

Cardiovascular Arousal and QEEG

Analysis of Quantitative Electroencephalographic and Cardiovascular Responses to Stress Among Low- and High-Hostiles

by

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(ABSTRACT)

This experiment was primarily designed to identify higher cortical correlates of cardiovascular arousal. Low- and high-hostile, right-handed, undergraduate men were identified using the Cook Medley Hostility Scale (CMHS). All participants (N = 30) completed the cold pressor paradigm. Cardiovascular (heart rate, systolic blood pressure, and diastolic blood pressure) and electroencephalographic (beta magnitude) data were collected before and after the stressor.

As predicted, high-hostiles showed greater increases of heart rate and systolic blood pressure to the stressor relative to low-hostiles. The primary findings of this research include significantly greater beta magnitude recorded by the T3, relative to F7, electrode among low-hostiles. This may suggest that low-hostiles experience left-frontal disinhibition of left-temporal regions, thereby strengthening cardiovascular regulation during the cold-pressor stress. In addition, irrespective of condition, high-hostiles evidenced significantly greater beta magnitude at

regions corresponding to the F7 and F8 electrodes. This perhaps suggests that high-hostiles have a relative inability to increase their rostral modulation of posterior systems related to cardiovascular reactivity/regulation.

Low- and high-hostiles did not, however, evidence reliable differences in their ability to monitor cardiovascular arousal to the cold-pressor stress. Findings are discussed in terms of a systems approach, and pertinent future research is recommended.

This research did not support the prominent neuropsychological theories of cardiovascular regulation proposed by Heilman et al. (1993) and Tucker and Williamson (1984). Rather, the results may suggest that right- and left-cerebral mechanisms may be primarily responsible for sympathetic and parasympathetic cardiovascular arousal, respectively.

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This work is dedicated to my future wife, Patricia Kelly.

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An act of violent crime (including murder, forcible rape, violent robbery, and aggravated assault) occurred every 22 seconds in the United States during 1994. (Federal Bureau of Investigation, 1994). An estimated 24,526 murders (9.5 per every 100,000 citizens), 104,806 forcible rapes (40.6/100,000), 659,757 violent robberies (255.8/100,000), and 1,135,099 aggravated assaults (440.8/100,000) victimized our nation's citizens during that year (Federal Bureau of Investigations, 1994). These staggering statistics represent a thirty-eight percent increase since 1984. The need for a better understanding of hostility is apparent and has resulted in increased attention towards its correlates over the past two decades.

This paper will review prominent theories of hostility and negative affective lability, in which one evidences any type of unusually strong negative emotional expression (like crying). As will be discussed, the right cerebrum is widely considered a central mediator of these phenomena. To study hostility and its correlates, many scientists incorporate stress paradigms in their experimental design. Physiological measures -- heart rate (HR), systolic blood pressure (SBP), and diastolic blood pressure (DBP) -- are commonly used dependent variables which generally suggest greater increases in brain activity to stress among high-hostiles. However, these physiological measures may be right-cerebrum mediated. Therefore, collected data may indicate heightened right cerebral reactivity among high- relative to low-hostiles.

This line of reasoning will be expanded and alternative dependent variables to assess brain laterality will be discussed. To conclude, an experiment that may help determine the anatomical regions which may mediate heightened physiological arousal among high-hostiles will be proposed.

Several lines of research have been pursued in the convergent literatures on the expressive (or behavioral) and physiological dimensions of hostility. In recent reviews, heightened physiological reactivity to a stressor has been a reliable and robust positive correlate of hostility level (e.g. Demaree & Harrison, 1996a; Hardy & Smith, 1988; Herridge, Harrison, & Demaree, 1997; Suarez & Williams, 1989; Weidner, Friend, Ficarotto, & Mendell, 1989). While these correlates are well described, much work remains to be completed in the transition from a previously behavioral or cognitive model of hostility into a neuropsychological model of emotion and emotional lability. A basic foundation for the integration and extension of this research may exist within the framework of the study of rage (an aggressive attack accompanied by autonomic arousal) and pseudobulbar palsy (marked by spontaneous extreme affective display, like crying) (e.g. Demakis, Herridge & Harrison, 1994), both examples of negative affective lability.

This review will identify major theoretical perspectives of negative affective lability. After identifying specific limitations to primarily cognitive models of hostility, a neuropsychological approach will be proposed. First, the role

of the right cerebrum in emotional processing will be elucidated. Second, neuroanatomical systems especially within the right cerebrum purported to mediate rage behavior and pseudobulbar palsy will be identified. Third, negative emotional processing, with an emphasis on hostility, will be seen primarily as a function of right cerebral arousal. Last, clinical and experimental research will be reviewed which may help identify behavioral and physiological correlates of a dynamic functional system, as linked to hostility. Last, procedures to help determine the anatomical regions possibly linked to heightened physiological reactivity to stress among high- relative to low-hostiles will be described.

Cognitive Models

Researchers have proposed primarily cognitive models to explain affective arousal and hostility. Krantz et al. (1986) discussed a model involving three primary factors -- stimulus characteristics, mediators, and physiological reactivity. Stimulus characteristics, such as facial valences of anger and fear expressed by others, produce a necessity for response. A complex interaction between cognition and biological predisposition occurs in the mediator (or organism), thereby producing a reaction to threatening stimuli. Different cognitions include fearful and/or angry thoughts while biological predisposition incorporates individual variance of hormonal (i.e., testosterone levels) or central nervous system variables (i.e. regional brain

arousal levels) activity. One effect of this interaction is physiological reactivity. Krantz et al. (1986) successfully accomplished their goal: To effectively incorporate cognitive components into a physiological model of arousal. However, their limited description of biological traits left this model somewhat lacking. Similar to Krantz et al. (1986), Williams (1986) proposed a biopsychosocial model that suggested that the brain is developed from an interaction between genes, personality, and the environment. The brain, then, sends messages to target organs to produce arousal when the environment or one's emotional state warrants it. Williams (1986), like Krantz et al. (1986), made theoretical advances when he cited the brain as a primary mediator of arousal. However, while these models (Krantz et al., 1986; Williams, 1986) are clearly interesting and deserving of empirical research, they primarily describe the brain as an "input-output" organ. Thus a greater understanding of the biological and brain processes related to physiological arousal may effectively complement these fascinating theories.

Contrada (1994) states that three cognitive processes -- primary appraisal, secondary appraisal, and coping -- impact hostility level and physiological reactivity to a stressor. *Problem representation* (which includes both primary and secondary appraisal processes) is the cognitive construction of the stressful interaction. This internal representation includes features of the stressor, potential harm of the

stressor, subjective and somatic reactions to the stressor (all subsumed under primary appraisal), and alternative responses to the stressor (a form of secondary appraisal). *Response generation* includes automatic and nonautomatic coping responses designed to counteract the stressful interaction, as represented by the individual. These coping strategies permit for continuous reassessment of the problem representation, perhaps leading to a modification of cognitions, behaviors, and response alternatives. Interestingly, and most important to this paper, Contrada (1994) states that problem presentation and response generation cannot be directly observed, but may be inferred from cardiovascular and neuroendocrine responses. Thus, greater physiological arousal among high-hostiles reflects a more negative representation of the stressful stimuli, requiring greater coping (Glass & Contrada, 1984). While this model is, again, interesting, it includes several brain-related phenomena -- including perceptions, cognitions, and cardiovascular reactivity. Thus, this model may benefit from a greater understanding of the brain's function within the context of this model.

Dominant right cerebral role in emotion

Viewing emotional processing from a neuropsychological perspective, the right cerebrum appears dominant in the perception and expression of emotion, and negative emotion in particular. Research of facial affect and vocal prosody

supports this notion. Identification of the dynamic right cerebral mechanisms integral to emotional processing highlights its possible role in negative emotional expression, including hostility.

Right cerebrum and emotional perception

Previous research suggests that the right cerebrum is dominant in the perception of visually-presented emotional stimuli. For example, Ley and Bryden (1979) tachistoscopically presented five extremely negative and five extremely positive emotional adult faces to "normal," intact right-handed undergraduate men without a history of head trauma. They found a significant left visual field (LVF; right cerebrum) advantage for emotional valence recognition. Subjects were both quicker and more accurate when identifying facial expressions presented to the left- relative to the right-visual field (RVF; left cerebrum). Similarly, Strauss and Moscovitch (1981) tachistoscopically presented a pair of emotional faces to the left- and to the right-visual fields of forty men and forty women undergraduates. Participants were significantly better at determining whether the facial expressions were the same (both positive or negative) or different (one positive, one negative) when presented to the LVF. In addition, participants were faster and more accurate in their determination of facial valence, positive or negative, in the LVF.

Some research, however, suggests that the right cerebrum is especially important in the mediation of *negative* affect perception. Mandel (1991) studied 10 right hemisphere damaged (RHD), 10 left hemisphere-damaged (LHD), and 10 normal controls (CTL). CTLs were significantly more accurate when labeling the emotional expression of each face relative to the LHD group. LHDs, in turn, were significantly better at this task in comparison to RHDs. Interestingly, a significant group by valence effect was noted. Specifically, compared to RHDs, LHDs had a greater advantage in the identification of negative relative to positive emotional valences. Hartley et al. (1991) found that tachistoscopically-presented neutral faces were processed faster in the left cerebrum while negative faces were processed more quickly in the right cerebrum. Last, Crews and Harrison (1994) studied 12 depressed and 12 non-depressed right-handed undergraduate men and women. Using tachistoscopic methods to present negative, positive, and neutral faces, they found that normal controls showed symmetry in the identification of positive valences. However, a significant right cerebral advantage in the processing of negative emotional faces was found among normals, irrespective of gender. Taken together, this research suggests relative right cerebral dominance for the perception of emotionally negative visual stimuli.

The perception of prosody may also be mediated by the right cerebrum. Prosody refers to the melody, pauses, stresses, and accents applied to verbal expression, and

frequently communicates the tone, or mood, of the speaker (Bunce & Harrison, 1991). "Since prosody adds such complexity and richness to language, one must consider it a very crucial part of communication" (Ross, 1985). Much research suggests that right temporal-parietal area lesions significantly reduce the ability to comprehend emotional prosody or to repeat emotional statements (Gorelick & Ross, 1987; Heilman et al., 1975; Ross, 1981; Tucker et al., 1977). For example, Starkstein et al. (1994) examined 59 patients with acute stroke lesions localized to either the right- or left-cerebrum. Patients with an inability to accurately perceive prosody (emotional comprehension aprosodia), identified using two standard batteries of auditorally-presented affective words and sentences, were significantly more likely to suffer from right- relative to left-cerebral infarct localized by CT scan. Indeed, research of both normal and brain-damaged patients robustly suggest that the right cerebrum is dominant in processing many individual prosodic aspects -- inflection, stress, pitch, intensity, timbre, cadence, frequency, amplitude, melody, duration, intonation, and emotional tone (Blumstein & Cooper, 1974; Bowers et al., 1987; Carmon & Nachshon, 1973; Heilman et al., 1975; Ley & Bryden, 1979; Mahoney & Sainsbury, 1987; Ross, 1981; Safer & Leventhal, 1977; Shapiro & Danley, 1985; Tucker et al., 1977). Because of its ability to process these vocal components, the right cerebrum can detect not only what a person feels about what they are saying, but why and in what context they are saying

it. Perhaps Joseph (1988) captures this advantage best, noting that only the right cerebrum can interpret the sentence, "Do you want to go outside?" as indicative of impending fun or altercation.

Some research, however, suggests that the right cerebrum is especially prepared in the perceptual processing of *negative* prosody. For example, Carmon and Nachshon (1973) dichotically presented nine different sounds to the left- (right cerebrum) and right-ear (left cerebrum) -- crying, laughing, and shrieking of an adult male, adult female, and a child -- to 25 undergraduate men. While they determined that there was a significant left-ear advantage for all emotional stimuli, the greatest lateralized advantage was for crying. Relatively little research exists in this area, however, which is ripe for further investigation.

Right cerebrum and emotional expression

The motor expression of affect appears central to right cerebral functioning, further supporting its role in emotional processing. Evidence was first introduced by Campbell (1978), who discovered that smiles were broader on the left side of the face, a region predominantly innervated by the right cerebrum. Borod and Caron (1980) replicated this finding by showing that both positive and negative posed facial expressions were more intense on the left hemiface, with no significant differences found between the participants' gender (26 men, 25 women) or handedness (31

right-handed and twenty left-handed). Borod et al. (1986), in their study of 27 brain-damaged individuals (12 right-brain-damaged and 15 left-brain-damaged), further found that left cerebrum lesions reduced the use of literal speech, whereas patients with right cerebrum dysfunction evidenced poorer facial affect perception and expression. Indeed, five empirical studies (Borod, Koff, Lorch, & Nicholas, 1985; Borod, Koff, Lorch, Nicholas, & Welkowitz, 1988; Blonder et al., 1993; Buck & Duffy, 1980; Ross & Mesulam, 1979), using a total of 31 right-brain-damaged, 30 left-brain-damaged, and 34 normal controls (all but one were men), independently found that right-brain-damaged patients were significantly less emotionally expressive in the face than both normal controls and left-brain-damaged participants.

The right cerebrum may, however, be particularly dominant for facial expression of negative affect. Borod, Koff, and Buck (1986) discovered that posed negative facial expressions were rated as more intense on the left hemi-face than were positive expressions. Further, Davidson et al. (1990) used quantitative electroencephalography (QEEG) recordings to conclude that spontaneous expressions of disgust and happiness correlated with heightened right- and left- fronto-temporal activity, respectively. This suggests that the right cerebrum may be dominant in negative affect expression whereas the left cerebrum may largely control positive affect expression.

The right cerebrum may additionally mediate prosodic expression. While the vast majority of researchers interested in verbal expression have investigated the role of the left cerebrum, Larsen et al. (1978) accidentally found that verbalization is largely mediated by the right cerebrum. These investigators, using direct unilateral intracarotid injection of radioactive ¹³³-xenon and its subsequent washout to determine the focal cerebral blood flow, found homologous bloodflow increases in both hemispheres among right-handed men and women instructed to count from 1 to 20. Not understanding the role of the right cerebrum in speech production, these researchers concluded that the right cerebral contribution to verbalization appears organized in a parallel fashion to propositional language within the left hemisphere (Larsen et al., 1978). If they had taken into account the research by Tucker et al. (1977), they may have suggested that the right cerebrum mediates prosodic expression. Tucker et al. (1977) examined a group of men and women comprised of 11 patients with right-parietal disease, 7 patients with left cerebral dysfunction, and 8 patients without intracranial disease. These investigators found that right-cerebrum damaged patients, relative to other patients, were significantly worse at inserting affective intonation into neutral statements either by request or imitation. Isotope scans revealed that these lesions were localized in the superior-posterior temporal and inferior parietal lobes within the right cerebrum. Ross and Mesulam (1979) followed

up this research by studying two individuals (one man, one woman) suffering ischemic infarctions in the right anterior-inferior parietal region verified by CT. Both participants indicated social difficulties from a relative inability to modulate their speech, affectively. One subject, a school teacher, reported monotonically, "I cannot put any emotion into my voice or actions, and the pupils do not know when I am angry and mean business."

Perhaps the best study investigating the right cerebral role in prosodic expression was performed by Ross (1981). This research was designed to test two hypotheses: Prosodic expression is 1) mediated by the right hemisphere and, 2) specifically mediated by regions adjacent to the right Sylvian fissure, tissue homologous to the left cerebral areas responsible for propositional speech expression. He studied ten consecutive medical center intakes who were diagnosed with right cerebral infarct using CT. Without previous knowledge of their lesion foci, he assessed the patients for their 1) spontaneous use of affective prosody, and 2) ability to repeat linguistically neutral statements with affective prosody. His hypotheses were supported: Patients with right cerebral damage localized near the right Sylvian fissure were significantly worse at the aforementioned tasks than those with right cerebral lesions located rostrally or caudally. Thus, it appears that prosodic expression is mediated by the right hemisphere, and near the Sylvian fissure in particular.

No known research exists which examines the role of the right hemisphere on negative prosodic expression. An area prime for exploration, it may be hypothesized that the right cerebrum plays a particularly dominant role for *negative* prosodic expression. Research suggesting that the right brain is particularly important to negative affective processing -- auditory and visual perception, as well as negative emotional expression -- may support this notion.

Although a relative dearth of investigation exists in this area too, some research suggests that affective gestures are largely controlled by the right cerebrum. Emotional gestures, while largely under-acknowledged, are important facets that enrich interpersonal communication. Ross and Mesulam (1979), in their aforementioned study, found that right frontal opercula lesions corresponded with a complete loss of spontaneous gestures. Similarly, Ross (1981) noted that right frontal lesions were associated with decreased use of affective gesture.

Right cerebrum and hostility

Given the above research implicating the right cerebrum as an important processor of emotion, and negative emotion in particular, it is not surprising that the right hemisphere has been linked to hostility expression. Lesion studies have been important contributors to our understanding of hostility. Several anatomical systems have been identified in which dysfunction at specific loci have been correlated with

emotional lability, including rage and/or pseudobulbar palsy (uncontrollable, extreme outbursts of emotion, including anger and crying). First, bilateral lesions of the bulbar motor nuclei, the neocortical upper motor neurons in particular, have produced pseudobulbar affect (Lieberman & Benson, 1977). Second, lesion of the diencephalon, basal forebrain, medial temporal lobe, or the brainstem tegmentum have yielded emotional lability (Brown, 1967; Contu & Drew, 1966; Sackheim et al., 1982). Ross (1993) also theorized that pathological affect may result from a combination of major depression and a right frontal opercula lesion, as supported by two case studies (Ross and Rush [1981], cases 1 and 3).

The hypothalamus also was discovered as an important mediator to rage production (Moyer, 1987; Panksepp, 1982). Ablation of sites in the hypothalamus result in sham rage, suggesting that pathways from the right frontal lobe to the hypothalamus may inhibit emotional lability. Previous experimental lesion studies with cats (Decsi & Nagy, 1974; Lu, Shaikh, & Siegal, 1992; Nagy & Decsi, 1974) and case studies with humans (Flynn, Cummings, & Tomiyasu, 1988; Sachdev, Smith, Matheson, & Last, 1992; Tonkonogy & Geller, 1992) support the hypothalamic role on rage and pseudobulbar palsy. According to this line of research, the right cerebrum appears central to hostility.

One neuropsychological explanation of hypothalamic rage suggests that the anterior right cerebrum may be responsible for the inhibition or regulation of autonomic functioning as

well as the expression of hostility. Activation of the frontal lobe, and orbital-frontal cortex in particular, appears to decrease hostility levels (Butter, 1970). The orbital-frontal cortex has extensive interconnections with the amygdala of the anterior temporal region via the uncinate tract, an inhibitory pathway (see Kandel et al., 1991). The amygdala has frequently been described as responsible for heightened hostility. Indeed, stimulation of the amygdala has produced a rage-like response (Ursin, 1960) whereas amygdala lesion has produced placid animals (Woods, 1956). Heilman et al. (1993) hypothesized that these two extensively interconnected regions interact with each other, via the uncinate tract, to yield a relatively conservative and stable aggression level among normals. Thus, ablation of right orbitofrontal regions or stimulation of right anteriomedial temporal regions may yield increased hostility. Passivity or flattened affective expression may result from stimulation of right orbitofrontal regions or ablation of right anteriomedial temporal regions (see Kalat, 1992). In support of this theory, we have found decreased orbital-frontal and increased right temporal beta activity in homicidal patients using QEEG (Demaree & Harrison, 1996b; Everhart, Demaree, & Harrison, 1996).

Right cerebrum and physiological regulation/arousal

While there exists a relative dearth of literature regarding physiological regulation, some evidence suggests

that physiological reactivity is mediated by the right cerebrum. Wittling (1990) experimentally showed 25 men and 25 women subjects an emotionally positive film independently to either the RVF and LVF. For all participants, right cerebrum (LVF) presentation of the film resulted in significantly higher blood pressure beyond that which followed presentation to the left cerebrum. However, this effect was significantly greater among men relative to women. While the authors described these data as evidence of right hemisphere dominance of physiological regulation, results may also be explained by the emotional perception literature. That is, the film's emotional context may have been better perceived by the right brain, thereby inducing greater BP reactivity. Herridge, Harrison, and Demaree (1997), using fifty right-handed male participants, found that subjects experienced greater Galvanic skin response (GSR) on the left hemibody, primarily innervated by the right hemisphere, when making posed emotional faces. Again suggestive of right cerebral control of physiology, one must likewise be cautious of inferring too much from this study. Greater left hemibody GSR reactivity may result from heightened right hemisphere arousal when processing emotional expression. Although very intriguing, both of these studies may confound physiological arousal with emotional perception or expression. From these studies, it may be premature to conclude that the right brain is dominant in physiological regulation.

Research on arousal, independent of emotion, may promote our understanding of physiological asymmetry. One such study, using sixty right-handed male undergraduates, was performed by Demaree and Harrison (1996a). Using a dichotic listening paradigm to assess cerebral asymmetry, these researchers found a significant relationship between right-cerebral arousal and heart rate (HR) reactivity to the cold-pressor test. Yokoyama et al. (1987) obtained similar results using an attention-demanding task as a stressor, HR as the indicator of arousal, and dichotic listening procedures for laterality assessment. Other research found that individuals with right- relative to left-temporoparietal lesions had reduced GSR response to startling stimuli independent of emotional content (Heilman et al., 1978; Schrandt et al., 1989). From their data, Heilman et al. (1978) posited that the right cerebrum plays a dominant role in physiological regulation. While these findings do not infer causality, they suggest that the right hemisphere may play a dominant role in physiological arousal. As suggested by the researchers, other deficits from right cerebral lesion may indicate hypoarousal. For example, several investigators found reductions in reaction time and diminished attentional functioning from right hemisphere infarct (DeRenzi & Faglioni, 1965; Heilman & Van Den Abell, 1979; Howes & Boller, 1975; Joseph, 1986; Weinstein, 1978).

Given the above research, it is not surprising that the right cerebrum has been found an important contributor to HR

elevation. Interestingly, however, decreased HR has corresponded with arousal of the left cerebrum, which has been theorized to suppress right hemisphere arousal and its subsequent elevating effect on HR (Zamrini et al., 1990). For example, using right-handed men and women, Hugdahl and colleagues (1983) presented visual stimuli to the right hemisphere (RH) to half of their participants and to the left hemisphere to the other half. They found an anticipatory HR acceleration in the RH group and an anticipatory deceleration in the LH group three to five seconds before stimulus presentation. Similarly, assessing male and female epilepsy patients subsequent to intracarotid barbiturate injection (WADA), Zamrini and colleagues (1989) found that injection to the left- and right-intracarotid artery corresponded with increased and decreased HR, respectively. Lane and colleagues (1989) used WADA testing on subjects with temporal lobe epilepsy. Like Zamrini et al. (1989), injection to the left intracarotid artery corresponded with increased HR. However, this effect was significant only among patients with focal left-, but not right-, temporal lobe epilepsy. These researchers concluded that both sodium amytal and a left temporal epileptic focus may have similar suppressing effects on the right hemisphere and HR. Similarly, Oppenheimer and colleagues (1992) found that, during surgery for epilepsy, stimulation of the left- and right-insular region was associated with decreased and increased HR, respectively.

The specific cerebral regions most important to autonomic regulation are the insula, medial prefrontal cortex, cingulate gyrus, and temporal poles (Cechetti & Saper, 1990; Mesulam, 1985). These anatomic regions appear central to HR regulation because of their direct projections to subcortical structures (including the hypothalamus, nucleus tractus solitarius, dorsal vagal nucleus, and nucleus ambiguus), as well as neurophysiologic studies showing HR changes with their stimulation or ablation (Lane & Jennings, 1995).

In accordance with the above research, two of the prominent neuropsychological explanations of heightened physiological arousal involve the right cerebrum. Tucker and Williamson (1984) proposed that the right cerebrum, which is moderated by the left cerebrum, may mediate physiological activity. Heilman (1982), to the contrary, speculated that the right cerebrum may have greater control of the subcortical systems, which largely regulate arousal.

Most consistent with Tucker and Williamson's (1984) theory, the right insular region may be especially important to autonomic regulation. Stimulation of this area produces autonomic and visceral changes consistent with sympathetic nervous system arousal (see Heilman, Bowers, & Valenstein, 1993). Insular impulses travel through the hypothalamus, ventrolateral pons, and medulla to the spinal cord. There, the sympathetic nervous system chain is completed through

relatively short, preganglionic, efferent cholinergic neurons producing widespread autonomic effects.

Hostility and its correlates

A basic understanding of the differing roles of the two hemispheres enables one to make specific predictions about the effects of heightened right cerebral arousal on behavior, emotional expression, and physiological reactivity to stress. Much of the relevant literature is clinical in nature although some experimental evidence is provided in the area of emotional perception.

Hostility has been theorized to be central to right cerebrum activity (see Kolb & Wishaw, 1990). Indeed, examination of lateralized neuropsychological impairments yielded a better than 84% correct identification rate of 25 aggressive psychopaths, 25 depressive patients, and 25 normal controls under double blind conditions (Yeudall, 1977). Criminal psychopaths tended to have left-cerebrum damage purportedly leading to a predominantly right-cerebrum mediation of affect.

Continued advances in neuropsychology have refined our knowledge of psychopaths. Krynicki (1978) compared assaultive and nonassaultive male adolescent delinquents using neuropsychological and EEG tests. He found that the former group had a heightened number of abnormal EEG recordings, particularly in the left fronto-temporal regions. Other research suggests that psychopathic men show greater motor

impulsiveness, poorer verbal memory, attention and concentration difficulties, poorer abstract thinking, and temporal sequencing problems (Berman and Siegal, 1976), indicative of frontal lobe dysfunction mainly attributable to the right hemisphere. Tucker and Frederick (1989) logically speculated that high-hostile behavior resulted from the dominant expression of the undamaged hemisphere.

Because the right cerebrum may be associated with both negative emotion and physiological lability, it is not surprising that hostility has been associated with increased physiological reactivity to stress. Suarez and Williams (1989) found that high hostile men experienced greater reactivity as measured by HR, BP, and blood flow during an anagram task accompanied by harassment than did low hostile men. Smith, Houston and Stucky (1984) found that high- and low-hostile men with low irritability differed in their physiological response, as measured by BP, to a challenging mental task. High hostile participants showed significantly increased reactivity to this cognitive stressor.

Schneider, Julius and Karunas (1989) echoed this finding by assessing high- and low-hostile men. High hostile men showed heightened HR and BP reactivity to a cognitive stressor test in comparison to low hostile men. Siegman et al. (1992) found a positive correlation between hostility level and BP reactivity among 41 undergraduate men during a serial subtraction task. This provided further evidence relating hostility and increased cardiovascular reactivity

during stress. Using an unsolvable anagram task with 51 undergraduate men and 53 undergraduate women, Weidner et al. (1989) found that high hostile men and women displayed greater SBP reactivity relative to their low-hostile counterparts. In research with greater external validity, Jamner et al. (1991) studied 33 paramedic men during a 24-hour period. Both defensiveness and cynical hostility were found to contribute to cardiovascular reactivity, as indicated by HR and BP, during occupational stress. These have been particularly robust findings, as supported by meta-analysis of the hostility-physiological reactivity relationship (Suls and Wan, 1993).

Hostility also has been associated with increased persistence (decreased rate of return to baseline measures; Kalat, 1992) on physiological measures after stress. We found perseveration on GSR activation after subjects posed angry faces (Herridge, Harrison, & Demaree, 1997). Further, examination of 28 Black men lead Ernst et al. (1990) to conclude that manifest hostility is negatively associated with recovery of cardiovascular baseline after stress, produced by repeated mental demands.

Role of stress

The cold pressor test has historically been used to induce stress and purportedly to produce a global increase in cerebral activity. One indicator of this heightened arousal

was increased physiological activity from cold pressor administration (see Kalat, 1990).

High hostiles tended to exhibit greater physiological arousal to the cold pressor test relative to low hostiles. For example, Dembroski, MacDougall, Herd, and Shields (1979) found that forty high-hostile undergraduate men showed significantly greater HR and BP reactivity to the cold pressor than their forty low-hostile counterparts. Glass, McKinney, Hofschire, and Fedorko (1990), using 22 men as participants, echoed these results by finding a positive association between hostility level and physiological reactivity (as indicated by HR, BP, total systemic resistance, and stroke volume index) to the cold pressor stress.

Stress clearly has differing effects on high- and low-hostile persons. However, because physiological measures may tap right hemisphere activity, it remains uncertain whether the effect of stress on hostile individuals is best accounted for by greater reactivity by the entire brain or solely the right hemisphere.

EEG as a dependent measure of hostility

EEG measures brain electrical activity from electrodes which are placed on the scalp, usually conforming to the International 10-20 System of electrode placement (Talbot, Hales, & Yudofsky, 1988). The electrical activity detected by these electrodes is presumed to originate primarily in the

cortical cell layers beneath each individual electrode. EEG data are usually analyzed within bandwidths corresponding to delta (less than 4 Hz), theta (4 to 8 Hz), alpha (8 to 13 Hz), and beta (13 to 25 Hz). Cortical arousal is thought to correspond to desynchronization of the record or with increased magnitude of the higher frequency bandwidths (Talbot, Hales, & Yudofsky, 1988). EEG data may be analyzed by identifying 1) dysrhythmias, such as isolated spikes or bursts of slow activity, 2) suppression of EEG amplitude, 3) EEG slowing, and most important to this research, 4) asymmetries of EEG data from comparable scalp locations.

Electrophysiological arousal, which may be measured using EEG technologies, has been shown to correlate modestly with behavioral and with physiological arousal indices. This may help researchers better understand the relationship between behavior and physiological function (Isaac, 1960). Research has robustly demonstrated that cortical arousal emanates from brain stem mechanisms, and the reticular activating system (RAS) in particular. Morruzi and Magoun (1949) were the first to show this relationship. They found that electrical stimulation of the RAS produced widespread electrophysiological activation in the brain. Arousal of the RAS via altered peripheral stimulation (Starzl, Taylor, & Magoun, 1951) and pharmacological agents (Bradley, 1958) were, likewise, found to have diffuse effects on cortical arousal. Behavioral changes corresponding to RAS arousal, and associated brain activity, included disturbed sleep/waking

cycles (Isaac, 1960), decreased reaction time (Isaac, 1960), as well as heightened locomotion (Isaac & Kallman, 1979), attention (Barrett, 1976, 1977), and vigilance (Delay & Issac, 1980).

QEEG techniques have been useful to better understand anatomical brain differences corresponding to different emotions and, more specifically, low- and high-hostiles. Machleidt et al. (1988), using right-handed undergraduate men as participants, examined whether EEG recordings of "sad" subjects could be distinguished from subjects with different emotions. Using a counterbalanced research paradigm, these researchers induced each subject into sad, happy, anxious, and aggressive states. Relative to other states, they found that sadness corresponded with heightened beta power. They concluded that EEG activity may correspond with actual emotional states. Important to the present research, McIntyre et al. (1976) compared 22 persons (both men and women were represented) with either left- or right-temporal lobe epilepsy diagnosed using the EEG record. They found that left lateralized participants showed a more reflective cognitive style whereas right lateralized participants showed more delayed-response deficits. Citing previous associations between impulsivity and aggression, these researchers concluded that heightened right temporal lobe arousal corresponds with hostile behavior.

EEG techniques have also been useful in understanding regional arousal levels associated with emotion in "normals."

In two studies using identical protocols, right-handed men were asked to watch short film clips designed to elicit happiness/amusement and disgust (Davidson et al., 1990; Ekman, Davidson, & Friesen, 1990). These researchers correlated EEG data with the valence of participants' facial expressions, assessed using Ekman and Friesen's (1978) Facial Action Coding System (FACS). These studies revealed greater right anterior activation, in both the frontal and anterior temporal regions, during negatively valenced expression. Interestingly, 100% of the participants showed this effect in the hypothesized direction. Similarly, using male and female infants born less than 72 hours before testing, Fox and Davidson (1986) associated EEG data to facial expressions of interest and disgust produced by feeding the infants sweet or sour solutions, respectively. They concluded that expressions of disgust were significantly correlated with greater right cerebral arousal.

Other EEG research supports the central role of the right cerebrum in hostility. For example, Edlund et al. (1987) described 6 patients (both men and women were represented) with atypical panic attacks involving hostility. While the participants did not exhibit right temporal lobe seizures, EEG recordings revealed right temporal abnormalities. Heath (1982) studied 76 men and women who were chronic psychosis patients and found that heightened right temporal lobe beta activity consistently corresponded with their violent aggressive behavior. Last, supporting a priori

hypotheses, we found reduced right-frontal and increased right-temporal beta activity in two high-hostile men (Demaree & Harrison, 1996b; Everhart, Demaree, & Harrison, 1996). These findings are consistent with the previously described neuropsychological correlates of hostility: reduced orbital-frontal and heightened amygdaloid arousal may increase the probability of overt aggression.

Rationale

As suggested by previous research, persons with high hostility levels should have heightened right cerebrum activity to a stressor relative to low hostiles (Schneider, Julius, & Karunas, 1989; Siegman et al., 1992; Smith, Houston, & Stucky, 1984). High- relative to low-hostiles have shown both heightened physiological and right-cerebral reactivity to stress, including the cold pressor test (Demaree & Harrison, 1996a). A direct extension of Demaree and Harrison's (1996a) research, this study will attempt to localize the aroused regions, rather than just the cerebrum, associated with heightened physiological reactivity among high-hostiles. Specifically, differences in arousal of the left- and right- temporal and frontal regions between low- and high-hostiles will be assessed. Similar to Demaree and Harrison's (1996a) dichotic listening results, one may expect heightened left- and right-cerebral arousal in the low- and high-hostile participants, respectively, following a stressor. These shifts in arousal may account for both hostility level (Heilman, Bowers, & Valenstein, 1993) and heightened physiological arousal among high-hostiles (Zamrini et al., 1990). This research, however, will investigate the validity of two prominent neuropsychological models of hostility. EEG data may help assess whether heightened hostility is associated with heightened right- relative to left-cerebral (Tucker & Williamson, 1984) or heightened

right-frontal relative to right-temporal arousal (Heilman, Bowers, & Valenstein, 1993).

Variables

The classifying variable will be self-reported hostility level (high or low).

Two classes of dependent variables will be used in this experiment. First, physiological measures will be SBP, DBP, and HR. Second, beta magnitude (13 to 25 Hz) within the right- and left-temporal lobes (from the T4 and T3 regions, respectively) and the right- and left-frontal poles (from the F8 and F7 regions, respectively) will be assessed using EEG.

Hypotheses

Please note that the Fp1 and Fp2 electrode sites originally used in these hypotheses have been changed to the F7 and F8 electrode sites, respectively. This decision logically follows from two important concerns. First, the prefrontal regions are located more proximal to the F7 and F8 electrode sites relative to the Fp1 and Fp2 electrodes. Second, the Fp1 and Fp2 electrodes record a greater amount of EMG and eye movement artifact relative to the F7 and F8 electrodes.

Hypothesis 1: High-hostile participants will show greater cardiovascular (SBP, DBP, HR) reactivity to stress relative to low-hostile subjects.

Hypothesis 2: Relative to low-hostiles, high hostile subjects will show a relatively greater increase of right-temporal (T4) beta magnitude to stress.

Hypothesis 3: Relative to low-hostiles, high-hostile subjects will show a relatively reduced increase in right-frontal (F8) beta magnitude to stress.

Hypothesis 4: Relative to low-hostiles, high-hostile subjects will show a relatively reduced increase of left-frontal (F7) beta magnitude to stress.

Hypothesis 5: Relative to low-hostiles, high hostile subjects will show a relatively reduced increase of left-temporal (T3) beta magnitude to stress.

Planned Comparisons

Comparison 1: The two groups will be compared by assessing anterior (Fp1, Fp2, F7, F3, F4, F8) and posterior (T5, P3, P4, T6, O1, O2) beta magnitude.

Comparison 2: The two groups will be compared by assessing left- (Fp1, F7, F3, T5, P3, O1) and right- (Fp2, F4, F8, P4, T6, O2) cerebral beta magnitude.

Method

Subjects. Participants included 143 right-handed men acquired from the undergraduate psychology pool. Participants self-reported no history of hearing aids, hearing problems (e.g. tubes in the ears or infections), major illness, head injury, or Reynaud's Syndrome. Only males were used because it is essential to ensure as much homogeneity as possible within the experiment to draw conclusions based solely on independent variable differences (Alba et al., 1985; Barneoud et al., 1987; Mayberg, Moran, & Robinson, 1990; Renoux et al., 1983). All participants received course credit for their involvement in this research. All identifying materials collected from participants were destroyed after data collection.

Participants must not have had a remarkable medical history to be eligible for inclusion in the experiment. Participants with sufficient hemibody preference based on the Coren, Porac, & Duncan laterality test, who additionally received the lowest or highest 15 scores on the Cook-Medley test, were assigned to low- and high-hostility level groups, respectively.

Self-Report

During group testing, participants were first required to read and sign an informed-consent form (Appendix A). A questionnaire assessing medical history (Appendix B) was also given. Participants then were administered the Coren, Porac,

and Duncan laterality test (Coren, Porac, & Duncan, 1979) (Appendix C) to determine hemibody preference. This self-report assessed right (-1) and left (+1) hemibody preference based on reported preferred use of either eye, ear, arm, and leg. Scores on the test range from a possible -13 to +13, indicating extreme left and right "handedness," respectively. A score of +5 was required for further participation in the experiment.

Participants were then administered the Cook-Medley Hostility Scale (CMHS) (Cook & Medley, 1954) (Appendix D). The Cook-Medley is the most often used measure of hostility and shows validity as a predictor of medical, psychological, and interpersonal outcomes (Contrada & Jussim, 1992).

One hundred forty three subjects participated in group testing. Among those receiving a score of +5 or greater on the laterality questionnaire and without a remarkable medical history, participants with the lowest or highest 15 scores on the Cook-Medley comprised the low- and high-hostility groups, respectively.

Apparati

The laboratory chamber was comprised of a sound-attenuated room (Controlled Acoustical Environments) containing a reclining chair. Located in this chamber was the EEG, physiological, and cold pressor apparati.

Physiological. SBP, DBP, and HR were assessed using the Norelco Healthcare Electronic Digital Blood Pressure / Pulse

Meter With Cuff. SBP and DBP were assessed using the Korotkoff method. Accuracy of HR was reported to be within 2% or 1 beat per minute while BP was +/- 3 mm Hg of those auscultated (Norelco, 1985).

Cold Pressor. The ice water for the CPT was maintained in a small ice cooler (Gott Corporation, model 1916/2) at 4 degrees Celsius. Water temperature was measured using a standard mercury thermometer (Fisher Scientific, model 14-985E).

EEG. Apparati necessary for the collection of EEG data included a lycra electrode cap (Electro-Cap International, Inc.), elastic straps (Electro-Cap International, Inc.), electrode impedance meter (Lexicor Medical Technology, Model 1089 MKII), body harness (Electro-Cap International, Inc.), electrode gel (Electro-Cap International, Inc.), SKINPREP (D. O. Weaver and Co.), cue tips (Rite Aid, Model No. CO-44149), sponge discs (Electro-Cap International, Inc.), syringe (Becton Dickenson and Co., Model No. W11807), rubbing alcohol (Roses Brand), grease pencil (Dixon, Model 80-Blue), measuring tape, vinyl gloves (Best Touch, Model No. BST 010) and reference electrodes (Electro-Cap International, Inc.). Data were recorded and analyzed using Lexicor Neurosearch-24 technology (Lexicor Medical Technology, 1992). The analogue EEG data were converted online to digital data with a Gateway 486 DX computer for display, storage, and analysis. The amplification factor was 32,000 with a sampling rate of 256 Samples/second.

Procedure

High- and low-hostility participants were invited back for further involvement in the experiment within a 1 month time period. Participants entered the laboratory chamber and were requested to read and sign another informed-consent form (Appendix E).

The experiment consisted of three (3) parts -- Prestress, Stress, and Poststress Phases.

Prestress Phase. Having the participant lean forward slightly while raising his arms, a body harness was placed around the participants chest and fastened in the back using Velcro straps. Then, the distance between theinion (the bony protrubance at the back of the skull) and the nasion (the juncture where the nose meets the forehead) was measured. Each participant's forehead was marked with a grease pencil ten percent of the measured distance above the nasion. The circumference of the participant's head was then measured by passing the measuring tape through the grease mark and inion. If the circumference was greater than 58 cm, a large electrocap was used. Otherwise, a medium electrocap was used. Wearing vinyl gloves, the researcher used an alcohol swab to wipe the participant's earlobes and forehead. Applying a small amount of skinprep to a cue tip, the researcher then lightly abraded the lateral surface of the participant's earlobes. In a firm yet comfortable manner, reference electrodes were placed over the participant's earlobes. After

placing two sponge discs over the two frontmost (Fp1 and Fp2) electrodes on the electrocap, the discs were placed on either side of the forehead grease mark. The participant was asked to hold the sponge discs in place while the researcher pulled the electrocap over the back of his scalp. The electrocap's Velcro straps were attached to the body harness to keep the cap firmly placed. Leads on the electrocap were attached to the reference leads on the electrocap, and the electrocap was plugged into the electroboard. Impedance was 5 kOhm or below. If not, the researcher reapplied the ear electrodes until the desired impedance was obtained. After attaching a blunt needle to the syringe, the syringe was filled with electrode gel. The syringe was then used to fill each of the electrodes in the electrocap. Using the electrode tester, each electrode must have had an impedance of 5 kOhm or less. If the desired impedance was not obtained for any electrode, techniques were used until the desired impedance had been obtained. EEG data was now ready for collection.

First, however, cardiovascular data was obtained. Participants were fitted for BP and HR readings. The blood pressure monitor was strapped to each participant's right upper-arm. The researcher then gave the following instructions: "Please take about two minutes to become accustomed to your surroundings and relax." After receiving the opportunity to establish a resting baseline, HR, SBP, and DBP data were collected twice in succession at the end of the two minute period. To determine the accurate reading, a third

reading was taken if the first two readings differed by 6 beats per minute (HR) or 10 mm Hg for either SBP or DBP.

EEG data were then collected from the participants. Each participant was first asked to relax and lie motionless in a near-supine position on a reclining chair within the sound-attenuated chamber. Specifically, the researcher instructed each participant to do the following:

Please sit back on this chair, in a relaxed and comfortable manner, with your eyes closed. Sometimes when we are drowsy or relaxed our eyes tend to roll somewhat when they are closed. Because this will affect the way your brainwaves look, try not to let this happen. Try to avoid moving your eyes during this procedure.

Using a high-pass filter to eliminate high frequency artifact caused by respiration and small head movement and a sampling rate of 256 samples/second, 120 1-second epochs were then recorded. The EEG record was collected exactly one minute after cardiovascular measures were obtained.

At the end of the two-minute recording period, the participant continued into the Stress Phase.

Stress Phase. Participants then were given the following instructions:

When you are 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. Although this may be difficult, please try your hardest to keep your hand in the water until instructed to take it out. Do you have any questions? O.K., begin.

After forty-five seconds, the subjects were asked to remove their hand from the water.

Poststress Phase. HR, SBP, and DBP arousal was then assessed in accordance with the Prestress Phase. While no preparation was necessary to collect EEG data, these measures were reassessed in the same manner as the Prestress Phase.

After the post-stress electroencephalographic recording, a self-report questionnaire (Appendix F) was administered to each participant. This questionnaire was designed to assess 1) how negative or positive each participant's cognitions were during the experiment, and 2) how reactive each participant thought they were to the cold-pressor stress on a) HR and b) SBP measures.

Participants were thoroughly debriefed and any questions were answered.

Analyses

T-tests were conducted to assess differences between low- and high-hostility participants on descriptive measures -- CMHS, and Coren, Porac, and Duncan's laterality questionnaire.

An independent multivariate analysis of variance (MANOVA) was performed on the cardiovascular variables (HR, SBP, DBP) to address reactivity to the cold pressor stress. This MANOVA used a mixed design with the independent factor of GROUP(2) and with the repeated measure of CONDITION(2).

Electroencephalographic data were also analyzed. In each condition, epochs contaminated by eye-blink and/or movement artifact were eliminated from the analyses. The remaining thirty substantially artifact-free epochs were averaged within each subject and condition.

An ANOVA was performed on the beta magnitude taken at electrode locations F7, F8, T3, and T4. The mixed design ANOVA consisted of the independent factor of GROUP(2) and the repeated measures of LOCATION(4) and CONDITION(2).

For planned comparisons, each subject's data were averaged to obtain left-anterior (Fp1, F7, and F3), left-posterior (T5, P3, and O1), right-anterior (Fp2, F8, and F4), and right-posterior (T6, P4, and O2) beta magnitude during each condition. One independent ANOVA was performed with the independent factor of GROUP(2) and with repeated measures of CONDITION(2), CEREBRUM(2) [left- and right-cerebrum] and LOCATION(2) [anterior and posterior].

Responses to their self-report questionnaire (Appendix F) were also analyzed. T-tests were used to assess differences between low- and high-hostility participants on their self-reported negativity or positivity of thought during the experiment.

To determine whether groups differed on the accuracy of their estimated cardiovascular reactivity to the stressor, t-tests were performed on the variance between participants actual and estimated cardiovascular reactivity. To do this, the following expressions were calculated:

$$\begin{aligned} \text{Var}(\text{HR}) &= (\text{EHRC} - \text{AHRC})^2 \\ &\text{and} \\ \text{Var}(\text{SBP}) &= (\text{ESBPC} - \text{ASBPC})^2 \end{aligned}$$

where $\text{Var}(\text{HR})$ = variance between estimated and actual heart rate change, EHRC = estimated heart rate change, AHRC = actual heart rate change, $\text{Var}(\text{SBP})$ = variance between estimated and actual systolic blood pressure change, ESBPC = estimated systolic blood pressure change, and ASBPC = actual systolic blood pressure change.

Results

Descriptive Measures

To compare low- and high-hostile groups on descriptive measures, t-tests were conducted on scores obtained on the Cook-Medley Hostility Scale, and Coren, Porac, and Duncan's Laterality Questionnaire. Table 1 provides a summary of group means and standard deviations for each measure.

Place Table 1 about here.

High-hostiles scored significantly higher on the CMHS ($M = 33.53$, $SD = 3.03$) than did low-hostiles ($M = 14.53$, $SD = 3.47$), $t(28) = 15.04$, $p < .05$. High-hostiles ($M = 9.80$, $SD = 2.43$) did not differ from low-hostiles ($M = 9.80$, $SD = 2.57$), $t(28) = .00$, $p > .05$ on the Coren, Porac, and Duncan Laterality Questionnaire.

Cardiovascular Measures

Group means and standard deviations on each cardiovascular measure are displayed in Table 2. A multivariate analysis of variance (MANOVA) was performed on the three cardiovascular variables -- SBP, DBP, and HR. The results of the MANOVA and independent ANOVAs are depicted in Table 3. All pairwise comparisons were made using Tukey's Studentized Range Test (Winer, 1971).

Table 1. Summary of Group Means and Standard Deviations for Descriptive Measures

Variable	<u>Low-Hostiles</u>		<u>High-Hostiles</u>	
	Mean	SD	Mean	SD
CMHO Questionnaire	14.53	3.47	33.53	3.03
Laterality Questionnaire	9.80	2.57	9.80	2.43

Place Table 2 about here.

Place Table 3 about here.

A two-factor, mixed design ANOVA with the independent factor of group (low- and high-hostile) and with repeated measure of condition (pre- and post-stress) was conducted to analyze HR (see Table 3). The main effect of group was found to be statistically significant, $F(1,28) = 6.26$, $p < .018$, with high-hostile subjects having a higher overall HR ($M = 68.6$, $SD = 10.12$), across conditions, relative to the low-hostiles ($M = 60.63$, $SD = 7.06$). The main effect of condition was reliable, $F(1,28) = 36.62$, $p < .01$, with participants having greater HR during the poststress condition (see Table 3). Finally, a group by condition interaction was reliable, $F(1,28) = 13.18$, $p < .001$ (see Table 3, Figure 1).

Place Figure 1 about here.

The minimum significant difference for heart rate data points on Figure 1 is 1.64. Thus, within each condition (pre- and post-stress), low- and high-hostiles evidenced significantly different heart rates. Each group also experienced a significant increase in heart rate subsequent to the cold-pressor condition.

Table 2. Summary of Group Means and Standard Deviations for Physiological Measures

Variable	<u>Low-Hostiles</u>				<u>High-Hostiles</u>			
	<u>Pre-Stress</u>		<u>Post-Stress</u>		<u>Pre-Stress</u>		<u>Post-Stress</u>	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Heart Rate	59.6	6.62	61.6	7.49	64.7	9.51	72.4	10.63
Systole(2)	124.1	5.94	128.0	5.98	124.8	8.11	133.3	10.35
Diastole(2)	75.8	7.07	77.8	6.92	76.7	9.19	78.5	8.18

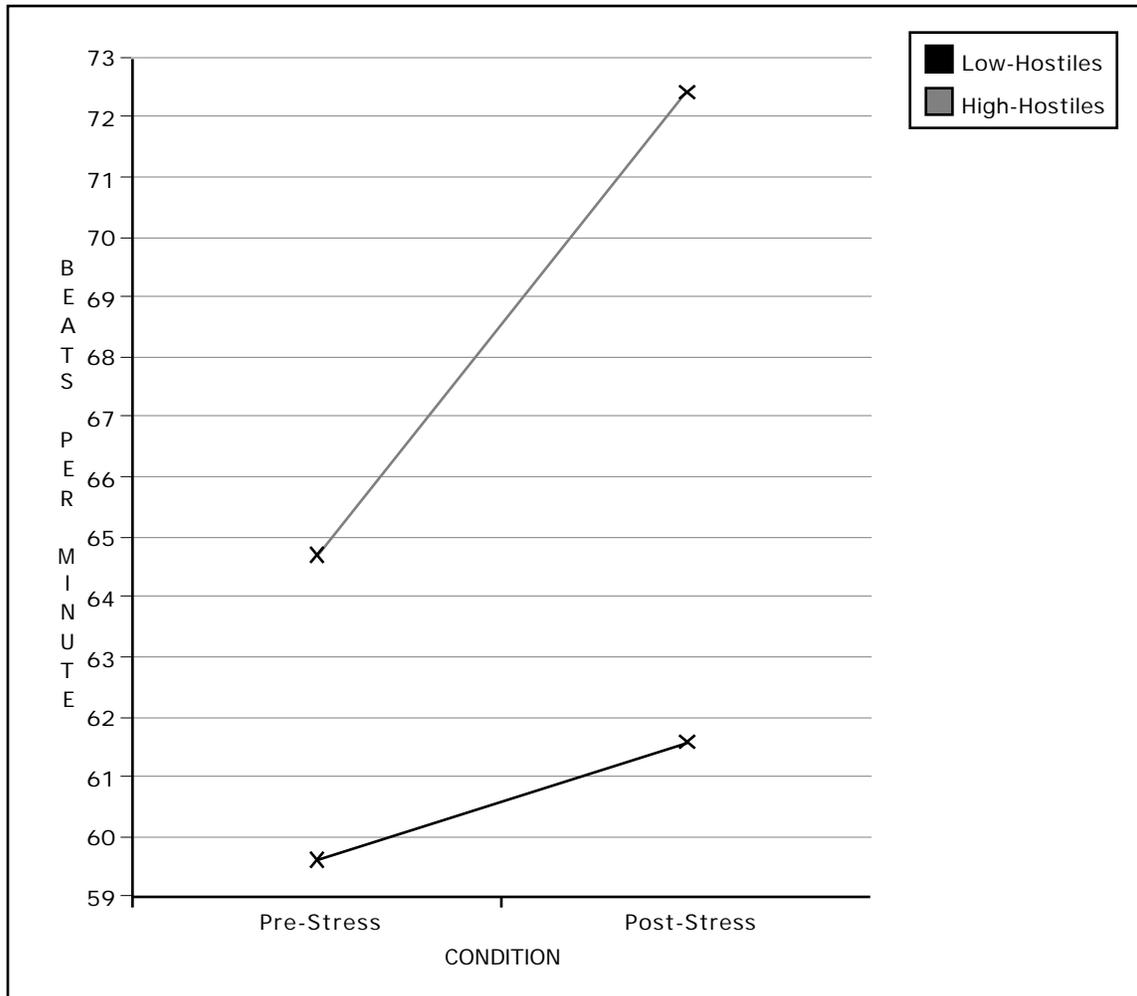
1 = beats per minute

2 = mm / Hg

Table 3. MANOVA Results for Physiological Measures

Source	df	SS	MS	F	p
MANOVA					
Hypothesis of an Overall Group Effect					
Hotelling-Lawley Trace	(3,26)				<.037
Independent ANOVAs					
Heart Rate					
Group	(1,28)	7616.13	7616.13	6.26	<.018
Condition	(1,28)	28.03	28.03	36.62	<.001
Group X Cond.	(1,28)	1009.20	1009.20	13.18	<.001
Systolic Blood Pressure					
Group	(1,28)	1009.20	1009.20	1.08	<.308
Condition	(1,28)	4465.20	4465.20	45.49	<.001
Group X Cond.	(1,28)	598.53	598.53	6.10	<.019
Diastolic Blood Pressure					
Group	(1,28)	76.80	76.80	.08	<.786
Condition	(1,28)	418.13	418.13	10.95	<.002
Group X Cond.	(1,28)	.53	.53	.01	<.906

Figure 1. Interaction Effect of Group by Condition on Heart Rate



For SBP, a two-factor, mixed design ANOVA was performed with the fixed factor of group and the repeated measure of condition. The main effect of condition was reliable, $F(1,28) = 45.49$, $p < .001$. That is, across groups, SBP was significantly higher during the poststress condition than during the prestress condition (see Table 2 and Table 3). In addition, the group by condition interaction was significant, $F(1,28) = 6.10$, $p < .019$ (see Figure 2). The main effect of group did not approach significance (see Table 3).

Place Figure 2 about here.

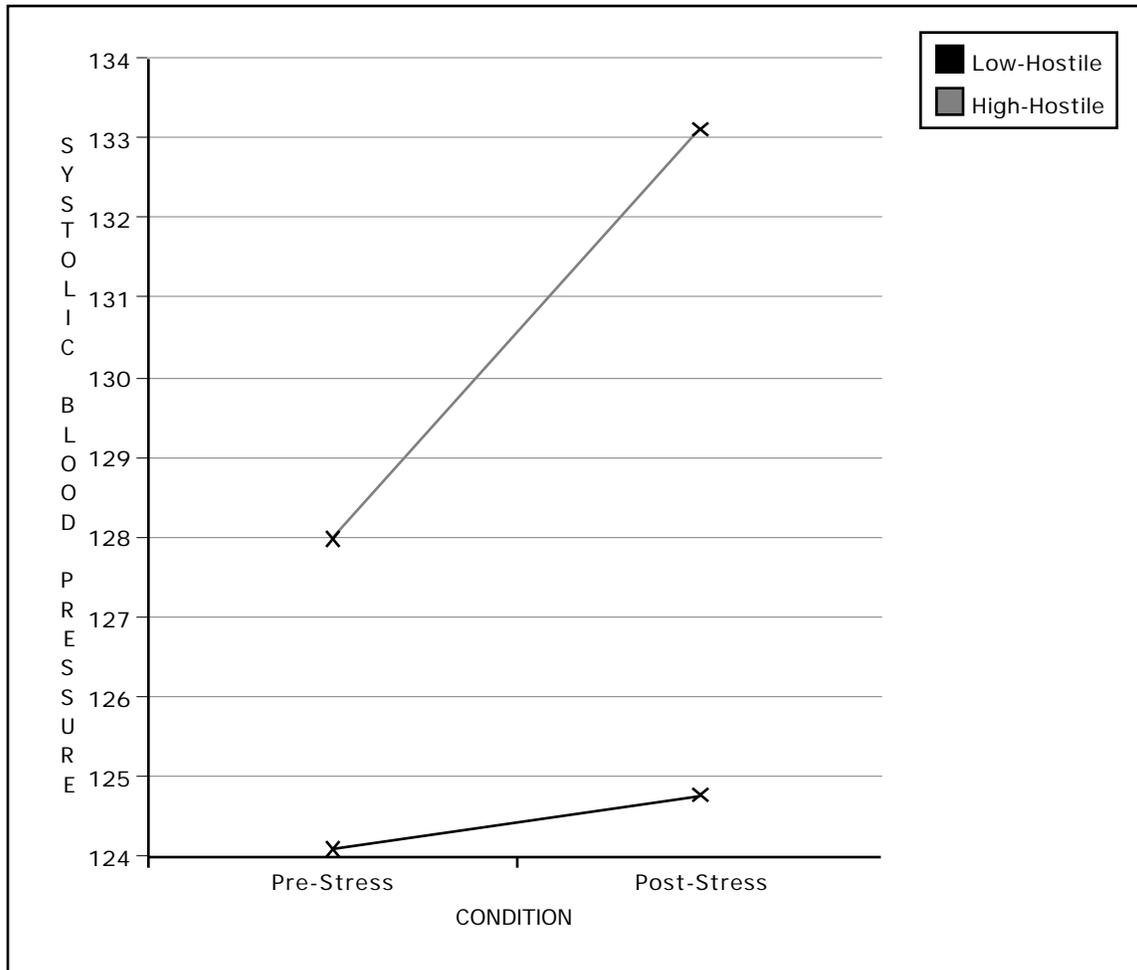
The minimum significant difference for systolic blood pressure data points on Figure 2 is 1.85. Thus, during the post-stress condition only, high-hostiles evidenced a significantly higher SBP relative to low-hostiles. Both groups, however, experienced a significant increase in systolic blood pressure after the cold-pressor stress.

The mixed design ANOVA for DBP with the fixed factor of group and the repeated measure of condition was performed (see Table 3). The main effect of condition was significant, $F(1,28) = 10.95$, $p < .002$, with participants having greater DBP during the poststress condition.

Quantitative Electroencephalographic Measures

Hypotheses two through five were assessed by performing a three factor, mixed-design ANOVA with a fixed factor of

Figure 2. Interaction Effect of Group by Condition on Systolic Blood Pressure



group (low- and high-hostile) and repeated measures of condition (pre- and post-stress) and region (F7, F8, T3, and T4). Means and standard deviations are presented in Table 4 and the ANOVA results are depicted in Table 5.

Place Table 4 about here.

Place Table 5 about here.

The interaction of group and region was significant, $F(3,84) = 2.79$, $p < .045$ (see Table 5, Figure 3).

Place Figure 3 about here.

The minimum significant difference for beta magnitude data points on Figure 3 was .93. Thus, across both conditions (both pre- and post-stress), low- and high-hostiles evidenced significantly different beta magnitudes at the F7 and F8, but not the T3 and T4, electrode sites.

Separate and more refined ANOVAs were performed for each group (low- and high-hostiles) using the same regions (F7, F8, T3, and T4) and conditions (pre- and post-stress). Using data for low-hostiles only, ANOVA results are shown in Table 6.

Place Table 6 about here.

Table 4. Summary of Group Means and Standard Deviations for Electroencephalographic Measures

Variable	<u>Low-Hostiles</u>				<u>High-Hostiles</u>			
	<u>Pre-Stress</u>		<u>Post-Stress</u>		<u>Pre-Stress</u>		<u>Post-Stress</u>	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
F7	10.04	4.29	10.28	3.69	11.84	3.70	11.57	3.41
F8	10.12	4.32	10.42	3.92	11.79	3.54	11.50	3.39
T3	11.39	4.71	11.73	4.48	10.92	3.12	11.93	3.90
T4	11.13	5.33	11.70	4.80	11.67	3.76	11.61	3.69

Table 5. Summary of the ANOVA Results for
Electroencephalographic Measures

Source	df	SS	MS	F	p
Group	(1,28)	33.97	33.97	.30	<.585
Condition	(1,28)	3.10	3.10	.83	<.370
Group X Cond.	(1,28)	1.02	1.02	.27	<.604
Region	(3,84)	19.15	6.38	1.71	<.171
Group X Region	(3,84)	31.24	10.41	2.79	<.045
Region X Cond	(3,84)	4.73	1.57	1.04	<.380
Group X Region X Condition	(3,84)	4.39	1.46	.96	<.413

Figure 3. Interaction Effect of Group by Region on Electroencephalographic Measures

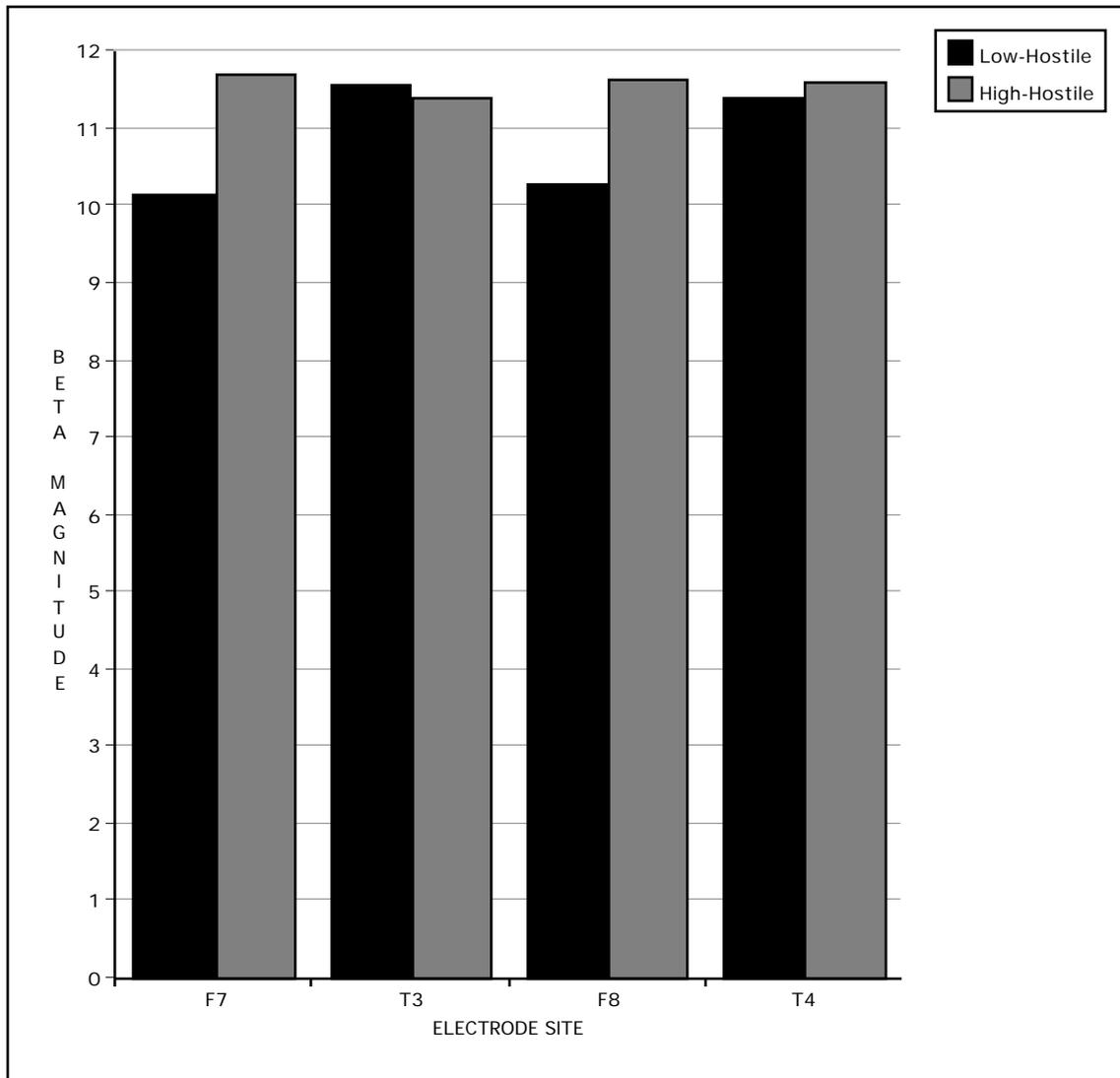


Table 6. Summary of ANOVA Results for
Electroencephalographic Measures Using Low-Hostiles

Source	df	SS	MS	F	p
Condition	(1,28)	3.85	3.85	.05	<.817
Region	(3,84)	49.01	16.33	5.36	<.002
Region X Cond	(3,84)	.47	.15	.05	<.984

For low-hostiles, the main effect of region was statistically significant, $F(3,84) = 5.36, p < .002$ (see Table 6).

Specifically, the beta magnitude recorded at the F7 electrode was significantly different from the beta magnitude recorded at the F8, T3, and T4 electrode sites. The record obtained at F8 differed reliably from that at the F7 and T3 electrodes. The record at T3 differed from that at the F7 and F8 electrodes. Finally, the record at T4 differed from that at the F7 electrode. No other main or interaction effects were significant (see Table 6).

For high-hostiles, ANOVA results are depicted in Table 7.

Place Table 7 about here.

No main or interaction effects were statistically significant using only the high-hostile participants (see Table 7).

Planned Comparisons

To help assess the role of the anterior, posterior, right-cerebral, and left-cerebral brain regions in cardiovascular regulation (planned comparisons 1 and 2), a four-factor, mixed-design ANOVA was performed with the fixed factor of group (low- and high-hostile) and with the repeated measures of condition (pre- and post-stress), cerebrum (left- and right-cerebrum) and location (anterior and posterior).

Table 7. Summary of ANOVA Results for
Electroencephalographic Measures Using High-
Hostiles

Source	df	SS	MS	F	p
Condition	(1,28)	.28	.28	.01	<.937
Region	(3,84)	1.38	.46	.21	<.890
Region X Cond	(3,84)	8.65	2.88	1.31	<.277

Group means and standard deviations are displayed in Table 8 and ANOVA results are displayed in Table 9.

Place Table 8 about here.

Place Table 9 about here.

The main effect of location, $F(1,28) = 17.16, p < .003$ was reliable (see Figure 4). Given the minimum significant difference of .57, beta magnitude at posterior regions were significantly greater relative to anterior regions.

Place Figure 4 about here.

Self-Report

At the completion of the experiment, participants A) rated their thoughts during the experiment (1 = thoughts were extremely negative in nature, 5 = thoughts were neutral, 9 = thoughts were extremely positive in nature), B) estimated how much their HR increased due to the cold-pressor stress, and C) estimated how much their SBP increased due to the cold-pressor stress.

To assess for group differences on self-reported thoughts, a t-test was performed on the participants ratings of their own cognitions. Low- ($M = 6.93, SD = .94$) and high-

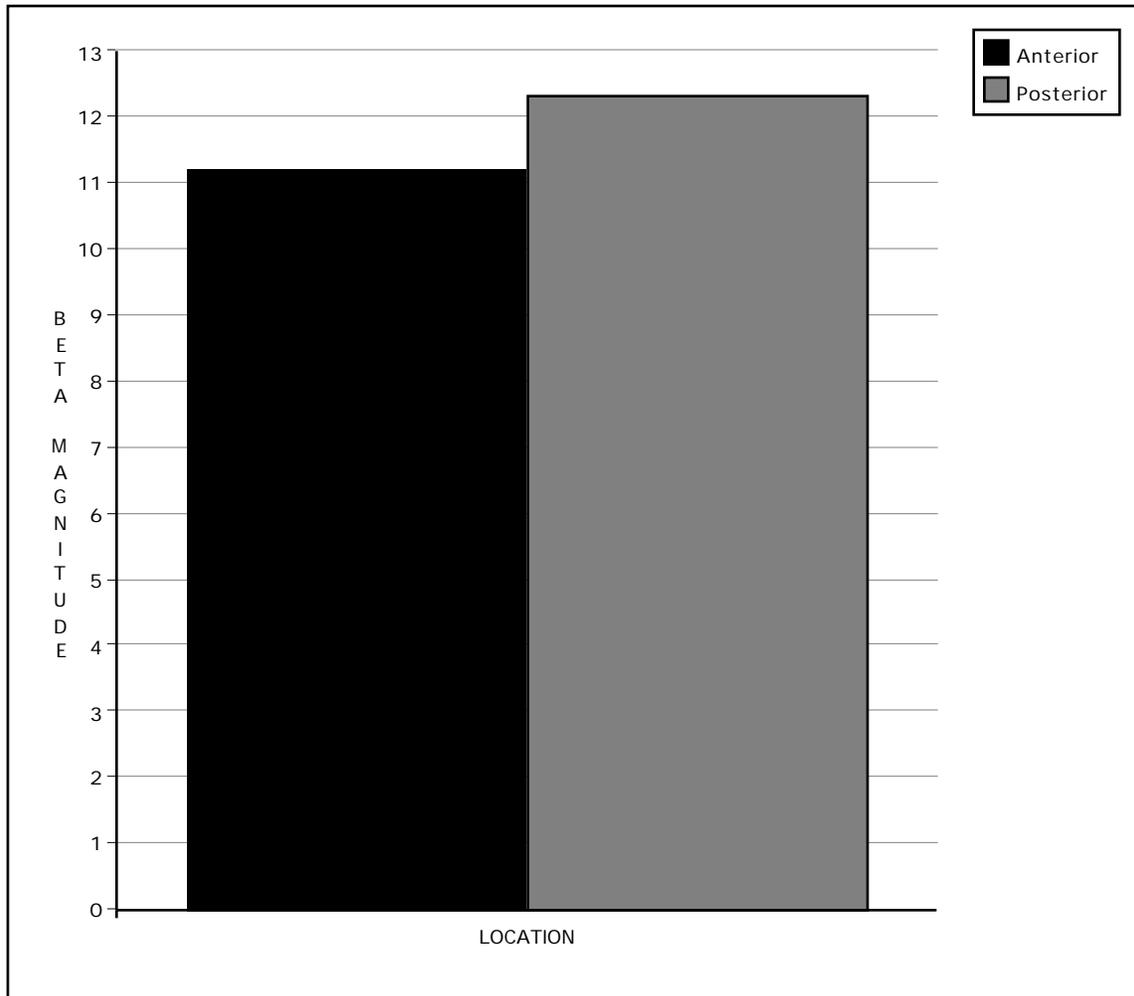
Table 8. Summary of Group Means and Standard Deviations for Regional Electroencephalographic Measures

Variable	<u>Low-Hostiles</u>				<u>High-Hostiles</u>			
	<u>Pre-Stress</u>		<u>Post-Stress</u>		<u>Pre-Stress</u>		<u>Post-Stress</u>	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Left Cerebrum	11.10	4.17	11.45	3.72	12.36	3.19	12.41	3.17
Right Cerebrum	10.94	4.19	11.35	3.67	12.27	3.16	12.29	3.41
Anterior	10.57	4.31	10.65	3.72	11.75	3.13	11.80	3.22
Posterior	11.47	4.00	12.14	3.51	12.89	3.11	12.90	3.28

Table 9. Summary of ANOVA Results for Regional Electroencephalographic Measures

Source	df	SS	MS	F	p
Group	(1,28)	75.86	75.86	.80	<.379
Condition	(1,28)	2.56	2.56	1.19	<.284
Group X Cond	(1,28)	1.80	1.80	.84	<.367
Cerebrum	(1,28)	.80	.80	.54	<.466
Group X Cereb	(1,28)	.01	.01	.01	<.935
Location	(1,28)	80.58	80.58	17.16	<.003
Group X Locat	(1,28)	.09	.09	.02	<.890
Cond X Cereb	(1,28)	.00	.00	.02	<.900
Group X Cond X Cerebrum	(1,28)	.03	.03	.22	<.641
Cond X Locat	(1,28)	1.10	1.10	2.34	<.137
Group X Cereb X Location	(1,28)	.17	.17	.21	<.648
Cond X Cereb X Location	(1,28)	.17	.17	.48	<.496
Group X Cond X Cereb X Loc	(1,28)	.66	.66	1.81	<.189

Figure 4. Main Effect of Location on Regional
Electroencephalographic Measures



hostiles ($\underline{M} = 6.67$, $\underline{SD} = .97$) did not differ on the positivity of their self-reported thoughts during the experiment, $\underline{t}(28) = .24$, $\underline{p} > .05$.

To determine whether groups differed on the accuracy of their estimated physiological reactivity to the stressor, the variance between their actual and estimated cardiovascular changes were calculated. That is, the following mathematical expressions were performed:

$$\begin{aligned} \text{Var}(\text{HR}) &= (\text{EHRC} - \text{AHRC})^2 \\ &\text{and} \\ \text{Var}(\text{SBP}) &= (\text{ESBPC} - \text{ASBPC})^2 \end{aligned}$$

where $\text{Var}(\text{HR})$ = variance between estimated and actual heart rate change, EHRC = estimated heart rate change, AHRC = actual heart rate change, $\text{Var}(\text{SBP})$ = variance between estimated and actual systolic blood pressure change, ESBPC = estimated systolic blood pressure change, and ASBPC = actual systolic blood pressure change.

T-tests were performed on these variances to assess group differences in the accuracy of their estimated physiological changes to the stressor. The effect of group on the accuracy of estimated heart rate changes approached significance, $\underline{t}(28) = 3.01$, $\underline{p} = .094$, with high-hostiles ($\underline{M} = 33.00$, $\underline{SD} = 9.42$) being less accurate than low-hostiles ($\underline{M} = 12.60$, $\underline{SD} = 7.68$). The effect of group on the accuracy of estimated systolic blood pressure changes was insignificant, $\underline{t}(28) = 1.31$, $\underline{p} > .05$.

Discussion

The present research compared low- and high-hostile men from an undergraduate population on self-report, cardiovascular, and quantitative electroencephalographic measures. The participants were assigned to the low- and high-hostile groups by using extreme scores on the CMHS. The robust finding of increased cardiovascular reactivity to the cold-pressor stress was supported by this research. The primary findings of this research include significantly greater beta magnitude recorded by the T3, relative to F7, electrode among low-hostiles. This may suggest that low-hostiles experience left-frontal disinhibition of left-temporal regions, thereby strengthening cardiovascular regulation during the cold-pressor stress. In addition, irrespective of condition, high-hostiles evidenced significantly greater beta magnitude at regions corresponding to the F7 and F8 electrodes. This perhaps suggests that high-hostiles have a relative inability to increase their rostral modulation of posterior systems related to cardiovascular reactivity/regulation. Regardless, there was a significant difference among the groups with heightened right cerebral arousal found for high-hostile participants.

As predicted, significant main effects of condition were found on the dependent measures of HR, SBP, and DBP. The participants, irrespective of group, showed greater physiological arousal after the cold-pressor stress. Further, significant group by condition interaction effects were found

on HR and SBP data. These results suggest that high-hostiles experienced significantly greater cardiovascular reactivity to the cold-pressor stress on these indicators of cardiovascular arousal (see Table 2 and Table 3). This finding of reliable cardiovascular reactivity was restricted to the HR and SBP data. Group differences in DBP reactivity were not reliable. This may partially result from initial value differences between the groups (LIV; Wilder, 1931, 1957), with high-hostile participants showing reduced reactivity due to higher initial values (Furedy & Scher, 1989).

Hypotheses two through five were assessed via a three-factor, mixed-design ANOVA using the beta magnitude record obtained by the F7, F8, T3, and T4 electrodes. These analyses revealed a significant group by region interaction (see Table 5, Figure 3). Low-hostiles, irrespective of condition, evidenced decreased beta magnitude in comparison with the high-hostiles at the regions corresponding to the F7 and F8 electrode sites. This finding of reduced prefrontal beta magnitude among low-hostiles may suggest that they experience a decreased inhibition of temporal beta magnitude (Butter, 1990; Demaree & Harrison, 1996b; Heilman et al., 1983; Ursin, 1960; Woods, 1956). Right- and left-temporal lobe disinhibition has been theorized to produce increased (DeRenzi & Faglioni, 1965; Heilman & Van Den Abell, 1979; Howes & Boller, 1975; Joseph, 1986; Weinstein, 1978) and decreased cardiovascular arousal (Demaree & Harrison, 1996a;

Hugdahl et al., 1983; Lane & Jennings, 1995; Oppenheimer et al., 1992; Zamrini, 1989), respectively.

Perhaps the most interesting results of this research were found by independently analyzing beta magnitude recorded by the F7, F8, T3, and T4 electrode sites by group. An independent ANOVA using only the high-hostile's data revealed no main (condition or region) or interaction (condition by region) effects (see Table 4 and Table 7). By contrast, an identical analysis using only the low-hostile data revealed a significant main effect of region (see Table 4 and Table 6). Specifically, low-hostiles evidenced significantly lower beta magnitude between regions corresponding to the F7 and T3 electrode sites. This finding may suggest that low-hostiles experience decreased left-frontal beta magnitude, thereby disinhibiting left-temporal beta magnitude (Butter, 1990; Demaree & Harrison, 1996b; Heilman et al., 1983; Ursin, 1960; Woods, 1956). As suggested by a growing literature base (Demaree & Harrison, 1996a; Hugdahl et al., 1983; Lane & Jennings, 1995; Oppenheimer et al., 1992; Zamrini, 1989), this left-temporal disinhibition may produce decreased cardiovascular arousal to stress among low-hostiles. Interestingly, both low- and high-hostiles experienced a nonsignificant difference on inspection and analysis of beta magnitude between regions corresponding to the F8 and T4 electrode sites (see Table 4 and Table 5). This may suggest that both low- and high-hostiles experienced similar right-brain beta magnitudes, thereby producing like tendencies

towards increased cardiovascular arousal to the cold-pressor stress.

At least two possible conclusions may be drawn from the low-hostile's relatively reduced beta magnitude over the F7 and F8 electrode sites (see Table 4, Table 5, and Figure 3). First, during the pre-stress phase, high-hostiles may have approached a "ceiling" on anterior beta magnitude. This ceiling effect may reduce the possibility of further regulation of the posterior systems to the cold-pressor stress among high-hostiles. That is, low-hostiles may have an increased capacity for anterior modulation of relatively caudal systems. Second, the relative hypoarousal of higher cortical anterior regions among low-hostiles may directly influence subcortical arousal levels, thereby inhibiting cardiovascular reactivity.

To my knowledge, this possible sequence is not well understood. Still, the anatomical tracks emanating from the left frontal regions make the rostral inhibition of cardiovascular arousal possible. The left frontal lobe, and prefrontal cortex in particular, has extensive interconnections with subcortical areas including the paralamellar portion of the dorsomedial nucleus (DM) of the thalamus and the centromedian-parafascicularis (CM-Pf) complex (Akert & Von Monakow, 1980; Kievert & Kuypers, 1977). The paralamellar region of the DM, in turn, has bidirectional bundles with the medulla along the fronto-cortico-dentato-rubro-thalamic track. The medulla, through the cranial

nerves, controls a number of vital reflexes -- breathing, sneezing, coughing, vomiting, and heart rate (Herkenham et al., 1990). Thus, it appears possible that decreased beta magnitude of the left frontal regions may directly influence subcortical structures controlling physiological arousal. In support of this possibility, research robustly suggests that left frontal hypoarousal corresponds with decreased cardiovascular arousal among depressed individuals (Davidson, 1984; Starkstein & Robinson, 1986).

It should be noted, however, that no significant region by condition interaction effects were found in the single group independent ANOVAs using beta magnitude data recorded by the F7, F8, T3, and T4 electrodes. Given the differential cardiovascular effects of the stressor between groups, this finding is initially difficult to interpret. However, it appears plausible that cardiovascular response to stress may have been induced *before* the administration of the cold-pressor test. Indeed, low- and high-hostiles had significantly different HR and SBP levels during the pre-stress phase, a finding inconsistent with previous research (see Table 2, Table 3, Figure 1, and Figure 2) (Demaree & Harrison, 1996a, Dembroski, MacDougall, Herd, & Shields, 1979; Glass, McKinney, Hofschire, & Fedorko, 1990). Previous literature indicates that the preparation of subjects for electroencephalographic recordings may significantly increase their cardiovascular arousal (Knox, 1980). Therefore, the significant group differences in F7, F8, T3, and T4 beta

magnitude recordings during both pre- and post-stress conditions may reflect disparate higher cortical effects between low- and high-hostiles to the "EEG preparation" stressor. Specifically, relative to high-hostiles, low-hostiles may evidence reduced beta magnitude in the left-frontal region before the cold-pressor stress (see Table 4, Table 5, and Figure 3). This relative hypoarousal may have a disinhibitory effect on left temporal regions (Butter, 1990; Demaree & Harrison, 1996b; Heilmen et al., 1983; Ursin, 1960; Woods, 1956), thereby suppressing physiological arousal (Demaree & Harrison, 1996a; Hugdahl et al., 1983; Lane & Jennings, 1995; Oppenheimer et al., 1992; Zamrini, 1989). Indeed, I suspect that the methodology used for EEG preparation may produce pain, anxiety, and cardiovascular arousal.

Review of the planned comparisons revealed a significant effect of location (see Table 9 and Figure 4). This may suggest that, among all participants, posterior regions had significantly greater beta magnitude relative to anterior regions. This is not surprising, and is typical among "normal," intact participants (Talbot, Hales, & Yudofsky, 1988). This finding may additionally reflect the role of posterior regions, and the reticular activating system in particular, in cardiovascular arousal (Morruzi & Magoun, 1949). This main effect of location may be particularly robust if participants generated cardiovascular responses to

"the EEG preparation" stressor experienced before the pre-stress phase (Knox, 1980).

This experiment is consistent with prior, related research (Demaree & Harrison, 1996a). Demaree and Harrison (1996a) found that, after administration of the cold-pressor stress, low- and high-hostiles experienced reduced and increased right-cerebral arousal, respectively. Cerebral lateralization, however, was assessed using a dichotic listening paradigm. This paradigm most accurately reflects arousal corresponding to the T3 and T4 regions. The current finding supports the notion that low- and high-hostiles, irrespective of condition, experience greater beta magnitude at regions assessed by the T3 and T4 electrodes, respectively (see Table 4 and Figure 3). While these group differences in beta magnitude are unreliable, they may produce statistically significant perceptual differences that may be tapped by the dichotic listening paradigm. These differences may, additionally, produce significant emotional, cognitive, and/or behavioral phenomena that were not assessed during this experiment. This investigation benefits from the use of technology which specifically reflects beta magnitude of distinct regions, rather than cerebral asymmetry more generally.

The results of this experiment do not appear to be confounded by outside group variables assessed during this research. Low- and high-hostiles did not differ on their mean responses to the Coren, Porac, and Duncan Laterality

Questionnaire (see Table 1), which may suggest that hemispheric dominance did not contribute to group differences on cardiovascular or electroencephalographic measures. Groups also did not differ on the negativity or positivity of their thoughts during the experiment. Interestingly, low- and high-hostiles were statistically equivalent in the accuracy of identifying their own HR and SBP increases to the cold-pressor stress (Appendix F). This does not support previous literature which provides evidence of decreased self-awareness among high-hostiles (Demaree & Harrison, 1996a, Demaree & Harrison, 1996c; Epstein & Cluss, 1982; Scheier et al., 1974). However, this is the first known attempt to determine whether low- and high-hostiles differ in their ability to effectively monitor their own cardiovascular arousal. Refinement of the questionnaire used in this investigation (Appendix F) may be beneficial to future research.

This research revealed differential cardiovascular and electroencephalographic reactivity to the cold-pressor stress between low- and high-hostiles. Taken together, one may speculate that low-hostile's reduced left-frontal beta magnitude may disinhibit left-temporal arousal and produce a suppressive effect on cardiovascular reactivity to the cold-pressor stress. This research may be important to our understanding of the positive relationship between hostility level and cardiovascular disease (Kubany et al., 1994; Treiber et al., 1989; Lee and Cameron, 1987). Indeed,

knowledge of the higher cortical correlates associated with cardiovascular arousal/regulation may be helpful in devising methods to reduce heightened cardiovascular lability among high-risk populations (e.g. high-hostiles).

While the results of this experiment were interesting, this research may be improved in three important ways. First, because HR and SBP data were collected during pre- and post-stress conditions, the group by condition interactions may be attributable to greater cardiovascular perseveration, rather than reactivity, among high-hostiles. This interpretation is consistent with previous hostility research (Herridge, Harrison, & Demaree, 1997; Ernst et al., 1990). Future research may benefit from cardiovascular data collection concurrent with the stress condition. Second, it appears plausible from the results of this research, as well as prior investigation, that subjects may evidence cardiovascular arousal from the experimental preparation for electroencephalographic recording. To better evidence higher cortical beta magnitude changes associated with cardiovascular reactivity, a relaxation period between electroencephalographic preparation and pre-stress measurement of participants' cardiovascular and beta magnitude baseline appears warranted. Last, a more valid questionnaire to assess subjects' perceptions of their cardiovascular reactivity may assist future research efforts to understand a possible relationship between hostility level and cardiovascular self-awareness.

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Appendix A

INFORMED CONSENT FOR PARTICIPANTS OF INVESTIGATIVE PROJECTS

Title of Project: Quantitative Electroencephalographic Analyses of Low- and High-Hostiles to Stress.

Experiment Number: 96-123

1. PURPOSE OF EXPERIMENT

You are invited to participate in a study to obtain data regarding your medical history, handedness, and hostility level.

2. PROCEDURE TO BE FOLLOWED IN THE STUDY

To accomplish the goals of this study, you will be asked to complete three questionnaires related to your medical history, handedness, and hostility level. Later, you may be called by telephone and asked to participate further in this research.

3. ANONYMITY OF SUBJECTS AND CONFIDENTIALITY OF RESULTS

Identifying information will be kept strictly confidential. At no time will the researchers release your personal information from 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 a subject number will identify you during analyses and any written reports of the research.

4. DISCOMFORTS AND RISKS FROM PARTICIPATING IN THE STUDY

You may feel some embarrassment from answering the questionnaires. You may omit any questions that you feel embarrassing. If, after you have left the experiment, you have any problems associated with this study please call Dr. David W. Harrison, Ph.D. (231-4422) so that he may assist you directly or direct you to appropriate services.

5. EXPECTED BENEFITS

Your participation in the project will help determine scores that may identify normal individuals as having relatively high or low levels of hostility for future research.

No guarantee of benefits has been made to encourage you to participate.

6. FREEDOM TO WITHDRAW

You are free to withdraw from this study at any time without penalty. If you choose to withdraw, you will not be penalized by reduction in points or grade for Psychology 2004. There are alternative choices for receiving extra credit for your course.

7. EXTRA CREDIT COMPENSATION

For participation in this study you will receive one point extra credit for Psychology 2004.

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. SUBJECT'S PERMISSION

I have read and understand the above description of the study. I have had an opportunity to ask questions and have had them all answered. I hereby acknowledge the above and give my voluntary consent for participation in this study.

I further understand that if I participate I may withdraw at any time without penalty.

I understand that if I have any questions regarding this research and its conduct, I should contact any of the persons named below.

<u>Heath A. Demaree</u> Primary Researcher	<u>231-6914</u> Phone
---	--------------------------

<u>David W. Harrison, Ph.D.</u> Faculty Advisor	<u>231-4422</u> Phone
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<u>Richard M. Eisler, Ph.D.</u> Chair, HSC	<u>231-7001</u> Phone
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<u>Ernest R. Stout</u> Chair, IRB	<u>231-9359</u> Phone
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Subject's Signature: _____ Date: _____

Subject's ID: _____ Subject's Telephone #: _____

Appendix B

Medical History Questionnaire

NAME: _____ I.D. #: _____

Have you ever experienced or been diagnosed with any of the following, or are you experiencing any of the following at present? Please circle the appropriate response and explain "Yes" answers below.

- | | | |
|---|-----|----|
| 1. Severe head trauma/injury | Yes | No |
| 2. Stroke | Yes | No |
| 3. Learning disabilities (problems with reading, writing, or comprehension) | Yes | No |
| 4. Epilepsy or seizures | Yes | No |
| 5. Paralysis | Yes | No |
| 6. Neurological surgery | Yes | No |
| 7. Other neurological/nervous system problems | Yes | No |
| 8. Alcohol or drug problems | Yes | No |
| 9. Using alcohol or drugs (other than for prescribed purposes) at present | Yes | No |
| 10. Past psychological/psychiatric problems | Yes | No |
| 11. Are you currently taking any prescription medications/drugs? | Yes | No |
| 12. Are you currently suffering from any medical conditions or illnesses? | Yes | No |
| 13. Arthritis | Yes | No |
| 14. Any head or lung problems | Yes | No |
| 15. Reynaud's Syndrome | Yes | No |
| 16. Hearing problems | Yes | No |
| 17. Fear of small or enclosed spaces | Yes | No |
| 18. Visual Problems | Yes | No |

Please explain "Yes" responses:

Appendix C

Handedness Questionnaire

Circle the appropriate number after each item:

	<u>Right</u>	<u>Left</u>	<u>Both</u>
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 wanted to pick up a pebble with your toes, which foot would you use?	1	-1	0
If you had to step up onto 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 their chest?	1	-1	0

Into which ear would you place the
earphone of a transistor radio? 1 -1 0

of Right + # of Left = Total Score

_____ + _____ = _____

Is mother left or right hand dominant? _____

Is father left or right hand dominant? _____

Appendix D

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.
Please give a response to every statement.

- | | | | |
|-----|---|---|---|
| 1. | When I take a new job, I like to be tipped off on who should be gotten next to. | T | F |
| 2. | When someone does me wrong I feel I should pay him back if I can, just for the principle of the thing. | T | F |
| 3. | I prefer to pass by school friends, or people that 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 through the fear of getting caught. | T | F |
| 10. | Most people will use somewhat unfair means to gain profit or an advantage, rather than to lose it. | T | F |
| 11. | I commonly wonder what hidden reason another person may have for doing something nice for me. | T | F |
| 12. | It makes me impatient to have people ask my advice or otherwise interrupt me when I am working on something important. | T | F |
| 13. | I feel that I have often been punished without cause. | T | F |
| 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 anyone for trying to grab everything he 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 which I consider wrong. | T | F |
| 21. | It is safer to trust nobody. | T | F |
| 22. | I do not blame a person for taking advantage of someone who lays himself open to it. | T | F |
| 23. | I have often felt that strangers were looking at me critically | T | F |
| 24. | Most people make friends because friends are likely to be useful to them. | T | F |
| 25. | I am sure I am being talked about. | T | F |
| 26. | I am not likely to speak to people until they speak to me. | T | F |
| 27. | Most people inwardly dislike putting themselves out to help other people. | T | F |
| 28. | I tend to be on guard with people who are somewhat more friendly than I had expected. | T | F |
| 29. | I have sometimes stayed away from another person because I feared saying or doing 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. | I would certainly enjoy beating a crook at his own game. | T | F |
| 36. | It makes me think of failure when I hear of the success of someone I know well. | 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 that 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 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 about things related to her sex. | T | F |
| 45. | I do not try to cover up my poor opinion or pity of a person so that he won't know how I feel. | T | F |
| 46. | I have frequently worked under other people who seem to have things so they can get credit for good work but are able to pass off mistakes onto those under them. | T | F |
| 47. | I strongly defend my own opinions as a rule. | T | F |
| 48. | People can pretty easily change em even though I thought that my mind was already made up on a subject. | T | F |
| 49. | Sometimes I am sure that other people can tell what I am thinking. | T | F |
| 50. | A large number of people are guilty of bad sexual conduct. | T | F |

Appendix E

INFORMED CONSENT FOR PARTICIPANTS OF INVESTIGATIVE PROJECTS

Title of Project: Quantitative Electroencephalographic Analyses of Low- and High-Hostiles to Stress.

Experiment Number: 96-123

1. PURPOSE OF EXPERIMENT

You are invited to participate in a study about the effects of stress. This research attempts to determine the effects of hostility on both physiological and brain-related reactivity in response to the cold pressor task.

You will be asked to place your hand in ice water (known as the cold-pressor test) which usually results in heightened physiological arousal. In addition, four minutes of quantitative electroencephalographic (QEEG) data will be recorded during this experiment. QEEG purportedly taps the arousal of different regions in your brain.

Last, you will be asked to complete a questionnaire which will assess your thoughts about the experiment, as well as how physiologically reactive you thought you were to the cold-pressor test.

2. PROCEDURE TO BE FOLLOWED IN THE STUDY

You will also be asked to undergo a stress condition called the cold pressor test. During this task, you will be asked to keep your hand in ice water for 45 seconds. The cold pressor paradigm is the primary method used to increase HR and has been a component in over 1000 research articles. It is the most widely accepted method to induce cardiovascular changes in humans.

You will also be hooked up to heart rate and blood pressure equipment, which will be monitored and recorded before and after the cold pressor test. This will help determine physiological reactivity to the cold pressor test.

Before and after the cold pressor test, electroencephalographic (EEG) data will be recorded from your scalp. This will help the researchers determine arousal of various brain regions. To obtain EEG data, you will wear an electrode cap which has electrodes permanently placed in the cap. The cap resembles a swimming cap, and may be slightly uncomfortable, as it is attached to a harness which is fastened loosely around your chest to keep the cap in place. You will also have electrodes placed on your earlobes.

You must undergo several procedures to place the proper equipment on your scalp. You will first be asked to lie in a supine position on a reclining chair. You will then be asked to lean forward slightly, while a body harness is placed around your chest and fastened in the back via Velcro straps. The distance between your inion (the bony protrubance at the back of your skull) and nasion (the juncture where your nose meets your forehead) will be measured. A grease mark will then be placed on your forehead. By placing a measuring tape from the grease mark to your inion, the circumference of your head will be measured. While wearing vinyl gloves, the researcher will then use an alcohol swab to wipe your earlobes and forehead. The researcher will then lightly abrade your earlobes with skinprep (a Vaseline-type substance). Reference electrodes will then be placed on your earlobes. The researcher will place two sponge discs on your forehead which will help to hold a cap of electrodes (known as an electro-cap) on your head. These electrodes will be used to collect electrical activity from your scalp. Velcro-straps will be connected from the electro-cap to the aforementioned body harness to help keep the cap in place. Electrode gel will be placed in each electrode on your scalp using a blunt syringe. This gel will be used to help record electrical activity from your scalp.

3. ANONYMITY OF SUBJECTS AND CONFIDENTIALITY OF RESULTS

Identifying subject information will be kept strictly confidential. At no time will the researchers release your personal information from 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 a subject number will identify you during analyses and any written reports of the research.

4. DISCOMFORTS AND RISKS FROM PARTICIPATING IN THE STUDY

You may feel some discomfort during this experiment due to your participation in the "cold pressor" procedure. The water will be cold and may be painful. You may also experience some discomfort related to the inflation of the blood pressure cuff.

The placement of and inflation of blood-pressure equipment on your arm may produce discomfort.

Participation in EEG data collection may also produce discomfort. Specifically, the placement of ear electrodes may pinch your ears and be painful. Wearing the electro-cap may produce pressure around your head that is uncomfortable, or even painful. Other risks include slight discomfort from the abrasive cleaning and a trivial risk of infection. To ensure your safety from infection, the experimenter has thoroughly sanitized the electrodes and washed the electrocap. The experimenter will wear clean rubber or vinyl gloves while attaching the electrodes.

Safeguards that will be used to minimize your discomfort include the continuous opportunity to terminate the experiment without penalty to yourself (losing your extra credit points) should you ever feel uncomfortable. A thorough debriefing discussing any issues that may be of concern to you will also be provided at the end of the experiment. At that time you will be given ample opportunity to ask any additional questions about the research that you may feel were inadequately addressed by our debriefing. If, after the experiment, you have any problems associated with this study please call Dr. David W. Harrison, Ph.D. (231-4422) so that he may either assist you directly or direct you to appropriate services.

5. EXPECTED BENEFITS

Your participation in the project may help identify several correlates of hostility. The effects of hostility on physiological, and brain-related reactivity to stress will be assessed.

No guarantee of benefits has been made to encourage you to participate.

You may receive a synopsis or summary of this research when completed. Please leave a self-addressed envelope if you are interested in receiving this information.

6. FREEDOM TO WITHDRAW

You are free to withdraw from this study at any time without penalty. If you choose to withdraw, you will not be penalized by reduction in points or grade for Psychology 2004. There are alternative choices for receiving extra credit for the course evaluation.

7. EXTRA CREDIT COMPENSATION

For participation in this study you will receive two points extra credit for Psychology 2004.

If as a result of this project, you or the investigator determine that you should seek counseling or medical treatment, the following is available: the University Health Center, the University Counseling Center.

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. SUBJECT'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 Reynaud's syndrome, head injury, epilepsy, or any cardiac problems. Report to the experimenter current regular medications.

10. SUBJECT'S PERMISSION

I have read and understand the above description of the study. I have had an opportunity to ask questions and have had them all answered. I hereby acknowledge the above and give my voluntary consent for participation in this study.

I further understand that if I participate I may withdraw at any time without penalty.

I understand that if I have any questions regarding this research and its conduct, I should contact any of the persons named below.

<u>Heath A. Demaree</u> Primary Researcher	<u>231-6914</u> Phone
---	--------------------------

<u>David W. Harrison, Ph.D.</u> Faculty Advisor	<u>231-4422</u> Phone
--	--------------------------

<u>Richard M. Eisler, Ph.D.</u> Chair, HSC	<u>231-7001</u> Phone
---	--------------------------

<u>Ernest R. Stout</u> Chair, IRB	<u>231-9359</u> Phone
--------------------------------------	--------------------------

Subject's Signature: _____

Date: _____

Subject's ID: _____

Appendix F

Did you generally experience negative or positive thoughts while participating in this experiment?

1 2 3 4 5 6 7 8 9
Extremely negative Neutral Extremely Positive

On average, Virginia Tech undergraduates have been found to have an 8-point increase of systolic blood pressure during the cold-pressor test (dipping your hand in ice water). How much do you think your systolic blood pressure increased due to the cold-pressor test?

1 2 3 4 5 6 7 8
9 10 11 12 13 14 15 16

On average, Virginia Tech undergraduates have been found to have a 4-point (beats per minute) increase of heart rate during the cold-pressor test. How much do you think your heart rate increased due to the cold-pressor test?

1 2 3 4
5 6 7 8

April, 1997

Vita

Heath A. Demaree

PERSONAL INFORMATION

Born: May 13, 1971, Bronxville, NY
Social Security Number:
Marital Status: Single
Business Address: Department of Psychology
Virginia Polytechnic Institute
and State University
Blacksburg, Virginia 24061-0436
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EDUCATION

B.A. Bachelor of Arts
Degree conferred, May, 1993 (cum laude)
Princeton University
Major field of study: Psychology
Title of thesis: Motivation in baseball.
Major advisor: Dr. Nancy Cantor
M.S. Master of Science candidate, 1993-1995
Degree conferred, May, 1995
Virginia Polytechnic Institute and State University
Title of thesis: Behavioral, physiological, and
neuropsychological correlates of hostility.
Major advisor: Dr. David W. Harrison

Ph.D. Doctor of Philosophy candidate, 1995-
Degree expected, May, 1998

Major field of study: Clinical
Psychology/Neuropsychology

Virginia Polytechnic Institute and State University

Preliminary Paper: A neuropsychological model
relating hostility to self-awareness (completed).

Title of dissertation: Analysis of Quantitative
electroencephalographic and Cardiovascular
Responses to Stress Among Low- and High-Hostiles.

Major advisor: Dr. David W. Harrison

HONORS AND AWARDS

1997 Received special commendation for Graduate Teaching
excellence

1997 Nominated by the Virginia Polytechnic Institute
Psychology Department for the Graduate Teaching
Excellence Award

1996 Nominated by the Virginia Polytechnic Institute
Psychology Department for the APA Dissertation
Award

1996 Nominated by the Virginia Polytechnic Institute
Psychology Department for the APA Researcher
Fellowship

1996 Nominated by the Virginia Polytechnic Institute
clinical psychology track for the APA Researcher
Fellowship

1996-1997 Virginia Tech Graduate Student Liaison, elected

1995-1996 Virginia Tech Graduate Student Liaison, elected

1993 Cum laude, Princeton University

1991-1993 Princeton Departmental Representative, elected

1989 Who's Who in American Colleges and Universities

CLINICAL TRAINING

1996-1997 Graduate Supervisor
Neuropsychological Assessment Practicum Team
Psychological Services Center
Virginia Polytechnic Institute and State University
Blacksburg, Virginia
Graduate supervisor of practicum team
specializing in the assessment and treatment
of neuropsychological disorders resulting from
cerebrovascular, convulsive, neoplastic, and
traumatic disorders of the brain.

Approximate number of hours: 240
Supervisor: Dr. David W. Harrison

1995 Extern
summer Salem Veterans Administration Medical Center
Salem, Virginia
Paid therapist working primarily in Behavioral
Medicine Unit with additional training in the
Psychological Services Center. Specialized in
the assessment and treatment of anger and
depression problems, as well as the
rehabilitation of individuals with cerebral
dysfunction from stroke, dementia, and head
injury. Helped run smoking cessation and
cardiovascular risk groups.

Approximate number of hours: 500
Supervisors: Dr. Kim Ragsdale and Dr. M. K. Johnson

1994-1996 Neuropsychological Assessment Practicum Team
Psychological Services Center
Virginia Polytechnic Institute and State University
Blacksburg, Virginia
Graduate level practicum team specializing in
the assessment and treatment of
neuropsychological disorders resulting from
cerebrovascular, convulsive, neoplastic, and
traumatic disorders of the brain.

Approximate number of hours: 960
Supervisor: Dr. David W. Harrison

1994-1995 Clinical Practicum Team
Psychological Services Center
Virginia Polytechnic Institute and State University
Blacksburg, Virginia
Graduate level practicum team specializing in
the assessment and treatment of excessive
anger/aggression expression, marital conflict,
and relationship issues.

Approximate number of hours: 480
Supervisors: Dr. Richard Eisler, Dr. Ellie Sturgis

1994 Therapist
summer Psychological Services Center
Blacksburg, Virginia
Responsibilities included the assessment and
treatment of a variety of psychological
disorders including depression, anxiety,
learning disability (LD), attention deficit
disorder (ADD), and personality disorders.

Approximate number of hours: 500
Supervisor: Dr. Richard M. Eisler

1993-1994 Clinical Practicum Team
Psychological Services Center
Virginia Polytechnic Institute and State University
Blacksburg, Virginia
Graduate level practicum team specializing in
assessment and treatment of a variety of
psychological disorders including depression,
anxiety, relationship and marital problems,
LD, ADD, and school phobia.

Approximate number of hours: 240
Supervisors: Dr. Jack Finney, Dr. Robert Stephens

1992-1993 Therapist
summers Independent households, Westchester County, N. Y.
Responsible for the creation and
implementation of behavior modification
protocols for autistic children, ages 4 to 8.

Approximate number of hours: 200
Supervisor: Dr. Marcia Gables

1991
summer Volunteer Therapist, summer
Cornell Hospital -- New York Medical Center
White Plains, New York
Responsible for patient intakes and
discharges, leadership of group discussion
meetings, and assorted clerical duties.

Approximate number of hours: 500

TEACHING EXPERIENCE

1996-1997 University Instructor
Virginia Polytechnic Institute and State University
Blacksburg, Virginia
Instructor of Psychology 2054, entitled
Psychological Theories of Personality.
Received overall rating of 4.0/4.0 for Fall
semester.

1996
summer University Instructor
Virginia Polytechnic Institute and State University
Blacksburg, Virginia
Taught Psychology 2054, entitled Psychological
Theories of Personality, as well as one (1)
Advanced Social Psychology Laboratory.
Received overall teacher ratings of 3.8 and
3.9/4.0, respectively.

1995-1996 Teaching Assistant
Virginia Polytechnic Institute and State University
Blacksburg, Virginia
Taught four (4) Advanced Social Psychology
laboratories and received an average of
3.9/4.0 overall teacher ratings.

1993-1994 Teaching Assistant
Virginia Polytechnic Institute and State University
Blacksburg, Virginia
Taught four (4) Introduction to Psychology
laboratories and received an average of
3.8/4.0 overall teacher ratings.

EMPLOYMENT

(described, if in addition to above)

1994-1995 Supervisor
Psychological Services Center
Virginia Polytechnic Institute and State University
Blacksburg, Virginia
Responsible for therapeutic duties, including
the supervision of graduate clinician
records/progress and maintenance of Physiology
Laboratory.

- 1995 Extern
Salem Veterans Affairs Medical Center
Salem, Virginia
(previously discussed)
- 1994-1995 Co-Director
Virginia Tech Men's Anger Control Clinic
Virginia Polytechnic Institute and State University
Blacksburg, Virginia
Responsible for the creation and maintenance of clinic. Job entailed client recruitment from several locations (court-, undergraduate- and shelter-populations). Clinic specializes in neuropsychological, behavioral, and cognitive assessment, treatment, and research.
- Co-Directors: Dr. Richard Eisler, Dr. Ellie Sturgis
- 1994-Therapist, summer position
Psychological Services Center
Blacksburg, Virginia
(previously discussed)
- 1993-1996 Scholastic Tutor
Kaplan Testing Services
Blacksburg, Virginia
Taught courses in SAT and GRE preparation and test-taking strategies.
- Approximate number of hours: 6 hours per week.
- 1992-1993 Therapist, summers
Independent households, Westchester County, N. Y.
(previously discussed)
- 1991 Volunteer Therapist, summer
Cornell Hospital -- New York Medical Center
White Plains, New York
(previously discussed)

PROFESSIONAL ACTIVITIES

Reviewer, Neuropsychology Review

Membership

American Psychological Association (Division 40),
student affiliate
The National Academy of Neuropsychology, student affiliate
Sigma Xi, The Scientific Research Society, associate member
International Neuropsychological Society

CURRENT RESEARCH AND SCHOLARLY INTERESTS

Cortical, subcortical, and autonomic correlates of impulsivity and cardiac control, especially in relation to hostility; methods in neuropsychology, psychophysiology, and psychopharmacology.

REFEREED PUBLICATIONS

Refereed Articles

- Crews, W. D., Harrison, D. W., Rhodes, R. D., & Demaree, H. A. (1995). Hand fatigue asymmetry in the motor performances of women with depressed mood. Neuropsychiatry, Neuropsychology, and Behavioral Neurology, 8(4), 277-281.
- Demaree, H. A., Crews, W. D., & Harrison, D. W. (1995). Topographical brain mapping in depression following mild closed head injury: A case study. Journal of Neurotherapy, 1(1), 38-43.
- Demaree, H. A., Harrison, D. W., Everhart, D. E., & Shenal, B. V. (1996). An organic correlate of diagnosed autism: A case study. Manuscript submitted for publication, Brain and Cognition.
- Demaree, H. A., & Harrison, D. W. (1997). Analysis of electroencephalographic and cardiovascular responses to stress among low- and high-hostiles. Manuscript being readied for submission.
- Demaree, H. A., & Harrison, D. W. (in press). A Neuropsychological model relating self-awareness to hostility. Neuropsychology Review, 7(4).
- Demaree, H. A., & Harrison, D. W. (1996). Topographical brain mapping in hostility following mild closed head injury. The International Journal of Neuroscience, 87, 97-101.
- Demaree, H. A., & Harrison, D. W. (in press). Physiological and neuropsychological correlates of hostility. Neuropsychologia.
- Demaree, H. A., Higgins, D., & Harrison, D. W. (1997). The effect of hostility on dynamometer hand grip strength, perseveration, and fatigue. Manuscript being readied for submission.
- Demakis, G. J., Harrison, D. W., & Demaree, H. A. (1995). A neuropsychological investigation of verbal and nonverbal fluency: Perspectives on asymmetries in frontal lobe functioning. Manuscript being readied for submission.

- Everhart, D. E., Demaree, H. A., & Harrison, D. W. (1995). "Delusions" of space: A case study utilizing topographical brain mapping and QEEG. Manuscript submitted for publication, Journal of Abnormal Psychology.
- Gilmore, J. D., & Demaree, H. A. (1996). The assessment of hopelessness in chronic pain patients. Manuscript being readied for publication.
- Herridge, M. L., Harrison, D. W., & Demaree, H. A. (in press). Hostility, facial configuration, and bilateral asymmetry on galvanic skin response. Psychobiology.
- Herridge, M. L., Harrison, D. W., & Demaree, H. A. (1996). Hostility, stress, and bilateral asymmetry on galvanic skin response. Manuscript being readied for publication.
- McDowell, C. L., Harrison, D. W., & Demaree, H. A. (1994). Is hemispheric decline a function of aging? The International Journal of Neuroscience, 79, 1-11.

Refereed Abstracts and Conference Proceedings
(one-page publications)

- Demaree, H. A., & Harrison, D. W. (1996). Physiological and neuropsychological correlates of hostility. Journal of the International Neuropsychological Society, 2(1), 31-32.
- Demaree, H. A., Crews, W. D. Jr., & Harrison, D. W. (1995). Topographical brain mapping in depression following mild closed head injury: A case study. Archives of Clinical Neuropsychology, 53, 270.
- Everhart, D. E., Demaree, H. A., & Harrison, D. W. (1994). "Delusions" of space: A case study utilizing topographical brain mapping and QEEG. The Clinical Neuropsychologist, 9(3), 280.

PRESENTATIONS AND PAPERS

National Meetings

- Crews, Jr., W. D., Harrison, D. W., Rhodes, R. D., & Demaree, H. A. (1995). Hand fatigue asymmetry in the motor performances of women with depressed mood. Annual meeting of the Virginia Psychological Association, Alexandria, VA.

Demaree, H. A., & Harrison, D. W. (1996). Physiological and neuropsychological correlates of hostility. Annual meeting of the International Neuropsychological Society, Chicago, IL.

Demaree, H. A., Crews, W. D. Jr., & Harrison, D. W. (1994). Topographical brain mapping in depression following mild closed head injury: A case study. Annual meeting of the National Academy of Neuropsychology, Orlando, FL.

Everhart, D. E., Demaree, H. A., & Harrison, D. W. (1995). Topographical brain mapping: Hostility following closed head injury. Annual meeting of the American Psychological Association, New York, NY.

GRANTS SUBMITTED

- 1996 Quantitative electroencephalographic analyses of cardiac arousal. National Heart, Lung, and Blood Institute, \$50,000. Principal Investigator.
- 1994 Behavioral, physiological, and neuropsychological effects of alcohol consumption. National Institute of Mental Health, \$10,000. Principal Investigator.

RELEVANT COURSEWORK

Advanced Psychotherapy
Advanced Topics in Clinical Psychology: Ethics
Advanced Topics in Clinical Psychology: Neuropsychology
Adult Psychopathology
Assessment of Human Intelligence
Behavioral Assessment and Treatment
Biological Bases of Behavior
Child Psychopathology
Developmental Psychology
Independent Study: Quantitative Electroencephalography
Interventions in Psychological Systems
Neurochemical Control
Personality Assessment
Perspectives of Social Psychology
Research Methods
Statistics for Social Science Research I
Statistics for Social Science Research II