostile individuals. Because negative emotionality (e.g.: anger), sympathetic tone and cardiovascular reactivity are related to right hemisphere function, heightened hostility may be related to changes in right hemisphere function. Differences in right frontal lobe

function have been related to design fluency performance (Foster, Williamson, & Harrison, 2005), and intensity of arousal (Foster & Harrison, 2002) in processing emotional stimuli. Differences in right cerebral function among low and high hostile individuals can be expected to be especially exaggerated upon exposure to a stressor that requires right hemisphere regulation.

Arousal

Changes in arousal may be associated with changes in frontal lobe function in high hostile individuals. The primary assumption is that one of the many roles of the frontal lobes is to inhibit arousal systems (Heilman et al., 2003; Isaac, 1960). It follows that decline in frontal lobe function may lead to a lack of inhibitory influence over the arousal system. Shenal and Harrison (2003) found that men classified as high hostile demonstrated increased right posterior activation (as indicated through enhanced ability to detect emotional phonemes at the left ear) after exposure to a cognitive stressor. When taken together with Heller's model of emotional asymmetry, a decrease in activation of right frontal region may lead to increased levels of arousal in high hostile individuals.

The Role of the Frontal Systems in Task Completion

With respect to overall functioning, the frontal lobes have a largely inhibitory role over more posterior regions of the brain (Damasio & Anderson, 2003; Heilman et al., 2003). The frontal lobes regulate goal directed behaviors, orienting and startle responses related to stimulus significance, and motivational states that are not strongly related to immediate needs (Mesulam, 2000, Heilman et al., 2003). The prefrontal regions of the brain are heterogeneous in nature (Stuss & Benson, 1984; Mesulam, 2000) in part because they share rich regulatory connections with cortical, brainstem, and limbic

structures, especially in the attention-arousal domain (Heilman, 1995; Heilman et al., 2003). Therefore, examining frontal lobe function as it regulates or inhibits posterior cerebral regions is useful. This may be especially the case when determining the relative activation of oppositional cerebral regions involved in the completion of specific tasks or functions. In the present experiment, right frontal systems will be investigated using design fluency performance.

The association between frontal lobe function and fluency performance has been well established. Perhaps the first indication of this association was from lesion data (Jacobsen, 1936). Many studies have been done since then that relate a decline in task performance to frontal lesions (Alexander & Stuss, 2006; Ruff, Allen, Farrow, Neiman, & Wylie, 1994, Baldo, Delis, Wilkins, & Shimamura, 2004; Baldo, Shimamura, Delis, Kramer, & Kaplan, 2001). The general finding is that patients who have sustained frontal lobe lesions are impaired in performance on complex tasks. Briefly, patients with right frontal or bilateral frontal lesions were impaired at performance on a design fluency task compared to non-lesioned controls (Ruff et al., 1994; Baldo et al., 2001). Implications with respect to cerebral laterality as it relates to task completion will be discussed later.

Executive Tasks

Completion of complex tasks requires the involvement of multiple activation sites. Executive function may be conceptualized as a series of higher-order organizational abilities, including the ability to engage in purposive, goal-directed behavior (Busch et al. 2005; Salthouse, Atkinson, & Berish, 2003). Generally referred to as “higher level” regulatory functions, recent interest in executive function as it relates to frontal lobe function has increased. Over 2,500 articles have been published in the past

10 years regarding changes in executive function as it relates to frontal lobe function (Alvarez & Emory, 2006). Changes in task performance such as refreshing information (Johnson, Mitchell, Raye, & Greene, 2004), and organizing information on verbal learning tasks (Baldo et al., 2002) are some examples of executive function that are strongly related to frontal lobe function (Luria, 1973; Salthouse et al., 2003; Mesulam, 2000, Heilman, 1998). Further, deficits in sequential planning and set shifting (Ozonoff et al., 2004) have been seen in individuals diagnosed with autism and demonstrating documented decreases in activation in the frontal lobes.

The primary measure of executive function originated through inspection of performance on the Wisconsin Card Sorting Test (WCST), which has been shown to be sensitive to frontal lobe lesions (Baddeley, 1996; Alvarez & Emory, 2006; Lezak, 1995). Individuals who have sustained frontal lobe lesions perform significantly worse on the WCST than do normal controls (Stuss et al., 1983; van den Broek, Bradshaw, & Szabadi, 1993), and those with thalamic (Wallesch, Kornhuber, Kunz, & Brunner, 1983) and posterior (Stuss et al., 2000; Teuber, Battersby, & Bender, 1951) lesions.

With respect to attentional abilities, frontal lobe damage can lead to increased distractibility and a decline in the ability to focus attention to relevant task demands (Malmo, 1942; Heilman et al., 2003). Changes in emotion, attitudes, and personality are commonly seen in patients who have sustained damage to the frontal lobe (Damasio and Anderson, 2003). While there is strong evidence supporting the relationship between frontal lobe function and executive function, the problem of defining what is meant by executive function remains. Given the multidimensional definition of executive function,

operationally defining it with respect to performance on a specific task may serve to simplify the relationship between executive and frontal lobe function.

Design Fluency

Design fluency is a measure of generative output that is considered to be an executive function (Ruff, Light, & Evans, 1987; Foldi et al., 2003). Design fluency has also been demonstrated to require differential activation of the right frontal lobe (Ruff, 1988). The original measure of design fluency was the Design Fluency Test (DFT) developed by Jones-Gotman and Milner (1977). Although the DFT was useful in relating impairments in design fluency to decline in frontal lobe function, scoring of this test was exceptionally subjective (Ruff et al., 1987) as demonstrated by inconsistencies among raters regarding whether or not a generated design was unique. The Ruff Figural Fluency Test (RFFT) was developed to control for this by creating a more objective scoring method (Ruff, 1988; Ruff et al., 1987; Ruff, Light, Parker, & Levin, 1996). The RFFT contains 35 grids with five dots configured two-dimensionally in each grid. Participants are asked to create as many unique designs for each grid as possible by connecting these dots in different ways, and are given one minute to do this (Ruff et al., 1987). Using these parameters, the RFFT provides a more objective measurement of design fluency.

Frontal Lobe Involvement in Design Fluency

There is evidence that patients with right frontal lobe lesions demonstrate a decline in design fluency (Ruff et al., 1994; Alexander, & Stuss, 2006; Baldo et al., 2001) as indicated by a decrease in the number of unique designs generated in this task. There is also evidence that high hostile men do not perform as well on the RFFT as do low hostile men (Williamson & Harrison, 2003; Williamson, Harrison, & Walters, 2007;

Walters & Harrison, 2007). These observations provide evidence that the RFFT is sensitive to the effects of hostility and frontal lobe function through measurement of design perseverations and the number of unique designs generated.

The RFFT is a measure of executive function that is sensitive to changes in right frontal lobe function, and changes in fluency performance with hostility. There is also evidence that the RFFT and COWAT are sensitive to relative activation of the right (Foster, Williamson, & Harrison, 2005; Ruff et al., 1994) and the left (Baldo et al., 2001) frontal lobes, respectively. To discuss how these tests may be useful in determining relative right or left frontal lobe decline, it is necessary to discuss lateralization of design fluency. In the following section, cerebral asymmetry and its relation to fluency performance will be discussed.

Laterality of Design Fluency

The right and left frontal lobes have different roles in the completion of tasks measuring executive function. Fluency has been widely used in neuropsychological assessment (Lezak, 1995) and lesion studies (Gladsjo et al., 1999; Ruff et al., 1994). Patients with right frontal lesions show decreased performance on the RFFT, but preserved performance on the COWAT (Ruff et al., 1994). Conversely, patients who had sustained lesions in the left DLPFC were most impaired on verbal (phonemic) fluency compared to patients who had right dorsolateral prefrontal lesions and normal controls (Stuss et al., 1998).

Using quantitative EEG measures of relative left and right frontal activation, relationships between tonic resting EEG and verbal and design performance have been found. Men who performed poorly on the RFFT showed an increase in delta magnitude

over the right frontal region of the brain relative to those who performed well (Foster, Williamson, & Harrison., 2005). Other research indicates that younger men and women who performed well on the COWAT showed a decrease in alpha power over the left frontal region (indicating an increase in left frontal activation) compared to those who did not perform well (Hoptman & Davidson, 1998; Papousek & Schulter, 2004). In research using the Wadda technique, patients were better able to remember verbal information (words) during right hemisphere anesthesia (Kelley et al., 2002). Conversely, patients were better able to remember spatial information (faces) during left hemisphere anesthesia. Relating differences in performance on verbal and design fluency tasks to tonic levels of activation lends itself well to determining differences in cerebral asymmetry.

Delta Magnitude as a measure of Cerebral Dysfunction

Focal increases in delta magnitude have been indicative of abnormality in clinical settings (Misulis, 1997). The association between the occurrence of this slow-wave activity and the occurrence of ischemic lesions in the brain (Lukashevich et al., 1999; Murri et al., 1998) and decreases in cerebral blood flow (Nagata, 1988) have been consistently observed. Moreover, delta magnitude has been associated with changes in arousal (Fernandez-Bouzas et al., 1999). In nonlesioned healthy individuals, heightened delta magnitude over the right frontal region has been associated with poorer performance on the RFFT (Foster et al., 2005). This finding not only indicates that the RFFT is sensitive to changes in right frontal function but also that performance on the RFFT can be related to heightened delta activation over this focal region.

Rationale

As suggested by the preceding review, we have proposed a capacity model of hostility wherein high hostility results from the diminished ability to regulate sympathetic tone and negative emotion (anger) with corresponding perceptual and neurophysiological changes. Further, it has been evidenced that design fluency as a cognitive stressor is associated with right frontal activation. Using dual task procedures, the proposed research examined changes in right frontal activation during physiological stress (left sided cold pressor) and cognitive stress (RFFT). Specifically, this research examined EEG changes in the right frontal region in response to a design fluency task and exposure to a cold pressor stressor in low and high hostile men. While there is well-documented evidence suggesting that increased physiological arousal is related to heightened levels of hostility after exposure to stress, cerebral activation as a result of stress exposure using a dual concurrent task has not been examined. The current experiment builds on existing knowledge by examining changes in design fluency and magnitude of cerebral activation as a function of hostility level and exposure to stress.

Variables

Independent Variables

Group. The between-subjects grouping variable of low and high hostility level was based on scores from the Cook Medley Hostility Scale (Cook & Medley, 1954). In the current experiment, participants obtaining a score of 29 or higher were classified as high hostile. Participants obtaining a score of 18 or lower were classified as low hostile. These classifications represent the upper and lower 1/3 range of scores, respectively, and

have been used in previous research (Williamson & Harrison, 2003; Shenal & Harrison, 2004; Herridge et al., 2004; Rhodes et al., 2002).

Cognitive Arousal. The variable of Cognitive Arousal consisted of two levels and was comprised of low cognitive arousal (pre RFFT) and high cognitive arousal (post RFFT) measurements of EEG delta magnitude.

Physiological Arousal. The variable of Physiological Arousal consisted of two levels (pre and post cold pressor).

Location: The variable of Location (i.e.: electrode site) consisted of two levels and was comprised of the right lateral frontal (F8) and right temporal (T6) electrode sites, arranged according to the International 10/20 system.

Dependent Variables

Delta Magnitude. Magnitude (μV) of the delta bandwidth (1-4 Hz) was used to assess slow wave activity at the right anterior and posterior electrode sites.

RFFT Performance. Scores from the RFFT (i.e., the total number of unique designs generated and the total number of perseverations made) was used as a measure of design fluency.

Hypotheses: Ruff Figural Fluency Performance

1. It is predicted that diminished right frontal capacity will be evident through lowered performance on the RFFT in high hostile men.
2. The dual task demands of regulating pain perception over the left hemibody concurrent with right frontal cognitive task demands will reduce performance on the RFFT in high hostile men.

Hypotheses: Delta Magnitude

1. High hostile men are predicted to evidence heightened delta magnitude over the right frontal region after exposure to the cold pressor stressor.
2. The dual task demands of regulating pain perception over the left hemibody concurrent with right frontal cognitive task demands will result in heightened delta activation in high hostile men.

Method

Participants

Forty-seven undergraduate, right-handed men between the ages of 18 and 26 years were recruited for participation using the online group screening system (http://vt-psyc.sona-systems.com/). Three participants were excluded from further analysis due to inconsistent hostility classification for online laboratory measures. Two participants were excluded from further analysis due to admission of a head injury during the laboratory visit. An additional two participants were excluded from further analysis due to excessive EEG artifact. With these exclusions, 40 participants were included for final data analysis for the current experiment.

Research from our lab has consistently found effects between groups using sample sizes between 20 and 37 participants (Foster et al., 2005; Foster & Harrison, 2006; Everhart & Demaree, 2003) in examining neurophysiological and cardiovascular measures. Moreover, research from other labs examining EEG delta magnitude as an indication of abnormality have used sample sizes that include approximately 11 individuals (Fernandez-Bouzas et al., 2001; Stathopoulou & Lubar, 2004).

For the current research, women were not included for participation due to sex differences in cerebral laterality (Harrison & Gorelczenko, 1990; Crews & Harrison, 1994; Kimura, 1987). Men were classified as either high-hostile or low-hostile using scores obtained from the Cook-Medley Hostility Scale (Cook & Medley, 1954). Potential participants also completed a handedness questionnaire (Coren, Porac, & Duncan, 1979). Participants received 1 extra credit point for completing the online questionnaires. Two additional extra credit points were allocated upon completion of the laboratory session. Participants had the option of applying any extra credit points they earned toward the psychology class of their choice if the instructor permits.

Group Classification Measures

Participants meeting the requirements of the Medical History Questionnaire, Laterality Questionnaire, and Hostility Questionnaire were invited to participate in the second portion of the experiment. Participants who are invited into the lab were tested individually.

The Cook-Medley Hostility Scale

The CMHS has been the most frequently used measure of hostility and is a subset of the Minnesota Multiphasic Personality Inventory (Barefoot et al., 1989). The CMHS predicts incidents of psychological, interpersonal, and medical outcomes (Contrada & Jussim, 1992) of trait hostility. The CMHS also shows a high degree of reliability (α = .84) (Smith & Frohm, 1985) and convergent and discriminant validity (Raikkonen, Matthews, Flory, & Owens, 1999) with respect to physiological measures such as blood pressure regulation. Participants obtaining a score of 19 or lower on the Cook-Medley Hostility Scale (see Appendix A) (Cook and Medley, 1954) were classified as low-

hostile. Participants who obtained a score of 29 or higher on this scale were classified as high-hostile. These classifications are consistent with previous research examining physiological and neuropsychological correlates of trait hostility (Williamson & Harrison, 2003; Shenal & Harrison, 2000; Herridge, et al., 2004; Rhodes et al., 2002).

The Coren, Porac, and Duncan Laterality Questionnaire

Participants completing the Coren, Porac, and Duncan Laterality Questionnaire (see Appendix C) (Coren, Porac, & Duncan, 1979) obtained a score of +7 or higher to be included in the experiment, indicating a strong right hemibody preference. The Coren, Porac, and Duncan Laterality Questionnaire is a 13-item questionnaire that assesses four types of hemibody preference: the eye, ear, arm and leg. Scores on the test range from +13 to -13, with a score of +13 indicating strong right hemibody preference (Demaree & Harrison, 1997).

The Medical History Questionnaire

Only men with a medical history (see Appendix B) unremarkable for neurological disorders, head injuries, vision or hearing problems, or motor impairments were included for laboratory participation. Unremarkable histories for the above impairments is instrumental in insuring that differences seen in the current experimental protocol are due to the effects of the independent variables as opposed to differences in cerebral function within groups.

Apparatus

Participants were seated comfortably at a desk in a sound-attenuated laboratory chamber facing a white wall to control for extraneous stimuli during the experiment.

Located within the laboratory chamber were the appropriate EEG apparati and cold pressor equipment described below.

Design Fluency Measure

Participants completed the Ruff Figural Fluency Test (RFFT) (Ruff, Light, & Evans, 1987; Ruff, 1988) to obtain a measure of generative output for design fluency. The Ruff Figural Fluency Test (see Appendix F) is a paper and pencil test that contains 35 grids with five dots configured two-dimensionally in each grid. Participants were asked to create as many unique designs for each grid as possible by connecting these dots in different ways. Each participant was given one minute to generate designs for each of the five trials on the RFFT. The five trials are arranged with increasing levels of distraction and difficulty (Ruff et al., 1987). Scoring of the RFFT consisted of counting the total number of designs generated and then subtracting the number of perseverative errors for each trial. The total number of unique designs generated is considered to be this score. A perseverative error is defined as repetition of previously generated designs on that trial.

The RFFT has been established as an effective measure for right hemisphere function (Ruff, Allen, Farrow, Niemann, & Wylie, 1994; Baldo, Shimamura, Delis, Kramer, & Kaplan, 2001) in patients with lesions in the right frontal region of the brain. As previously mentioned, a relationship between right frontal capacity and performance on the RFFT has also been seen in healthy right-handed individuals (Foster et al., 2005). Specifically, individuals with low scores on the RFFT showed higher delta magnitude over the right frontal region.

Physiological

EEG

To ascertain right hemispheric activation, quantitative electroencephalography (EEG) activity will be measured using a NeuroSearch-24 system (Lexicor Medical Technology, Inc., Boulder, CO, USA). EEG was recorded and digitized at a sampling rate of 128 Hz, with a high-pass filter set at 0.5 Hz, and a low pass filter set at 32 Hz. Participants were fitted with an appropriately sized cap based on head circumference (Electrocap International). The impedance for each electrode was below 5 kOhms, and often was below 2.5 kOhms. The 19 electrode sites from which data was recorded was arranged in accordance with the International 10/20 System (see Appendix J). Linked ear references were used. Frequency bandwidths was classified as: delta = 1-4 Hz, theta = 5-8 Hz, alpha = 9-12 Hz, and beta = 13-24 Hz. Removal of eye and muscle artifacts from recorded EEG data was done manually before analysis.

Cold Pressor

To ascertain changes in right hemispheric activation and changes in performance on the RFFT, the cold pressor was administered to the left hand using a medium-sized cooler that was filled with ice water and maintained at zero degrees Celsius. A standard mercury thermometer was used to verify temperature stability. Exposure to the painful cold pressor stressor has been shown to cause an increase in right hemispheric activation as a function of hostility level (Shenal & Harrison, 2004; Demaree & Harrison, 1997; Herridge et al., 1997).

Procedure

High- and low-hostile men who meet the criteria indicated above were scheduled for a lab visit within two weeks of completing their online screening. Each participant

was tested individually. Upon entering the lab, participants were seated comfortably and the procedure was explained. Participants then signed the Informed Consent Form (see Appendix E) and were allowed to become familiar with the EEG apparatus. Participants then completed the CMHS a second time to insure reliability of their hostility classification. In other words, participants must maintain the same classification of hostility (high or low) on the online and lab administration of the CMHS. The experiment consisted of three conditions: Low Arousal Cognitive Stress (pre and post RFFT), Physiological Stress (pre and post cold pressor), and High Arousal Cognitive Stress (pre and post RFFT; post cold pressor).

Low Arousal Cognitive Stress Phase

Participants were fitted for EEG measurements. Appropriate cap size was determined through measurement of the circumference of the head. Two consecutive 1-minute baseline EEG recordings were taken. The researcher gave the following instructions:

> "Please sit still in the chair and face forward toward the wall in front of you. Allow yourself to become completely relaxed. When you are comfortable, please close your eyes until I ask you to open them."

EEG data was then collected, after which the participant was requested to open his eyes. To determine an accurate reading, the experimenter remained in the testing room with each participant to insure that no extraneous movement was observed during each recording.

The participant then completed the parts 1 through 5 of the RFFT. The experimenter gave the following instructions:

> "For this test I will ask you to draw different designs by connecting the dots in each box. One the first page, you will notice 3 boxes with 5 dots in each box. These are the practice items. Please connect 3 or more dots in each making a unique pattern every time. Now turn the page. When I say 'go' please connect 2 or more dots in each box making a unique pattern working as fast as you can."
>
> Scoring of the RFFT was done after the participant leaves the lab.

Physiological Stress Phase

Two consecutive 1-minute EEG recordings were taken to assess brain function from performance of the lateralized stressor, and before exposure to cold pressor stress. The researcher gave the following instructions before the recordings began:

> "I will now take another EEG recording. Please sit still in the chair and face forward toward the wall in front of you. Allow yourself to become completely relaxed. When you are comfortable, please close your eyes until I ask you to open them."

The cold pressor was then administered after the EEG recording. Participants were given the following instructions:

> "When instructed, please place your left hand in the water to a point about one inch above your wrist. You will be asked to keep your hand in the water for 45 seconds. You have the option to withdraw your hand at any time, but we prefer that you do not. Although this might be difficult, please try your hardest to keep your hand in the water until instructed to take it out. Do you have any questions? Ready, begin."

Immediately after the participant completed the cold pressor stressor, two consecutive 1-minute EEG recordings were taken to assess changes in brain function after exposure to the cold pressor stressor and before the second completion of the lateralized stressor. The researcher then gave the following instructions before the recordings began:

> "I will now take another EEG recording. Please sit still in the chair and face forward toward the wall in front of you. Allow yourself to become completely relaxed. When you are comfortable, please close your eyes until I ask you to open them."

High Arousal Cognitive Stress Phase

To assess changes in design fluency performance after exposure to the cold pressor, participants completed parts 1 through 5 of the RFFT a second time. The experimenter again gave the following instructions to insure each participant's understanding of the task:

> "For this test I will ask you to draw different designs by connecting the dots in each box. One the first page, you will notice 3 boxes with 5 dots in each box. These are the practice items. Please connect 3 or more dots in each making a unique pattern every time. Now turn the page. When I say 'go' please connect 3 or more dots in each box making a unique pattern working as fast as you can."

The results of the second administration of the RFFT were scored after the participant left the laboratory to assess changes in design fluency performance as a function of cold pressor stress. Finally, two consecutive 1-minute EEG recordings were

taken to assess potential changes in brain function after completion of the RFFT. The researcher gave the same instructions as outlined for previous administrations of EEG.

Upon completion of the experiment, each participant was asked to complete a Cold Pressor Intensity Rating Scale to indicate their perception of how painful the cold pressor stressor was (1= not intense, 3= moderately intense, and 5= extremely intense). A RFFT Difficulty Scale was then administered to assess their perception of how difficult the RFFT was to complete (1= not at all difficult, 3= moderately difficult, and 5= extremely difficult). Finally, participants were asked whether they thought they experienced low hostility or high hostility relative to other undergraduates in the undergraduate pool. This self-awareness variable (Demaree & Harrison, 1997) indicating their perception of their own hostility level was coded for accuracy (1= accurate identification, 0= inaccurate identification).

Results

Self-Report Questionnaire Analysis

A one-way ANOVA was used to compare group means on the Cook-Medley Hostility Scale (Cook & Medley, 1954). Results indicated that scores for the high and low hostile groups were significantly different ($F(1, 38) = 140.39$, $p < .0001$). The high hostile group scored significantly higher on the Cook Medley Hostility Scale (M = 31.84, SD = 1.09) than did the low hostile group (M = 14.1, SD = 1.03).

A separate one-way ANOVA was used to compare group means on the Coren, Porac, and Duncan Laterality Questionnaire (Coren Porac, & Duncan, 1979). The results indicated that the high and low hostile groups were statistically equivalent with respect to scores on the laterality questionnaire ($F(1, 38) = 0.18$, $p = 0.67$). The mean score for the

high hostile group (M = 9.74, SD = .53) on the laterality questionnaire was only marginally lower than scores on the laterality questionnaire for the low hostile group (M = 10.05, SD = .51).

A one-way ANOVA was used to examine group differences in self-reported pain experienced during cold pressor stress. A five-point Likert type scale was used (see Appendix H) with a rating of "5" indicating extremely intense pain, and a rating of "1" indicating a perception of "not at all intense." For this analysis, data from one high hostile man was excluded due to failure to obtain the rating score at the end of the experiment. Groups did not differ on the level of self-reported pain experienced during the cold pressor ($F(1, 37) = 0.36$, $p = .55$). The mean score for the high hostile group (M = 2.53, SD = 0.21) was only marginally higher than the mean score for the low hostile group (M = 2.35, SD = 0.21). Consequently, both high and low hostile groups indicated that they only experienced mild to moderate pain while undertaking the cold pressor.

A one-way ANOVA was used to examine group differences on the perceived difficulty of the Ruff Figural Fluency Test (Ruff, Light, & Evans, 1987). A five-point Likert-type scale was used (see Appendix G) to assess how difficult each participant found the Ruff Figural Fluency Test (RFFT) to complete. A rating of "1" indicated a rating of "not at all difficult" and a rating of "5" indicated that the participant found the task to be "extremely difficult." For this analysis, data from one high hostile man was excluded due to failure to obtain the rating score at the end of the experiment. Groups did not differ on the level of perceived difficulty during the Ruff Figural Fluency Test ($F(1, 37) = 0.49$, $p = .49$). The mean score for the high hostile group (M = 1.84, SD = 0.2) was only marginally higher than the mean score for the low hostile group (M = 1.65,

SD = 0.19). Mean scores indicated that both high and low hostile men perceived the test as relatively easy to complete.

Design Fluency Analysis

The following procedures were used to analyze data from the design fluency task. To determine changes in design fluency performance over the course of the five trials, three way mixed design ANOVAs with the fixed factor of Group (low and high hostile) and with the repeated measure of Condition (pre and post cold pressor) and Trial (trials 1, 2, 3, 4, and 5) were conducted. Separate ANOVAs were conducted to assess the mean number of unique designs produced and the mean number of perseverative errors made across the five trials of the RFFT.

For the number of unique designs generated, a main effect of Condition was found ($F(1, 38) = 48.14$, $p < .0001$) indicating a change in the number of unique designs produced as a function of cold pressor stress (see Figure 1). Post hoc comparisons indicated that the mean number of unique designs generated was greater at each trial in the post stress condition (M = 22.26, SD = 5.97) than in the pre stress condition (M = 19.73, SD = 5.04). A main effect for Trial was found ($F(4, 152) = 3.22$, $p > .05$), indicating that the number of unique designs generated differed across trials irrespective of group (see Figure 2).

A Condition x Trial interaction was found ($F(4, 152) = 6.02$, $p < .001$) indicating a reliable difference in the number of designs produced over trials before and after exposure to cold pressor stress. Post hoc comparisons using Tukey's HSD indicated a reliable increase in the number of designs produced across all 5 trials of the RFFT after exposure to cold pressor stress (see Figure 3).

Moreover, a Hostile x Condition x Trial interaction was found ($F(4, 152) = 5.13$, $p < .001$) indicating that the number of designs produced over trials differed as a function of group and condition. Post hoc comparisons revealed that both high and low hostile men show a significant increase in the total number of designs produced after experiencing physiological (cold pressor) stress (see Figure 4) with the exception of trial 4 in low hostile men. In the post stress condition, post hoc comparisons indicated that increases in the number of unique designs produced in low hostile men was greater for trial 1 (low hostile: M = 24.85, SD = 6.58; high hostile: M = 21.45, SD = 5.49), trial 2 (low hostile: M = 24.8, SD = 6.66; high hostile: M = 21.35, SD = 4.57), trial 3 (low hostile: M = 23.0, SD = 6.77; high hostile: M = 20.95, SD = 5.2), and trial 4 (low hostile: M = 24.85, SD = 6.58; high hostile: M = 21.45, SD = 5.49). Low hostile men demonstrated heightened design fluency as a function of the stressor beyond that demonstrated in the high hostile group. High hostiles were restricted in their capacity to effect the same enhancement in design fluency performance under stress.

The ANOVA source table for findings for unique designs produced across conditions is in table 1.

Using perseverative errors, a Hostile x Condition x Trial interaction was found ($F(4, 152) = 5.13$, $p < .001$), indicating a difference in the number of perseverative errors made across trials as a function of hostility and stress (see Figure 5). Post hoc comparisons revealed that for low hostile men, the number of perseverative errors did not significantly increase after stress with the exception of trial 4 (pre stress: M = 1.15, SD = 1.63; post stress: M = 1.9, SD = 2.15). High hostile men showed a significant increase in perseverative errors after stress in trial 1 (pre stress: M = 1.6, SD = 2.01; post stress: M =

3.2, SD = 4.59) and trial 3 (pre stress: M =1.9, SD = 2.07; post stress: M = 2.5, SD = 5.24). Conversely, high hostile men showed a significant reduction of perseverative errors in trial 4 (M = 1.65, SD = 3.26). Although high hostile men made less perseverative errors on trial 5 in the post stress condition relative to pre stress, this effect was not significant using Tukey's HSD. No other main effects or interactions were found using perseverative errors as the dependent variable.

The ANOVA source table for findings regarding perseverative errors made across conditions is in table 2.

To further determine changes in design fluency performance over the course of individual trials, separate ANOVAs were conducted for the first and last trials of the RFFT. A three way mixed design ANOVA with the fixed factor of Group (low and high hostile) and with the repeated measures of Condition (pre and post cold pressor) and Trial (trials 1, and 5) was conducted. Separate ANOVAs were conducted to assess the number of unique designs generated and the total number of perseverative errors made across all five trials of the RFFT.

A main effect for condition was found ($F(1, 38) = 50.07$, $p < .0001$) indicating that the number of designs generated in trials 1 and 5 differed before and after exposure to cold pressor stress (see Figure 6). Post hoc comparisons revealed that the number of designs produced increased after stress (Condition 2; M = 22.53) over that which was recorded before stress (Condition 1; M = 19.3). A Condition x Trial interaction was found ($F(1, 38) = 9.7$, $p < .01$) indicating that there was a reliable difference in the number of designs produced in trials 1 and 5 as a function of cold pressor stress. Post

hoc comparisons revealed a significant increase in the number of designs produced on trial 1 and trial 5 in the post stress condition (see Figure 7).

Moreover, a Hostile x Condition x Trial interaction was found ($F(1, 38) = 7.03$, $p < .05$) indicating that the number of designs produced in trials 1 and 5 varied as a function of hostility and stress (see Figure 8). Post hoc comparisons revealed that high hostiles showed a significant increase in the number of designs produced at trial 1 in the post stress condition (pre stress: M = 18.8, SD = 4.53; post stress: M = 21.45, SD = 5.49) and this effect was also seen at trial 5 (pre stress: M = 19.2, SD = 5.06; post stress: M = 21.5, SD = 5.45). Low hostile men showed a similar increase in design fluency in the post stress condition for trial 1 (pre stress: M = 18.75, SD = 5.48; post stress: M = 24.85, SD = 6.58) and trial 5 (pre stress: M = 20.55, SD = 5.41; post stress: M = 22.3, SD = 5.96). Compared using Tukey's HSD, low hostiles in the post stress condition were significantly better at producing more designs in trial, although the increase did not reach significance for trial 5.

For perseverative errors made on trial 1 and 5, a Hostile x Trial interaction was found ($F(1, 38) = 4.74$, $p < .05$) indicating that the number of perseverative errors made on the first and last trials of the RFFT changed as a function of hostility. Post hoc comparisons revealed that high hostile men showed significantly more perseverative errors at trial 1 (see Figure 9). Comparison between groups was not significant at trial 5 using Tukey's HSD. Post hoc comparisons did reveal that low hostile men made more perseverative errors in trial 5 (M = 1.83, SD = 2.61) than at trial 1 (trial 1: M = 1.1, SD = 1.37). No other main effect or interaction effect was found for perseverative errors on trial 1 and trial 5.

Design Fluency Performance Pre Stress

To further examine design fluency performance before exposure to cold pressor stress, an ANOVA was conducted to assess performance across the five trials of the RFFT. A two way mixed design ANOVA with the fixed factor of Group (low and high hostile) and with the repeated measure of Trial (trials 1, 2, 3, 4, and 5) was conducted. Separate ANOVAs were conducted to assess the number of unique designs generated and the number of perseverative errors made across all five trials of the RFFT.

A main effect for trial was found ($F(4, 152) = 3.43$, $p < .05$) indicating that the number of designs generated differed across trials (see Figure 10). Post hoc comparisons revealed that the number of designs produced increased over trials (trial 5; M = 19.88; trial 1; M = 18.77). No other main effect or interaction effect was found for design fluency performance prior to exposure to cold pressor stress.

Design Fluency Performance Post Stress

To further examine design fluency performance after exposure to cold pressor stress, an ANOVA was conducted for all five trials of the RFFT. A two way mixed design ANOVA with the fixed factor of Group (low and high hostile) and with the repeated measure of Trial (trials 1, 2, 3, 4, and 5) was conducted. Separate ANOVAs were conducted to assess the number of unique designs generated and the number of perseverative errors made across all five trials of the RFFT.

For the number of unique designs generated, a main effect for Trial was found ($F(4, 152) = 5.27$, $p < .005$) indicating that the number of designs generated varied reliably across the five trials (see Figure 11). Additionally, a Hostile x Trial interaction was found ($F(4, 152) = 2.92$, $p < .05$) indicating that high and low hostile men differed

reliably in the number of designs generated across trials of the RFFT (see Figure 12). Post hoc comparisons revealed that high hostiles produced significantly fewer designs during the earlier trials (trials 1, 2, and 3) than those produced by the low hostile group. While high hostile men did produce fewer designs at trials 4 and 5 as well, between groups differences were not significant when compared with Tukey's HSD.

For the ANOVA conducted on perseverative errors in design fluency post cold pressor stress, a Hostile x Trial interaction was found ($F(4, 152) = 4.57$, $p < .01$), indicating a difference in perseverative errors made in high and low hostile men across trials. Post hoc comparisons revealed a reliable between groups difference in perseverative errors made across the first three trials of the RFFT. High hostile men produced significantly more perseverative errors across the first three trials compared to low hostile men. For the later trials (trials 4 and 5), between groups differences in perseverative errors made was not significant when compared with Tukey's HSD (see Figure 13). No other main effects or interactions were seen for perseverative errors made in the post stress condition.

QEEG Analysis

Raw EEG data underwent Fast Fourier Transform and the magnitude for each bandwidth was specified and computed. QEEG waveform data for each participant were manually artifacted offline. Artifacting involved visual inspection of EEG waveforms in 2-second time epochs. Epochs containing artifact from eye or muscle movement were excluded from data analysis. QEEG samples contained no less than 50 seconds (25 epochs) of artifact-free data. QEEG data were collected from each participant eight times over the course of the experiment for no less than 1 minute for each time period.

Delta Magnitude (1-4 Hz)

A three way mixed design ANOVA was performed on the physiological variable of EEG delta magnitude (1-4 Hz). There was a fixed factor of Group (low and high hostile) and repeated measures of Condition (1, 2, 3, 4, 5, 6, 7, and 8) and Location (electrode sites F8 and T6). Pairwise comparisons were conducted for each ANOVA using Tukey's HSD. An a priori significance level was set at $p \leq .05$.

A main effect for Location was found ($F(1, 38) = 29.18$, $p < .0001$), indicating a difference in delta magnitude at the frontal (F8) and temporal (T6) electrode sites (see Figure 14). Post hoc comparisons revealed heightened delta magnitude at the temporal location (M = 7.59, SD = 2.31) and reduced delta magnitude at the frontal location (M = 5.82, SD = 1.34) irrespective of condition. Moreover, a Condition x Location interaction was found ($F(7, 264) = 2.18$, $p < .05$) indicating a change in delta magnitude over the frontal and temporal electrode sites as a function of condition. Post hoc comparisons indicate a reduction in delta magnitude over the right frontal site not found over the right temporal site across conditions (see Figure 15).

The ANOVA source table for findings across conditions is in table 3.

Group differences in delta magnitude with each experimental manipulation were independently assessed. Separate three way mixed design ANOVAs were performed with a fixed factor of Group (low and high hostile), and with the repeated measures of Condition (pre and post cold pressor; pre and post RFFT performance) and Location (electrode sites F8 and T6). The first ANOVA was conducted to assess delta magnitude as a function of performance on the design fluency test in the low arousal cognitive stress condition (before cold pressor stress). Specifically, delta magnitude was examined over

conditions 2 and 3 (pre and post RFFT 1). A main effect for Location was found ($F(1, 38) = 28.22$ $p < .0001$), indicating a difference in delta magnitude over the frontal and temporal electrode sites. Post hoc comparisons indicated that delta magnitude was greater over the temporal site (M = 7.7, SD = 2.33) than the frontal site (M = 5.93, SD = 1.32). No other main effects were observed for the right frontal and right temporal electrode sites.

The ANOVA source table for findings for the low arousal cognitive stress phase is in table 4.

A second ANOVA was conducted to assess delta magnitude as a function of physiological stress (before and after the cold pressor). A main effect for Location was found ($F(1, 38) = 25.57$ $p < .0001$), indicating a difference in delta magnitude over the frontal and temporal electrode sites. Post hoc comparison indicated that delta magnitude was greater over the temporal site (M = 7.55, SD = 2.4) than the frontal site (M = 5.73, SD = 1.18). No other main effects were observed for the right frontal and right temporal electrode sites.

The ANOVA source table for findings for the physiological stress phase is in table 5.

A third ANOVA was conducted to assess changes in delta magnitude for the high arousal cognitive stress phase (cognitive stress from the second RFFT post cold pressor). A main effect for Location was found ($F(1, 38) = 24.93$ $p < .0001$), indicating a difference in delta magnitude over the frontal and temporal electrode sites. Post hoc comparison indicated that delta magnitude was greater over the temporal site (M = 7.48,

SD = 2.16) than the frontal site (M = 5.64, SD = 1.16). No other main effects were observed for the right frontal and right temporal electrode sites.

The ANOVA source table for findings for the high arousal cognitive stress phase is in table 6.

Delta (1-4 Hz) Extended Analyses of Different Conditions

More refined analyses were conducted to determine delta magnitude as a function of stressor type. Separate three way mixed design ANOVAs were performed with a fixed factor of Group (low and high hostile) and with the repeated measures of Condition (pre and post cold pressor; pre and post RFFT performance) and Location (electrode sites F8 and T6). The first ANOVA was conducted to assess delta magnitude as a function of the cognitive stressor (design fluency task) before the cold pressor stress. Specifically, delta magnitude was examined over conditions 1 and 3 (baseline and post RFFT 1). A main effect for Location was found ($F(1, 38) = 10.43$ $p < .01$), indicating a difference in delta magnitude over the frontal and temporal electrode sites. Post hoc comparison indicated that delta magnitude was greater over the temporal site (M = 7.73, SD = 2.3) than the frontal site (M = 6.06, SD = 1.5).

A Condition x Location interaction was found ($F(1, 38) = 7.62$ $p < .01$), indicating a change in delta magnitude over the frontal and temporal sites at baseline and after cognitive stress. Post hoc comparisons revealed a decrease in delta magnitude after cognitive stress over the right frontal site although this decrease was not significant using Tukey's HSD (see Figure 16). While delta magnitude remained stable in the baseline and post low arousal cognitive stress condition for the right temporal electrode site, this site showed significant increases in delta magnitude at baseline (frontal: M = 6.33, SD = 1.65;

temporal: M = 7.67, SD = 2.23) and post cognitive stress (frontal: M = 5.85, SD = 1.31, temporal: 7.79, SD = 2.39). No other main effect or interaction effect was observed for the right frontal and right temporal electrode sites for conditions 1 and 3.

The second ANOVA was conducted to assess delta magnitude as a function of cognitive stress (design fluency) before the cold pressor stress. Specifically, delta magnitude was examined over conditions 1 and 4 (baseline and pre cold pressor). A main effect for Condition was found ($F(1, 38) = 4.89$, $p < .05$), indicating a difference in delta magnitude at baseline and before the cold pressor test. Post hoc comparisons indicated that delta magnitude was greater at baseline (M = 6.99, SD = 2.06) than before the cold pressor (M = 6.68, SD = 2.02). A main effect for Location was found ($F(1, 38) = 21.61$, $p < .01$), indicating a difference in delta magnitude over the frontal and temporal electrode sites. Post hoc comparisons indicated that delta magnitude was greater over the temporal site (M = 7.58, SD = 2.27) than the frontal site (M = 6.09, SD = 1.45).

A Hostile x Condition interaction was found for conditions 1 and 4 ($F(1, 38) = 4.68$, $p < .05$), indicating a change in delta magnitude as a function of hostility at baseline and before cold pressor stress. Post hoc comparisons revealed a significant decrease in delta magnitude after cognitive stress in high hostile men, whereas low hostile men maintained stable activation for delta magnitude irrespective of exposure to the cognitive stressor (see Figure 17). Additionally, high hostile men showed a reliable increase in delta magnitude at baseline relative to low hostile men using Tukey's HSD. There were no significant differences between groups in the post cognitive stress condition.

A third ANOVA was conducted to assess delta magnitude as a function of design fluency after cold pressor stress. Specifically, changes in delta magnitude were examined

over conditions 1 and 5 (baseline and post cold pressor). A main effect for Condition was found ($F(1, 38) = 7.26$, $p < .05$), indicating a difference in delta magnitude at baseline and before the second design fluency test. Post hoc comparison indicated that delta magnitude was greater at baseline (M = 6.99, SD = 2.06) than before the cold pressor (M = 6.6, SD = 2.17). A main effect for Location was found ($F(1, 38) = 25.19$, $p < .0001$), indicating a difference in delta magnitude over the frontal and temporal electrode sites. Post hoc comparison indicated that delta magnitude was greater over the temporal site (M = 7.63, SD = 2.35) than the frontal site (M = 5.96, SD = 1.46).

A Condition x Location interaction was found ($F(1, 38) = 11.69$, $p < .01$), indicating a change in delta magnitude at the right frontal and temporal sites at baseline and after the cold pressor stress. Post hoc comparisons revealed a significant reduction in delta magnitude at the frontal (baseline: M = 6.33, SD = 1.65; post physiological stress: baseline: M = 5.59, SD = 1.16) but not temporal (baseline: M = 7.67, SD = 2.23; post physiological stress: baseline: M = 7.61, SD = 2.48) electrode sites (see Figure 18).

A fourth ANOVA was conducted to assess changes in delta magnitude as a function of design fluency at baseline and after cold pressor stress. Specifically, delta magnitude was examined over conditions 1 and 6 (baseline and pre RFFT 2). A main effect for Condition was found ($F(1, 38) = 11.65$, $p < .01$), indicating a difference in delta magnitude at baseline and before the second design fluency test. Post hoc comparison indicated that delta magnitude was greater at baseline (M = 7.0, SD = 2.06) than before the cold pressor (M = 6.48, SD = 1.95). A main effect for Location was found ($F(1, 38) = 24.88$, $p < .0001$), indicating a difference in delta magnitude over the frontal and

temporal electrode sites. Post hoc comparison indicated that delta magnitude was greater over the temporal site (M = 7.52, SD = 2.21) than the frontal site (M = 5.96, SD = 1.46).

A Hostile x Condition interaction was found for Conditions 1 and 6 ($F(1, 36) = 5.19$, $p < .05$), indicating a change in delta magnitude as a function of hostility at baseline and before the second design fluency test. Post hoc comparisons revealed a significant decrease in delta magnitude for high hostile men before the second RFFT whereas low hostile men maintained stable delta activation across these conditions (see Figure 19).

A Condition x Location interaction was also found ($F(1, 38) = 4.85$, $p < .05$), indicating a change in delta magnitude at the right frontal and temporal sites at baseline and before the second design fluency test. Post hoc comparisons revealed a significantly greater decrease in delta magnitude at the right frontal site before the second design fluency test than at baseline (see Figure 20). Delta magnitudes at the temporal location showed no significant decrease.

A fifth ANOVA was conducted to assess changes in delta magnitude as a function of design fluency after cold pressor stress. Specifically, changes in delta magnitude were examined over conditions 1 and 7 (baseline and post RFFT 2). A main effect for Location was found ($F(1, 38) = 30.05$, $p < .0001$), indicating a difference in delta magnitude over the frontal and temporal electrode sites. Post hoc comparisons indicated that delta magnitude was greater over the temporal site (M = 7.62, SD = 2.17) than the frontal site (M = 6, SD = 1.46).

A Condition x Location interaction was found ($F(1, 38) = 11.02$, $p < .01$), indicating a change in right frontal delta magnitude across baseline and after the second design fluency test. Post hoc comparisons revealed a significant decrease in delta

magnitude at the right frontal site after the second design fluency test in comparison with that recorded at baseline (see Figure 21). Delta magnitudes at the temporal location showed no significant decrease. No other main effect or interaction effect was found.

Delta (1-4 Hz): Changes in Laterality over the Frontal Electrode Sites

More refined analyses were conducted to determine changes in delta magnitude between the left (F7) and right (F8) frontal electrode sites. Separate three way mixed design ANOVAs were performed with a fixed factor of Group (low and high hostile) and with the repeated measures of Condition (pre and post cold pressor; pre and post RFFT performance) and Hemisphere (left and right). The first ANOVA was conducted to assess delta magnitude as a function of laterality, hostility, and stress. Specifically, delta magnitude was examined at the left and right frontal electrode sites in high and low hostile men across conditions. A main effect for Condition was found ($F(7, 265) = 5.59$, $p < .0001$), indicating a difference in delta magnitude over the left and right frontal electrode sites across conditions. A main effect for Hemisphere was found ($F(1, 38) = 21.75$, $p < .0001$), indicating a difference in delta magnitude in the left and right frontal electrode site (see Figure 22). Post hoc comparison indicated that delta magnitude was greater over the left frontal site (M = 6.27, SD = 1.56) than the right frontal site (M = 5.82, SD = 1.34).

A Hostile x Hemisphere interaction was found ($F(1, 38) = 3.94$, $p = .05$), indicating a change in delta magnitude over the left and right frontal sites as a function of hostility (see Figure 23). Post hoc comparisons revealed a significant increase in delta magnitude over the left frontal electrode site for high and low hostile men. Interestingly,

delta magnitude was significantly greater at the left frontal electrode site for high hostile men (M = 6.38, SD = 1.83) than for low hostile men (M = 6.16, SD = 1.23).
No other main effects were observed for the right and left frontal electrode sites across conditions.

The ANOVA source table for findings at the frontal electrode sites across conditions is in table 7.

For conditions 2 and 3 (pre and post RFFT1), a main effect for Hemisphere was found ($F(1, 38) = 13.75$, $p < .001$), indicating a difference in delta magnitude in the left and right hemisphere. Post hoc comparison revealed that delta magnitude was greater over the left frontal site (M = 6.37, SD = 1.49) than the right frontal site (M = 5.93, SD = 1.32).

The ANOVA source table for findings at the frontal electrode sites for the low arousal cognitive stress phase is in table 8.

For conditions 4 and 5 (pre and post cold pressor), a main effect for Condition was found ($F(1, 38) = 5.96$, $p < .05$), indicating a difference in delta magnitude over the frontal locations before and after cold pressor stress. Post hoc comparison revealed greater delta magnitude before cold pressor stress (M = 6.03, SD = 1.21) than after cold pressor stress (M = 5.8, SD = 1.26). A main effect for Hemisphere was also found ($F(1, 38) = 10.89$, $p < .005$), indicating a difference in delta magnitude in the left and right frontal electrode sites. Post hoc comparison indicated that delta magnitude was greater over the left frontal site (M = 6.1, SD = 1.27) than the right frontal site (M = 5.73, SD = 1.18).

A Hostile x Hemisphere interaction was found ($F(1, 38) = 6.75$, $p = .05$), indicating a change in delta magnitude over the left and right frontal electrode sites as a function of hostility. Post hoc comparisons revealed a significant increase in delta magnitude over the left frontal electrode site for high hostile men but not for low hostile men (see Figure 24). Conversely, low hostile men demonstrated a significant increase in delta magnitude at the right frontal electrode site (M = 5.9, SD = 1.15) relative to high hostile men (M = 5.74, SD = 1.51) using Tukey's HSD. No other main effects were observed for the right and left frontal electrode sites across all conditions.

The ANOVA source table for findings at the frontal electrode sites for the physiological stress phase is in table 9.

For conditions 6 and 7 (pre and post RFFT 2), a main effect for Hemisphere was found ($F(1, 38) = 19.68$, $p < .0001$), indicating a difference in delta magnitude in the left and right frontal electrode sites. Post hoc comparison indicated that delta magnitude was greater over the left frontal site (M = 6.04, SD = 1.33) than the right frontal site (M = 5.64, SD = 1.16). Moreover, a Hostile x Hemisphere interaction was found ($F(1, 38) = 4.0$, $p = .05$), indicating a change in delta magnitude over the left and right frontal electrode sites as a function of hostility. Post hoc comparisons revealed a significant increase in delta magnitude over the left frontal electrode site relative to the right frontal electrode site for high and low hostile men (see Figure 25). Moreover, low hostile men showed a significant increase in delta magnitude at the right frontal electrode site (M = 5.77, SD = 1.04) relative to high hostile men (M = 5.51, SD = 1.27) using Tukey's HSD. No other main effects were observed for the right and left frontal electrode sites.

The ANOVA source table for findings at the frontal electrode sites for the high arousal cognitive stress phase is in table 10.

Delta (1-4 Hz): Changes in Laterality over the Temporal Electrode Sites

More refined analyses were conducted to determine changes in delta magnitude between the left (T5) and right (T6) temporal electrode sites. Separate three way mixed design ANOVAs were performed with a fixed factor of Group (low and high hostile), and repeated measures for Condition (pre and post cold pressor; pre and post RFFT performance) and Hemisphere (left and right). The first ANOVA was conducted to assess changes in delta magnitude as a function of laterality, hostility, and stress. Specifically, delta magnitude was examined at the left and right temporal electrode sites in high and low hostile men across conditions. A main effect for Hemisphere was found ($F(1, 38) = 18.43$, $p < .0001$), indicating a difference in delta magnitude among the left and right temporal electrode sites. Post hoc comparison indicated that delta magnitude was greater over the right temporal site (M = 7.59, SD = 2.31) than the left temporal site (M = 6.73, SD = 1.68).

The ANOVA source table for findings at the temporal electrode sites across conditions is in table 11.

For conditions 2 and 3 (pre and post RFFT1), a main effect for Hemisphere was found ($F(1, 38) = 17.39$, $p < .001$), indicating a difference in delta magnitude among the left and right temporal electrode sites. Post hoc comparison indicated that delta magnitude was greater over the right temporal site (M = 7.7, SD = 2.33) than the left temporal site (M = 6.83, SD = 1.49). A disordinal Hostile x Condition interaction was found ($F(1, 38) = 3.87$, $p = .05$), indicating a diametrically opposite change in delta

magnitude as a function of cold pressor stress in high and low hostile men (see Figure 26). Post hoc comparisons revealed a significant increase in delta magnitude for high hostile men post cognitive stress. Moreover, significant increases in delta magnitude were found across the temporal electrode sites for high hostile men (M = 7.5, SD = 2.2) relative to low hostile men (M = 7.14, SD = 1.86).

The ANOVA source table for findings at the temporal electrode sites for the low arousal cognitive stress phase is in table 12.

For conditions 4 and 5 (pre and post cold pressor), a main effect for Hemisphere was found ($F(1, 38) = 20.88$, $p < .0001$), indicating a difference in delta magnitude in the left and right hemisphere. Post hoc comparison indicated that delta magnitude was greater over the right temporal site (M = 7.55, SD = 2.39) than the left temporal site (M = 6.51, SD = 1.45). No other main effect or interaction effect was observed for the right and left temporal electrode sites before and after exposure to cold pressor stress.

The ANOVA source table for findings at the temporal electrode sites for the physiological stress phase is in table 13.

For conditions 6 and 7 (pre and post RFFT 2), a main effect for Hemisphere was found ($F(1, 38) = 14.76$, $p < .005$), indicating a difference in delta magnitude in the left and right hemisphere. Post hoc comparisons indicated that delta magnitude was greater over the right temporal site (M = 7.48, SD = 2.15) than the left temporal site (M = 6.69, SD = 1.62). No other main effect or interaction effect was observed for the right and left temporal electrode sites before and after the second design fluency task.

The ANOVA source table for findings at the temporal electrode sites for the high arousal cognitive stress phase is in table 14.

Discussion

The current experiment examined cerebral activation to physiological (cold pressor) and cognitive (design fluency) stress in high and low hostile men using a dual-task paradigm. By using this dual task approach, high and low hostile men were asked to maintain a consistent level of right cerebral activation and fluency performance in high and low arousal conditions. Cerebral activation was measured physiologically using quantitative electroencephalography (QEEG) and cognitively using the Ruff Figural Fluency Task (RFFT). High hostiles demonstrated heightened right frontal activation (as indicated through delta suppression) relative to low hostiles after exposure to physiological (cold pressor) stress. Heightened right frontal arousal occurred along with diminished capacity for right frontal cognitive task completion. Specifically, high hostiles showed increased executive deficits in the high arousal condition as evidenced by measurements of perseverative errors on the RFFT. Moreover, performance improvements demonstrated by low hostiles in the high cognitive arousal condition occurred with minimal delta suppression over the right frontal lobe.

Originally, heightened delta magnitude was predicted in the physiological and high arousal cognitive stress conditions as delta magnitude is an indication of cerebral dysfunction in clinical settings (Murri et al., 1998; Nagata, 1988). Moreover, low design fluency has been found concurrent with heightened right frontal delta magnitude in normal populations (Foster et al., 2005). In the current experiment, a dual-task paradigm was used to assess changes in delta magnitude as a function of stress. Delta magnitude suppression was found after exposure to physiological (cold pressor) stress. This was especially the case for high hostile men.

The hypothesis that high hostile men would demonstrate reduced performance on the RFFT after exposure to cold pressor stress was partially supported. Results from the design fluency task indicated that high hostile men failed to produce a comparable increase in design fluency after exposure to physiological stress, which is potentially consistent with an interpretation of diminished right frontal capacity in this group. With increased arousal, high hostile men do show increases in design fluency. However, this increased output in design fluency is significantly below the increments seen in low hostile men under high arousal conditions and this is suggestive of capacity limitations in high hostile men.

Moreover, although high hostiles showed an increase in design fluency in the high arousal cognitive stress condition, it was at the cost of an increase in executive errors as seen in the analysis of perseverative errors (see Figure 5). In other words, high hostile men were unable to regulate right frontal activation as a result of physiological (cold pressor) pain and perform at the level of low hostile men on a right lateralized cognitive stressor. Taken together, findings from behavioral (RFFT) and physiological (QEEG) measures of cerebral activation are supportive of diminished right frontal capacity in high hostile men.

For low hostile men, enhanced performance on the RFFT in the high arousal cognitive stress condition occurred with minimal delta suppression over the right frontal lobe as a function of physiological stress. Overall, it appears that the role of arousal in a dual task paradigm is larger than previously suspected. This will be increasingly more evident in the following sections as behavioral and physiological indices of cerebral activation will be discussed with respect to hostility and stress.

It is important to note the effect of trial in the current experiment as it relates to vigilance and habituation. Vigilance and habituation are integral components of arousal (Heilman & Valenstein, 2003). As mentioned in the introduction, changes in arousal may be associated with changes in frontal lobe function and this is especially the case for high hostile individuals (Shenal & Harrison, 2003; Holland et al., 2007). A decrease in design fluency production across trials was seen after exposure to cold pressor stress. This indicates habituation in later trials of the RFFT. It is interesting to note that this effect was not present for the low arousal cognitive stress condition where habituation to a physiological stressor is not a factor in design task completion.

With respect to habituation as evidenced by perseverative errors, high hostile men showed a decrease in perseverative errors across trials post stress, whereas perseverative errors for low hostile men remained stable. This finding is an indication that high hostile men are habituating to stress across trials. This finding does not indicate that high hostile men are managing stress effectively, as the overall number of perseverative errors made across trials is significantly higher than those of low hostile men. In other words, the addition of stress produces more right frontal executive deficits with a provision of recovery time in the high hostile group, but not to the level seen in the low hostile group.

The hypothesis that performance on the RFFT in high hostile men would be reduced when they are required to regulate pain perception over the left hemibody and concurrently manage right frontal cognitive stress was supported. As previously mentioned, high hostile men produced fewer designs than low hostile men. With the addition of stress, low hostile men showed a significant increase in the number of designs produced while showing no reliable increase in perseverative errors. Conversely, high

hostile men showed limited capacity to produce the same increase in unique designs post stress and made more perseverative errors.

With respect to habituation effects and perseverative errors, two findings are worth mentioning. High hostile men showed an increase in perseverative errors made over the first three trials after exposure to physiological stress relative to low hostile men (see Figure 12). The number of perseverative errors made for low hostile men remained relatively stable after exposure to physiological stress. As with the finding for unique designs produced, a reduction in perseverative errors across trials for high hostile men may be an indication of habituation to the physiological cold pressor stress. It is important to mention that high hostile men did not show the same reduction in perseverative errors made across trials as that seen in low hostile men. This is an indication that high hostile men showed a limited capacity for right frontal regulation of stress in the high cognitive arousal conditions. In contrast, low hostile men demonstrated enhanced right frontal capacity through reduced perseverative errors across the high and low cognitive arousal conditions.

For the analyses concerning cerebral activation across the right frontal (F8) and right temporal (T6) locations, the overall finding was reduced delta magnitude (μV) over the right frontal region and increased delta magnitude over the right temporal region across all 8 conditions. As previously mentioned, delta magnitude is inversely related to cerebral activation. Any reduction in delta magnitude indicates an increase in cerebral activation and any increase in delta magnitude is consistent with reduced functional capacity (Foster et al., 2005; Foster et al., 2008). For the current research, changes in delta magnitude reflect changes in cerebral activation as a function of arousal.

To further determine the effects of stress on hostility and cerebral activation, the relationship between right frontal (F8) and right temporal (T6) delta magnitude at baseline and after each stress condition was examined. Significant reductions in delta magnitude were found at the frontal electrode site but not at the temporal electrode site. It is interesting to note the delta magnitude reduction at the right frontal location in the post physiological and high arousal cognitive stress conditions. This observation is consistent with the prediction for right frontal inhibition of the right temporal region during exposure to a right-lateralized cognitive stressor.

With respect to hostility, two interactions are worth further discussion. The Hostile x Condition interaction that was found for the baseline and pre-cold pressor conditions (Figure 17) provided information on cerebral arousal with respect to hostility and possibly the anticipation of physiological stress. The second Hostile x Condition interaction that was found for the baseline and pre RFFT 2 conditions (Figure 19) provided information on cerebral arousal with respect to hostility and possibly the anticipation of cognitive stress. In both cases, high hostile men showed heightened delta magnitude during the baseline condition, and significant reductions in delta magnitude in the later conditions. Low hostile men showed no significant change in delta magnitude across conditions. Because all participants were informed of the experimental protocol before participation, possible anticipatory reactions may be relevant in explaining these findings.

In addition, these findings support the capacity model in that low hostile men appear to maintain stable activation over the right frontal (F8) and right temporal (T6) regions as indicated by consistent measures for delta magnitude irrespective of exposure

to cognitive stress. High hostile men, on the other hand, show a significant reduction in delta magnitude before exposure to physiological stress and before cognitive stress in the high arousal condition. While high hostile men were initially predicted to show heightened delta magnitude after exposure to cognitive and physiological stress, the observed inability to maintain stable measures of cerebral activation is an indication that high hostile men are less able to regulate activation in the right hemisphere while managing cognitive and physiological stress. This finding of increased right cerebral activation in high hostile men is consistent with previous research (Harrison & Gorelczenko, 1990; Demaree & Harrison, 1997; Everhart, Demaree, & Harrison, 2008).

Results from the QEEG data collected across all conditions provide information on cerebral laterality as well. To determine the effects of hostility and stress on cerebral laterality, extended analyses were conducted examining delta magnitude at the right frontal (F8) and left frontal (F7) electrode sites. Overall, delta magnitude was greater over the left frontal electrode site in high and low hostile men across conditions. This finding will be discussed in greater detail in the following sections.

The Hostile x Hemisphere interaction (Figure 24) found for the physiological stress condition (conditions 4 and 5) provides additional information regarding changes in cerebral laterality with hostility after cold pressor stress. High hostile men experienced heightened delta magnitude at the left frontal location relative to the right frontal location whereas low hostile men maintained consistent measures of delta magnitude at the left and right frontal electrode sites. Moreover, high hostile men showed significantly higher delta magnitude at the left frontal location and significantly reduced delta magnitude at

the right frontal location. This finding may be an indication that high hostile men are underaroused with relative activation of the right brain compared to low hostile men.

The second Hostile x Hemisphere interaction of interest was found for the high arousal cognitive stress condition (conditions 6 and 7). A similar pattern of activation was found for the high arousal cognitive stress condition (Figure 25) as that recorded during the physiological stress condition. High hostile men showed heightened delta magnitude at the left frontal electrode site relative to the right. In the high arousal stress condition, low hostile men also showed relative left frontal delta magnitude increases. Cerebral activation at the right frontal region, however, remained significantly higher in high hostile men relative to low hostile men. This finding supports the idea that high hostile men are underaroused with relative activation of the right brain. Everhart, Demaree, and Harrison (2008) found that high hostile men were consistently under aroused across all electrode sites during an affective learning task. Moreover, high hostile men evidenced relative activation at the right hemisphere on this task. This appears consistent with the current finding that high hostile men show a greater increase in delta magnitude over the left frontal site and relative delta suppression at the right frontal site.

Of equal importance, a Hostile x Hemisphere interaction was *not* found for the low arousal cognitive stress condition (conditions 2 and 3). The effects described above indicate that high hostile men evidence reduced activation across the left frontal site and increased activation across the right frontal site. This selective increase in right frontal activation and reduction in left frontal activation in high hostile men seems to be evident only in task conditions where high hostile men are concurrently regulating the effects of

the lateralized cognitive stressor and pain. This further supports the usefulness of the capacity model in providing physiological indices of changes in arousal as a function of hostility and stress.

To determine the effects of hostility and stress on cerebral laterality for posterior cerebral regions, extended analyses were conducted examining delta magnitude at the right temporal (T6) and left temporal (T5) electrode sites. Overall, delta magnitude was greater over the right temporal electrode site in high and low hostile men across conditions. The Hostile x Condition interaction that was found (Figure 26) for the low arousal cognitive stress condition (conditions 2 and 3) provides information regarding changes in cerebral arousal at the posterior sites. High hostile men experienced heightened delta magnitude at the left and right temporal location after the first design fluency task relative to low hostile men. Low hostile men maintained consistent measures of delta magnitude at the left and right temporal electrode sites before and after cognitive stress.

Interestingly, this interaction was not found for the physiological and high arousal cognitive stress conditions. The reason why high hostile men did not show changes in delta magnitude at the temporal region during later stress conditions is not entirely understood. Reductions in functional capacity appear to be consistent over the right temporal region across conditions of the experiment.

The major hypotheses made for the current experiment were based on prior research from our lab. Foster, Williamson, and Harrison (2005) found that participants who obtained lower scores on the RFFT also evidenced heightened right frontal delta magnitude. However, the current delta magnitude results are suggestive of heightened

right frontal activation (as evidenced through reduced delta magnitude) after completing the RFFT. While these results appear to be contradictory to those found in Foster et al. (2005), further examination suggests that increased task demands for the current experiment may be producing opposite patterns in right frontal delta magnitude as a function of stress. Moreover, this effect was evident only in high hostile men, which is an extension of the work done by Foster and colleagues.

It is important to note that a classification of high hostility may be an indicator of poor performance on the RFFT. However, poor performance on the RFFT may not necessarily be indicative of a high hostility classification. The between groups variable of hostility is proposed in this model to be directly influenced by arousal level. Arousal level in turn is expected change as a function of stress condition. Results from the current experiment suggest that there was increased activation of the right frontal region. This right frontal increase in activation in high hostile men is suggestive of a relative inability to maintain stable levels of activation across physiological and cognitive stress conditions in comparison with low hostile men.

Recent research using dual task paradigms have found similar patterns of activation in high hostile men. Using a dual task paradigm, Holland and colleagues (2007a) found that high hostile men evidenced reduced systolic blood pressure (SBP) after exposure to two right frontal stressors. High and low hostile men were asked to complete the RFFT and estimate short passages of time, a task theorized to be regulated by the right frontal region. Because increases in SBP are typically associated with reductions in right frontal activation (Foster & Harrison, 2002; Shenal & Harrison, 2003), this reduction in SBP may be indicative of increased right frontal activation in high

hostile men in a dual task setting. Moreover, Mollet (2006) reported that high hostile men failed to show increases in delta magnitude after exposure to cold pressor stress and the completion of an emotional dichotic listening task. Ultimately, it appears that the effects of arousal change as a function of hostility, and this change is effected by the type of stressor used.

The current findings have implications for the capacity model with respect to hostility. As mentioned, there was a reliable increase in design output at the cost of increased perseverative errors in high hostile men. This finding may indicate that high hostile men are likely to be more ballistic and labile in stress conditions that bring about this increased output. Individuals are asked to maintain sympathetic tone while completing tasks that are necessary for every day life. High hostile men may have difficulty maintaining stable levels of arousal and concurrently carry out a task that requires right hemisphere activation. As a result, high hostile men are likely to show reduced functional capacity in dual task settings where both tasks require right frontal regulation.

A potential limitation of the current experiment may be that QEEG data were not recorded during cold pressor exposure or during completion of the RFFT in the low and high cognitive arousal conditions. Rather, QEEG data were recorded immediately before and after each experimental manipulation. While the experiment was structured to assess changes in delta magnitude (μV) pre and post cognitive and physiological stress, observing changes during task completion may have provided insight into the nature of change in right frontal and right temporal activation while completing each stressor. In not gathering QEEG data during RFFT task completion, the possibility of recording other

activity that may be associated with it (i.e.: differences in strategy for design task completion) is reduced. With respect to exposure to cold pressor stress, motor movement from face or jaw tension is typically observed during this task. QEEG recordings during this time are likely to have increased artifact from these movements.

Another potential limitation of this experiment should be mentioned. While data from electrophysiological measures of cortical arousal were collected, data from peripheral measures of sympathetic activation (i.e.: systolic blood pressure and heart rate) were not collected. While determining potential relations among central and peripheral measures of arousal would have been useful, the relationship between central and peripheral measures of arousal has been established in previous research (Foster & Harrison, 2006; Shenal & Harrison, 2004). Increases in right temporal lobe activation have been associated with increases in systolic blood pressure (Foster & Harrison, 2006). Moreover, measures of central and peripheral activation have been conceptualized in models of cerebral activation and emotion as complementary in nature (Foster & Harrison, 2006; Heilman, 1997; Heller, 1993; Wittling et al., 1998). Collecting heart rate (HR) and blood pressure (BP) data was not carried out due to the establishment of this relationship in addition to time constraints for the experimental protocol. Moreover, the possibility of introducing error from collecting data from multiple dependent measures was reduced.

Perhaps the most notable limitation is the fact that a single dissociation was used as opposed to a double dissociation. The current findings would have provided stronger support for the hypotheses had a left frontal task (i.e.: verbal fluency) been used as a means for comparison to examine left frontal activation. Foster and colleagues (2008)

found evidence for a relationship between delta magnitude at the left temporal location (T6) and performance on a verbal learning task. Specifically, Foster and colleagues found heightened delta magnitude to be associated with poor performance on a verbal leaning task. This finding provides an indication that the right and left hemispheres are differentially affected depending on the type of task used. For the current experiment, a double dissociation was not used as the main focus of the investigation involved right cerebral activation as a function of hostility and stress in a dual task setting.

Findings from the current research call for a need for future research examining arousal in a dual concurrent setting. While the current research showed changes in delta magnitude and design fluency that were not in the expected direction, support for the capacity model of hostility was still demonstrated. Primary findings suggest an inability to maintain stable levels of cerebral activation at the right frontal region as indicated by changes in delta magnitude across the right frontal region, and a failure to show increases in design fluency that are comparable to low hostile men. Moreover, high hostile men showed an increase in perseverative errors regardless of low and high arousal condition. This is consistent with previous research examining the effects of hostility in a dual concurrent setting (Williamson & Harrison, 2003). This is another indication of reduced functional capacity for design fluency in high hostile men.

Directions for future research may include the use of experimental designs that employ a double-dissociative technique in examining changes in cerebral activation in a dual task setting. Everhart, Demaree, and Harrison (2008) found reductions in cerebral activation across electrode sites over the left hemisphere in high hostile men, indicating the importance of further examining the role of the left hemisphere in task completion.

This finding may be an indication that the left hemisphere may be recruited for tasks that are typically regulated by the right frontal region for high hostile men. As a result, the left hemisphere may be experiencing reductions in arousal as a function of stress.

Findings from the current research may help determine underlying changes in cerebral activation as a function of hostility and stress. The current research found changes in right hemisphere activation as a function of stressor type (physiological and cognitive stress) and hostility. It may be beneficial to use an emotional stressor (i.e.: baby cries) to examine changes in right cerebral activation as a function of hostility. This type of stressor has been effective in creating changes in right cerebral activation in previous research (Holland et al., 2007b) and may be useful for determining changes in cerebral activation with hostility. Additionally, collecting SBP and HR measures in dual concurrent settings may be useful in further determining the strength and direction of the relationship between the frontal and temporal lobes as a function of hostility.

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Table 1

ANOVA source table for the total number of unique designs produced on the design task across conditions

Source	*df*	*SS*	*MS*	*F*	*p*
Group	1, 38	275.56	275.56	1.10	0.30
Condition	1, 38	640.09	640.09	48.14	<0.001
Trial	4, 152	77.54	77.54	3.22	0.01
Hostile x Condition	1, 38	25.00	25.00	1.88	0.17
Hostile x Trial	4, 152	12.04	12.04	0.50	0.74
Condition x Trial	4, 152	101.96	25.49	6.02	<0.001
Hostile x Condition x Trial	4, 152	86.90	21.73	5.13	<0.001

Table 2

ANOVA source table for the total number of perseverative errors made on the design task across conditions

Source	*df*	*SS*	*MS*	*F*	*p*
Group	1, 38	124.26	124.26	1.18	0.28
Condition	1, 38	381.31	381.31	48.23	<0.001
Trial	4, 152	17.56	17.56	2.88	0.09
Hostile x Condition	1, 38	9.51	9.51	1.20	0.28
Hostile x Trial	4, 152	0.31	0.31	0.05	0.82
Condition x Trial	4, 152	66.31	66.31	12.75	0.001
Hostile x Condition x Trial	4, 152	61.26	61.26	11.77	0.001

Table 3

ANOVA source table for the delta bandwidth across the F8 and T6 electrode sites: All Conditions

Source	*df*	*SS*	*MS*	*F*	*p*
Group	1, 38	0.11	0.11	0.00	0.95
Condition	7, 265	14.60	2.09	2.55	0.01
Location	1, 38	495.84	495.84	29.18	<0.001
Hostile x Condition	7, 265	7.44	1.06	1.30	0.25
Hostile x Location	1, 38	2.75	2.75	0.16	0.69
Condition x Location	7, 265	6.77	0.96	2.18	0.03
Hostile x Condition x Location	7, 265	1.98	0.28	0.64	0.72

Table 4

ANOVA source table for the delta bandwidth across the F8 and T6 electrode sites:

Low Arousal Cognitive Stress Condition

Source	*df*	*SS*	*MS*	*F*	*p*
Group	1, 38	0.11	0.11	0.01	0.91
Condition	1, 38	0.002	0.002	0.00	0.94
Location	1, 38	124.96	124.96	28.22	<0.001
Hostile x Condition	1, 38	0.07	0.07	0.15	0.70
Hostile x Location	1, 38	0.81	0.81	0.18	0.67
Condition x Location	1, 38	1.19	1.19	2.78	0.10
Hostile x Condition x Location	1, 38	1.06	1.06	2.47	0.12

Table 5

ANOVA source table for the delta bandwidth across the F8 and T6 electrode sites: Physiological Stress Condition

Source	*df*	*SS*	*MS*	*F*	*p*
Group	1, 38	1.12	1.12	0.13	0.71
Condition	1, 38	0.27	0.27	0.52	0.48
Location	1, 38	132.13	132.13	25.57	<0.001
Hostile x Condition	1, 38	0.17	0.17	0.32	0.57
Hostile x Location	1, 38	0.78	0.78	0.15	0.70
Condition x Location	1, 38	1.44	1.44	3.02	0.09
Hostile x Condition x Location	1, 38	0.21	0.21	0.44	0.51

Table 6

ANOVA source table for the delta bandwidth across the F8 and T6 electrode sites: High Arousal Cognitive Stress Condition

Source	*df*	*SS*	*MS*	*F*	*p*
Group	1, 38	1.42	1.42	0.20	0.66
Condition	1, 38	0.99	0.99	1.26	0.27
Location	1, 38	134.54	134.54	33.58	<0.001
Hostile x Condition	1, 38	0.60	0.60	1.26	0.27
Hostile x Location	1, 38	0.30	0.30	0.07	0.79
Condition x Location	1, 38	0.06	0.06	0.16	0.69
Hostile x Condition x Location	1, 38	0.26	0.26	0.66	0.42

Table 7

ANOVA source table for the delta bandwidth across the F7 and F8 electrode sites: All Conditions

Source	*df*	*SS*	*MS*	*F*	*p*
Group	1, 38	0.15	0.15	0.01	0.93
Condition	7, 265	46.76	6.68	5.59	<0.001
Hemisphere	1, 38	32.19	32.19	21.75	<0.001
Hostile x Condition	7, 265	5.88	0.84	0.70	0.67
Hostile x Hemisphere	1, 38	5.82	5.82	3.94	0.05
Condition x Hemisphere	7, 265	1.89	0.26	1.00	0.43
Hostile x Condition x Location	7, 265	1.37	0.19	0.73	0.65

Table 8

ANOVA source table for the delta bandwidth across the F7 and F8 electrode sites:

Low Arousal Cognitive Stress Condition

Source	*df*	*SS*	*MS*	*F*	*p*
Group	1, 38	0.01	0.01	0.00	0.96
Condition	1, 38	0.39	0.39	0.41	0.52
Hemisphere	1, 38	7.97	7.97	13.75	<0.001
Hostile x Condition	1, 38	2.28	2.28	2.41	0.13
Hostile x Hemisphere	1, 38	1.24	1.24	2.14	0.15
Condition x Hemisphere	1, 38	0.17	0.17	0.60	0.44
Hostile x Condition x Location	1, 38	0.56	0.19	1.91	0.17

Table 9

ANOVA source table for the delta bandwidth across the F7 and F8 electrode sites: Physiological Stress Condition

Source	*df*	*SS*	*MS*	*F*	*p*
Group	1, 38	0.01	0.01	0.00	0.95
Condition	1, 38	2.18	2.18	5.96	0.01
Hemisphere	1, 38	5.29	5.29	10.89	0.002
Hostile x Condition	1, 38	0.13	0.13	0.34	0.56
Hostile x Hemisphere	1, 38	3.28	3.28	6.75	0.01
Condition x Hemisphere	1, 38	0.06	0.06	0.28	0.59
Hostile x Condition x Location	1, 38	0.09	0.09	0.44	0.51

Table 10

ANOVA source table for the delta bandwidth across the F7 and F8 electrode sites: High Arousal Cognitive Stress Condition

Source	*df*	*SS*	*MS*	*F*	*p*
Group	1, 38	0.23	0.23	0.05	0.83
Condition	1, 38	0.49	0.49	0.44	0.51
Hemisphere	1, 38	6.28	6.28	19.68	<0.001
Hostile x Condition	1, 38	0.35	0.35	0.31	0.58
Hostile x Hemisphere	1, 38	1.27	1.27	4.00	0.05
Condition x Hemisphere	1, 38	0.05	0.05	0.32	0.57
Hostile x Condition x Location	1, 38	0.01	0.01	0.09	0.77

Table 11

ANOVA source table for the delta bandwidth across the T5 and T6 electrode sites:

All Conditions

Source	*df*	*SS*	*MS*	*F*	*p*
Group	1, 38	7.59	7.59	0.16	0.69
Condition	7, 265	13.23	1.89	1.40	0.20
Hemisphere	1, 38	114.75	114.75	16.88	<0.001
Hostile x Condition	7, 265	11.75	1.68	1.25	0.27
Hostile x Hemisphere	1, 38	2.04	2.04	0.30	0.58
Condition x Hemisphere	7, 265	3.54	0.51	1.47	0.18
Hostile x Condition x Location	7, 265	1.39	0.19	0.58	0.77

Table 12

ANOVA source table for the delta bandwidth across the T5 and T6 electrode sites:

Low Arousal Cognitive Stress Condition

Source	*df*	*SS*	*MS*	*F*	*p*
Group	1, 38	0.70	0.70	0.05	0.82
Condition	1, 38	0.53	0.53	1.03	0.31
Hemisphere	1, 38	30.27	30.27	17.39	<0.001
Hostile x Condition	1, 38	1.98	1.98	3.87	0.05
Hostile x Hemisphere	1, 38	0.07	0.07	0.04	0.83
Condition x Hemisphere	1, 38	0.17	0.17	0.63	0.43
Hostile x Condition x Location	1, 38	0.01	0.01	0.05	0.83

Table 13

ANOVA source table for the delta bandwidth across the T5 and T6 electrode sites: Physiological Stress Condition

Source	*df*	*SS*	*MS*	*F*	*p*
Group	1, 38	0.52	0.52	0.04	0.84
Condition	1, 38	0.05	0.05	0.05	0.82
Hemisphere	1, 38	43.58	43.58	20.88	<0.001
Hostile x Condition	1, 38	0.43	0.43	0.52	0.47
Hostile x Hemisphere	1, 38	0.79	0.79	0.38	0.54
Condition x Hemisphere	1, 38	0.22	0.22	0.57	0.46
Hostile x Condition x Location	1, 38	0.05	0.05	0.12	0.73

Table 14

ANOVA source table for the delta bandwidth across the T5 and T6 electrode sites:

High Arousal Cognitive Stress Condition

Source	*df*	*SS*	*MS*	*F*	*p*
Group	1, 38	0.06	0.06	0.01	0.94
Condition	1, 38	3.72	3.72	2.35	0.13
Hemisphere	1, 38	25.19	25.19	14.76	<0.001
Hostile x Condition	1, 38	1.40	1.40	0.89	0.35
Hostile x Hemisphere	1, 38	1.26	1.26	0.74	0.39
Condition x Hemisphere	1, 38	0.15	0.15	0.45	0.51
Hostile x Condition x Location	1, 38	0.08	0.08	0.25	0.62

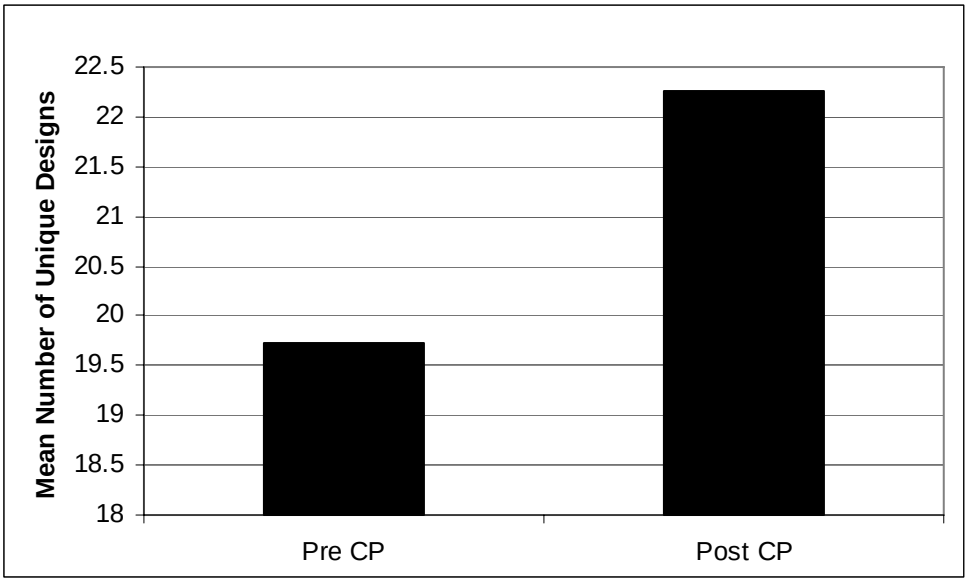


Figure 1. Main effect for the Number of Unique Designs Produced Pre and Post Stress across all trials

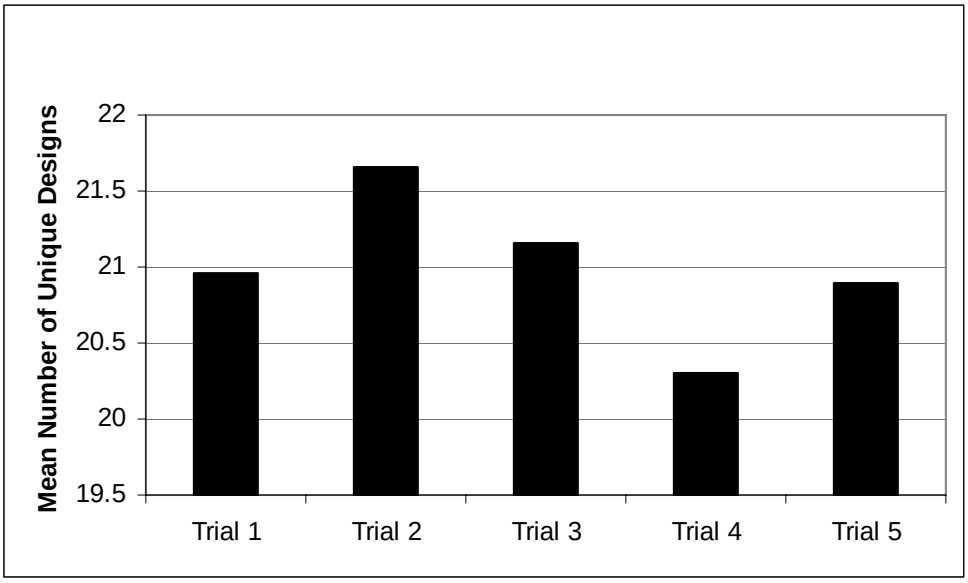


Figure 2. Main effect for the Number of Unique Designs Produced as a function of Trial

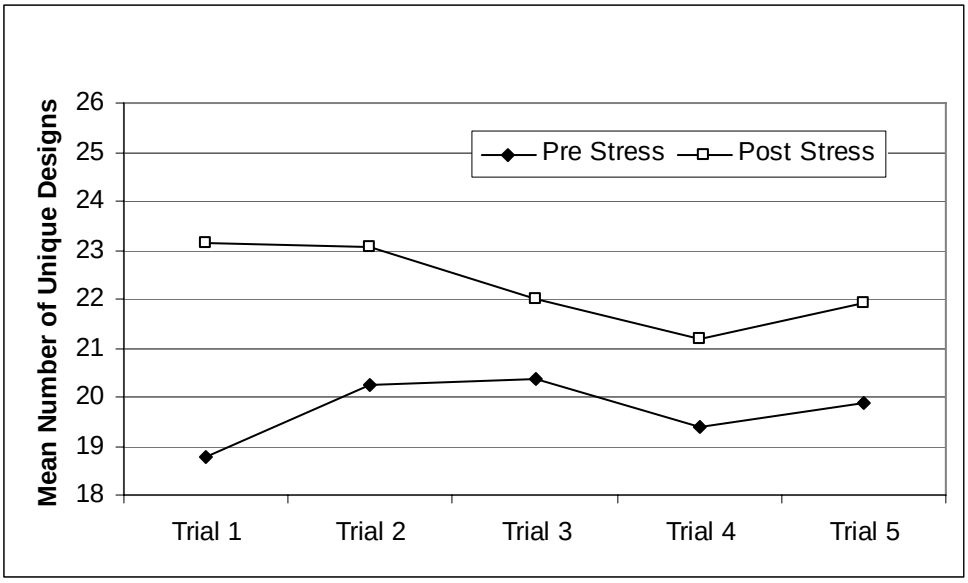


Figure 3. Condition x Trial interaction for the Number of Unique Designs Produced

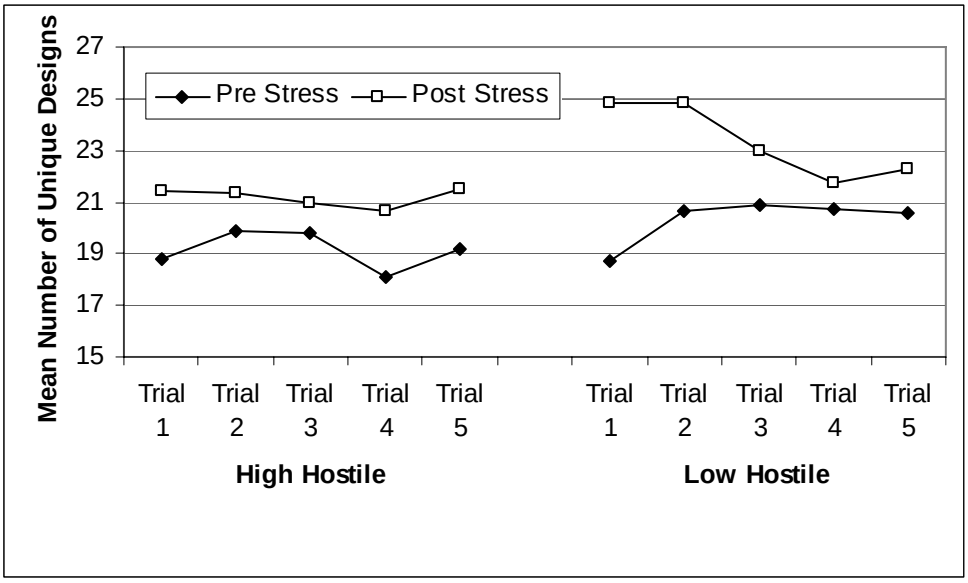


Figure 4. Hostile x Condition x Trial interaction for the Number of Unique Designs Produced

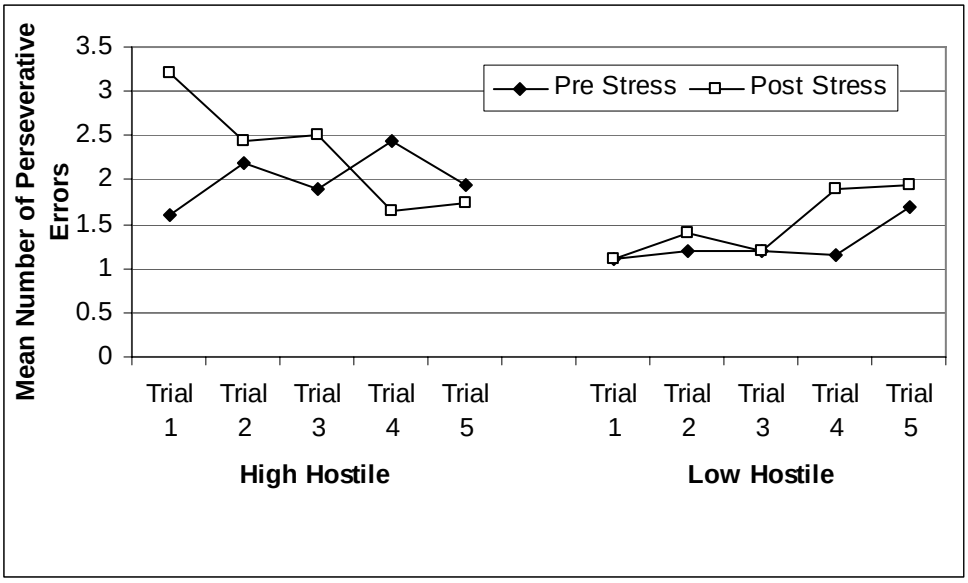


Figure 5. Hostile x Condition x Trial interaction for the Number of Perseverative Errors Made

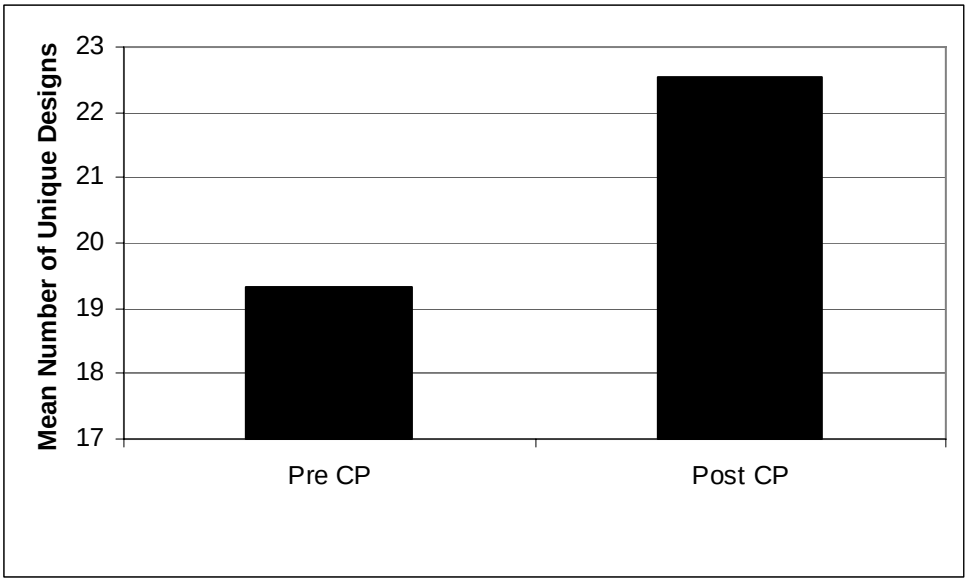


Figure 6. Main effect for the Number of Unique Designs Produced Pre and Post Stress on the first and last trials

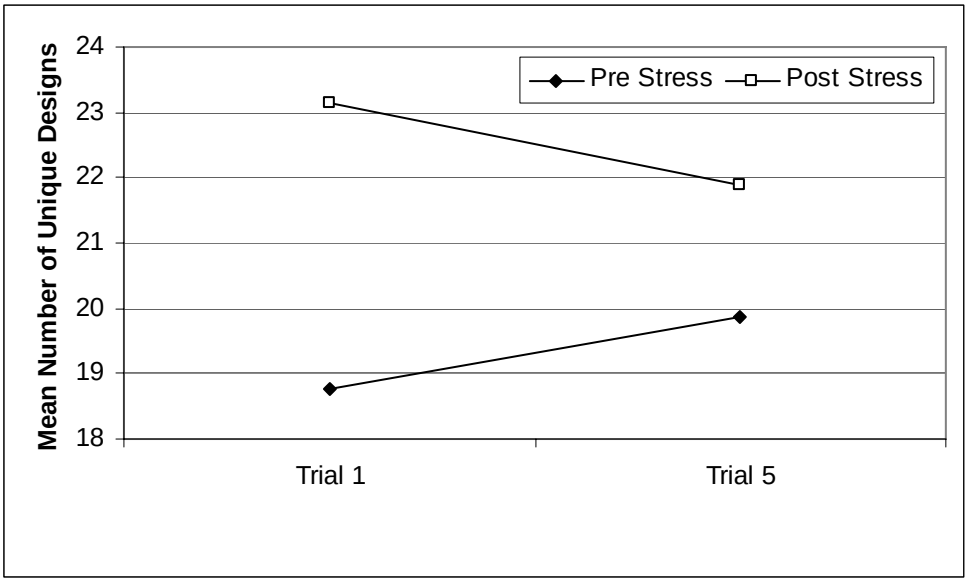


Figure 7. Condition x Trial interaction for the Number of Unique Designs Produced on the first and last trials

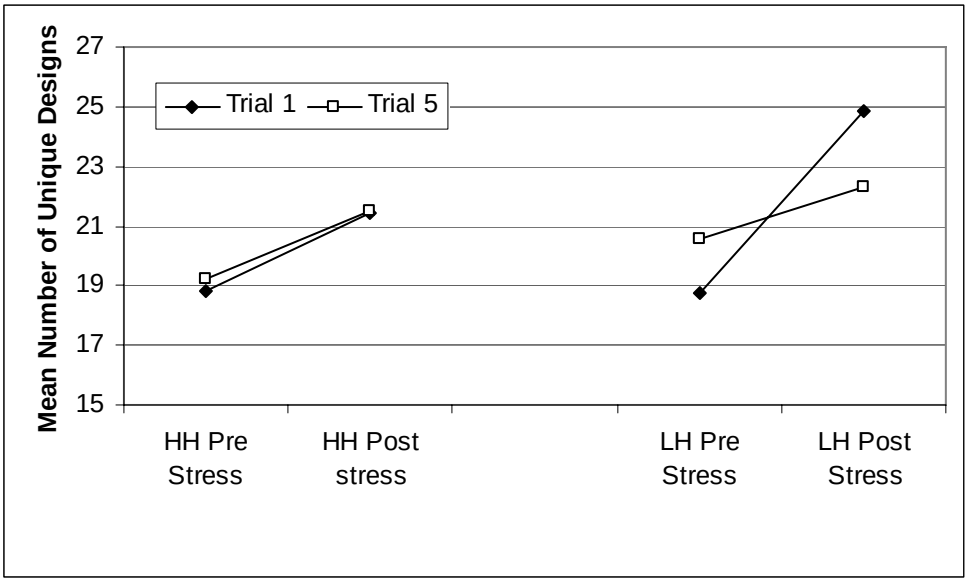


Figure 8. Hostile x Condition x Trial interaction for the Number of Unique Designs Produced on the first and last trials

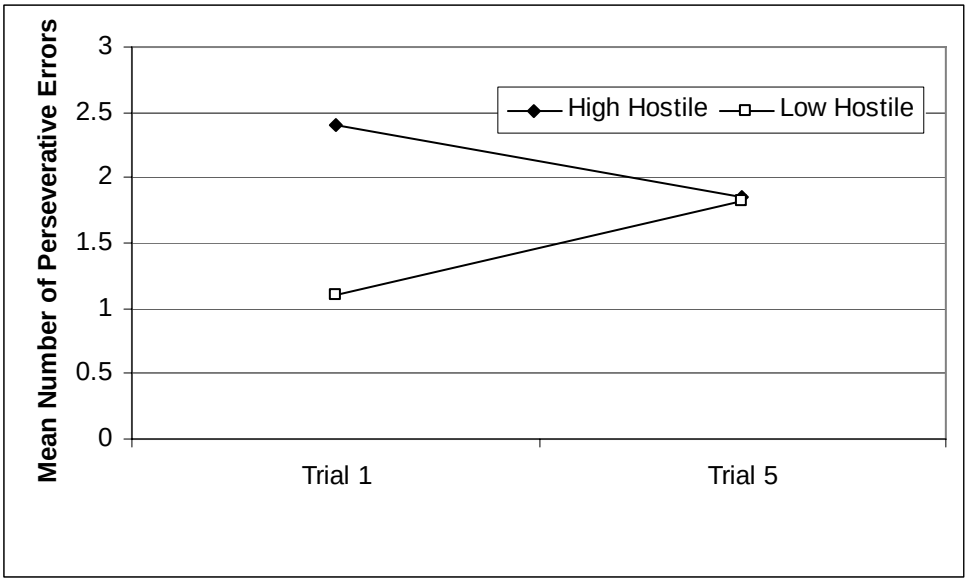


Figure 9. Hostile x Trial interaction for the Number of Perseverative Errors Made on the first and last trials

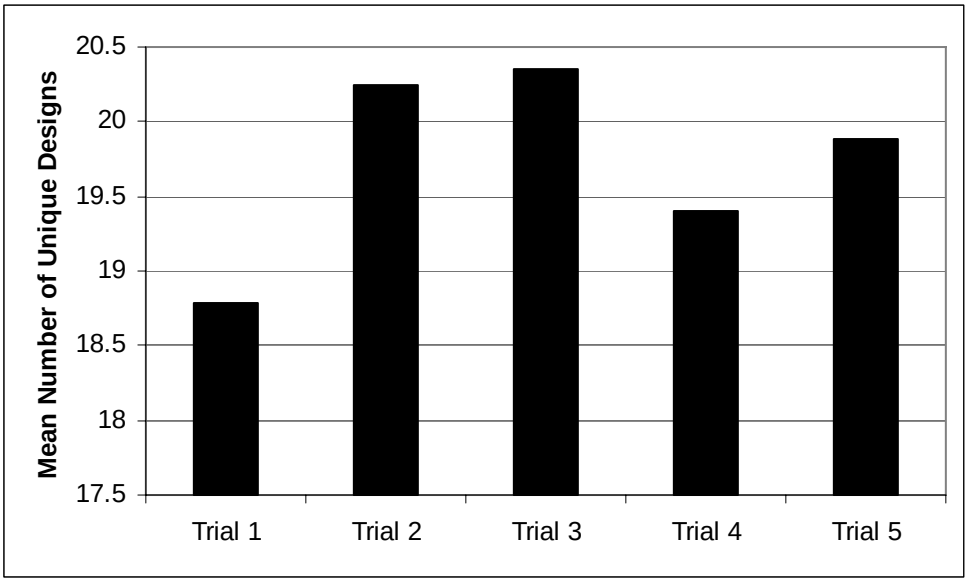


Figure 10. Main effect for Trial for the Number of Unique Designs Produced

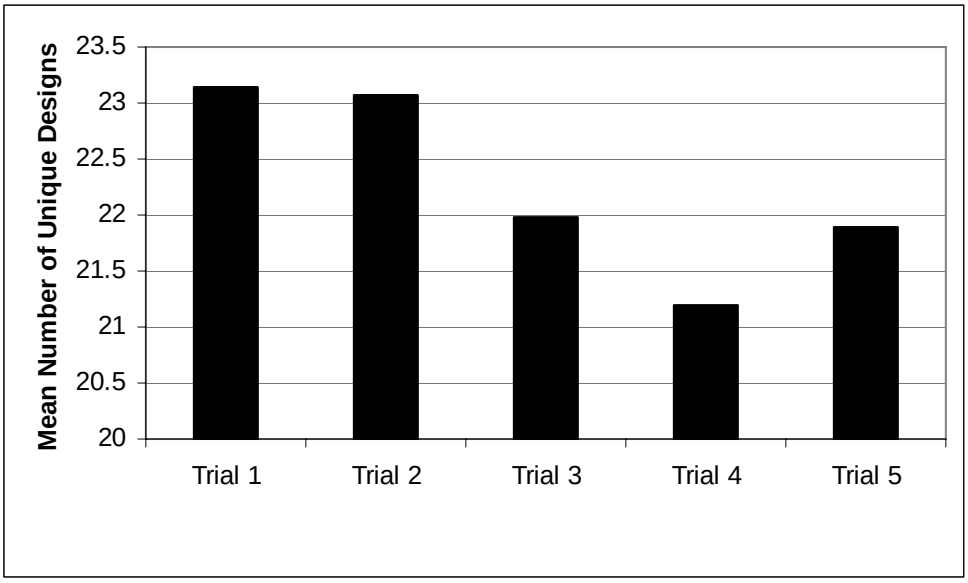


Figure 11. Main effect for Trial for the Number of Unique Designs Produced in the post stress condition

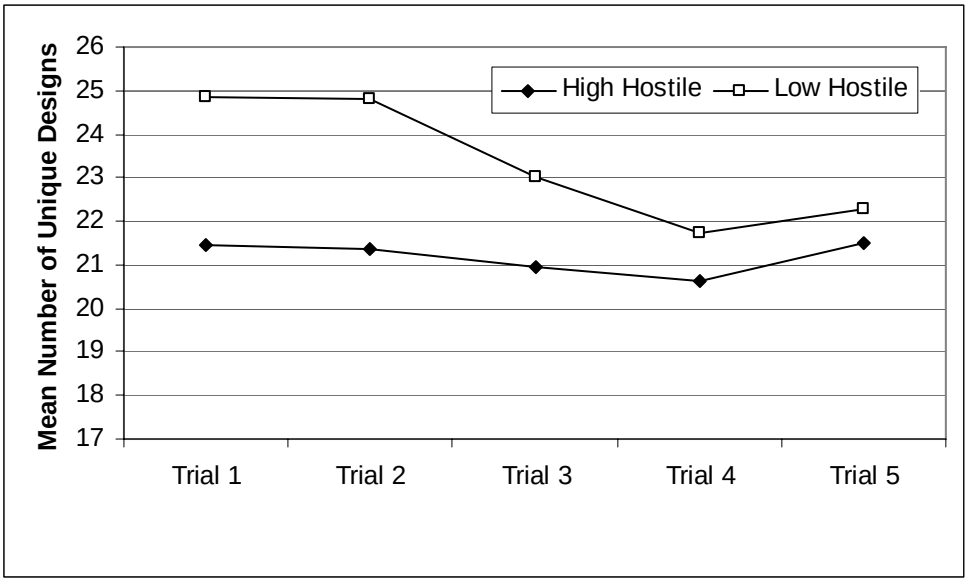


Figure 12. Hostile x Trial interaction for the Number of Unique Designs Produced in the post stress condition

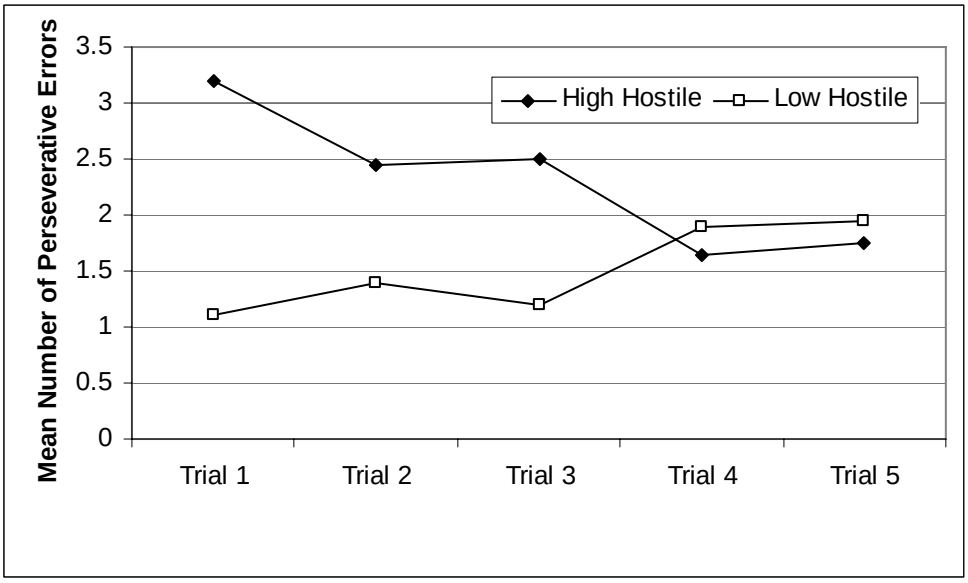


Figure 13. Hostile x Trial interaction for the Number of Perseverative Errors Made in the post stress condition

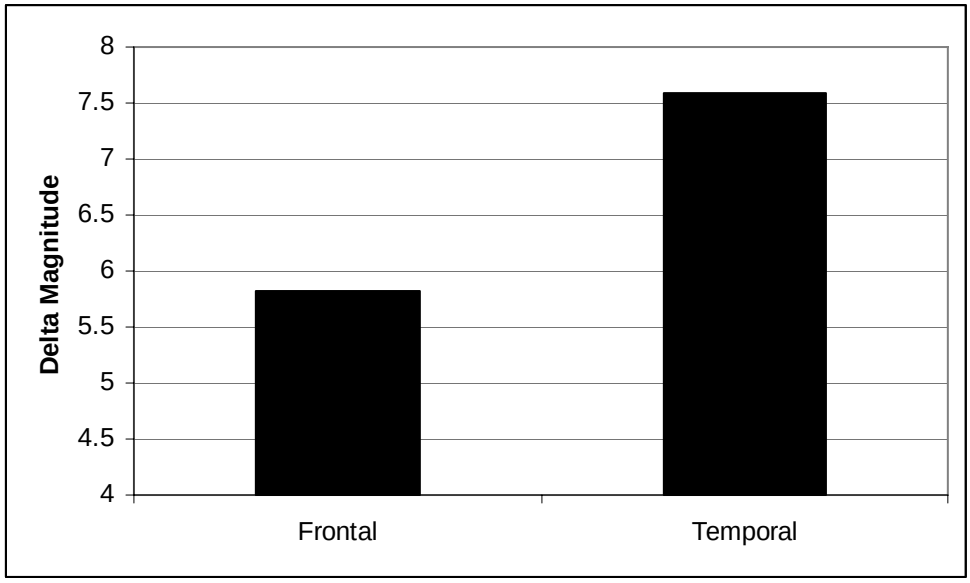


Figure 14. Main effect for Location for the Delta Bandwidth

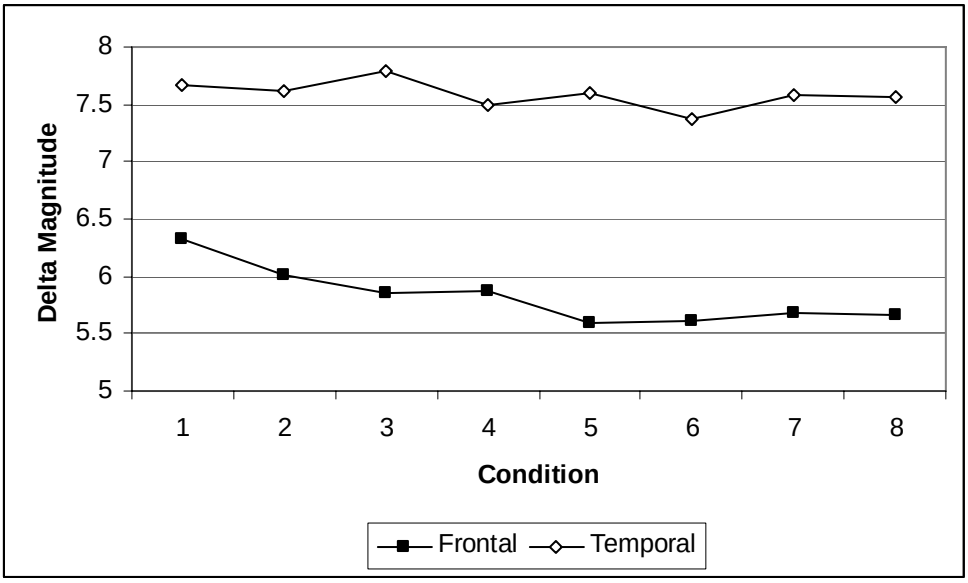


Figure 15. Condition x Location interaction for the Delta Bandwidth at the F8 and T6 electrode sites across conditions

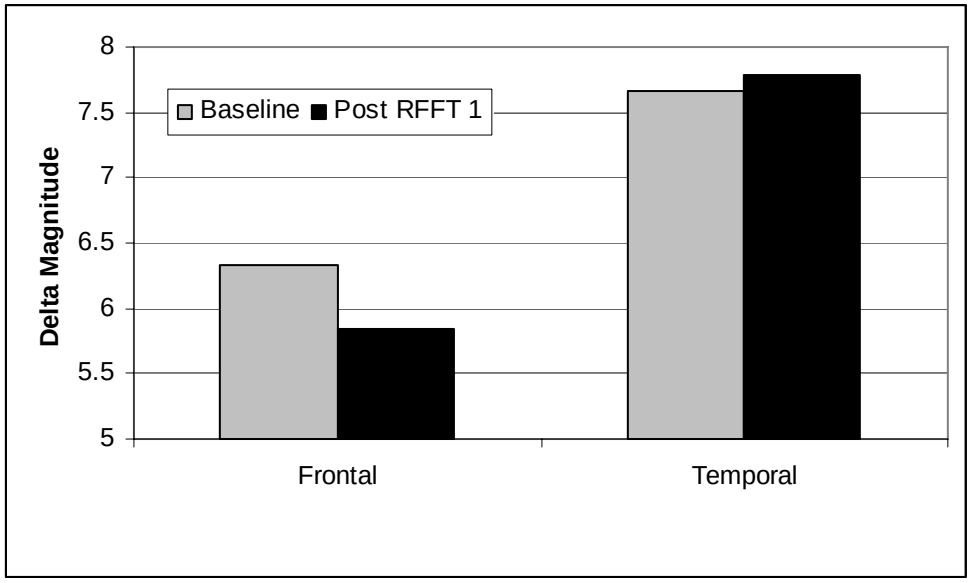


Figure 16. Condition x Location interaction for the Delta Bandwidth at the F8 and T6 electrode sites pre and post Low Arousal Cognitive Stress

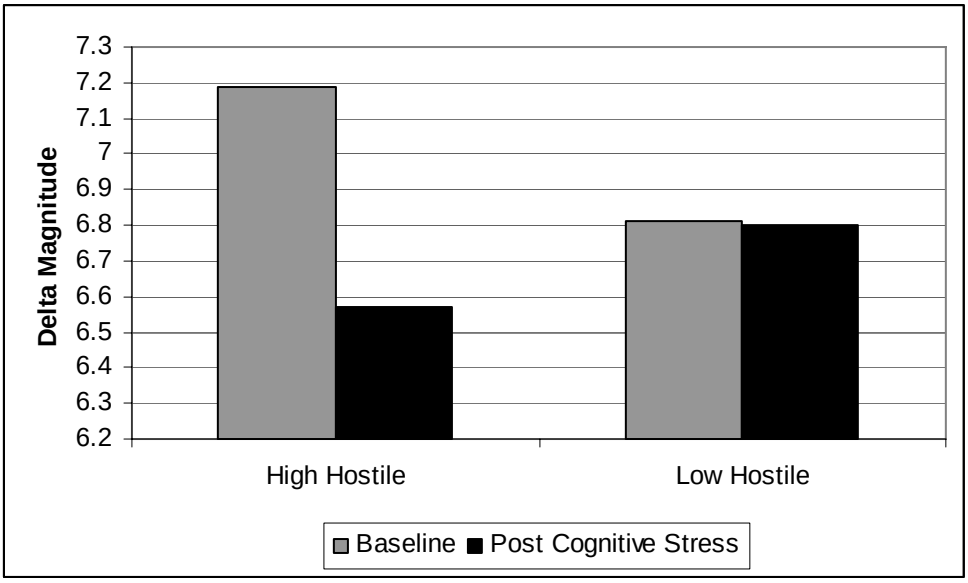


Figure 17. Hostile x Condition interaction for the Delta Bandwidth at the F8 and T6 electrode sites at baseline and post cognitive stress

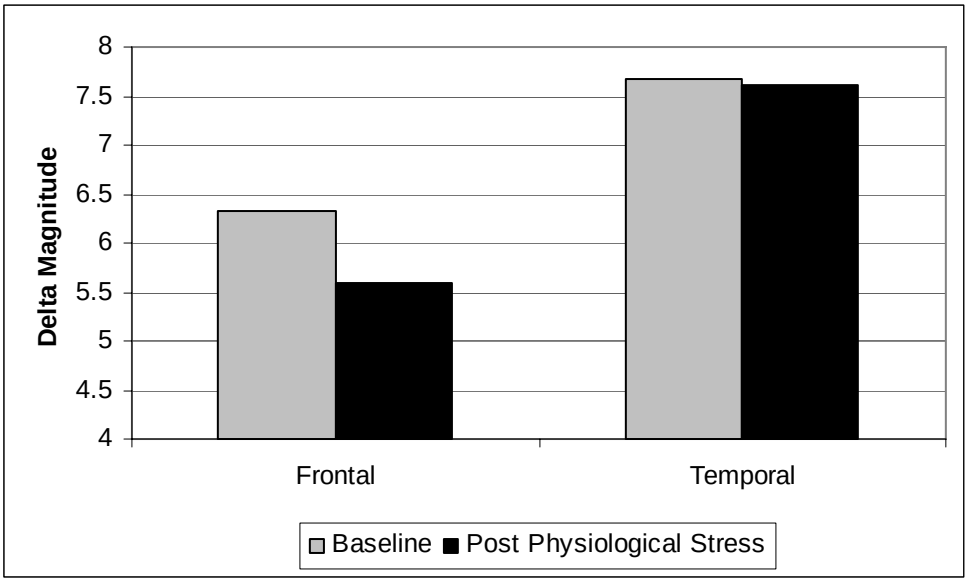


Figure 18. Condition x Location interaction for the Delta Bandwidth at the F8 and T6 electrode sites at baseline and after cold pressor stress

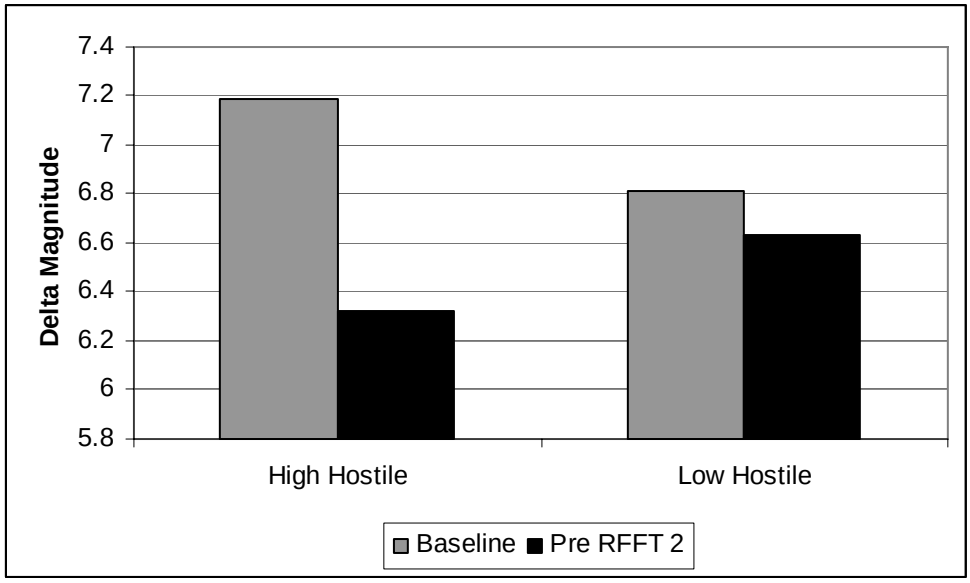


Figure 19. Hostile x Condition interaction for the Delta Bandwidth at the F8 and T6 electrode sites at baseline and before the second design fluency test

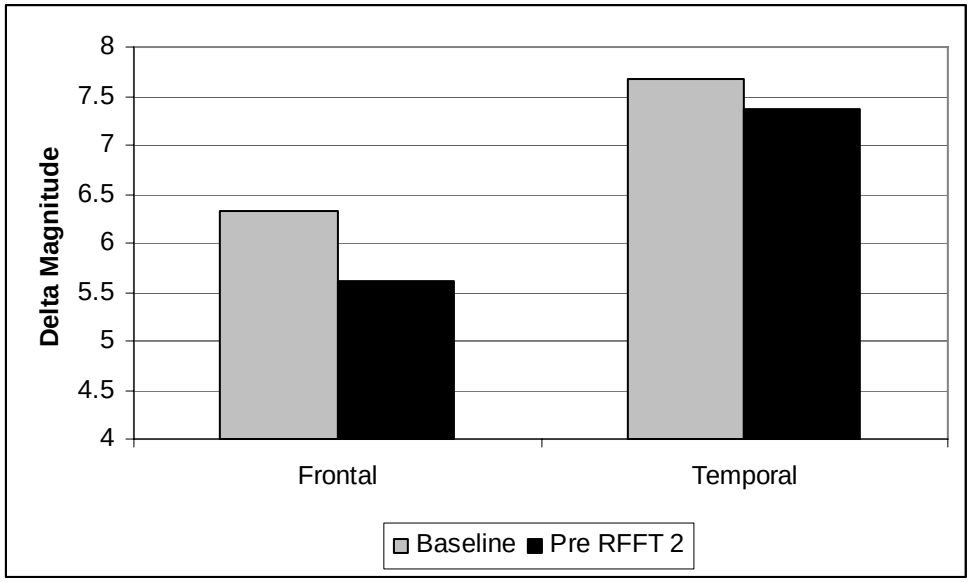


Figure 20. Condition x Location interaction for the Delta Bandwidth at the F8 and T6 electrode sites pre and before the second design fluency test

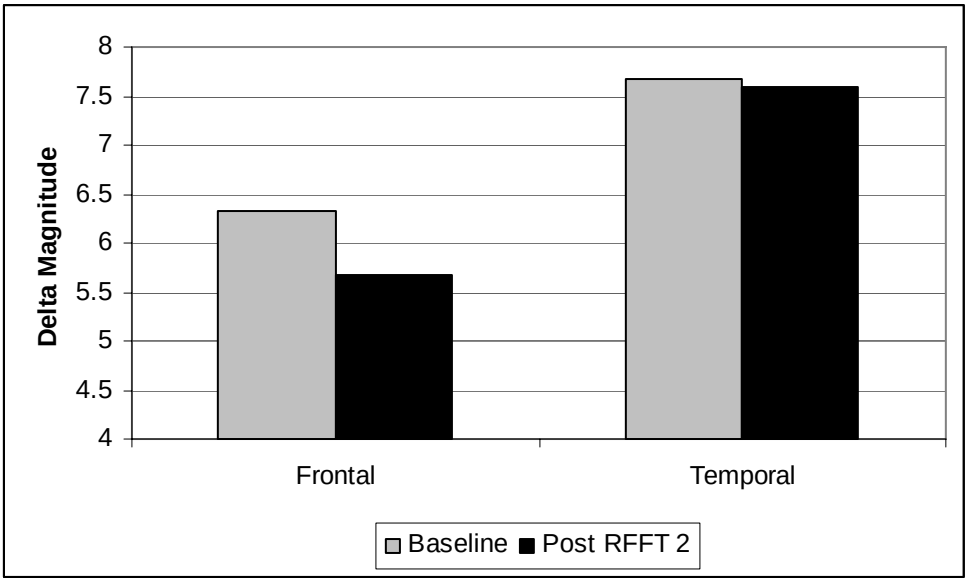


Figure 21. Condition x Location interaction for the Delta Bandwidth at the F8 and T6 electrode sites pre and after the second design fluency test

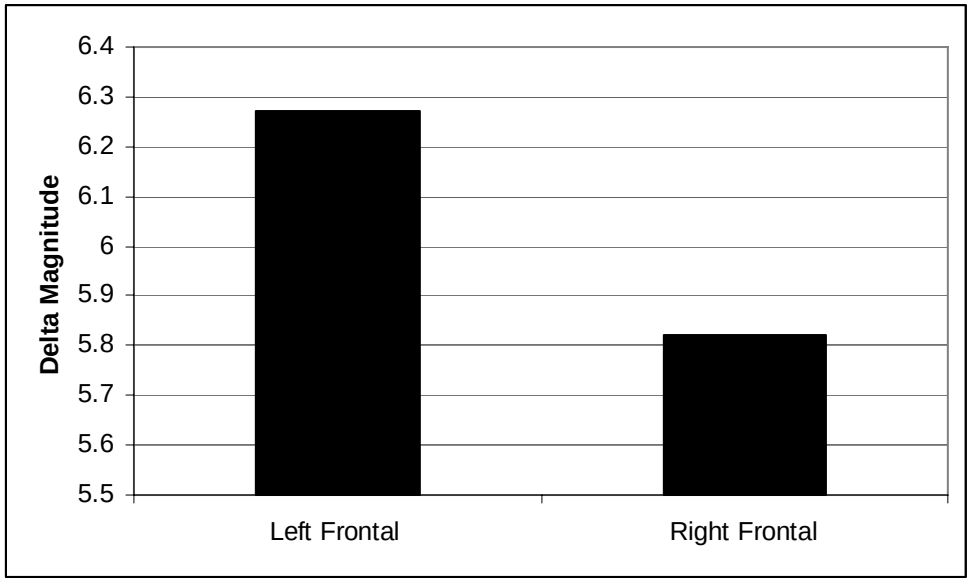


Figure 22. Main effect for Hemisphere for the Delta Bandwidth at the F7 and F8 electrode sites

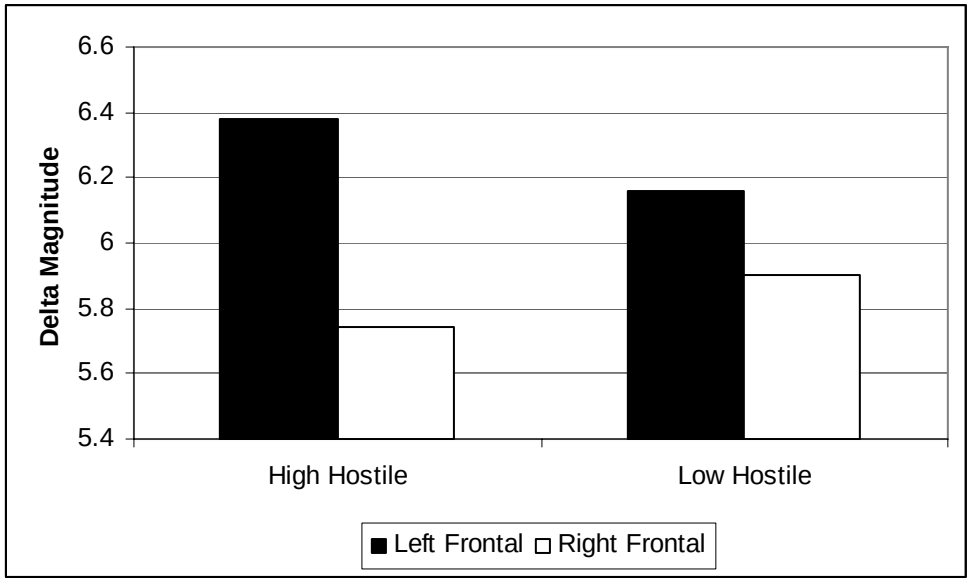


Figure 23. Hostile x Hemisphere interaction for the Delta Bandwidth at the F7 and F8 electrode sites across all conditions

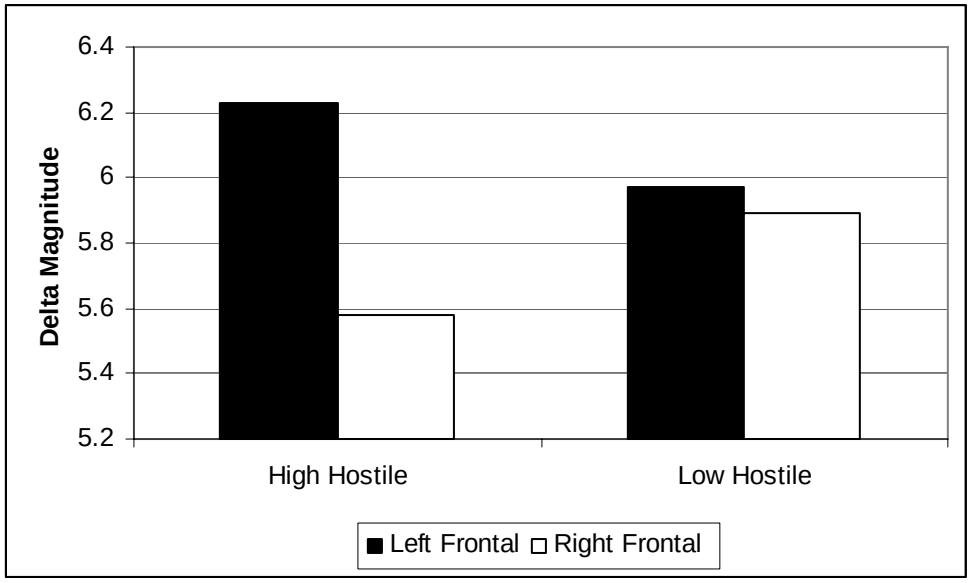


Figure 24. Hostile x Hemisphere interaction for the Delta Bandwidth at the F7 and F8 electrode sites before and after cold pressor stress

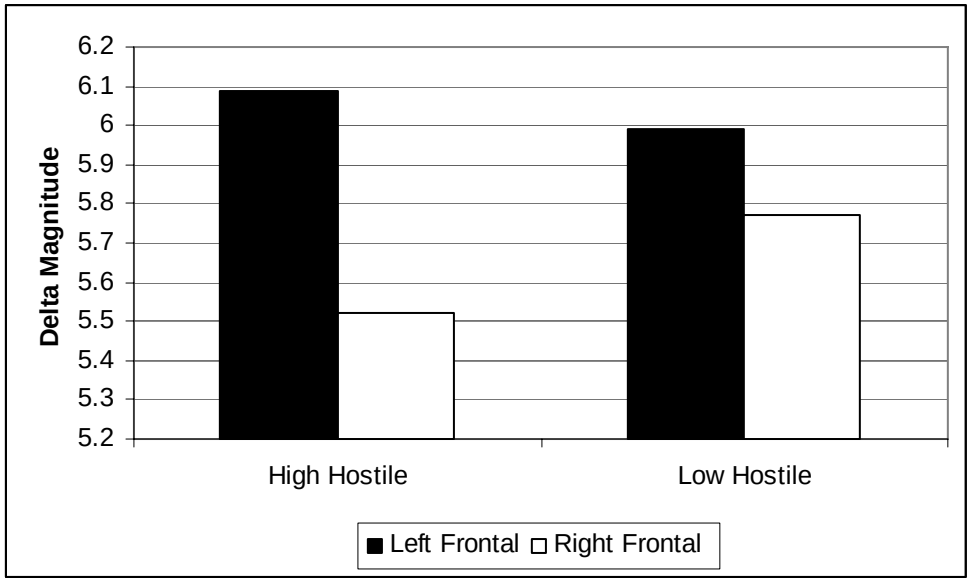


Figure 25. Hostile x Hemisphere interaction for the Delta Bandwidth at the F7 and F8 electrode sites before and after the second design fluency task

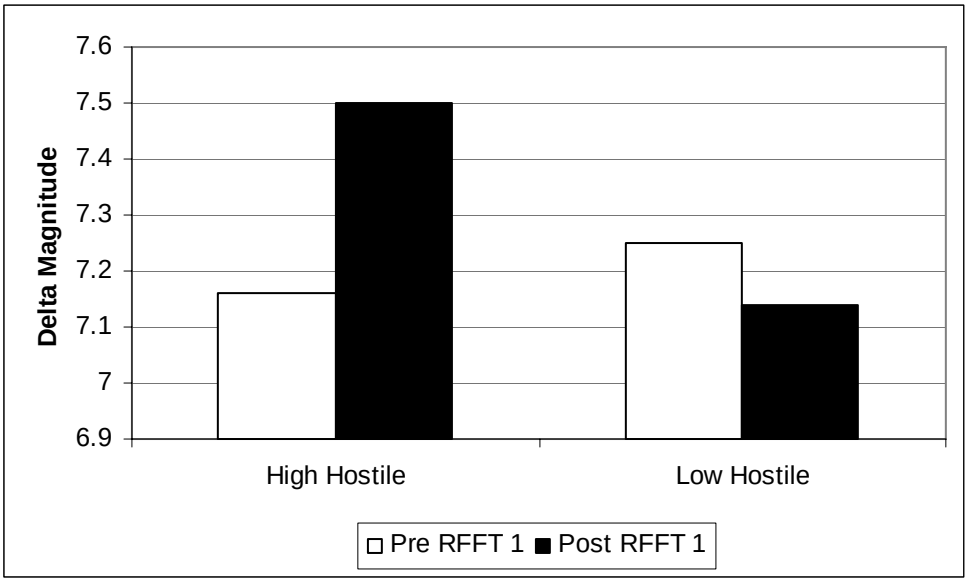


Figure 26. Hostile x Condition interaction for the Delta Bandwidth at the T5 and T6 electrode sites before and after the first design fluency test

Appendix A:

CMHO

Directions: 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 find out who it is important to be nice to.	T	F
2. When people do me wrong, I feel I should pay them 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 have 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 would lie to get ahead.	T	F
8. Someone has it in for me.	T	F
9. Most people are honest chiefly because they are afraid of being 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 often 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
14. I am against giving money to beggars.	T	F
15. Some of my family have habits that bother and annoy me very much.	T	F
16. My relatives are nearly all in sympathy with me.	T	F
17. My way of doing things is apt to be misunderstood by others.	T	F
18. I don’t blame people for trying to grab everything they can get in this world.	T	F
19. No one cares much 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 people who leave themselves 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 my 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 were supposed to be experts who were no better than I.	T	F
35. It makes me feel like a failure when I hear of the success of someone I know well.	T	F
36. I would certainly enjoy beating criminals at their own game.	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 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 people so that they 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 my mind even when I have made a decision about something	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

Appendix B:

Medical History Questionnaire

1	Do you have any history of congenital or developmental problems?	Yes	No
2	Do you have any history of learning disabilities or special education?	Yes	No
3	Do you have any history of hypoglycemia (low blood glucose)?	Yes	No
4	Do you have any history of hyperglycemia (diabetes)?	Yes	No
5	Are you experiencing blood glucose problems at present?	Yes	No
6	Do you have any history of hypertension? (high blood pressure)	Yes	No
7	Do you have any history of hypotension? (low blood pressure)	Yes	No
8	Do you have any history of hyperthyroidism?	Yes	No
9	Do you have any history of hypothyroidism?	Yes	No
10	Have you ever suffered a head injury resulting in a hospital stay longer than 24 hours?	Yes	No
11	Have you ever been knocked out or rendered unconscious (more than 5 minutes)?	Yes	No
12	Have you ever suffered "black-out" or fainting spells?	Yes	No
13	Do you have a history of other neurological disorders (e.g. stroke or brain tumor)?	Yes	No
14	Have you ever received psychiatric/psychological care or counseling?	Yes	No
15	Have you ever been hospitalized in a psychiatric facility/hospital?	Yes	No
16	Have you ever been diagnosed with a psychiatric/psychological disorder?	Yes	No
17	Have you ever been administered any (neuro)psychological tests or measures?	Yes	No
18	Do you have a history of substance abuse or alcohol abuse?	Yes	No
19	Do you have any history of heart disease?	Yes	No
20	Do you have any history of pancreatic disease?	Yes	No
21	Are you currently taking any prescription blood-thinning medications?	Yes	No
22	Do you have a history of high blood pressure?	Yes	No
23	Do you have any uncorrected visual or hearing impairments?	Yes	No
24	Are you able to read, write, and speak English effectively?	Yes	No
25	Do you consume three or more alcoholic more than two nights a week?	Yes	No
26	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, Huntington's Chorea, Alzheimer's dementia, Schizophrenia, or Bipolar Disorder?	Yes	No
27	Have you ever used smoked or used tobacco products?	Yes	No
28	Do you use any unprescribed or "illegal/street" drugs?	Yes	No
29	Are you taking any of the following medications: antidepressant, antianxiety, antipsychotic?	Yes	No
30	Are you taking any allergy or cold medication?	Yes	No
31	Do you frequently experience migraine headaches?	Yes	No

32	Do you have a history of chronic earache that lasted more than a month?	Yes	No
33	Do you often experience pressure in the inner ear?	Yes	No
34	Do you frequently hear a persistent ringing, buzzing, or hissing sound?	Yes	No
35	Have you ever been diagnosed with any of the following vestibular disorders: Orthostatic dysregulation, Meniere's Disease, Cogan's syndrome, Labyrinthine Infarct, Neurolabyrinthitis?	Yes	No
36	Do you have a history of panic attacks or agoraphobia?	Yes	No

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

_____

_____

_____

_____

Appendix C:

Coren, Porac, and Duncan Laterality Questionnaire

Participant #: _____

Date: _____

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
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
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 you earphone of a transistor radio?	1	-1	0

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

Is mother right or left hand dominant? _____

Is father right or left hand dominant? _____

Appendix D:

INFORMED CONSENT I FOR PARTICIPANTS OF INVESTIGATIVE PROJECTS

Title of Project: Changes in Right Frontal Lobe Delta Activity and Fluency Performance as a Function of Stress.

IRB Approval Number: _____

1. PURPOSE OF THE EXPERIMENT
You are invited to participate in a study about the effects of stress on brain activity and design performance. This research will attempt to determine physiological and brain-related changes in response to exposure to a cold pressor test.

2. PROCEDURE TO BE FOLLOWED IN THE STUDY
You will be asked to complete three questionnaires, including one in which you indicate your dominant hand, and one in which you describe your health history. The third questionnaire will ask you to indicate your views on other people.

3. ANONYMITY OF SUBJECTS AND CONFIDENTIALITY OF RESULTS
Identifying participant information will be kept strictly confidential. At no time will the researchers release your personal information for the study to anyone other than individuals working on the project without your written consent. The information you provide will have your name removed and only the participant number will identify you during analyses and any written reports of the research. If you indicate that you intend to harm yourself or someone else, you will be encouraged to seek help, confidentiality will be broken, and the appropriate agencies will be informed.

4. DISCOMFORTS AND RISKS FROM PARTICIPATING IN THE STUDY
There are no potential health risks for completing the online survey. Some of the questionnaires may contain material that you find embarrassing or uncomfortable to answer. You may omit any questions that you find embarrassing or uncomfortable. If you have any questions after leaving the experiment or have any problems associated with the study, you may contact the researcher or Dr. David W. Harrison (231-4422) and he will assist you directly or direct you to appropriate services.

5. EXPECTED BENEFITS
Your participation in this project may help identify the effects of exposure to a cold pressor test on brain-related reactivity. No guarantee of benefits has been made to encourage you to participate.

You may receive a synopsis of the research findings when completed. If you are interested in being informed about the results of the study, please email the researcher (Kate Holland - akhollan@vt.edu) for more information.

6. FREEDOM TO WITHDRAW
You are free to withdraw from this study at any time without penalty. If you withdraw, extra credit points are awarded based on time spent on this effort in half-hour increments.

7. EXTRA CREDIT COMPENSATION
For participation in this study you will receive one point of extra credit to be awarded in the psychology class of your choosing if the instructor will accept the points.

8. USE OF RESEARCH DATA
The information from this research may be used for scientific or educational purposes. It may be presented at scientific meetings and/or published and reproduced in professional journals or books, or used for any other purpose that Virginia Tech's Department of Psychology considers proper in the interest of education, knowledge, or research.

9. APPROVAL OF RESEARCH
This project has been approved by the Human Subjects Committee of the Department of Psychology and by the Institutional Review Board of Virginia Tech.

10. PARTICIPANT'S RESPONSIBILITIES
I know of no reason I cannot participate in this study. I have the following responsibilities: Report to the experimenter any history of head injury, epilepsy, cardiac problems, or hearing impairments; Report to the experimenter current regular medications.

11. PARTICIPANT'S PERMISSION
I have read and understand the above description of the study. I have had the opportunity to ask questions and all have been answered in an appropriate manner. I hereby acknowledge the above and voluntarily give my consent to participate in this study. I realize that I may withdraw at any time without penalty and that I may contact one of the people listed below at any time if I have questions regarding the study.

Kate Holland, M.A. 231-3235
Primary Researcher

David W. Harrison, Ph.D. 231-4422
Faculty Advisor

Bob Stephens, Ph.D. 231-6670
Department Chair, Department of Psychology

David Moore, Ph.D. 231-4991
Institutional Review Board Chair, Research Division

Participant's Signature: _____ Date: _____

Participant's Student ID: _____ Participant Phone #: _____

Appendix E

INFORMED CONSENT II FOR PARTICIPANTS OF INVESTIGATIVE PROJECTS

Title of Project: Changes in Right Frontal Lobe Delta Activity and Fluency Performance as a Function of Stress.

IRB Approval Number: _____

1. PURPOSE OF THE EXPERIMENT
You are invited to participate in a study about the effects of stress on brain activity and design performance. This research will attempt to determine brain-related changes in response to exposure to a cold pressor test.

2. PROCEDURE TO BE FOLLOWED IN THE STUDY
You will be asked to complete a questionnaire in which you answer questions pertaining to your view of others. You will then be fitted with a cap that collects electroencephalographic (EEG) information from the scalp. Once fitted with the cap, you will then be asked to complete a design fluency task that is 5 minutes in length. After completion of the design fluency task, your left hand will be immersed in cold water for 45 seconds. You will then complete the design fluency task once more. EEG activity will be measured several times throughout this experiment.

3. ANONYMITY OF SUBJECTS AND CONFIDENTIALITY OF RESULTS
Identifying participant information will be kept strictly confidential. At no time will the researchers release your personal information for the study to anyone other than individuals working on the project without your written consent. The information you provide will have your name removed and only the participant number will identify you during analyses and any written reports of the research. If you indicate that you intend to harm yourself or someone else, you will be encouraged to seek help, confidentiality will be broken, and the appropriate agencies will be informed.

4. DISCOMFORTS AND RISKS FROM PARTICIPATING IN THE STUDY
You may experience some discomfort when the EEG cap is being fitted. Some of the questionnaires may contain material that you find embarrassing or uncomfortable to answer. You may omit any questions that you find embarrassing or uncomfortable.

If you have any questions after leaving the experiment or have any problems associated with the study, you may contact the researcher or Dr. David W. Harrison (231-4422) and he will assist you directly or direct you to appropriate services.

5. EXPECTED BENEFITS
Your participation in this project may help identify the effects of exposure to a cold pressor test on brain-related reactivity. No guarantee of benefits has been made to encourage you to participate.

You may receive a synopsis of the research findings when completed. If you are interested in being informed about the results of the study, please email the researcher (Kate Holland-akhollan@vt.edu) for more information.

6. FREEDOM TO WITHDRAW
You are free to withdraw from this study at any time without penalty. If you withdraw, extra credit points will be awarded based on time spent on this effort in half-hour increments.

7. EXTRA CREDIT COMPENSATION
For participation in this study you will receive two points of extra credit to be awarded in the psychology class of your choosing if the instructor will accept the points.

8. USE OF RESEARCH DATA
The information from this research may be used for scientific or educational purposes. It may be presented at scientific meetings and/or published and reproduced in professional journals or books, or used for any other purpose that Virginia Tech's Department of Psychology considers proper in the interest of education, knowledge, or research.

9. APPROVAL OF RESEARCH
This project has been approved by the Human Subjects Committee of the Department of Psychology and by the Institutional Review Board of Virginia Tech.

10. PARTICIPANT'S RESPONSIBILITIES
I know of no reason I cannot participate in this study. I have the following responsibilities: Report to the experimenter any history of head injury, epilepsy, cardiac problems, or hearing impairments. Report to the experimenter current regular medications.

11. PARTICIPANT'S PERMISSION
I have read and understand the above description of the study. I have had the opportunity to ask questions and all have been answered in an appropriate manner. I hereby acknowledge the above and voluntarily give my consent to participate in this study.

I realize that I may withdraw at any time without penalty and that I may contact one of the persons listed below at any time if I have questions regarding the study.

Kate Holland, M.A. 231-3235
Primary Researcher

David W. Harrison, Ph.D. 231-4422
Faculty Advisor

Bob Stephens, Ph.D. 231-6670
Department Chair, Department of Psychology

David Moore, Ph.D. 231-4991
Institutional Review Board Chair, Research Division

Student Signature Date

_____ _____

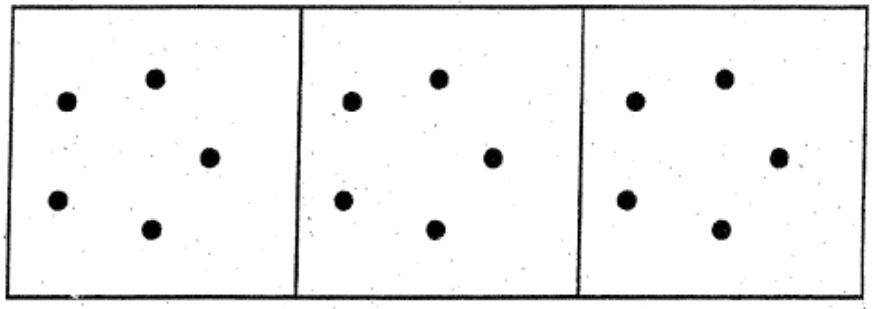
Student Name (printed) Student ID Number

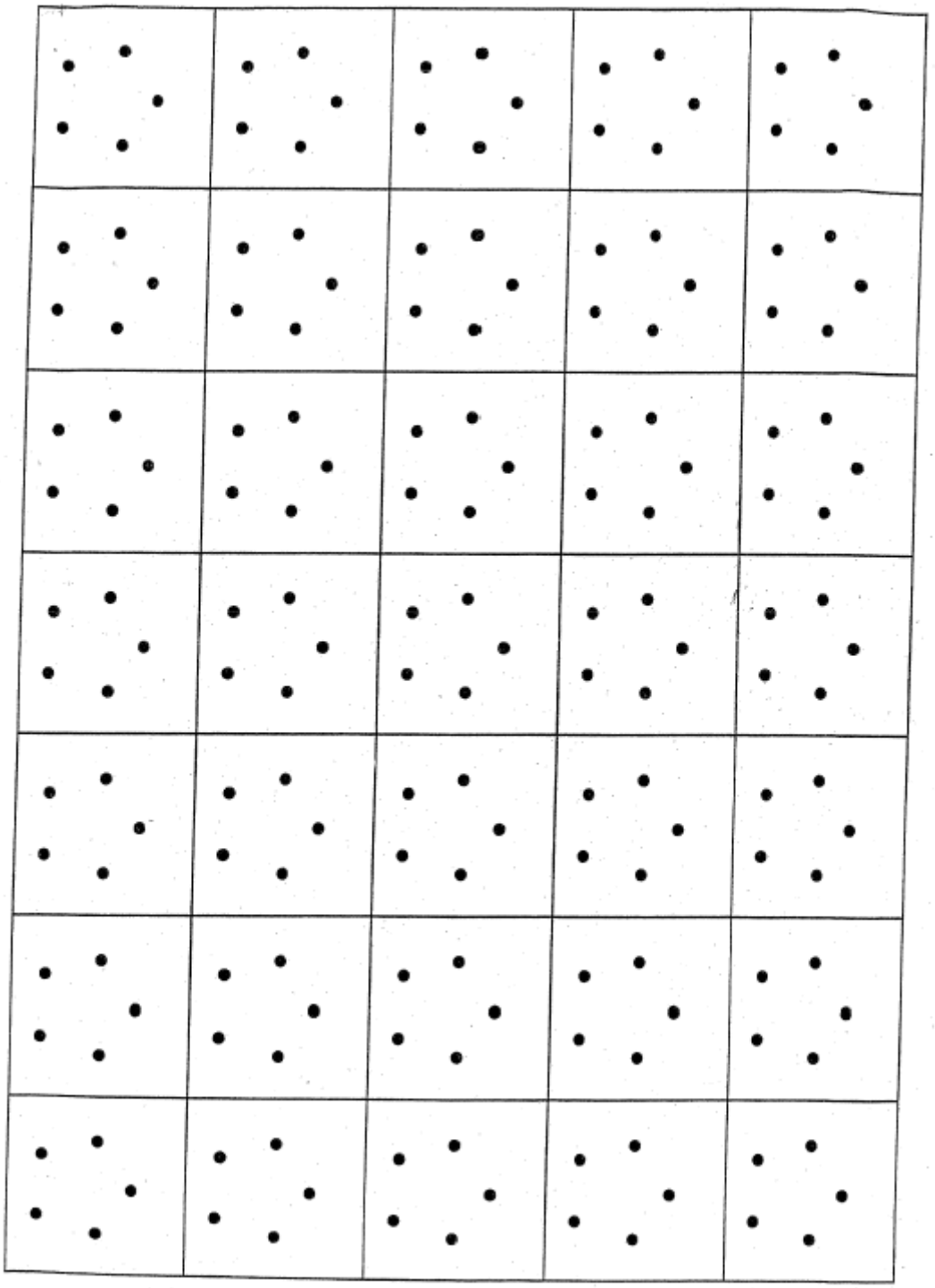
_____ _____

Appendix F:

Ruff Figural Fluency Test

Part 1





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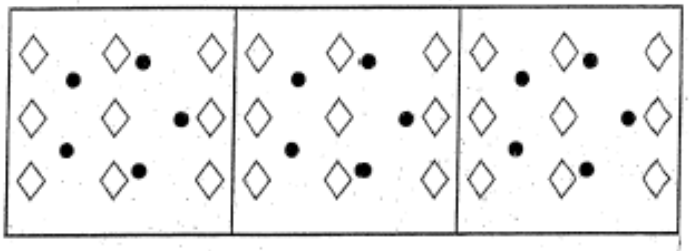

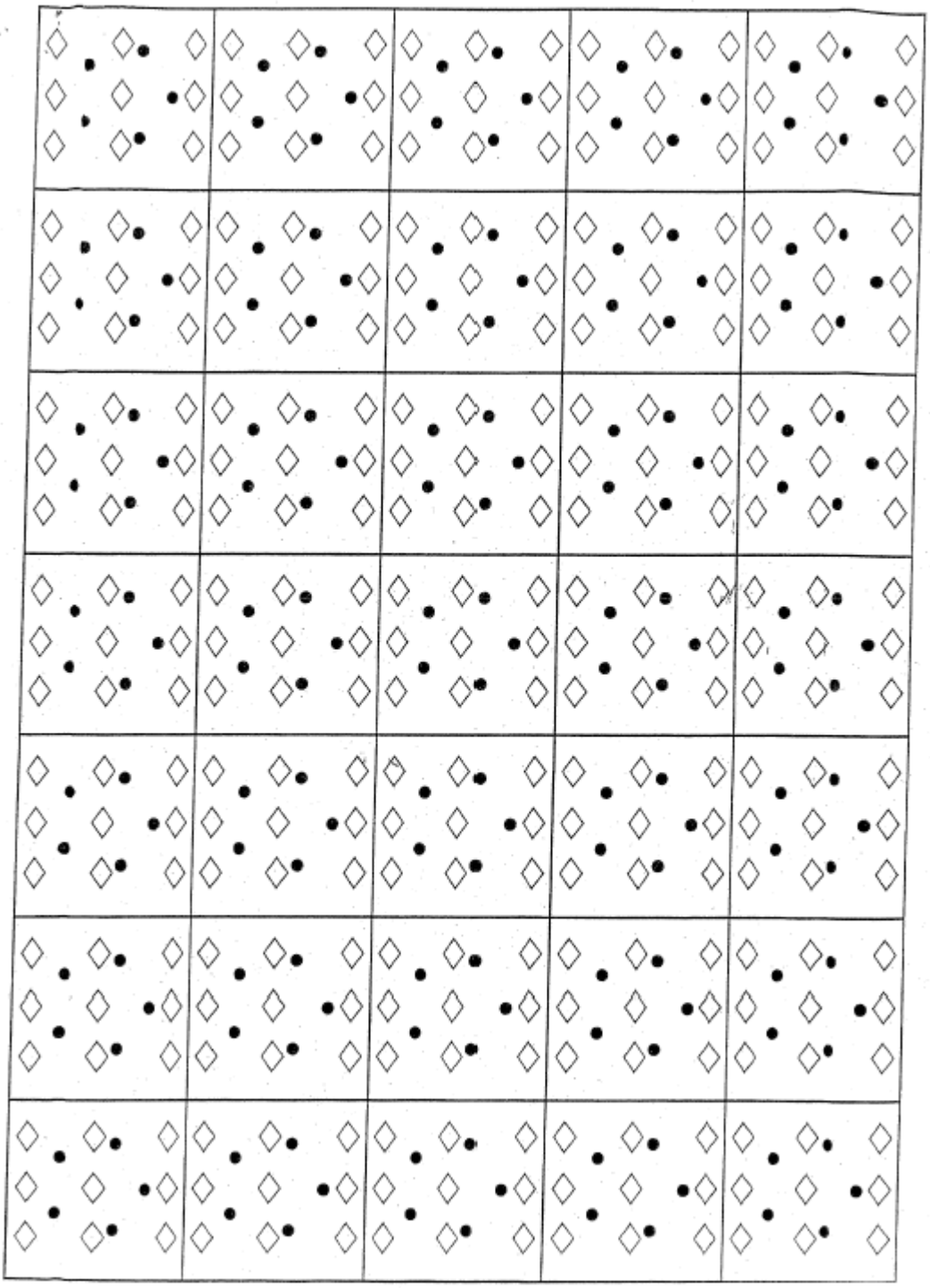

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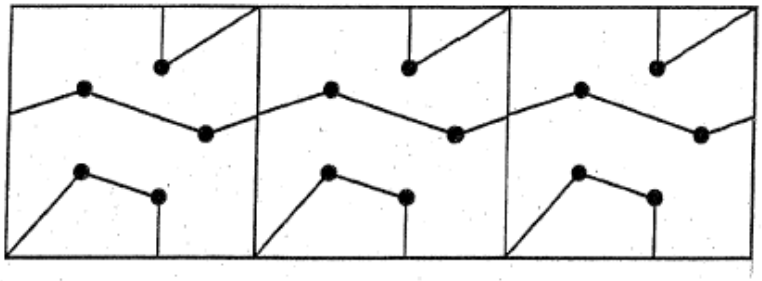
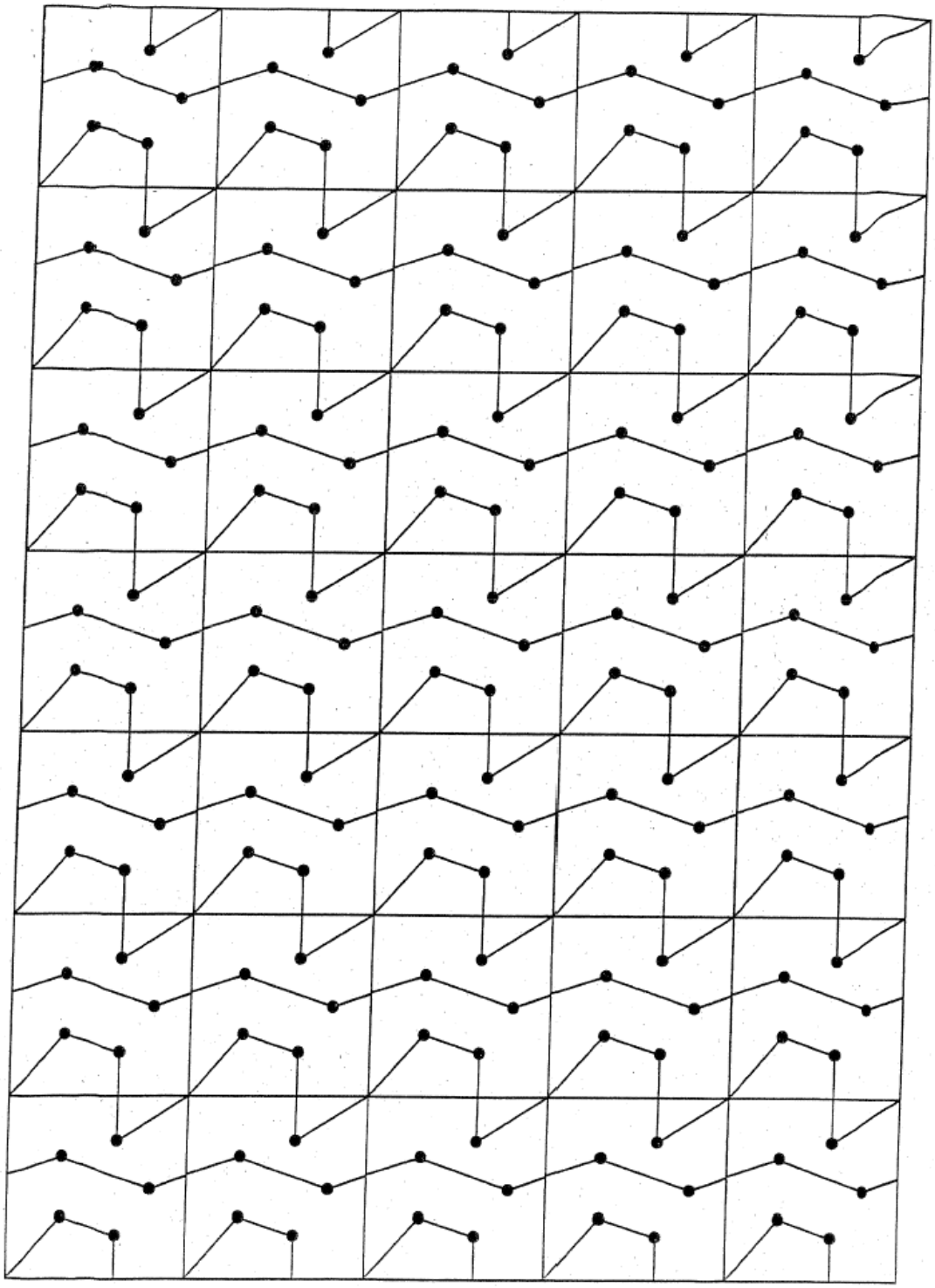
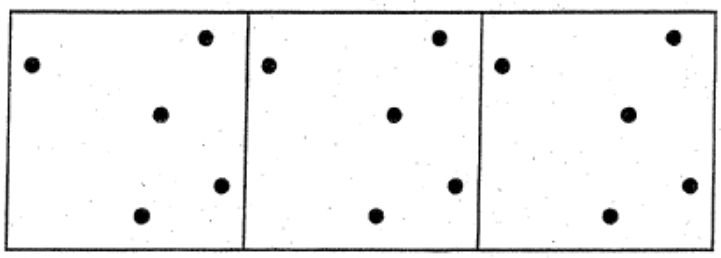
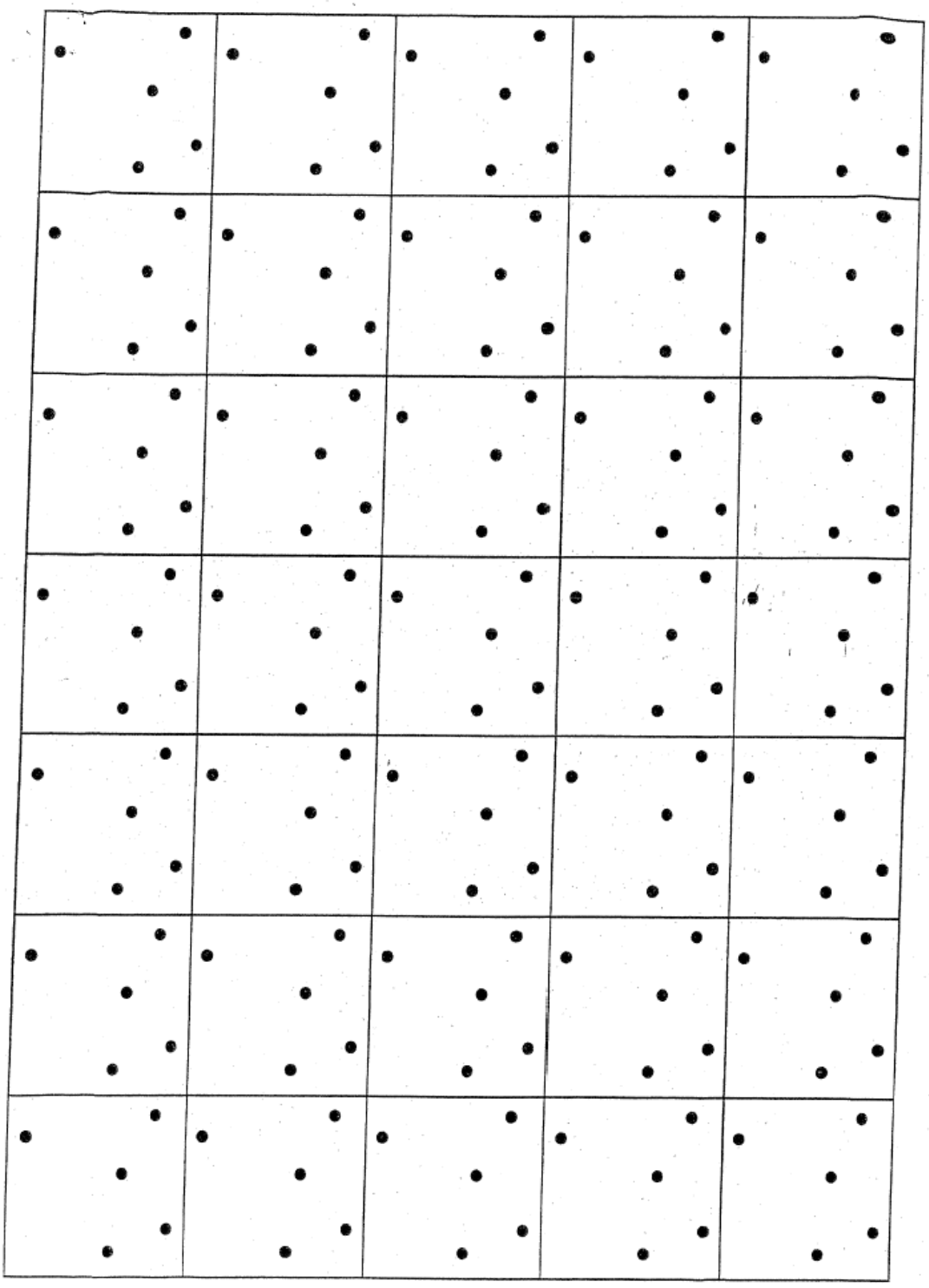


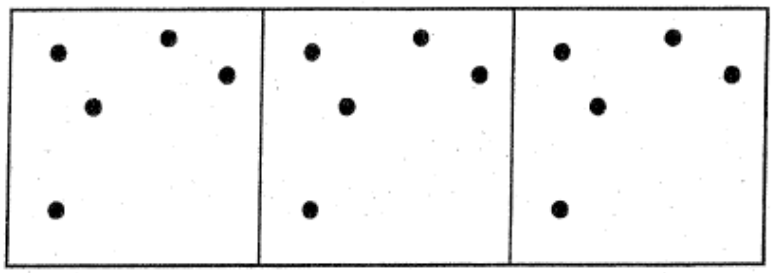


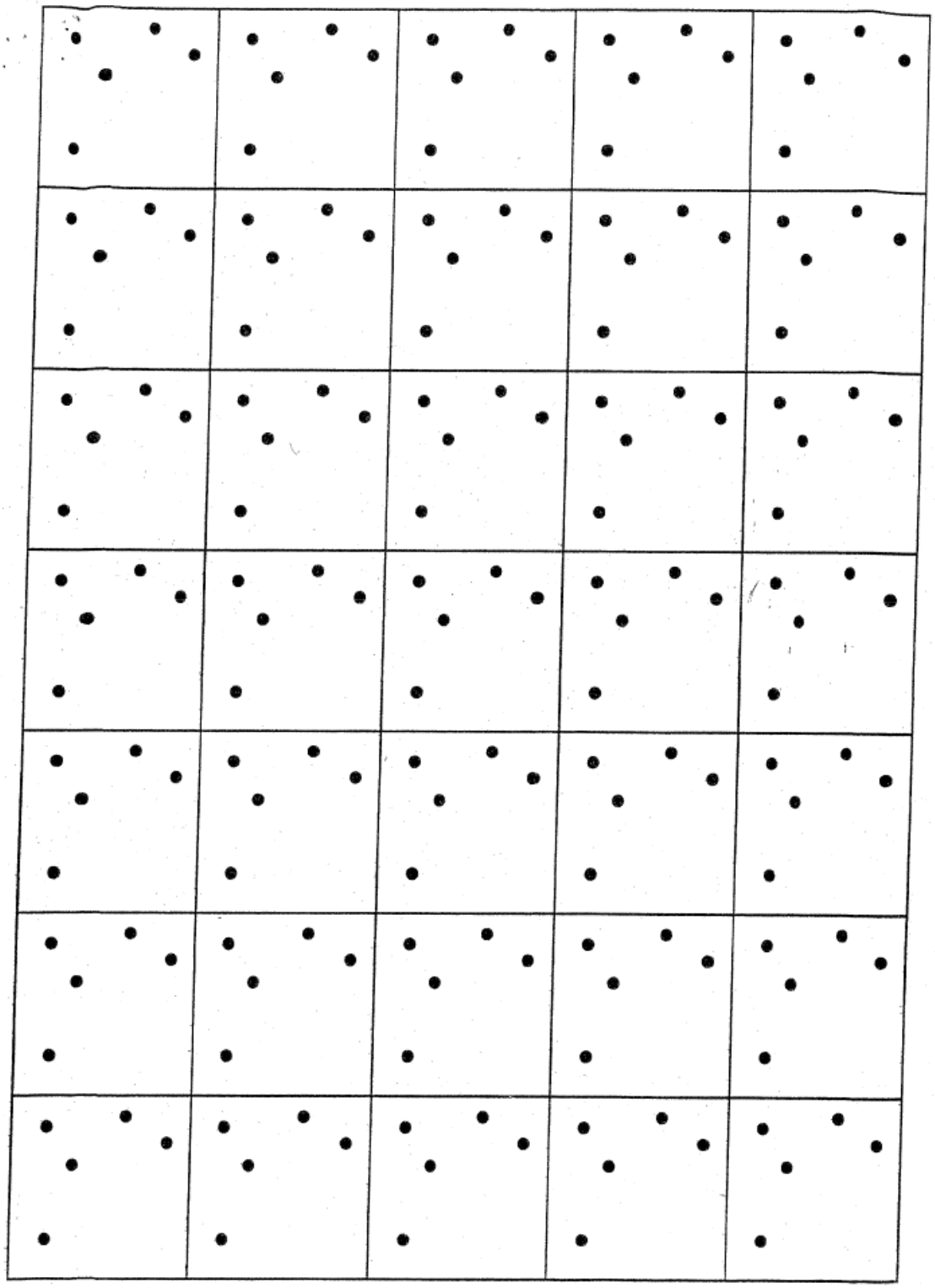
Part 4





Part 5





Appendix G

RFFT Difficulty Scale

Please rate how difficult the design task was:

1	2	3	4	5
Not at all difficult		moderately difficult		extremely difficult

Appendix H

Cold Pressor Intensity Scale

Please rate the level of pain you perceived when your hand was immersed in ice water:

1	2	3	4	5
Not at all intense		moderately intense		extremely intense

Appendix I

Hostility Rating Scale

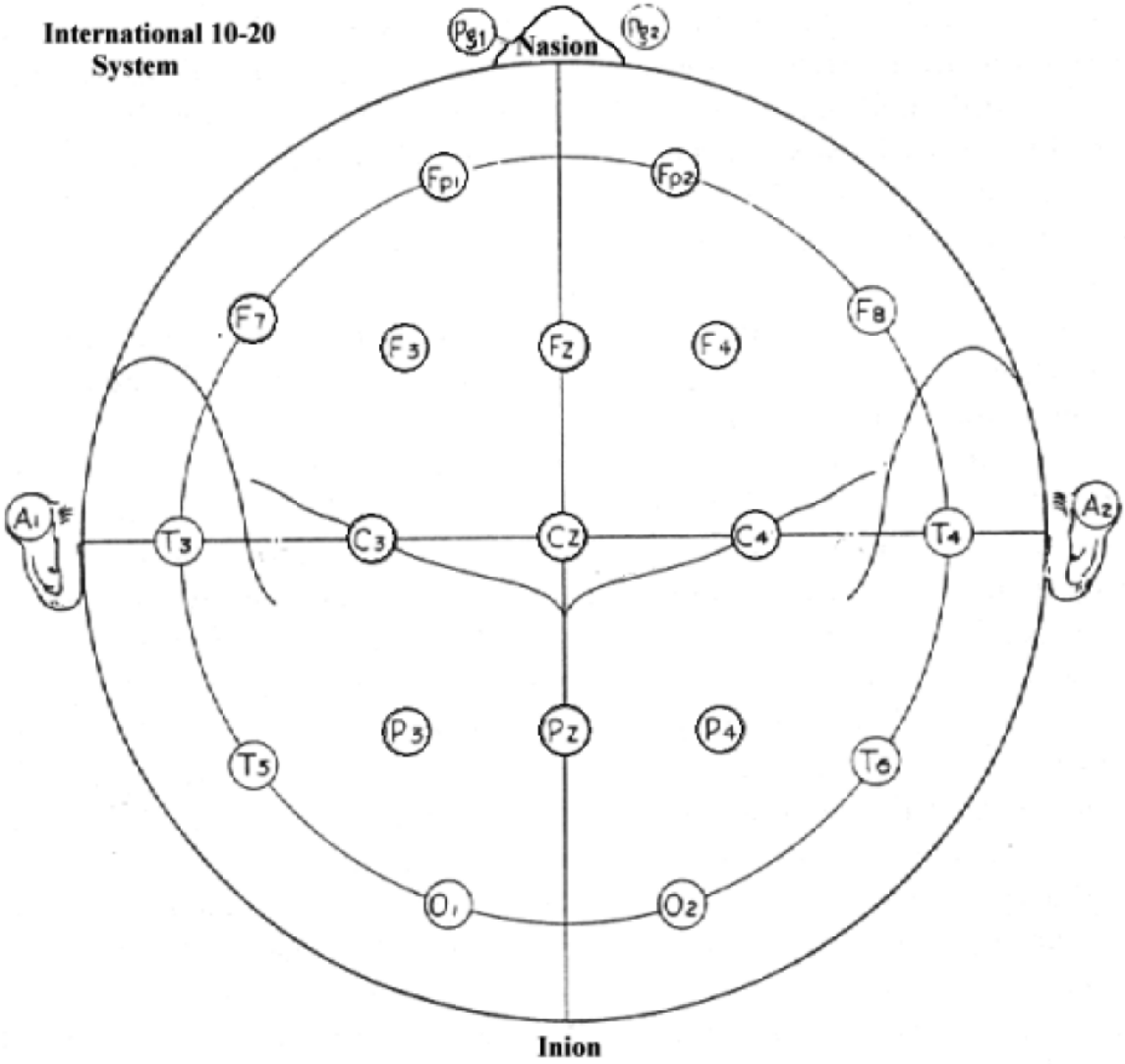
Please indicate whether you think you experienced low-hostility or high-hostility by circling the option that best describes you:

High Hostile

Low Hostile

Appendix J

The International 10/20 System



Appendix K

IRB Approval Letter

VirginiaTech

Office of Research Compliance
Institutional Review Board
2000 Kraft Drive, Suite 2000 (0497)
Blacksburg, Virginia 24061
540/231-4991 Fax 540/231-0959
e-mail moored@vt.edu
www.irb.vt.edu

FWA00000572(expires 1/20/2010)
IRB # is IRB00000667

DATE: July 20, 2007

MEMORANDUM

TO: David W. Harrison
Kate Holland

Approval date: 7/20/2007
Continuing Review Due Date:7/5/2008
Expiration Date: 7/19/2008

FROM: David M. Moore

SUBJECT: **IRB Expedited Approval:** "A Functional Cerebral Systems Approach to Hostility: Changes in Right Frontal Lobe Delta Activation and Fluency Performance as a Function of Stress" , IRB # 07-358

This memo is regarding the above-mentioned protocol. The proposed research is eligible for expedited review according to the specifications authorized by 45 CFR 46.110 and 21 CFR 56.110. As Chair of the Virginia Tech Institutional Review Board, I have granted approval to the study for a period of 12 months, effective July 20, 2007.

As an investigator of human subjects, your responsibilities include the following:

1. Report promptly proposed changes in previously approved human subject research activities to the IRB, including changes to your study forms, procedures and investigators, regardless of how minor. The proposed changes must not be initiated without IRB review and approval, except where necessary to eliminate apparent immediate hazards to the subjects.
2. Report promptly to the IRB any injuries or other unanticipated or adverse events involving risks or harms to human research subjects or others.
3. Report promptly to the IRB of the study's closing (i.e., data collecting and data analysis complete at Virginia Tech). If the study is to continue past the expiration date (listed above), investigators must submit a request for continuing review prior to the continuing review due date (listed above). It is the researcher's responsibility to obtained re-approval from the IRB before the study's expiration date.
4. If re-approval is not obtained (unless the study has been reported to the IRB as closed) prior to the expiration date, all activities involving human subjects and data analysis must cease immediately, except where necessary to eliminate apparent immediate hazards to the subjects.

Important:
If you are conducting **federally funded non-exempt research**, this approval letter must state that the IRB has compared the OSP grant application and IRB application and found the documents to be consistent. Otherwise, this approval letter is invalid for OSP to release funds. Visit our website at http://www.irb.vt.edu/pages/newstudy.htm#OSP for further information.

cc: File

Appendix L

Schematic