Dynamic Effects of Stress and Hostility: Group Differences In Cardiovascular Regulation and Learning

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(ABSTRACT)

This experiment tested hypotheses linking the right cerebral regulation of hostility and cardiovascular reactivity. First, replication of previous research supporting heightened cardiovascular reactivity (mean arterial pressure, systolic blood pressure, diastolic blood pressure, and heart rate) among high-hostile participants was attempted. Second, dynamic variations in functional cerebral asymmetry in response to pain (cold pressor stressor) and affective verbal learning (positive and negative valenced word lists) were measured.

High- and low-hostile participants (n = 64) were identified using the Cook Medley Hostility Scale. Participants completed either the cold-pressor stressor condition or the no-stress control condition as well as the negative and the positive affective verbal learning test. Cardiovascular measures (MAP, SBP, DBP, and HR) before and after the stress or no-stress condition and before and after the negative and the positive affective learning tasks were recorded.

The results demonstrated that high-hostiles had difficulty processing emotional stimuli. High-hostiles were reliably impaired in emotional word learning. Further, results suggested that negative affective learning produced proactive interference for the learning or recall of subsequently presented information. Positive affective learning produced diametrically opposite effects with retroactive interference for the recall of previously presented information. Also, high-hostiles' cardiovascular reactivity to a physical stressor was independent of the valence of the learning task. In contrast, low-hostiles' cardiovascular reactivity was valence dependent with activation to both the positive list concurrent with stress and to the negative list concurrent with no stress. Finally, the results indicated that the effect of the affective learning lists, on the heart, is stress dependent. Neuropsychological theories of ANS regulation and emotion are discussed in relation to the primary findings and a new model of lateralized regulation is proposed.

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This work is dedicated to my son, Benjamin Shenal. He is quite simply the coolest kid in the world and I wish him a life of joy and happiness.

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Dynamic Effects of Stress and Hostility:

Group Differences In Cardiovascular Regulation and Learning

Cardiovascular diseases are the predominant cause of death and disability in the United States and in most industrialized nations (Kaplan, Sallis, & Patterson, 1993). Approximately 60 million Americans have cardiovascular diseases and about 50 million Americans, about one-fourth of the adult population, have hypertension (NHLBI, 1993). More than 40 percent of the deaths in the United States are related to some form of heart disease. Further, in the United States alone, about 950,000 people die each year of heart disease, 1.25 million people suffer heart attacks, and over 6 million people display heart disease symptoms (Kaplan et al., 1993; Satcher, 1997; Wardlaw, Insel, & Seyler, 1992). Moreover, one person dies from cardiovascular disease approximately every 32 seconds and about 200,000 of those deaths are people of working age, younger than 65 (Kaplan et al., 1993). The economic burden of cardiovascular disease to society, including medications, health professional services, and lost productivity due to death and disability, is over $260 billion per year (Satcher, 1997). A better understanding of risk factors, such as stress and emotion, and how these factors affect individuals, may lead to improved preventative measures and decreased heart disease (American Heart Association, 1988).

Recent research has examined hostility and its correlates with cardiovascular reactivity to provide a better understanding of risk factors. Cardiovascular reactivity is a psychophysiological construct referring to the magnitude and mechanisms of cardiovascular responses associated with exposure to psychological stress. It is often used to refer to the propensity for an individual to exhibit an alteration in cardiovascular reactivity during exposure to some external stimulus (Sherwood & Turner, 1992). As the relevant literature has evolved, hostility has been implicated as a primary risk factor for cardiovascular disease. This paper describes pertinent theories of hostility and emotion, recent developments within the literature on the neuropsychology of negative emotions, and an integration of these literatures to investigate the neuropsychological underpinnings of hostility and cardiovascular reactivity or lability. It is suggested that such research affords an increased understanding of hostility and cardiovascular risk. With an increased understanding of the neuropsychological mechanisms involved in the mediation of hostility and cardiovascular disregulation, future programs of education, prevention, and treatment may be developed and practiced.

Following the review of pertinent literature, the experimental design is described. The experiment addresses four principle questions. First, relative to low-hostiles, do high-hostile individuals demonstrate heightened autonomic nervous system (ANS) arousal following negative affect induction? Second, relative to low-hostiles, do high-hostiles demonstrate a heightened sensitivity to proactive interference as evidenced by a primacy effect on list learning during negative affect induction? Third, what is the effect of stress induction on high-hostiles' ANS arousal and affective list learning performance? Fourth, what implications follow for high- and low-hostile functional cerebral asymmetry and dynamic functional cerebral laterality? These questions are theoretically addressed through the critical review of the relevant neuropsychological literature and then scientifically tested.

Neuropsychology of Emotion

Early research on the biological substrates of emotion focused on affect and changes within the autonomic nervous system. Subcortical limbic system circuits were described which mediate specific emotional behaviors (e.g., Heilman, Bowers, & Valenstein, 1985). As a result of more recent research, however, it is clear that the cerebral cortex plays an important role in emotional behavior (see Davidson, 1993, for review).

Neuropsychological research provides a useful framework to study emotions and their correlates. Accordingly, neuropsychological theories that describe cerebral asymmetries in the processing of emotion are presented. The preponderance of research to date has implicated the right cerebrum in the reception, comprehension, expression, and regulation of negative emotions (Heilman et al., 1985). Additional research has begun to investigate differential effects of the right anterior and posterior regions of the cerebrum in order to provide a more accurate representation of emotional processing. Alternate accounts exist which rely on cerebral balance (e.g., Tucker & Frederick, 1989), or a proportionate degree of processing capability by either cerebrum contingent upon the valence of the emotion at hand (Harrison & Gorelczenko, 1990). For example, Tucker and Fredrick (1989) proposed that the left cerebrum differentially processes positive emotions while the right cerebrum differentially processes negative emotions. These theories, and others, are presented and critically evaluated in order to provide a review of current neuropsychological theories of emotion. Finally, relevant research is presented in order to investigate the validity of the reviewed neuropsychological theories of emotion.

Right Hemisphere Model of Emotion

One neuropsychological theory of emotion proposes that emotions, irrespective of valence, are processed preferentially within the right cerebrum. Past studies of emotional perception have supported the contention that emotion, in general, is differentially controlled by the right cerebrum (Borod, Koff, Lorch, & Nichols, 1986; Etcoff, 1989). These studies suggested that the right posterior region of the cerebrum is specialized for the perception of emotional information, regardless of valence.

Heilman (1982) speculated that the right cerebrum may have greater control of the subcortical systems, which are largely responsible for arousal and emotion. Heilman and Van Den Abell (1979) demonstrated the superiority of the right cerebrum for arousal through the facilitation of reaction times following left-visual-field warning lights (right cerebrum). Although left-visual-field warning lights improved reaction times reliably compared to right-visual-field warning lights, improvement occurred at both the left and at the right hand. Following these results, researchers suggested that the right cerebrum differentially controls general arousal and attention and may lead to overall cerebral activation (see Tucker & Williamson, 1984, for review). Heilman, Bowers, and Valenstein (1993) argued that activation of the posterior cerebral regions, particularly the right parietal region and the right temporal region, is likely to result in emotional responses. Further, Heilman et al. (1985) used the Galvanic skin response (GSR) to demonstrate that right cerebrum dysfunction yielded altered levels of arousal and reactivity. Lesions within the right cerebrum were found to produce decrements in arousal as measured using GSR in response to provocative stimuli (Heilman et al., 1985). These findings offered additional support for the right cerebral mediation of emotion via increased arousal.

Balance Model of Emotion

In contrast to Heilman et al. (1985), Tucker and Frederick (1989) described a balance model of emotion. While Heilman et al. (1993) primarily considered the effects of lesions on emotions, Tucker and Frederick (1989) were more concerned with relative activation using electroencephalographic indices. Tucker and Frederick (1989) acknowledged that lesions and activation may relate to each other; however, their perspective differs from the right hemisphere model of emotion. According to the balance model, the left cerebrum primarily processes positive

emotions and the right cerebrum primarily processes negative emotions. Additionally, it was proposed that deactivation of one cerebrum leads to increased relative activation of the opposite cerebrum and, therefore, an increased expression of the dominant cerebrum's primary response pattern. Therefore, deactivation of the left cerebrum may result in an increase of negative emotion while deactivation of the right cerebrum may result in an increase of positive emotion.

Tucker acknowledged that the brain's processing of emotion is complex and likely involves the interaction of multiple systems that are only partially understood. Tucker cited comparative studies (e.g., Ploog, 1981) and human studies (e.g., Rinn, 1984) and suggested that emotional processing must integrate reflexive emotional displays that originate in the brainstem with ongoing behaviors. Emerson, Harrison, and Everhart (1999) indicated that this may occur by way of corticolimbic mechanisms which integrate ongoing experiences and behavior with reflexive affective representations in order to recruit emotional significance. A weakness of Tucker's model, however, is that it does not adequately address the resultant behavioral patterns following deactivation of both hemispheres. Further, there is no conclusive evidence within the literature that deactivation in one cerebrum necessarily leads to increased activation of the other cerebrum. In fact, it is possible that both the left and the right cerebrum may demonstrate concordant deactivation. Moreover, generalized arousal deficits have been found with right cerebral deactivation alone.

Circumplex Model of Emotion

Heller (1993) proposed a model of cerebral activation during emotional processing. Heller's circumplex model divides the brain into four quadrants, defined by the valence axis (pleasant or unpleasant) and the arousal axis (high and low). Most emotions fall within the four quadrants of these axes. Further, these dimensions correspond to distinct physiological responses (Heller, Nitschke, & Miller, 1998). For example, heart rate is related to valence and skin conductance is related to arousal. These dimensions are characterized by distinct cortical brain activation patterns. Specifically, the valence dimension is associated with the anterior regions of the brain and the arousal dimension is associated with the right posterior region.

Similarly, Davidson (1992) proposed that the anterior left cerebrum processes positive emotional expression and that the anterior right cerebrum processes negative emotional expression (Davidson, 1992; Kinsbourne & Bemporad, 1984). Davidson, Ekman, Saron, Senulis,

and Friesen (1990) demonstrated that disgust is associated with less alpha power (i.e., more activation) in the right frontal region and anterior temporal region than was happiness, while happiness was associated with less alpha power (more activation) in the left frontal region. Additionally, Tomarken, Davidson, Wheeler, and Doss (1992) found that individuals with extreme left frontal activation on baseline EEG reported more positive affect and less negative affect than those who demonstrated right frontal activation.

The reviewed neuropsychological theories of emotion are debated within the current literature. Additional experimental data are needed in order to test these contrasting models and to provide confirmation/disconfirmation of each model. The proposed experiments are designed to afford an increased understanding of these models. Results from the proposed experiments are discussed relative to each of the reviewed models.

Right Hemisphere Regulation of the ANS

The ANS is thought to be a regulatory system which transmits information from the central nervous system (CNS) to the effector organs of the body. The sympathetic nervous system (SNS), a branch of the ANS, produces a pattern of increased arousal in the body, with heightened heart rate and blood pressure. Axons of the parasympathetic (PNS) branch run to the same organs; however, their activation leads to quiescent effects (heart rate and blood pressure are reduced). Under stress, the SNS produces elevations in heart rate and blood pressure, while the PNS counters this effect when stress is absent (Green, 1994).

A comprehensive review of the ANS and subcortical systems is beyond the scope of this manuscript. Therefore, the reader is referred to Wittling (1995) for extensive information regarding lateralized anatomical projections of the ANS and comparative research.

The literature, which has examined changes in cardiac functioning following brain damage, is extensive. Caltagirone, Zoccolotti, Originale, Daniele, and Mammucari (1989) measured heart rate changes in 40 right-handed patients with unilateral right or left brain damage and in 20 normal controls. Participants viewed 20-second sequences of either an emotionally negative film or a neutral film. Left-brain damaged patients and control participants displayed a reliably higher decelerative response to the emotionally negative film than to the neutral film. In contrast, right brain-damaged patients did not differ in their cardiac responses to the films and had a clearly smaller decelerative response to the emotional film than both other groups.

Studies of unilateral hemispheric inactivation following intracarotid amobarbital injection also suggest asymmetrical ANS regulation. Rosen, Gur, Sussman, Gur, and Hurtig (1982) found higher heart rate increases in patients following left hemisphere inactivation than following right hemisphere inactivation. Similarly, Lane and Schwartz (1990) and Zamrini et al. (1990) assessed heart rate in patients with epilepsy undergoing unilateral intracarotid amobarbital injections and also found that heart rate increased during left hemisphere inactivation.

Differences in cardiovascular functioning have also been observed following lateralized presentation of stimuli. Hugdahl, Franzon, Andersson, and Walldebo (1983) exposed subjects either to a fear-related or to a neutral slide, presented repeatedly to the left (LVF) or to the right visual half-field (RVF) by means of tachistoscopic exposure. Subjects were told that the slide would always appear in the same visual hemifield. Anticipatory heart rate acceleration prior to slide onset was observed only if the stimuli were expected to appear in the left hemifield (right hemisphere). Dimond and Farrington (1977) also demonstrated heightened right hemispheric activation and enhanced sympathetic influence on heart rate by presenting a film of a surgical operation to the left and right hemispheres. Presentation of the film to the right hemisphere was associated with greater increments in heart rate than left hemisphere processing of the same film.

Wittling and Schweiger (1992) presented an emotionally aversive film to the left or right visual hemifield in 60 right handed subjects. Results suggest a higher correspondence between heart rate changes and changes in subjective arousal if the film is presented to the right versus the left hemisphere. Accordingly, Wittling (1997) developed a model of brain asymmetry and autonomic control of the heart. Consistent with Hugdahl's (1995) model, cortical control of autonomic cardiac regulation may be related to a division of responsibility between the cerebral hemispheres. The right hemisphere predominately controls sympathetic activity, whereas the left hemisphere predominately controls parasympathetic activity. The role of the right hemisphere in the modulation of sympathetic activity follows from its strong impact on heart rate acceleration. The role of the left hemisphere in the control of parasympathetic activity has been observed during lateralized film presentation and heart rate deceleration (Wittling, 1997; Yoon, Morillo, Cechetto, & Hachinski, 1997).

Wittling (1997) further described the neuroanatomical model of autonomic cardiac control. It is believed that ipsilateral pathways connect brainstem areas with heart ganglia,

whereas cortical areas involved in cardiac control may tend to project bilaterally to the preganglionic neurons in the brainstem (Gatti, Johnson, & Massari, 1996; Massari, Johnson, & Gatti, 1995). Loewy's (1990) model also posited that descending fibers connecting higher and lower brainstem areas and cardiac ganglia predominately pass on the ipsilateral side. Further, there is evidence that cortical areas such as the medial frontal and the insular cortex are involved in autonomic modulation of cardiac activity. These areas have substantial bilateral projections to brainstem areas that are important for efferent modulation of sympathetic and parasympathetic outflow (Shipley, 1982; Shipley & Sanders, 1982; Terreberry & Neafsey, 1983).

Hemispheric Response Systems

Wittling, Block, Schweiger, and Genzel (1998) postulate that, while the two hemispheres differ in emotion-related processing, it is the unique role of the cerebral cortex to modulate the lower brain stem areas in coordination with behavioral interactions, internal needs, and other cognitive and emotional responses. Accordingly, differences in the hemisphere's relationships and attitudes towards internal and external environments should be reflected in asymmetric hemispheric organization (Wittling, Block, Genzel, & Schweiger, 1998). While hemispheric differences are not restricted to single response parameters, both hemispheres have unique response systems that differ from one another (Davidson & Hugdahl, 1995).

The right hemisphere's response system appears to deal with the challenges of the external environment, defending the individual against potential threat, and preparing the organism to respond to external challenges in an efficient way. Thus, Hellige (1995) argued that the right hemisphere is endowed with efficient perceptual mechanisms to analyze sensory information. Further, neural systems mediating arousal, vigilance, and outward-directed attention are represented in the right hemisphere (Heilman, 1995). This hemisphere has been found to be activated by stress and emotional situations of negative emotions and tries to cope through intense feelings like anger and fear (Davidson, 1995). Further, the right hemisphere appears to predominately control the sympathetic modulation of the heart and the main stress mechanisms (Wittling, 1997). In addition, the right hemisphere appears to also regulate arterial blood pressure, renal sympathetic nerve discharge, and cardiac arrhythmias (Wittling 1997). Taken together, the right hemispheric control of cardiac reactivity is consistent with a response system that efficiently prepares an individual to respond to external challenges.

Related to the cognitive and language facilities, the left hemisphere's response system is more engaged in the internal symbolic representation of environmental events and seems to promote disengagement to undesirable external events (Wittling, 1997). Similarly, the left hemisphere responds less spontaneously, abruptly, and vividly to negative emotional situations and shows more signs of emotional indifference (Gainotti, 1989; Wittling, 1995). The left hemisphere is closely associated with reward and incentive events and responds strongly to these situations (Davidson, 1995). Further, the left hemisphere contributes to reducing the effects of sympathetic activation, by exerting control over parasympathetic outflow to the heart and enhancing immune responsiveness of T cell-dependent immune parameters (Kang et al., 1991; Wittling, 1997). Accordingly, the left hemisphere's response system primarily maintains homeostasis, counteracts environmental stress, and promotes defense responses.

In summary, research implicates asymmetrical hemispheric regulation of heart rate and other functions of the ANS (i.e., blood pressure, GSR, and skin temperature). Wittling (1990) experimentally showed subjects an emotionally positive film to either their RVF or LVF. Right-hemisphere presentation of the emotionally-laden film resulted in heightened systolic and diastolic blood pressure beyond that which followed presentation to the left-hemisphere. As perception of emotions is hypothesized to reflect right posterior cerebral processes, the perception of emotionally laden films by the right posterior regions may have served to increase blood pressure. These results are consistent with the current model of asymmetrical hemispheric regulation of the ANS, with specialization of the right hemisphere for sympathetic activation and specialization of the left hemisphere for parasympathetic activation.

Neuropsychology of Hostility

Hostility and its physiological correlates have been studied frequently in the health psychology literature. Although most of the relevant literature is clinical in nature, some experimental evidence is provided in the area of emotional perception. Smith (1994) offers the following description of the construct of hostility:

> "It [hostility] has been defined previously as the tendency to wish to inflict harm on others or the tendency to feel anger toward others (Chaplin, 1982), although these definitions clearly blur the distinctions among emotion, cognition, and behavior. In part, hostility entails a negative attitude toward others, consisting of enmity,

> denigration, and ill will. Cynicism is a closely related belief that other people are motivated by selfishness rather than by concern for others or by similar higher motives. Mistrust is an associated expectancy that people are unlikely to fulfill obligations and are frequent sources of mistreatment, provocation, and harm, despite the fact that they might appear to be friendly and cooperative. Thus, as an enduring, general trait, hostility connotes a devaluation of the worth and motives of others, an expectation that others are likely sources of wrong doing, a relational view of being in opposition toward others, and a desire to inflict harm or see others harmed" (p. 26).

Hostility has also been defined as a combination of angerability, cynicism (Houston, 1992), and antagonistic behaviors (Williams & Williams, 1993), as well as heightened physiological reactivity (Smith & Allred, 1989).

Recently, research has begun to focus on the physiological correlates of hostility and the cerebral mediation of hostility. This research has provided evidence for an increased understanding of hostility from a neuropsychological perspective. Hostility has been theorized to be central to right cerebrum activation (Demaree & Harrison, 1997a, in press; Herridge, Harrison, & Demaree, 1997). In a recent review of hostility and self-awareness, Demaree and Harrison (1997b) suggested that hostility and self-awareness are strongly linked to the right cerebrum, and hostility and self-awareness may be negatively related. Earlier research demonstrated the relationship between the orbital-frontal and anterior temporal regions in the regulation of emotion expression. Specifically, the right orbital-frontal cortex appears to decrease hostility levels (Butter, Snyder, & McDonald, 1970). Activation of the anterior temporal region has been found to yield heightened anger or rage behaviors. This area has extensive interconnections with the orbital-frontal cortex. Activation of the right orbital-frontal region has been found to reduce anger or rage behaviors. It has been hypothesized that the anterior temporal region and the orbital-frontal cortex interact to form a constant, conservative aggression level among normal individuals (Heilman et al., 1993).

Furthermore, if the construct of hostility continues to hold up within the neuropsychological framework, then we may be able to develop therapies that have implications

for the mediation of hostility. Cognitive tasks may be developed that are useful in mediating hostility. Continued clinical and experimental research will build upon what little is now known about the neuropsychology of hostility.

Supportive Research

In the past, clinical research has offered support for the theory of right cerebral mediation of hostility. In order to learn more about hostility, past research has focused on constructs such as aggression, rage, and negative affect. Cummings and Mendez (1984) reported that individuals with right cerebrum infarctions displayed affect and mood changes. Similarly, and more recently, decreased orbital-frontal and increased right temporal beta activation has been shown in a patient with anger-control difficulties and a homicidal patient using electroencephalographic techniques. This evidence supports the theory of the right cerebrum's role in mediating hostility (Demaree & Harrison, 1996). Continued clinical research which is relevant to the neuropsychology of hostility is necessary for an increased understanding of the cerebrum's mediation of hostility.

Experimental investigations of hostility and its effects on the bilateral processing and recognition of stimuli have led to research on emotional sensation and emotional perception. Research has shown that there is faster affect perception by the right cerebrum (LVF) than the left cerebrum (RVF), and that there are hemispheric differences related to affective valence (Harrison, Gorelczenko, & Cook, 1990). In this experiment, a forced-choice (i.e., happy or angry) tachistoscopic method was used presenting happy, angry, or neutral faces. The hostile participants evidenced an angry bias in the left visual field. Specifically, neutral faces were perceived as angry by the hostile participants. This bias was restricted to the left visual field, suggesting increased activation of the right cerebral systems among high-hostiles. Likewise, Herridge et al. (1997) found evidence that hostile participants less accurate at assessing a neutral affective face in their left visual field than low-hostile participants. This experiment demonstrated that hostile individuals show diminished accuracy with angry faces subsequent to a painful cold-pressor (CP) stimulus. Further, a negative affect bias was found for hostile individuals.

Continued experimental research on hostility will afford a better and more comprehensive understanding of the correlates of hostility. High-hostile individuals differ from low-hostile individuals in affect perception. Likewise, high-hostile individuals demonstrate increased cardiovascular reactivity compared to low-hostile individuals. Continued research examining the

role of hostility, affect perception, and cardiovascular reactivity will provide an increased understanding of cardiovascular risks.

Cardiovascular Reactivity and Hostility

Coronary heart disease and stress have been the focus of many research projects. Historically, research has focused on Type A individuals with elevated risks for coronary heart disease. Increased reactivity to stress has been shown in Type A individuals through numerous cardiovascular measures including blood pressure (BP) and heart rate (HR) (Dembroski, MacDougall, Herd, & Shields, 1983; Glass & Contrada, 1982; Harbin, 1989). Numerous studies have indicated that hostility may be the primary "toxic" component of Type A behavior pattern (see Matthews & Haynes, 1986 for review). Specifically, Krantz, Manuck, and Wing (1986) suggest that cardiovascular responses are related to an individual's perception. Therefore, an affective event produces a cardiovascular response if the individual's perception of the event is hostile, regardless of whether or not the event is in fact hostile. Harrison and Gorelczenko (1990) utilized a tachistoscopic method, which was sensitive to hemispheric processing differences, to investigate functional cerebral asymmetries for the identification of happy and angry faces for high- and low-hostile men and women. Results of this project suggested that high- and low-hostile individuals differ in their perception of affective stimuli.

Hostility from a Functional Cerebral Distance Model

Historically, cerebral functioning has been conceptionalized as the study of static, localized "centers." Researchers have described these areas as fixed functional areas. Recently, however, researchers have begun to view the role of the cerebral hemispheres and brainstem from a system's viewpoint. The mediational role of the cerebral hemispheres are beginning to be conceptualized as a dynamic interplay of regional cerebral activation.

The functional cerebral distance (FCD) principle was introduced to address how the brain may organize and concurrently process dual- or multiple-task requirements (Kinsbourne & Hicks, 1978). This principle stems from understanding the brain as a differentiated neural network. Localized networks, specialized for different functions, are connected with all other local networks (Kinsbourne & Hiscock, 1983). Therefore, activation in one functional region may spread throughout much of the cerebral cortex via the unifying dendritic network (Purpura, 1967).

The functional cerebral distance principle affords the following three principles. First, localization of function refers to the ability of particular regions of the cortex to generate specific patterns of neuronal firing in response to task demands. Second, regions that generate similar neuronal patterns are highly interconnected. Third, interconnections allow for efficient representation of many variations within a category; however, this interconnection among similar local networks also makes it difficult to perform two activities concurrently.

Kinsbourne (1970) demonstrated the FCD principle in a left hemisphere priming task. Participants were requested to detect the presence of gaps in squares displayed in two conditions. The verbal load condition required rehearsal of six one-syllable words during the gap detection task, whereas no rehearsal was performed in the standard condition. While the hemispheres were equally accurate in gap recognition in the standard condition, the performance of the left hemisphere exceeded that of the right hemisphere during the verbal load condition (a left hemisphere priming task).

Recently, the FCD principle has been refined to distinguish priming effects from interference effects. With light demands, concurrent tasks spread activation and facilitate processing (priming). More demanding tasks, however, may tax a functional system and decrease processing capacity for other tasks (i.e., interference) (Kinsbourne & Hiscock, 1983). For example, Harrison (1991) found that reading interfered more with right than left hand and elbow tapping, presumably due to competition for similar functional cerebral space. Also, Demakis and Harrison (1994) found evidence of overloading of the left hemisphere's affective perceptual capabilities in an affective subvocalization and tachistoscopic study.

It is thought that interference occurs when two concurrent tasks (e.g., speaking and right hand motor performance) compete for similar functional cerebral areas. A number of experiments provide support for both priming and interference effects on numerous tasks (Bouma, 1987; Demakis & Harrison, 1994; Harrison, 1991; Hellige & Cox, 1976; Hellige, Cox, & Litvac, 1979; Kinsbourne & Byrd, 1985).

A number of experiments indicate that affective stimuli may preferentially prime the right hemisphere. For example, Brody, Goodman, Holm, Krinzman, and Sebrechts (1987) used affective stimuli as primes and found reliable reaction time improvements for affective target

stimuli presented to the right hemisphere. Other studies have also examined the use of affective stimuli in priming the right hemisphere (Bryden & Ley, 1980,1983; McKeever & Dixon, 1981).

One purpose of this experiment is to apply the FCD principle to the functional cerebral regulation of the autonomic nervous system. Specifically, the brain's ability to regulate autonomic nervous system functioning and how it may relate to affective verbal learning tasks is explored. Recent research implicates the proposed role of the right hemisphere in the regulation of autonomic nervous system functioning, including heart rate and blood pressure. As Kinsbourne and Byrd (1985) demonstrated, the effort or load of a task is integral to explain priming or interference effects. It is hypothesized that cold- pressor (CP) stress for high-hostile individuals produces an interference effect with ongoing cardiovascular control, leading to cardiovascular disregulation. The relationship between hostility and the decompensation in cardiovascular regulation is suggestive of a dual-task interference effect. Under dual-task demands (i.e., hostility and cardiovascular control) the secondary task of cardiovascular control may suffer.

Role of Stress

In order to study hostility and cardiovascular reactivity, many researchers have utilized stress induction. Cardiovascular measures such as heart rate (HR), mean arterial pressure (MAP), systolic blood pressure (SBP), and diastolic blood pressure (DBP) have served as traditional dependent measures to establish an indicant of heightened reactivity to stress (e.g., Schneider, Julius, & Karunas, 1989; Turner, Sherwood, & Light, 1992) and a predictor indicant for cardiovascular risks (e.g., Suls & Wan, 1993). A variety of stimuli have been shown to be associated with increased cardiovascular reactivity and are generally considered to impose stress of a psychological nature. Stimuli range form laboratory tasks, such as aversive reaction time tasks (Obrist et al., 1978), mental arithmetic tasks (Brod, Fencl, Hejl, & Jirka, 1959), and video games (Turner, Carroll, & Courtney, 1983) to interpersonal stressors as presented by interview procedures (Dimsdale, Stern, & Dillon, 1988) or public speaking (Gliner, Bunnell, & Horvath, 1982). Several studies have indicated that the sympathetic nervous system plays the primary role in mediating cardiovascular response during psychological stress (Dimsdale & Moss, 1980; Langer et al., 1985; Sherwood, Allen, Obrist, & Langer, 1986; Wittling, 1997).

It has been suggested that cardiovascular reactivity to stress may be differentially mediated by the right and left cerebrum (Demaree & Harrison, 1997a, in press). The view of the right

cerebrum's role in hostility will be incorporated with implications for the role of the right cerebrum in cardiovascular reactivity with incremental heart rate and blood pressure for high-hostile individuals. Left cerebral activation, using a dichotic listening procedure (Demaree & Harrison, 1997a; Shenal & Harrison, 1999) and quantitative electroencephalography (Demaree & Harrison, in press), has been implicated in cardiovascular stability and in decreased blood pressure. The latter finding corresponds with positive emotional experiences and with the learning of a positive verbal list in recent research (Snyder, Harrison, & Shenal, 1998).

Recently, cerebral lateralization has been investigated in response to physical and cognitive stressors (Demaree & Harrison, 1997a; Snyder et al., 1998). Findings indicate that hostile individuals evidence right cerebrum activation following stress while low-hostile individuals evidence a diametrically opposite activation of the left cerebrum following stress (Demaree & Harrison, in press). The present experiment continues the study of the dynamic cerebral lateralization effect and potential applications for education and the prevention of cardiovascular risks.

Stress Induction

In order to study the role of painful stress on cardiovascular reactivity, stress has traditionally been induced by methods such as the cold-pressor task. This procedure, which requires participants to place their hand in cold water, has been shown to heighten arousal and to increase cardiovascular activity (e.g., Demaree & Harrison, 1997a, in press; Glass, McKinney, Hofschire, & Fedorko, 1990). This stressor is the most widely studied stressor in the "passive" coping category which is typically associated with sympathetic activation leading to pressor responses mediated by the vasoconstrictive action of alpha-adrenergic receptor stimulation (Peckerman et al., 1991). Other, "active coping" tasks (Obrist, 1976), are sometimes chosen that elicit responses mediated by the sympathetic nervous system and resemble the defense reaction which lead to stimulation of cardiac and vascular beta-adrenergic receptors (Sherwood & Turner, 1992).

It has been demonstrated that high-hostile individuals show greater cardiovascular reactivity (BP and HR) to the cold-pressor task than low-hostiles individuals (Dembroski, MacDougall, Herd, & Shields, 1979). It is suggested that the painful cold-pressor task activates the right cerebrum in these individuals thereby increasing cardiovascular reactivity. Past research

has supported the notion of right cerebrum involvement during stress and during arousal inducing conditions (Heilman & Van Den Abell, 1979; Tucker, Roth, Arneson, & Buckingham, 1977). Tasks, such as the painful cold-pressor, which have been shown to induce painful stress, provide a method for testing these hypotheses.

Affective Auditory Verbal Learning

Additional tasks thought to induce stress and arousal have recently been pursued and developed. Snyder and Harrison (1997) recently developed the Affective Auditory Verbal Learning Test (AAVL) consisting of lists of positively and negatively valenced words. The negative and positive emotional lists were designed to be analogous to those on the Rey Auditory Verbal Learning Test (RAVL) but to vary on the affect dimension. The RAVL has been used extensively by researchers to study the acquisition of verbal information, as well as primacy, recency, and interference effects (Rey, 1964). The new version consists of three word lists: a negatively valenced word list, a positively valenced word list, and the original RAVL (neutral list).

The AAVL was designed in order to provide an objective measure of auditory affective verbal processing. Since verbal processing is predominantly a left hemisphere task (Hugdahl & Anderson, 1987) and emotional processing has also been reported to be lateralized (Bryden & MacRae, 1989), the AAVL was designed to investigate differences in affective learning among groups such as high- and low- hostile participants (Snyder & Harrison, 1997). The positive and negative lists were developed using an index of word norms (Toglia & Battig, 1978), which provides a rating of words on a 7-point Likert Scale for attributes including familiarity and pleasantness. To assess the suitability of the AAVL as a test of affective induction, list learning patterns (e.g., acquisition, primacy, and recency) for the original RAVL word list were compared with the new affective lists. Snyder and Harrison (1997) found a heightened primacy effect for participants learning the negative list (potential persistence of negative affect) and a heightened recency effect for participants learning the positive list (potential impersistence of positive affect).

AAVL and ANS Arousal

In an experiment subsequent to the Affective Auditory Verbal Learning Test development, the affective lists were shown to alter cardiovascular functions (Snyder et al., 1998). This experiment assessed the utility of the AAVL in leading to psychophysiological patterns characteristic of the experience of an emotion. Ax (1953) initially demonstrated that specific

ANS patterns might occur for specific emotions. Subsequently, Schacter (1957) and Roberts and Weerts (1982) replicated the findings of Ax (1953), with blood pressure and skin conductance evidencing greater increases following angry and fearful imagery scenes compared to happy imagery scenes.

Research demonstrating specific patterns of ANS reactivity often utilizes techniques whereby participants are asked to recite affective words. Schwartz, Weinberger, and Singer (1981) requested participants to verbally describe emotional imagery. Weerts and Roberts (1976), as well as Izard (1977), requested participants to verbalize feelings associated with emotional events in their life. Hence, stating emotional words or verbally describing emotional imagery purportedly results in specific ANS patterns (heart rate, blood pressure, skin conductance, respiration rate, etc.) for specific emotional valences. Reciting negatively valenced words is associated with greater pulse rate and blood pressure reactivity, with slower recovery to baseline levels than that resulting from positive or neutral verbalizations (Izard, 1977; McNaughtan, 1989; Schwartz et al., 1981; Weerts & Roberts, 1976). Conversely, reciting positively valenced words is associated with less reactivity and faster recovery of baseline, than negative or neutral verbalizations (Izard, 1977; McNaughton, 1989).

Snyder et al. (1998) hypothesized that since the verbalization of affectively valenced words is associated with differential pulse rate and blood pressure patterns, it was plausible that attempting to learn emotionally valenced words and being prompted to recall them verbally would lead to differential ANS patterns. Listening to the experimenter read affective words, subvocally rehearsing the affective words, and then verbally recalling the affective words was hypothesized to produce differential ANS pulse rate and blood pressure patterns for specific emotions. The negative affective list learning task was shown to increase blood pressure; whereas the positive affective list was shown to decrease blood pressure. It was hypothesized that the increased cardiovascular reactivity may be accounted for by right cerebrum activation (Snyder et al., 1998). This reactivity may be accounted for by dual-task processing of affect and cardiovascular reactivity with decrements of the regulation occurring on the latter. These results suggested that the AAVL may be useful as an empirical tool for emotion/arousal induction, since blood pressure significantly increased following the learning of the negative word list and significantly decreased following the learning of the positive list (Snyder et al., 1998).

Rationale

Findings of the previously reviewed research indicate that physical stress (e.g., pain), as well as cognitive stress (e.g., affective verbal learning), may induce heightened right cerebral activation for high-hostiles. As Demaree and Harrison (1997a, in press) point out, the use of methods more sensitive to brain lateralization may produce additional useful data about dynamic cerebral lateralization. Snyder et al. (1998) demonstrated that the negative affective learning task increased cardiovascular reactivity and that the positive affective learning task decreased cardiovascular reactivity. Specifically, individuals' heart rate and blood pressure increased following the negative affective list learning task and decreased following the positive affective list learning task. It was suggested that the cognitive challenge of negative list learning was an analogous stressor to the physical cold-pressor stressor, whereas, the positive list learning served an opposite function. An increased understanding of the effect of affective list learning on cardiovascular reactivity may lead to future prevention and therapeutic techniques for cardiovascular disease. For instance, minimizing negative affective cognitive tasks and rehearsing positive affective verbal word lists may be shown to decrease cardiovascular reactivity. These methods of decreasing cardiovascular reactivity may provide an efficient and effective prevention method. Further, if the negative affective list learning task is demonstrated to be analogous to the painful cold-pressor stressor, then affective list learning may provide researchers with a cognitive task that is an effective stressor, yet one that does not require an invasive, painful procedure.

The purpose of the following experiment was to learn more about the role of stress as a risk factor of heart disease by improving the understanding of the neuropsychological explanations of chronicity and pervasiveness of a hostile disposition. Further, this experiment was designed to demonstrate dynamic cerebral lateralization to affective auditory verbal learning and to physical cold-pressor stress by comparing high- and low-hostile individuals' cardiovascular reactivity and affective word recall (both positive and negative affective valence) following stress.

Experimental Overview

The following experiment examined the principle of functional cerebral distance (FCD) with respect to hostility. This study adds valuable information to further the understanding of hemispheric activation among hostile individuals through the investigation of the right

hemisphere's proposed role in the regulation of ANS activation, negative and positive affective word learning tasks, and performance on neuropsychological tasks under a laboratory stressor.

The experimental model describes a system where stress mediates the individual's affective word recall performance and cardiovascular reactivity. The experimental group was comprised of high- and low-hostile participants. Half of the participants in each group were assigned to the no-stress condition (no cold-pressor) and half to the stress condition (cold-pressor). The painful cold-pressor task was used to induce stress. Participants completed both the negative and positive affective list learning tasks. Cardiovascular measures were recorded following the stress or no-stress condition as well as before and after each list learning task. These data were used to investigate how high- and low-hostile participants differ on affective learning and cardiovascular reactivity. Further, the effect of stress on each group and condition was investigated.

This experiment investigated autonomic reactivity (HR, MAP, SBP, DBP) to a positive and negative affective list learning task among high- and low-hostile individuals. Based on previous findings of heightened ANS reactivity among high-hostile individuals, it was hypothesized that high-hostiles would demonstrate heightened autonomic reactivity to the negative list learning task (see Appendix A). This study was considered novel given that previous studies of ANS arousal and the AAVL had not investigated the reactivity to this neuropsychological test with high-hostiles. This experiment also investigated functional cerebral asymmetry using performance on a neuropsychological test (AAVL) among high- and low-hostile individuals. A positive list (possible left hemisphere task) and negative list (possible right hemisphere task) were used to assess hemispheric functioning among high- and low-hostile individuals.

The experiment also assessed ANS functioning and performance on lateralized neuropsychological tasks in the face of an ANS stressor. The effects of the cold-pressor stimulus on ANS reactivity and positive and negative list-learning were examined. It was hypothesized that administration of the cold-pressor would differentially affect the high-hostile group's performance on the negative list learning task when compared to low-hostile individuals. ANS activation was also monitored throughout the duration of this task. High-hostile individuals were expected to display more reactivity to both stimuli (cold-pressor and negative list learning task) than the low-hostile individuals.

Variables

Participants were classified as high- or low-hostile based on their scores on the Cook-Medley Hostility Scale (CMHS).

Two categories of dependent variables were used in this experiment. First, cardiovascular indicators were be SBP, DBP, MAP, and HR. Second, learning performance was measured as the total number of words recalled, as well as the total number of words recalled from the first five words of each trial (primacy effect), the middle five words of each trial, and the last five words of each trial (recency effect) on each of the five trials for both affectively valenced lists.

Method

Participants

Group testing at Virginia Tech, a large state university, yielded the experimental groups composed of high-hostile and low-hostile men. Volunteer participants were recruited from the Virginia Tech undergraduate pool. A sign-up folder, explaining the experiment and scheduled screening times, was made available to undergraduates. The participants were informed that this experiment includes two phases (group screening phase and experimental phase) and that they would receive one credit point for each session that they attended. Further, they were informed that participation in the group screening phase did not guarantee inclusion in the experimental phase. 183 participants were screened to complete the final participant pool needed for the experiment (n = 64).

Inventories and Questionnaires

The screening sessions involved groups of 10 - 30 participants. An Informed Consent Form (Appendix B) was completed by all participants describing the experiment and indicating that they may be requested for further testing. This consent form included pertinent information on the nature, purpose, and procedures of the experiment, as well as a description of potential risks and benefits of the experiment. Further, the Informed Consent Form stated that the participants' identity would remain confidential and that they were free to withdraw at any time.

A Lateral Preference Questionnaire (Appendix C), consisting of thirteen items assessing four types of lateral preference (hand, foot, eye, and ear), was then distributed to the participants (Coren, Porac, & Duncan, 1979). This questionnaire was developed from several preexisting laterality inventories, as well as behavioral tests of lateral preference. The items were self report, and were scored as a +1 for "right", -1 for "left", and 0 for "both." The criterion for right hand dominance and inclusion in the experiment was a total score of +6 or greater on this questionnaire.

For inclusion, participants must have self-reported no history of hearing difficulties, major illness, or major head injury on the 18 question Medical History Questionnaire (Appendix D). Participants who did not meet the above criteria for handedness and medical history were informed that they would not be asked to participate in the experimental phase of the project.

The Cook-Medley Hostility Scale (CMHS) (Appendix E) was then administered to the group (Cook & Medley, 1954). This scale consists of a 50 question, true - false inventory assessing hostility which has been used as a valid predictor of medical, psychological, and interpersonal outcomes (Contrada & Jussim, 1992). Participants who scored 20 or below on the scale (CMHS), and who met the above criteria, were asked to continue with the experimental phase and comprised the "low-hostile" group (n = 32). Likewise, participants who scored 28 or above on the scale (CMHS), and who met the above criteria, were asked to continue with the experimental phase and comprised the "high-hostile" group (n = 32). Participants who scored in the range of 21 to 27 points on the scale (CMHS) during group screening were notified that they would not be asked to participate in the experimental phase of the experiment. These self-report cutoffs replicate those of previous similar research projects (Demaree & Harrison, 1997a; Shenal & Harrison, 1999).

The participants chosen for the experimental phase (n = 64) were contacted and scheduled for further testing. They were informed that they would receive another credit point for their participation and that the experiment would take approximately 45 minutes.

Apparatus

The participants were seated in a sound-attenuated room. The automated programming equipment and the experimenter were located in an adjacent room. The participants were monitored through a one-way observation window and were prompted via an intercom. All instructions and auditory stimuli were prerecorded on a Verbatim Data Life Plus recordable compact disc. Stimuli were presented at about 75 dB by a JVC PC-X300 CD Portable System compact disc player using Koss Pro/4X Plus headphones.

Cardiovascular

Systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial pressure (MAP), and heart rate (HR) were assessed using the Industrial and Biomedical Sensors Corporation SD-700A Blood Pressure / Pulse Monitor. Systolic blood pressure, diastolic blood pressure, and mean arterial pressure were assessed using the Korotkoff method. A preset inflation pressure setting of 180 mmHg was used for all subjects. The exhaust rate was 3 mmHg/sec. Exhaust was performed automatically following the reading. The procedure adhered to the basic requirements of the Association for the Advancement of Medical Instrumentation and

the American Heart Association (see Harrison, Gorelczenko, & Kelly, 1988). The left arm of the participants was partially extended, supported, and positioned at about the fourth intercostal space with the palmar surface facing upward. The cuff was positioned on the left upper arm with the cuff connector located over the brachial artery about 2.5 cm above the antecubital space. Arterial location was determined by palpation. In the event of an error reading, the cuff was removed and replaced to assure correct placement over the brachial artery.

Cold-Pressor (Autonomic Nervous System Prime)

The cold-pressor (CP) stimulus consisted of an insulated container (18" x 12" x12") filled with crushed ice and water. The container was maintained at approximately the same level as the heart. A constant temperature, below 5°C, was maintained. Participants were asked to place their left hand and wrist in the container for a period of 45 seconds when instructed. Following each CP trial, the participant was asked to rest his hand in a position over the open container. Participants were also instructed that they could remove their hand from the ice water in the event of heightened discomfort, and that they would not be penalized in any fashion should this occur. All participants that received the cold-pressor completed the experiment. Participants in the no-stress condition completed the same procedures; however, the participants placed their hand and wrists in an empty container instead of a container containing ice water.

Affective Auditory Verbal Learning Test

The affective auditory verbal learning (AAVL) procedure consists of three (positive, negative, neutral) separate 15-item word lists (Snyder & Harrison, 1997; Snyder et al., 1998). The negatively valenced word list (Appendix F) and the positively valenced word list (Appendix G) were used in this experiment. Construction of the negative affective list of the AAVL consisted of selecting 15 words having the lowest mean pleasantness (PLS) rating from the subset of all words with a familiarity (FAM) rating of at least 5.0 or greater (Toglia & Battig, 1978). An equal proportion of one syllable and two syllable affective words were chosen. Construction of the positive affective list of the AAVL consisted of selecting 15 words having the highest mean pleasantness (PLS) rating from the subset of all words with a familiarity (FAM) rating of at least 5.0 or greater (Toglia & Battig, 1978). An equal proportion of one syllable and two syllable affective words were chosen. Refer to Appendix H for the FAM and PLS ratings of each of the words in the positively and negatively valenced word lists. Snyder et al., (1998) demonstrated

that the negative affective learning task increased ANS arousal and that the positive affective learning task decreased ANS arousal. Specifically, individuals' heart rate and blood pressure increased following the negative affective list learning task and decreased following the positive affective list learning task.

Procedure

No-Stress Condition

Following group classification procedures, 16 high-hostile and 16 low-hostile participants were assigned to participate in the no-stress condition. Upon arrival, all participants were habituated to the laboratory and upright seated position for five minutes. Participants were informed that they would be asked to place their left hand in an empty container (no-CP); that their blood pressure and heart rate would be recorded; and that they would be asked to complete two tasks (positive and negative affective lists). Heart rate (HR), systolic blood pressure (SBP), and diastolic blood pressure (DBP) data was recorded. Immediately following recording of the cardiovascular data, the participant was asked to place his left hand in the container, leaving it there until the experimenter requested removal. The participant was required to leave his hand in the container for 45 seconds. Upon removal of the left hand, SBP, DBP, and HR was recorded again with the use of the automated monitoring equipment. Participants were then administered instructions for completion of either the positive or the negative list. Participants received the following instructions:

> "I am going to read you a list of words. Please listen carefully. When I stop, you are to say back as many words as you can remember. Say the words in any order you can remember. Just try to remember as many as you can."

Instructions for Trial 2 through Trial 5 were as follows:

> "Now I'm going to read the same list again. When I stop, again I want you to tell me as many words as you can remember, including words you said the first time. It doesn't matter what order you say them. Just say as many words as you can remember whether or not you said them before."

Participants were then administered the positive or negative list for a series of five trials. Words were read to the participants at a rate of approximately one word per second. When the participant could no longer recall additional words, the next trial began. Participants' responses

were recorded on the data sheet (see Appendix I). Order of administration of the lists was alternated across participants in a partially counterbalanced fashion in order to control for potential order effects. Cardiovascular measurements (SBP, DBP, HR) were taken immediately following completion of each positive/negative affective list, and three minutes post-completion of the task. Presentation of the second task (either positive or negative list) proceeded in an identical fashion, with cardiovascular measurements taken at identical periods of time. Upon completion of both list learning tasks, participants were debriefed and excused from the experiment.

Stress Condition

Following group classification procedures, 16 high-hostile and 16 low-hostile participants were assigned to participate in the stress condition. Upon arrival, all participants were habituated to the laboratory and upright seated position for five minutes. Participants were informed that they would be asked to place their left hand in ice water (CP); that their blood pressure and heart rate would be recorded; and that they would be asked to complete two tasks (positive and negative affective lists). Heart rate (HR), systolic blood pressure (SBP), and diastolic blood pressure (DBP) data were recorded. Immediately following recording of the cardiovascular data, the participant was asked to place his left hand in the ice water, leaving it there until the experimenter requested removal. The participant was required to leave his hand in the water for 45 seconds. Participants were also told that they could remove their hand from the water at any time without penalty should the discomfort become too painful. Upon removal of the left hand, SBP, DBP, and HR were recorded again with the use of the automated monitoring equipment. Participants were then administered instructions for completion of either the positive or the negative list. Participants received the following instructions:

> "I am going to read you a list of words. Please listen carefully. When I stop, you are to say back as many words as you can remember. Say the words in any order you can remember. Just try to remember as many as you can."

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It doesn't matter what order you say them. Just say as many words as you can remember whether or not you said them before."

Participants were then administered the positive or negative list for a series of five trials. Words were read to the participants at a rate of approximately one word per second. When the participant could no longer recall additional words, the next trial began. Participants' responses were recorded on the data sheet (see Appendix I). Order of administration of the lists was alternated across participants in a partially counterbalanced fashion in order to control for potential order effects. Cardiovascular measurements (SBP, DBP, HR) were taken immediately following completion of each positive/negative affective list, and three minutes post-completion of the task. Presentation of the second task (either positive or negative list) proceeded in an identical fashion, with cardiovascular measurements taken at identical periods of time. Upon completion of both list learning tasks, participants were debriefed and excused from the experiment.

Analysis

Descriptive Measures

T-tests were conducted to assess differences among high-hostile and low-hostile participants on the CMHS hostility questionnaire and the CPD handedness questionnaire. A priori criterion for reliability was $p \leq .05$ for all analyses.

Cardiovascular Reactivity

In order to test the cardiovascular reactivity hypotheses, data analysis consisted of separate four factor mixed design analyses of variance (ANOVAs) consisting of Group x Condition x (Valence x Trial x Subject) for the cardiovascular dependent measures of MAP, SBP, DBP, and HR. Fixed effects consisted of Group (high- and low-hostile participants) and Condition (no cold-pressor stressor and cold-pressor stressor). Repeated measures were Valence (positive list and negative list) and Trial (Trial 1, Trial 2, Trial 3, and Trial 4). Trial 1 represents the cardiovascular data immediately prior to the stress/no-stress condition, whereas Trial 2 represents the cardiovascular data immediately following the stress/no-stress condition. Trial 3 represents the cardiovascular data immediately prior to the affective list learning task, whereas Trial 4 represents the cardiovascular data immediately following the affective list learning task.

Affective List Learning

In order to test the affective list learning hypotheses, data analysis consisted of an analysis of variance (ANOVA) for the affective list learning variable of total words per location for each trial. Data analysis consisted of a five factor mixed design analysis of variance (ANOVA) consisting of Group x Condition x (Valence x Trial x Location x Subject). Fixed effects consisted of Group (high- and low-hostile participants) and Condition (no cold-pressor stressor and cold-pressor stressor). Repeated measures consisted of Valence (positive list and negative list), Trial (Trial 1, Trial 2, Trial 3, Trial 4, Trial 5), and Location (first third, middle third, and last third of the list).

Results

Descriptive Measures

T-tests were conducted on scores obtained on the Cook-Medley Hostility Scale and the Coren, Porac, and Duncan Laterality Questionnaire in order to compare groups (high- and low-hostiles) on the descriptive measures. Table 1 provides a summary of the group means and the standard deviations for each measure.

High-hostiles scored significantly higher on the CMHS (M = 31.84, SD = 2.94) than low-hostiles (M = 14.78, SD = 3.07) , $t(62) = 22.72$, $p \leq .05$.

On the Coren, Porac, and Duncan Laterality Questionnaire, high-hostiles (M = 10.69, SD = 2.68) did not reliably differ from the low-hostiles (M = 10.38, SD = 2.30), $t(62) = .50$, $p \geq .05$.

Cardiovascular Reactivity

Group means and standard deviations of cardiovascular measures (MAP, SBP, DBP, and HR) are displayed in Table 2. Separate ANOVAs with the fixed factors of Group and Condition and with the repeated measures of Valence and Trial were performed on each of the four cardiovascular variables - MAP, SBP, DBP, and HR. All pairwise comparisons were made using Tukey's Studentized Range Test (see Winer, 1971).

Mean Arterial Pressure

The ANOVA with the fixed factors of Group and Condition and with the repeated measures of Valence and Trial was performed on the MAP data. The results are represented in Table 3. The main effect of Trial was significant, F (3, 180) = 3.00, $p \leq .05$, indicating that MAP was higher during Trial 2 and Trial 4 relative to Trial 1 and Trial 3. The results are represented in Figure 1.

The Condition X Trial interaction was significant, F (3, 180) = 4.32, $p \leq .05$. The results are represented in Figure 2. Post hoc analyses using Tukey's HSD Test revealed that participants in the stress condition evidenced a significantly higher MAP relative to participants in the no-stress condition at Trial 1, Trial 2, and Trial 3. Also, MAP increased reliably for the participants in the no-stress condition at Trial 4. In contrast, MAP increased for the participants in the stress condition at Trial 2 and decreased reliably at Trial 3.

The Condition X Valence X Trial interaction was also statistically significant, F (3, 180) = 4.44, $p \leq .05$. This interaction is depicted in Figure 3. Post hoc analyses revealed that

participants in the stress condition evidenced a significantly higher MAP, relative to participants in the no-stress condition, at Trial 2. Further, MAP increased significantly at Trial 2 for participants in the stress condition. For participants in the stress condition, MAP increased significantly following the negative list and MAP decreased significantly following the positive list. Conversely, for participants in the no-stress condition, MAP increased significantly following the positive list.

The Group X Condition X Valence interaction was also reliable, F (1, 60) = 6.87, $p \leq .05$. The results are represented in Figure 4. Post hoc analyses revealed that, for both the positive and negative list learning blocks, MAP was elevated for high-hostiles in the stress condition relative to high-hostiles in the no-stress condition. Also, MAP was significantly higher during the positive list learning block for low-hostiles in the stress condition, relative to low-hostiles in the no-stress condition. Also, low-hostiles in the stress condition evidenced heightened MAP during the positive list learning block, relative to the negative list learning block. In contrast, low-hostiles in the no-stress condition evidenced heightened MAP during the negative list learning block, relative to the positive list learning block.

Systolic Blood Pressure

The ANOVA with the fixed factors of Group and Condition and with the repeated measures of Valence and Trial was performed on the SBP data. ANOVA results are represented in Table 4. The main effect of Valence was significant, F (1, 60) = 3.89, $p \leq .05$, indicating that SBP was higher during the positive list learning block relative to the negative list learning block. These results are represented in Figure 5. The main effect of Trial was also significant, F (3, 180) = 3.75, $p \leq .05$, indicating that SBP was higher at Trial 2 and Trial 4 relative to Trial 1 and Trial 3. The results are represented in Figure 6.

The Condition X Trial interaction was also reliable, F (3, 180) = 5.88, $p \leq .05$. These results are depicted in Figure 7. Post hoc analyses revealed that participants in the stress condition evidenced a significantly higher SBP relative to participants in the no-stress condition at Trial 2 and Trial 3. Further, SBP for participants in the stress condition increased significantly from Trial 1 to Trial 2 and decreased significantly from Trial 2 to Trial 3. Also, SBP for participants in the no-stress condition increased significantly from Trial 3 to Trial 4.

The Condition X Valence interaction was also significant, F (1, 60) = 4.61, $p \leq .05$. This interaction is depicted in Figure 8. Post hoc analyses revealed that SBP was higher for participants in the stress condition, relative to participants in the no-stress condition, during the positive as well as negative list learning blocks. Further, for the participants in the stress condition, SBP was reliably higher during the positive list learning block, relative to the negative list learning block.

The Group X Condition X Valence interaction was also significant, F (1, 60) = 3.80, $p \leq .05$. This interaction is represented in Figure 9. Post hoc analyses revealed that, for both the positive and negative list learning blocks, SBP was elevated for high-hostiles in the stress condition relative to high-hostiles in the no-stress condition. Further, for high-hostiles in the stress condition, SBP was higher during the positive list learning block, relative to the negative list learning block. Also, SBP was significantly higher during the positive list learning block for low-hostiles in the stress condition, relative to low-hostiles in the no-stress condition. Further, low-hostiles in the stress condition evidenced heightened SBP during the positive list learning block, relative to the negative list learning block. In contrast, low-hostiles in the no-stress condition evidenced heightened SBP following the negative list learning block, relative to the positive list learning block.

Diastolic Blood Pressure

The ANOVA with the fixed factors of Group and Condition and with the repeated measures of Valence and Trial was performed on the DBP data. ANOVA results are represented in Table 5. The Condition X Valence X Trial interaction was significant, F (3, 180) = 2.86, $p \leq .05$. The results are displayed in Figure 10. Post hoc analyses revealed that participants in the stress condition with the positive list and participants in the stress condition with the negative list evidenced significantly higher DBP than participants in the no-stress condition with the positive list at Trial 1, Trial 2, and Trial 3. Further, participants in the stress condition with the positive list evidenced higher DBP than participants in the no-stress condition with both the positive and negative lists at Trial 2. Also, participants in the stress condition, during the negative list learning block, evidenced higher DBP at Trial 4, relative to other trials. In contrast, participants in the no-stress condition, during the positive list learning block, evidenced higher DBP at Trial 4, relative to other trials.

Heart Rate

The ANOVA with the fixed factors of Group and Condition and with the repeated measures of Valence and Trial was performed on the HR data. ANOVA results are represented in Table 6. The Condition X Trial interaction was significant, F (3, 180) = 4.70, $p \leq .05$. This interaction is represented in Figure 11. Post hoc analyses revealed that participants in the stress condition evidenced significantly higher HR relative to participants in the no-stress condition at Trial 1 and Trial 4. Further, HR for participants in the stress condition was significantly lower at Trial 2 relative to Trial 1 and Trial 4.

The Group X Valence X Trial interaction was also significant, F (3, 180) = 2.76, $p \leq .05$. This interaction is represented in Figure 12. Post hoc analyses revealed that high-hostile participants evidenced significantly higher HR than low-hostile participants across all trials. Further, HR was significantly higher at Trial 4 for high-hostiles during the positive list learning block relative to the negative list learning block. For low-hostiles, HR was higher at Trial 1 in the positive list learning block, relative to the negative list learning block. Also, HR was significantly lower for low-hostiles within the positive list learning block at Trial 2, relative to Trial 1.

Affective List Learning

Group means and standard deviations of total words recalled per location for each affective list learning trial are displayed in Table 7. Analysis of variance (ANOVA) with the fixed factors of Group and Condition and with the repeated measures of Valence, Trial, and Location was performed. All pairwise comparisons were made using Tukey's Studentized Range Test (see Winer, 1971).

The main effect of Group was significant, F (1, 60) = 4.38, $p \leq .05$, indicating that high-hostile participants recalled fewer affective words than low-hostile participants. Results are displayed in Figure 13. The main effect of Valence was also significant, F (1, 60) = 23.07, $p \leq .05$, indicating that there were more negatively valenced affective words recalled, relative to positively valenced affective words. This effect is represented in Figure 14. The main effect of Trial was also significant, F (1, 60) = 4.38, $p \leq .05$, indicating that the number of affective words recalled increased significantly across each trial from Trial 1 to Trial 4. The results are depicted in Figure 15. The main effect of Location was also significant, F (2, 120) = 73.52, $p \leq .05$, indicating that the number of words recalled within the first third and last third of each affective

list was significantly higher than the number of words recalled within the middle third of each list. This effect is displayed in Figure 16.

The Group X Trial interaction was reliable, F (4, 240) = 2.69, $p \leq .05$. This interaction is represented in Figure 17. Post hoc analyses revealed that low-hostile participants recalled significantly more affective words at Trial 2, Trial 4, and Trial 5, relative to high-hostile participants. Additionally, low-hostile participants recalled significantly more words at each trial from Trial 1 to Trial 4. In contrast, high-hostiles recalled significantly more words at each trial from Trial 1 to Trial 3 and significantly more words at Trial 5 than Trial 4.

The Valence X Trial interaction was also reliable, F (4, 240) = 2.47, $p \leq .05$. The results are displayed in Figure 18. Post hoc analyses revealed that there were significantly more negative words recalled than positive words at each trial from Trial 2 to Trial 5. Further, there were significantly more words recalled at each trial from Trial 1 to Trial 5 for the positive and negative affective words.

The Valence X Location interaction was also significant, F (2, 120) = 12.68, $p \leq .05$. This interaction is depicted in Figure 19. Post hoc analyses revealed that for the positive list, there were significantly fewer words recalled within the middle third location, relative to the first third and last third location. Further, for the positive list, there were significantly more words recalled within the last third location, relative to the first third and middle third. Also, for the negative list, there were significantly fewer words recalled within the middle third location, relative to the first third and last third location. Further, for the negative list, there were significantly more words recalled within the first third location, relative to the middle third and last third. There were significantly more negative words recalled within the first third, relative to all other valence and location combinations.

The Trial X Location interaction was also significant, F (8, 480) = 6.20, $p \leq .05$. The results are represented in Figure 20. Post hoc analyses revealed that relative to the first third and the last third, there were significantly fewer words recalled within the middle third at each trial from Trial 1 to Trial 5. For the first third location, there were significantly more words recalled for each trial from Trial 1 to Trial 3 and from Trial 3 to Trial 5. For the middle third location, there were significantly more words recalled for each trial from Trial 1 to Trial 5. For the last third location, there were significantly more words recalled at each trial from Trial 1 to Trial 4.

The Valence X Trial X Location interaction was also significant, F (8, 480) = 3.95, $p \leq$ .05. This interaction is represented in Figure 21. Post hoc analyses revealed that there were significantly more words recalled at the first third location for the negative list than all other combination across all trials. Also, for the negative list, significantly more words were recalled from the first third of the list than the middle third of the list and the last third of the list across all trials. Additionally, for the negative list, there were significantly more words recalled from the last third of the list than the middle third of the list for each trial from Trial 1 to Trial 4. For the positive list, significantly more words were recalled from the last third of the list than the middle third of the list across all trials. Also, for the positive list, significantly more words were recalled from the last third of the list than the first third of the list across each trial from Trial 1 to Trial 4. Further, for the positive list, there were significantly more words recalled from the fist third of the list than the middle third of the list across each trial. Finally, the middle third of the positive list was significantly greater than the middle third of the negative list at Trial 1; however, the middle third of the negative list was significantly greater than the middle third of the positive list at Trial 3, Trial 4, and Trial 5.

Discussion

To the author's knowledge, this experiment is novel in three distinct ways. This is the first experiment that investigated the performance of high-hostile and low-hostile participants on the AAVL. This is also the first experiment that investigated the AAVL by providing direct comparisons of learning under stress and no-stress conditions. Finally, this is the first experiment that concurrently investigated both cardiovascular reactivity and learning differences to the affective lists in order to provide integrated physiological and learning results. This experiment was designed to partially replicate the cardiovascular procedures used by Snyder et al. (1997). MAP data (the average of SBP and DBP) provided the most robust results in the initial study of cardiovascular reactivity to the AAVL. In order to remain consistent with the initial experiment, MAP is discussed as the primary indicant of cardiovascular reactivity in the present experiment. When appropriate, SBP, DBP, and HR results will also be discussed in order to provide a more refined representation of cardiovascular functioning.

Investigation of the Hypotheses

This experiment tested four hypotheses (Appendix A). First, it was hypothesized that high-hostiles would demonstrate heightened autonomic reactivity to the negative list. Specifically, it was expected that results would demonstrate a significant Group X Valence interaction for the cardiovascular dependent variables. The results of the present experiment did not support this a priori hypothesis. For MAP, SBP, DBP, and HR, no significant Group X Valence interaction was found. Second, it was hypothesized that high-hostile individuals' performance on the negative list would differ from that of low-hostile individuals. A significant Group X Valence interaction for the number of affective words recalled was expected to support this hypothesis. The non-significant results of this interaction in the present experiment did not support this valence dependent hypothesis. However, high-hostile individuals' overall performance on both valences of the affective list learning task was significantly lower than low-hostile individuals' performance. Third, it was hypothesized that the administration of the cold-pressor stressor would differentially affect the high-hostiles' performance on the negative list when compared to the low-hostiles' performance. Specifically, it was expected that results would demonstrate a significant Group X Condition X Valence interaction for the number of affective words recalled. The results of the present experiment did not support this a priori hypothesis. For the number of words recalled, no

significant Group X Condition X Valence interaction was found. Finally, it was hypothesized that high-hostiles would display more ANS reactivity to both the cold-pressor and to the negative list than low-hostiles. It was expected that results would demonstrate a significant Group X Condition X Valence interaction for the cardiovascular dependent variables. This hypothesis was partially supported by the significant Group X Condition X Valence interaction for MAP. High-hostiles evidenced significant cardiovascular reactivity to the cold-pressor stressor alone. However, this hypothesis was not fully supported since the high-hostile individuals did not demonstrate heightened ANS reactivity to both the cold-pressor and the negative list, relative to low-hostile individuals.

Additional Findings

Although the hypotheses were not fully supported, this experiment yielded the following informative findings: First, high-hostiles had difficulty processing emotional stimuli. The high-hostile participants recalled fewer affective words than their low-hostile counterparts. This extends a line of research across function cerebral systems in which high-hostiles typically demonstrate slowness and inaccuracy of processing negative emotions. Second, there was a significant "primacy effect" for the negative affective list and a significant "recency effect" for the positive affective list. Third, the cold-pressor stressor served as a valid stressor as indicated by increased cardiovascular reactivity following the stressor. Fourth, high-hostile participants evidenced increased cardiovascular reactivity following stress, whereas, low-hostile participants evidenced increased cardiovascular reactivity with the positive list and stress as well as with the negative list and no stress. Therefore, high-hostiles' cardiovascular reactivity to stress was independent of the valence of the learning task. In contrast, low-hostiles' cardiovascular reactivity was valence dependent with activation to both the positive list concurrent with stress and to the negative list concurrent with no stress. Finally, under stress, the positive affective list learning task resulted in decreased cardiovascular reactivity, while the negative affective list learning task resulted in increased cardiovascular reactivity. In contrast, with no stress, the positive affective list learning task resulted in increased cardiovascular reactivity. Thus, the effect of the affective list on the heart is stress dependent. Each of these additional findings are discussed in more detail.

High Hostiles Have Trouble with Affective Learning

The first additional result indicated that high-hostile participants recalled fewer affective words, relative to the low-hostile participants. This is both interesting and important. Past research has shown that high-hostiles inaccurately identify neutral faces as angry at the left hemispace (Harrison & Gorelczenko, 1990) and that high-hostiles evidence diminished self-awareness and impaired accuracy for social appraisals (Demaree & Harrison, 1997b). High-hostiles are typically slower (Demaree & Harrison, 1997a, in press) and less accurate (Herridge & Harrison, 2000) than low-hostiles for the processing of negative emotions. This experiment demonstrated that high-hostiles, relative to low-hostiles, are impaired in their ability to recall affective words, regardless of emotional valence. This result is of particular importance. Recall that Krantz, Manuck, and Wing (1986) suggested that cardiovascular responses are related to an individual's perception. Therefore, an affective event produces a cardiovascular response if the individual's perception of the event is hostile, regardless of whether or not the event is in fact hostile. This experiment suggests that high-hostiles' misappraisal of emotional information, possibly including threat, may be related to their diminished ability to process affective information, regardless of valence.

Affective Lists Produce Differential Primacy and Recency Effects

The second additional result indicated that there was a significant primacy effect for the negative affective list and a significant recency effect for the positive affective list. These findings are consistent with the results of Snyder and Harrison (1997), which demonstrated that the learning of positive and negative words produced differential patterns of acquisition as a function of list location. Snyder and Harrison (1997) demonstrated a diametrically opposite effect (primacy and recency) of negative and positive words on acquisition processes in learning. The negative list learning task produced a greater primacy effect (proactive interference), whereas the positive list learning task produced a greater recency effect (retroactive interference). Historically, proactive interference refers to the disruptive effect of previous learning on the recall of new information, whereas retroactive interference refers to the disruptive effect of new information on the recall of previous information (Underwood, 1957). The results of the present experiment also suggest that there are reliable differences in learning for positive and negative affective words.

Within the AAVL results, there was also a typical learning curve. This finding is consistent with research using the RAVL which has also commonly reported a typical learning curve during acquisition, with the total number of words recalled increasing with each successive trial (Ryan & Geisser, 1986). Also, results of this experiment demonstrated that more negative words were recalled than positive words and that the middle third set of words were recalled less frequently than the first and the last third.

The Cold-Pressor was a Valid Stressor

The third additional result indicated that the cold-pressor stressor elevated MAP. This finding indicated that the cold-pressor served as a valid stressor for those participants in the stress condition (participants who placed their hand in a cooler of ice water with a constant temperature below 5°C). In contrast, participants in the no-stress condition (participants who placed their hand in an empty cooler) did not evidence heightened MAP following the no-stress trial. These results are consistent with past experiments utilizing the cold-pressor stressor, which have also demonstrated heightened blood pressure following the stressor (Demaree & Harrison, 1997a, in press; Glass et al., 1990). Similarly, SBP was elevated following the stress task, but did not change following the no-stress task. Interestingly, MAP at baseline was significantly different for participants in the stress condition, relative to participants in the no-stress condition. It is likely that the difference at baseline represents anticipatory reactivity, since the participants in the stress condition knew that they would receive the cold-pressor before the baseline measure was recorded.

While MAP and SBP increased reliably following the stressor, HR decreased following the cold-pressor stressor. This finding may seem to contradict the conclusion that the cold-pressor was a valid stressor in the present experiment. However, Sherwood and Turner (1992) demonstrated that HR varies primarily with vagal and beta-adrenergic sympathetic influences on the sinoatrial node (main pacemaker cells), whereas, peripheral resistance and alpha-adrenergic sympathetic influences are primarily correlated with blood pressure variability. The cold-pressor stressor is typically associated with sympathetic activation leading to pressor responses mediated by the vasoconstrictive action of alpha-adrenergic receptor stimulation (Peckerman et al., 1991). Therefore, it is not surprising that only the MAP and SBP indicants of cardiovascular functioning increased reliably following the cold-pressor.

These results are consistent with past experiments that demonstrated increased blood pressure following the cold-pressor. However, the discrepant results between MAP, SBP, and HR may warrant the use of a clearer definition of cardiovascular reactivity to the cold-pressor. It is likely that blood pressure data will provide the most robust cardiovascular findings following the cold-pressor, in future experiments. Accordingly, future experiments with the cold-pressor should include independent a priori hypotheses with respect to blood pressure and heart rate.

High- and Low-Hostile Groups Differ in Cardiovascular Reactivity

The fourth additional result of the present experiment indicated that high-hostile participants evidenced increased cardiovascular reactivity following stress, whereas, low-hostile participants evidenced increased cardiovascular reactivity with both the positive list concurrent with stress and with the negative list concurrent with no stress. In the presence of a physical stressor, high-hostile participants maintained elevated MAP, regardless of the valence of the list learning task. In essence, the high-hostile individuals may react to a physical stressor with increased right hemisphere activation and resultant heightened blood pressure. Past research has shown that elevated blood pressure persists longer for high-hostile than low-hostile individuals and high-hostiles often demonstrate perseverative dysfunction on frontal lobe tasks (Demaree & Harrison, 1997a, in press). The present experiment may indicate that high-hostile participants not only maintain their elevated ANS arousal longer than low-hostiles, but that their blood pressure is less influenced by cognitive tasks once they are aroused. In contrast, low-hostile individuals demonstrated diametrically opposite effects on MAP with the positive list concurrent with stress and with the negative list concurrent with no stress. It is possible that low-hostiles are more capable of dynamically shifting cerebral activation following affective cognitive tasks, relative to high-hostiles. Moreover, low-hostiles may be more sensitive to subtle affective manipulation because high-hostiles are already highly aroused.

High-hostiles have historically been more reactive to stress than low-hostiles (Dembroski, MacDougall, Herd, & Shields, 1983; Glass & Contrada, 1982; Harbin, 1989). This experiment supports this notion. However, this experiment also demonstrates that the high-hostile participants are not as capable of dynamically shifting cerebral activation following different cognitive challenges and affective valences. This may help to explain how low-hostiles maintain lower levels of blood pressure: they are more able to shift cerebral activation following

environmental and cognitive challenges. This hypothesis is consistent with Wittling's (1997) account of the left hemisphere's and the right hemisphere's response system. Low-hostiles may be able to recruit the left-hemisphere response system and maintain ANS arousal more readily than high-hostiles resulting in a more effective ability to recruit parasympathetic ANS activation and the resultant acquiescent effects on blood pressure. However, these results warrant further investigation since significant results were only found when all trials of each affective block were combined. These results could be more useful if the effects were found to be significant from the pre-list trial to the post-list trial only.

Cardiovascular Reactivity to Affective Learning is Stress Dependent

The final set of additional results from the present experiment are very exciting. Specifically, these results may indicate weaknesses in the current neuropsychological models of laterality and ANS reactivity. Following the discussion of the results and a brief review of the neuropsychological models, an integrative model is specified. Results of the present experiment indicated that following stress, the positive affective list learning task resulted in decreased cardiovascular reactivity while the negative affective list learning task resulted in increased cardiovascular reactivity. In contrast, with no stress, the positive affective list learning task resulted in increased cardiovascular reactivity. These results partially replicate the results of Snyder et al. (1997). However, the present experiment provides a more refined understanding of cardiovascular reactivity with the AAVL. Consistent with the Snyder et al. (1997) experiment, MAP increased following the negative list and decreased following the positive list in the present experiment. However, these results were only reliable following stress induction. Without the presence of a stressor, MAP only increased following the positive list. Also consistent with the results of Snyder et al. (1997), DBP increased following the negative list in the presence of the stressor. However, consistent with the MAP results of this experiment, DBP increased following the positive list only in the absence of the stressor. These exciting results suggest that cardiovascular reactivity to the AAVL may be dynamic. Specifically, the effect of the affective lists on the heart appears to be stress dependent.

Limitations of the Reviewed Models of Emotion and ANS Regulation

This experiment was designed to investigate dynamic cerebral laterality to stress and to investigate whether or not current neuropsychological models of emotion and ANS activation

adequately reflect the hypothesized dynamic laterality. Wittling's (1997) model of brain asymmetry and autonomic control of the heart proposed that cortical control of autonomic cardiac regulation may be related to a division of responsibility between the cerebral hemispheres. The right hemisphere predominately controls sympathetic activation, whereas the left hemisphere predominately controls parasympathetic activation (Wittling, 1997; Yoon, Morillo, Cechetto, & Hachinski, 1997). It was expected that the positive list learning task would prime the left hemisphere. Since the left hemisphere has been associated with parasympathetic ANS arousal, it was predicted that this task would increase parasympathetic activation resulting in decreased blood pressure. In contrast, it was expected that the negative list learning task would prime the right hemisphere. Since the right hemisphere is more associated with sympathetic ANS arousal, it was predicted that this task would increase sympathetic activation, resulting in increased blood pressure.

Heilman (1982) speculated that the right cerebrum may have greater control of the subcortical systems, which are largely responsible for arousal and emotion. More recently, Heilman, Bowers, and Valenstein (1993) argued that activation of the posterior cerebral regions, particularly the right parietal region and the right temporal region, is likely to result in emotional responses. This model does not adequately account for the role of the left hemisphere in the regulation of parasympathetic activation (resulting in decreased blood pressure). Since the positive affective list was hypothesized to increase left hemisphere activation, and proved to reliably decreased blood pressure in the present experiment, this right cerebral model of ANS regulation may be too simplistic to account for these results.

In contrast to Heilman et al. (1985), Tucker and Frederick (1989) described a balance model of emotion. Tucker and Frederick (1989) proposed that deactivation of one cerebrum leads to increased relative activation of the opposite cerebrum and, therefore, an increased expression of the dominant cerebrum's primary response pattern. Therefore, deactivation of the left cerebrum may result in an increase of negative emotion while deactivation of the right cerebrum may result in an increase of positive emotion. A weakness of Tucker's model, however, is that it does not adequately address resultant behavioral patterns following deactivation or activation of both hemispheres. Specifically, results of the present experiment demonstrated that MAP deceased in the stress condition (right hemisphere prime) concurrent with the positive list

(left hemisphere prime). Tucker and Frederick's (1989) model does not adequately account for this finding of concurrent activation of both the left and right cerebral hemispheres.

Heller (1993) proposed a model of cerebral activation during emotional processing. Heller's circumplex model divides the brain into four quadrants, defined by the valence axis and the arousal axis. It is proposed that most emotions fall within the four quadrants of these axes and correspond to distinct physiological responses (Heller, Nitschke, & Miller, 1998). Further, activation of one quadrant following emotional induction results in an increase of the corresponding physiological response. As with Tucker and Frederick's (1989) balance model, Heller's (1993) model does not adequately account for the dynamic cardiovascular results found in the present experiment with concurrent activation of multiple quadrants. Also, Heller's (1993) model does not adequately explain why MAP decreased with the positive list concurrent with stress but increased with the positive list concurrent with no stress.

Proposed Model of Emotion and ANS Regulation

The results of the present experiment are both exciting and complex. As such, the previously reviewed neuropsychological theories may be too simplistic and may not adequately explain these findings. Tucker and Frederick (1989) acknowledged that the brain's processing of emotion is complex and likely involves the interaction of multiple systems that are only partially understood. A revised model of dynamic cerebral lateralization that combines Tucker and Frederick's (1989) balance model with Kinsbourne's FCD theory may provide an improved account of the results of this experiment. The functional cerebral distance (FCD) principle was introduced to address how the brain may organize and concurrently process dual- or multiple-task requirements (Kinsbourne & Hicks, 1978). This principle stems from understanding the brain as a differentiated neural network. Accordingly, activation in one functional region may spread throughout much of the cerebral cortex via the unifying dendritic network (Purpura, 1967). Recently, the FCD principle has been refined to distinguish priming effects from interference effects. With light demands, concurrent tasks spread activation and facilitate processing (priming). More demanding tasks, however, may tax a functional system and decrease processing capacity for other tasks (interference) (Kinsbourne & Hiscock, 1983). The FCD principle is similar to arousal theory (Duffy, 1962; Easterbrook, 1959; Harrison & Pavlik, 1983; Hebb, 1955; Linsdsley, 1951), which suggests that increased arousal is associated with improved task

proficiency up to an optimal level, at which point further increases in arousal may impair performance.

The proposed combination of the balance model with FCD theory affords an explanation for the significant finding of increased cardiovascular reactivity to stress in the present experiment. At baseline, cerebral activation patterns of an individual may represent roughly equivalent activation of the left and right cerebral hemispheres. In this state, the functional system maintains appropriate arousal and cardiovascular reactivity by regulating sympathetic and parasympathetic activation via the right and left hemisphere, respectively (Wittling, 1997). With stress, this system becomes dynamic. Activation shifts to the right hemisphere, leading to decreased relative activation of the left hemisphere. Cardiovascular reactivity increases as a result of this dynamic shift. Specifically, the heightened right hemisphere activation following stress may reflect a dual-task effect with the regulation of the sympathetic ANS. This dual-task may produce an interference effect, since the right hemisphere may be taxed by the increased activation necessary to respond to the heightened stress (Kinsbourne & Hiscock, 1983). Concurrently, the resultant relative deactivation of the left hemisphere (Tucker & Frederick, 1989) may promote less parasympathetic activation. Therefore, increased blood pressure following stress, as supported by the results of the present experiment, may result from the interference effect of sympathetic ANS regulation following the relative activation of the right hemisphere as well as a diminished parasympathetic response, following the relative deactivation of the left hemisphere.

This model may also explain the dynamic effects of cardiovascular reactivity to the positive and negative affective list learning task in the presence and absence of stress. In the present experiment, blood pressure reliably increased with the negative list learning task combined with the cold-pressor stressor. Concurrent with stress, the negative list learning task may increase the dual-task requirements of the right hemisphere. This pattern of activation may result in a heightened interference effect for sympathetic ANS control, leading to higher blood pressure (Demaree & Harrison, 1997a, in press; Kinsbourne & Hiscock, 1983). The increased right hemisphere activation following the negative list learning task in the presence of a stressor may also produce a relative decrement in left hemisphere activation, thus reducing the left hemispheres' ability to promote parasympathetic activity (Wittling, 1997). Thus, the administration of the negative list in the presence of a stressor may increase blood pressure by dynamically affecting

both the right and left hemispheres. Results of the present study are consistent with this hypothesized model.

The proposed model may also explain the finding of decreased cardiovascular reactivity following the positive list learning task concurrent with stress. Since the left hemisphere exhibits decreased activation relative to the right hemisphere under stress, the addition of a positive list learning task may increase activation of the left hemisphere. This priming effect may produce heightened parasympathetic ANS activation, thus reducing blood pressure (Wittling, 1997). Similarly, with the increase in left hemisphere activation following the positive list learning task, the right hemisphere may exhibit decreased relative activation. Therefore, the relative right hemisphere deactivation following the positive list may then alleviate the interference effect that existed with the initial right hemisphere activation to stress (Kinsbourne & Hiscock, 1983). The combined effect of heightened left hemisphere relative activation and relative decreased right hemisphere activation may then lower blood pressure by increasing parasympathetic ANS activation and reducing sympathetic ANS activation.

This experiment also yielded the exciting result that MAP increased reliably following the positive list learning task in the no-stress condition. This appears to be inconsistent with the previously reviewed models of lateralization and ANS regulation. However, the proposed model provides a plausible explanation for this effect. With no stress, it is likely that the left and right hemispheres have roughly equivalent activation. It is possible that the positive list learning task shifted cerebral activation to the left hemisphere. Just as heightened activity of the right hemisphere may have produced an interference effect with sympathetic regulation, this heightened activation of the left hemisphere may interfere with the left cerebrum's ability to promote parasympathetic activation. Thus, this dual-task interference effect (Kinsbourne & Hiscock, 1983) could lead to heightened blood pressure following a lack of parasympathetic activation. Further, it is plausible that with this left hemisphere activation, the right hemisphere would evidence decreased relative activation. Thus, the right hemisphere's ability to regulate the sympathetic ANS may be diminished, adding to the overall effect of increased blood pressure following the positive list concurrent with no stress.

The proposed model may also provide an explanation of the previously discussed group difference in cardiovascular reactivity. Recall that high-hostiles' cardiovascular reactivity to stress

was independent of the valence of the learning task. In contrast, low hostiles' cardiovascular reactivity was valence dependent with increased activation to both the positive list concurrent with stress and to the negative list concurrent with no stress. With stress, high-hostiles may evidence an interference effect due to the dual-task of stress and cardiovascular regulation in the right hemisphere. High-hostiles tend to maintain heightened right hemisphere activation (Demaree & Harrison, 1996). A strong and persistent interference of cardiovascular regulation may result from this heightened right hemisphere activation, especially with the additional burden on the right hemisphere in the presence of stress. It is possible that high-hostiles maintain elevated cardiovascular reactivity with stress following the negative list and following the positive list because of the tremendous interference of hostility and stress with the ability to regulate cardiovascular functioning. In other words, high-hostiles' cardiovascular reactivity to stress is independent of valence because the immense interference effect of hostility and stress persists regardless of positive or negative emotional processing.

Similarly, the proposed model may account for the low-hostiles' heightened MAP with stress concurrent with the positive affective list. This increased cardiovascular reactivity may result from increased activation in both the left and right hemispheres. With stress and the concurrent positive list learning task, both hemispheres may have heightened activation. This bilateral increase in activation may interfere with low-hostiles' cardiovascular regulation. Specifically, the right cerebrum's control of sympathetic ANS activity may be taxed in the presence of stress yielding a resultant increase in blood pressure. Further, the left cerebrum's ability to promote parasympathetic activity may be diminished as a result of the dual-task requirements of processing the positive list while attempting to maintain parasympathetic activation.

The proposed model may also account for the low-hostiles' heightened MAP with no stress concurrent with the negative affective list. Just as stress may tax the right hemisphere, the negative list learning task may diminish the low-hostiles' ability to regulate sympathetic activation as a results of a right hemisphere dual-task. Thus, the negative list learning task may tax the right hemisphere, leading to interference with sympathetic cardiovascular regulation and a resultant increase in blood pressure.

This model is a logical extension of Tucker and Frederick's (1989) balance model and Kinsbourne and Hiscock's (1983) FCD theory. It also provides a plausible account of the cardiovascular results of the present experiment. Further, this proposed model addresses the dynamic cerebral lateralization and resultant ANS responses to the cold-pressor stressor and to the affective list learning tasks.

Future Directions

Although the results of this experiment were very interesting, the primary hypotheses were largely unsupported. This research may be improved through the repetition of the present experiment with modifications. First, the subject size should be increased to include a group of individuals that placed their right versus left hand in the cold-pressor stimulus. As preliminary reports in our lab have shown, there may be differences in lateralized activation concurrent with hand use. Second, it seems possible from the results of this experiment, as well as prior investigation, that participants may evidence cardiovascular arousal from the expectation of receiving the cold-pressor stressor. To better evidence cardiovascular changes associated with the cold-pressor administration, a longer relaxation period between experiment instructions and pre-stress measurement of cardiovascular measures appears warranted. Third, although high-hostiles recalled significantly fewer affective words than low-hostiles, the present experiment does not allow for a comparison of list learning performance for affective and non-affective words. To better investigate differences in learning among high- and low-hostiles, a neutral list (the original RAVL list) should be administered to both groups along with the positive and negative affective word lists in subsequent investigations. Fourth, the sample used in the current experiment represents 18 to 25-year-old participants that were healthy and intelligent. Further, the participants in the present experiment were all college students who were likely to tolerate stress well within social norms. Future experimenters may consider using participants from other settings such as medical centers, captive populations, and other settings in which differences between high- and low-hostiles may be more robust. Finally, as the literature suggests that it is likely that cerebral regulation for women differs than that of cerebral regulation for men (Heller, 1993) , it is also important that future experiments include women participants.

This experiment demonstrated that high-hostiles have difficulty processing emotional information. These results should be combined with other psychological theories in future

research projects. For example, several studies have demonstrated that problem-focused coping strategies tend to work best in situations where the individual has the ability to control a situation. In contrast, emotion-focused coping strategies may work best in the absence of control (Aldwin & Revenson, 1987; Folkman, 1984; Strentz & Auerbach, 1988). It would be very interesting to investigate if this is also true for high-hostiles. This experiment suggests that high-hostiles have difficulty with emotional processing. Therefore, it is plausible that high-hostiles would have a difficult time successfully implementing effective emotion-focused coping strategies in situations with little control. Thus, high hostiles may be limited to problem-focused coping strategies and may lack an adequate mechanism to cope with situations that are beyond their control.

While the proposed model adequately addresses the results of the present experiment, it should be tested further by comparing participants' performance on additional behavioral tasks (tachistiscopic and dichotic presentations, hand grip strength, verbal versus nonverbal fluency investigation, etc.) and neuroimaging techniques (QEEG, fMRI, etc.). Further experimentation would afford a means to directly test the proposed model. This is important since controversy remains in the current literature about the cerebral control of ANS regulation and emotion. For example, alternative theories from the psychophysiology literature suggest that the right hemisphere regulates both sympathetic and parasympathetic activation (Porges, 1994, 1995). Continued testing of the neuropsychological theories, psychophysiological theories, and the proposed model appears warranted.

Although it is beyond the scope of this project, it is likely that the anterior and posterior regions of each hemisphere interact with one another to promote cardiovascular control. As Tucker and Frederick (1989) suggested, the functional cerebral system for the control of emotional processing and cardiovascular reactivity is complex and additional information about differences in the anterior and posterior regions' role in cardiovascular reactivity may continue to improve the proposed model.

Summary and Conclusions

The present experiment compared high- and low-hostile men from an undergraduate population on self-report, cardiovascular, and affective verbal learning measures. The primary

hypotheses were largely unsupported by the results of this project. However, additional results demonstrated that high-hostiles had difficulty processing emotional stimuli. Further, results suggested that negative affective learning produced proactive interference for the recall of new information while positive affective learning produced retroactive interference for the recall of previously learned information. Also, high-hostiles' cardiovascular reactivity to a valid physical stressor was independent of the valence of the learning task, although low-hostiles' cardiovascular reactivity was valence dependent with activation to both the positive list concurrent with stress and to the negative list concurrent with no stress. Finally, results suggested that the effect of the affective lists on the heart is stress dependent.

As proposed, this experiment addressed four primary questions. First, relative to low-hostiles, do high-hostile individuals demonstrate heightened autonomic nervous system (ANS) arousal following negative affect induction? High-hostiles clearly demonstrated heightened ANS arousal to physical stress; however, their cardiovascular reactivity was valence independent. Interestingly, low-hostiles' reactivity to stress was valence dependent, indicating that they may dynamically shift cerebral activation as a result of processing positive or negative valenced emotional information.

Second, relative to low-hostiles, do high-hostiles demonstrate a heightened sensitivity to proactive interference as evidenced by a primacy effect on list learning during negative affect induction? Although high-hostiles did not demonstrate a heightened sensitivity to proactive interference, a primacy effect on the negative list and a recency effect on the positive list was found across all participants. These results replicate the findings of Snyder et al. (1997).

Third, what is the effect of stress induction on high-hostiles' ANS arousal and affective list learning performance? High-hostiles learned fewer emotional words than low-hostiles and physical stress clearly elevated high-hostiles' ANS arousal; however, physical stress did not impact their emotional learning. This may be related to the "law of initial values" since the low-hostiles evidenced poor emotional word recall prior to the stressor (Wilder, 1931, 1957). Therefore, the methodology of the present experiment may have a reduced ability to detect a significant decrease in affective word recall following the stressor as a result of a potential floor effect with the low initial vales.

Fourth, what implications follow for high- and low-hostile functional cerebral asymmetry and dynamic functional cerebral laterality? The results of the present experiment are not consistent with any one current neuropsychological model of lateralization. However, by combining the balance model with FCD theory, the results of the present experiment, including group difference, may be adequately addressed. It is likely that the continued study of the proposed model will provide additional useful contributions to this important program of research in cardiovascular reactivity and emotion.

The present experiment advances our current understanding of cerebral functioning among hostile individuals and raises new questions about the hemispheric lateralization and cardiovascular reactivity. Future experiments should continue to investigate these issues and their importance for the treatment of cardiovascular disease, especially in high-hostile individuals. Further, the reciprocal relationship between the left hemisphere and the right hemisphere with parasympathetic and sympathetic ANS activation should continue to be studied. Finally, the proposed model should be subjected to additional theoretical review and scientific investigation in order to continue to advance the neuropsychological understanding of emotion and cardiovascular reactivity.

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Appendix A

Hypotheses

- High-hostile individuals will demonstrate heightened autonomic reactivity to the negative list.
- High-hostile individuals' performance on the negative list should differ from that of low-hostile individuals.
- Administration of the cold pressor will differentially affect the high-hostiles' performance on the negative list when compared to the low-hostiles' performance.
- High-hostiles are expected to display more ANS reactivity to both the cold pressor and to the negative list than low-hostiles.

Appendix B

Informed Consent

Title of Experiment: Dynamic Effects of Stress and Hostility

Experiment Number: HSC # 99-32, IRB # 99-133

Principle Investigator: Brian V. Shenal

1. **Purpose of this Research:** You are invited to participate in a study about blood pressure. This study will involve blood pressure correlates of hostility.
2. **Procedures:** To accomplish the goals of this study, you will be asked to do both written and behavioral tests which will take approximately 1 hour. There are no risks or potential harm associated with participants in this study.
3. **Benefits of this project:** Your participation in this research will help clinical psychologists better understand physiological correlates of emotion. No promise of benefits has been made to encourage you to participate. You may receive a synopsis or summary of this research when it is completed. Please give a self addressed stamped envelope to the experimenter if you wish for a synopsis.
4. **Anonymity and Confidentiality:** The results of this study will be strictly confidential. At no time will the researchers release your results to anyone other than the individuals working with the project without your written consent. The information you provide will have your name removed and only a subject number will identify you during the analysis and write up.
5. **Discomforts/Risks:** Although you may experience some discomfort with the blood pressure measurement there are no apparent risks to you for participation in this study.
6. **Compensation:** You may receive one extra credit point for the psychology class you are enrolled in. For alternative methods of receiving extra credit talk to your professor. If, as a result of this procedure, you should seek counseling or medical treatment, treatment will be made available at the Psychological Services Center and the University Counseling Center.

7. **Freedom to Withdraw:** You are free to withdraw from this study at any time without penalty. If you choose to withdraw, you will still receive the extra credit and will not be penalized by any reduction in points. Talk to your professor if alternative sources of extra credit are desired.

8. **Use of the Research Data:** the information from this project may be used for scientific or educational purposes. It may be used for scientific meetings or be published in professional journals or books, or used for any other purpose which Virginia Tech's department of psychology considers proper in the interest of education, knowledge, or research.

9. **Approval of the Research:** This research has been approved by the Human Subjects Committee of the department of psychology and the Institutional Review Board of Virginia Polytechnic Institute and State University.

10. **Subjects Responsibilities:**

I know of no reason I cannot participate in this study.

11. **Subjects Permission:**

I have read and understand the informed consent and conditions of this project. I have all my questions answered. I hereby acknowledge the above and give my voluntary consent for participation in this project. If I participate, I may withdraw at any time without penalty. I agree to abide by the rules of this project. Should I have any questions about this research or its conduct, I will contact:

Brian V Shenal Investigator	231-6914 Phone
David W. Harrison, Ph.D. Faculty Advisor	231-4422 Phone
David W. Harrison, Ph.D Chair, IRB Research Division	231-4422 Phone
H. T. Hurd Chair, HSC	231-5281 Phone

Participant's Signature: _____ Date: _____

Participant's ID: _____ Participant's Telephone: _____

Appendix C

Handedness Questionnaire

Subject #_____

Circle the appropriate number after each item.

	Right	Left	Both
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

Is mother left or right hand dominant? ____
Is father left or right hand dominant? ____

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

Appendix D

Neurological History Questionnaire

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 any "Yes" answers below.

1. Visual difficulties, blurred vision, or eye disorders	Yes	No
2. Blindness in either eye	Yes	No
3. If Yes to either of the above, have problems been corrected	Yes	No
4. Severe head trauma/injury	Yes	No
5. Stroke	Yes	No
6. Learning disabilities (problems of reading, writing, or comprehension)	Yes	No
7. Epilepsy or seizures	Yes	No
8. Paralysis	Yes	No
9. Neurological surgery	Yes	No

Please explain any "Yes" responses:

_____

_____

_____

_____

Appendix E

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 F

Negative List

	Trials				
	1	2	3	4	5
Morgue	_____	_____	_____	_____	_____
Murder	_____	_____	_____	_____	_____
Kill	_____	_____	_____	_____	_____
Pimple	_____	_____	_____	_____	_____
Gun	_____	_____	_____	_____	_____
Greedy	_____	_____	_____	_____	_____
Lice	_____	_____	_____	_____	_____
Measles	_____	_____	_____	_____	_____
Slay	_____	_____	_____	_____	_____
Deface	_____	_____	_____	_____	_____
Cruel	_____	_____	_____	_____	_____
Failing	_____	_____	_____	_____	_____
Hate	_____	_____	_____	_____	_____
Ache	_____	_____	_____	_____	_____
Grave	_____	_____	_____	_____	_____

Appendix G

Positive List

	Trials				
	1	2	3	4	5
Smile	_____	_____	_____	_____	_____
Freedom	_____	_____	_____	_____	_____
Cheerful	_____	_____	_____	_____	_____
Friend	_____	_____	_____	_____	_____
Music	_____	_____	_____	_____	_____
Joy	_____	_____	_____	_____	_____
Happy	_____	_____	_____	_____	_____
Wisdom	_____	_____	_____	_____	_____
Blossom	_____	_____	_____	_____	_____
Laugh	_____	_____	_____	_____	_____
Beauty	_____	_____	_____	_____	_____
Peace	_____	_____	_____	_____	_____
Sunset	_____	_____	_____	_____	_____
Garden	_____	_____	_____	_____	_____
Beach	_____	_____	_____	_____	_____

Appendix H

Mean FAM and PLS Ratings for the AAVL Test
Taken From Toglia and Battig (1978)

	Familiarity Rating (FAM)	Pleasantness Rating (PLS)
Positive List		
SMILE	6.41	6.23
FREEDOM	6.40	6.30
CHEERFUL	6.15	5.90
FRIEND	6.50	6.16
MUSIC	6.77	6.22
JOY	6.27	6.00
HAPPY	6.84	6.10
WISDOM	6.34	6.03
BLOSSOM	5.85	5.91
LAUGH	6.68	6.00
BEAUTY	5.03	6.07
PEACE	6.45	6.00
SUNSET	6.25	6.04
GARDEN	5.51	6.07
BEACH	6.27	5.81
Negative List		
MORGUE	5.48	1.87
MURDER	6.08	1.75
KILL	6.23	1.95
PIMPLE	6.31	1.81
GUN	6.28	1.98
GREEDY	5.94	1.88
LICE	5.38	2.02
MEASLES	5.61	2.00
SLAY	5.55	2.03
DEFACE	5.64	2.10
CRUEL	5.95	2.06
FAILING	5.85	2.15
HATE	6.29	2.06
ACHE	5.97	2.16
GRAVE	5.89	2.07

Appendix I

Data Sheet

Participant # _____

Experiment: 1

Presentation: 1-2; 2-1

Self Report
Laterality _____
CMHO _____

Physio Measures:

I. 1) Baseline1 a)SBP____ DBP ____ HR____
b)SBP____ DBP ____ HR____
c)SBP____ DBP ____ HR____
2) List1 a)SBP____ DBP ____ HR____
b)SBP____ DBP ____ HR____
c)SBP____ DBP ____ HR ____
3) Baseline2 a)SBP____ DBP ____ HR ____
b)SBP____ DBP ____ HR____
c)SBP____ DBP ____ HR____
4) List2 a)SBP____ DBP ____ HR____
b)SBP____ DBP ____ HR____
c)SBP____ DBP ____ HR____
5) Recovery a)SBP____ DBP ____ HR____
b)SBP____ DBP ____ HR____
c)SBP____ DBP ____ HR____

Experiment: 2

Presentation: 1-2; 2-1

Self Report
Laterality _____
CMHO _____

Physio Measures:

I. 1) Baseline1 a)SBP____ DBP ____ HR____
b)SBP____ DBP ____ HR____
c)SBP____ DBP ____ HR____
2) C-Press1 a)SBP____ DBP ____ HR____
b)SBP____ DBP ____ HR____
c)SBP____ DBP ____ HR ____
3) List 1 a)SBP____ DBP ____ HR ____
b)SBP____ DBP ____ HR____
c)SBP____ DBP ____ HR____
4) Baseline2 a)SBP____ DBP ____ HR____
b)SBP____ DBP ____ HR____
c)SBP____ DBP ____ HR____
5) C-Press2 a)SBP____ DBP ____ HR____
b)SBP____ DBP ____ HR____
c)SBP____ DBP ____ HR____
6) List2 a)SBP____ DBP ____ HR____
b)SBP____ DBP ____ HR____
c)SBP____ DBP ____ HR____
7) recovery a)SBP____ DBP ____ HR____
b)SBP____ DBP ____ HR____
c)SBP____ DBP ____ HR____

Table 1. Means and Standard Deviations for CMHS and CPD.

	Low-Hostile		High-Hostile	
Questionnaire	MEAN	SD	MEAN	SD
CMHS	14.78	3.07	31.84	2.94
CPD	10.38	2.30	10.69	2.68

Table 2. Means and Standard Deviations of Cardiovascular Variables.

No-Stress Condition

	Low-Hostile		High-Hostile	
Positive List	Mean	SD	Mean	SD
Trial 1				
MAP	99.66	8.75	98.09	8.80
SBP	126.94	13.38	122.63	11.51
DBP	72.38	8.49	73.56	9.19
HR	71.44	15.85	74.06	18.21
Trial 2				
MAP	98.81	8.16	98.34	7.80
SBP	125.13	13.03	122.88	11.41
DBP	72.50	9.54	73.81	8.79
HR	69.31	15.26	76.00	18.68
Trial 3				
MAP	97.88	9.43	97.50	9.77
SBP	123.56	13.18	121.31	13.68
DBP	72.19	12.32	73.69	10.14
HR	69.81	17.19	76.50	17.83
Trial 4				
MAP	101.34	6.76	102.81	9.39
SBP	126.25	10.00	127.13	13.62
DBP	76.44	8.49	78.50	7.23
HR	68.94	13.71	76.25	18.39
Negative List				
Trial 1				
MAP	101.47	10.03	98.00	8.41
SBP	126.69	12.80	121.94	11.26
DBP	76.25	11.91	74.06	10.61
HR	67.88	14.27	76.00	19.52
Trial 2				
MAP	101.00	8.17	97.69	8.63
SBP	126.31	9.89	122.63	12.95
DBP	75.69	10.98	72.75	10.94
HR	69.63	14.12	76.69	16.38
Trial 3				
MAP	101.28	8.21	97.69	7.59
SBP	126.56	11.76	121.06	11.43
DBP	76.00	11.63	74.31	9.97
HR	70.13	14.73	76.25	15.58
Trial 4				
MAP	100.81	8.90	100.44	7.69
SBP	124.75	14.14	124.00	11.09
DBP	74.13	12.38	76.88	8.86
HR	71.25	14.71	74.06	16.96

Table 2. Continued.

Stress Condition

	Low-Hostile		High-Hostile	
Positive List	Mean	SD	Mean	SD
Trial 1				
MAP	101.06	7.92	102.50	7.96
SBP	127.25	14.75	128.13	15.68
DBP	74.88	8.76	76.88	11.35
HR	75.56	10.49	76.31	11.34
Trial 2				
MAP	105.03	8.49	104.28	11.68
SBP	132.81	15.54	131.81	17.62
DBP	77.25	11.45	76.75	11.11
HR	72.19	12.05	72.75	12.30
Trial 3				
MAP	102.47	8.55	101.34	10.51
SBP	129.19	13.19	127.06	16.34
DBP	75.75	10.67	75.63	9.29
HR	73.63	12.43	73.63	12.44
Trial 4				
MAP	101.00	9.36	100.31	10.60
SBP	127.13	15.41	124.75	14.14
DBP	74.88	13.44	75.88	12.21
HR	73.50	9.34	75.88	9.52
Negative List				
Trial 1				
MAP	98.88	8.75	101.88	9.96
SBP	123.63	12.59	125.44	17.11
DBP	74.13	14.25	78.31	7.93
HR	74.69	12.74	76.88	14.86
Trial 2				
MAP	101.31	10.19	102.63	10.06
SBP	127.56	16.54	128.19	14.60
DBP	75.06	11.99	77.06	11.55
HR	72.69	12.56	72.69	11.82
Trial 3				
MAP	99.00	9.17	101.84	8.69
SBP	124.13	13.09	125.75	14.31
DBP	73.88	13.77	77.94	9.17
HR	73.81	13.57	73.88	12.94
Trial 4				
MAP	101.38	7.85	103.16	10.19
SBP	125.50	10.21	127.06	14.28
DBP	77.25	11.75	79.25	11.30
HR	73.56	11.85	75.06	13.43

Table 3. Independent ANOVA Results for Mean Arterial Pressure.

Source	df	SS	MS	F	p	
Group	(1, 60)	7.51	7.51	.01	.9030	
Condition	(1, 60)	621.28	621.28	1.24	.2698	
GroupXCondition	(1, 60)	190.13	190.13	.38	.5401	
Valence	(1, 60)	8.00	8.00	.28	.5998	
GroupXValence	(1, 60)	.03	.03	.00	.9738	
ConditionXValence	(1, 60)	70.51	70.51	2.45	.1226	
GroupXConditionXValence	(1, 60)	197.51	197.51	6.87	.0111	**
Trial	(3, 180)	207.32	69.11	3.00	.0320	*
GroupXTrial	(3, 180)	33.61	11.20	.49	.6923	
ConditionXTrial	(3, 180)	298.39	99.46	4.32	.0058	**
GroupXConditionXTrial	(3, 180)	91.62	30.54	1.33	.2677	
ValenceXTrial	(3, 180)	24.92	8.31	.49	.6918	
GroupXValenceXTrial	(3, 180)	3.33	1.11	.07	.9783	
ConditionXValenceXTrial	(3, 180)	227.11	75.70	4.44	.0049	**
GroupXConditionXValenceXTrial	(3, 180)	15.21	5.07	.30	.8273	

** $p \leq .01$ * $p \leq .05$

Table 4. Independent ANOVA Results for Systolic Blood Pressure.

Source	df	SS	MS	F	p	
Group	(1, 60)	297.07	297.07	.24	.6236	
Condition	(1, 60)	919.13	919.13	.75	.3890	
GroupXCondition	(1, 60)	347.82	347.82	.28	.5955	
Valence	(1, 60)	200.00	200.00	3.89	.0531	*
GroupXValence	(1, 60)	.28	.28	.01	.9413	
ConditionXValence	(1, 60)	236.53	236.53	4.61	.0359	*
GroupXConditionXValence	(1, 60)	195.03	195.03	3.80	.0560	*
Trial	(3, 180)	401.95	133.98	3.75	.0120	**
GroupXTrial	(3, 180)	23.66	7.89	.22	.8817	
ConditionXTrial	(3, 180)	629.29	209.76	5.88	.0008	**
GroupXConditionXTrial	(3, 180)	101.88	33.96	.95	.4169	
ValenceXTrial	(3, 180)	60.23	20.08	.75	.5236	
GroupXValenceXTrial	(3, 180)	1.17	.39	.01	.9976	
ConditionXValenceXTrial	(3, 180)	193.48	64.49	2.41	.0685	
GroupXConditionXValenceXTrial	(3, 180)	63.70	21.23	.79	.4990	

** $p \leq .01$ * $p \leq .05$

Table 5. Independent ANOVA Results for Diastolic Blood Pressure.

Source	df	SS	MS	F	p	
Group	(1, 60)	138.20	138.20	.21	.6454	
Condition	(1, 60)	381.57	381.57	.59	.4452	
GroupXCondition	(1, 60)	79.70	79.70	.12	.7266	
Valence	(1, 60)	72.00	72.00	1.23	.2727	
GroupXValence	(1, 60)	.03	.03	.00	.9817	
ConditionXValence	(1, 60)	2.00	2.00	.03	.8542	
GroupXConditionXValence	(1, 60)	200.00	200.00	3.40	.0700	
Trial	(3, 180)	254.26	84.75	1.92	.1286	
GroupXTrial	(3, 180)	65.85	21.95	.50	.6854	
ConditionXTrial	(3, 180)	104.66	34.89	.79	.5017	
GroupXConditionXTrial	(3, 180)	83.76	27.92	.63	.5959	
ValenceXTrial	(3, 180)	33.48	11.16	.35	.7922	
GroupXValenceXTrial	(3, 180)	16.61	5.54	.17	.9155	
ConditionXValenceXTrial	(3, 180)	276.89	92.30	2.86	.0384	*
GroupXConditionXValenceXTrial	(3, 180)	61.98	20.66	.64	.5902	

** $p \leq .01$ * $p \leq .05$

Table 6. Independent ANOVA Results for Heart Rate.

Source	df	SS	MS	F	p	
Group	(1, 60)	1505.63	1505.63	1.00	.3224	
Condition	(1, 60)	253.13	252.13	.17	.6839	
GroupXCondition	(1, 60)	800.00	800.00	.53	.4699	
Valence	(1, 60)	.20	.20	.00	.9495	
GroupXValence	(1, 60)	.38	.38	.01	.9293	
ConditionXValence	(1, 60)	.03	.03	.00	.9798	
GroupXConditionXValence	(1, 60)	.28	.28	.01	.9394	
Trial	(3, 180)	119.98	39.99	2.10	.1020	
GroupXTrial	(3, 180)	2.29	.76	.04	.9893	
ConditionXTrial	(3, 180)	268.45	89.48	4.70	.0035	**
GroupXConditionXTrial	(3, 180)	73.14	24.38	1.28	.2829	
ValenceXTrial	(3, 180)	12.73	4.24	.23	.8773	
GroupXValenceXTrial	(3, 180)	154.23	51.41	2.76	.0439	*
ConditionXValenceXTrial	(3, 180)	5.86	1.95	.10	.9573	
GroupXConditionXValenceXTrial	(3, 180)	61.55	20.52	1.10	.3507	

** $p \leq .01$ * $p \leq .05$

Table 7. Means and Standard Deviations of AAVL Words by Location (first, second, and last third of each trial).

		Low-Hostile		High-Hostile	
No-Stress Condition		Mean	SD	Mean	SD
Positive List					
Trial 1	1st	2.44	.89	2.31	.79
	2nd	1.44	1.15	1.94	.93
	3rd	2.50	1.37	2.81	1.17
Trial 2	1st	2.69	1.14	2.88	1.15
	2nd	2.50	1.21	1.88	1.02
	3rd	3.69	1.14	3.06	1.00
Trial 3	1st	3.63	1.02	3.25	1.00
	2nd	2.50	1.10	2.63	1.15
	3rd	3.69	1.25	3.81	.83
Trial 4	1st	3.81	.83	3.88	1.26
	2nd	3.25	1.13	2.69	1.14
	3rd	4.19	.75	3.44	1.31
Trial 5	1st	3.88	.89	3.88	1.15
	2nd	3.38	.96	3.06	.77
	3rd	4.13	1.09	4.06	.85
Negative List					
Trial 1	1st	3.06	1.00	3.31	.79
	2nd	1.00	.82	1.00	.73
	3rd	2.69	1.45	2.50	1.03
Trial 2	1st	3.69	.70	3.88	.96
	2nd	2.31	1.54	2.19	1.17
	3rd	3.00	1.59	2.50	1.21
Trial 3	1st	4.13	.81	4.25	1.00
	2nd	3.13	1.02	3.19	1.17
	3rd	3.44	1.31	3.63	1.09
Trial 4	1st	4.06	1.00	4.13	.96
	2nd	3.44	1.03	3.44	1.26
	3rd	4.25	.68	3.69	.95
Trial 5	1st	4.25	.68	4.38	.62
	2nd	3.88	1.26	3.19	1.17
	3rd	4.06	1.12	4.19	.91

Table 7. Continued.

		Low-Hostile		High-Hostile	
Stress Condition		Mean	SD	Mean	SD
Positive List					
Trial 1	1st	2.50	.73	1.94	.77
	2nd	1.06	.85	1.56	.81
	3rd	2.88	1.09	2.19	1.05
Trial 2	1st	3.50	1.10	2.75	1.39
	2nd	2.25	1.00	2.13	.62
	3rd	3.44	.73	2.94	.85
Trial 3	1st	3.50	1.03	2.94	1.29
	2nd	2.63	1.09	3.06	.93
	3rd	4.06	.77	3.75	.93
Trial 4	1st	3.88	.89	3.13	1.26
	2nd	3.38	1.20	3.00	1.10
	3rd	4.31	.79	3.69	1.01
Trial 5	1st	3.94	.85	3.94	1.39
	2nd	3.44	1.36	2.75	1.00
	3rd	4.00	.89	4.06	.77
Negative List					
Trial 1	1st	3.13	1.02	2.88	1.09
	2nd	.94	.57	.63	.50
	3rd	2.75	1.06	2.38	1.20
Trial 2	1st	4.00	.82	3.44	.89
	2nd	2.56	1.09	2.06	1.06
	3rd	3.19	1.60	3.13	1.36
Trial 3	1st	4.06	.68	4.38	.89
	2nd	3.06	1.12	2.56	1.03
	3rd	3.88	1.26	3.19	1.17
Trial 4	1st	4.38	.81	4.31	1.25
	2nd	3.75	1.13	2.94	.93
	3rd	4.06	1.06	3.75	1.06
Trial 5	1st	4.56	.81	4.25	1.00
	2nd	4.00	.89	3.94	1.06
	3rd	4.19	.75	3.38	1.02

Table 8. Independent ANOVA Results for AAVL Words by Location.

Source	df	SS	MS	F	p	
Group	(1, 60)	20.21	20.21	4.38	.0405	*
Condition	(1, 60)	.44	.44	.10	.7590	
GroupXCondition	(1, 60)	8.94	8.94	1.94	.1689	
Valence	(1, 60)	28.76	28.76	23.07	<.0001	**
GroupXValence	(1, 60)	.44	.44	.35	.5556	
ConditionXValence	(1, 60)	.03	.03	.02	.8867	
GroupXConditionXValence	(1, 60)	.88	.88	.70	.4053	
Trial	(4, 240)	727.02	181.75	307.56	<.0001	**
GroupXTrial	(4, 240)	6.37	1.59	2.69	.0317	*
ConditionXTrial	(4, 240)	2.86	.72	1.21	.3069	
GroupXConditionXTrial	(4, 240)	.92	.23	.39	.8151	
ValenceXTrial	(4, 240)	4.31	1.08	2.47	.0454	*
GroupXValenceXTrial	(4, 240)	3.76	.94	2.15	.0748	
ConditionXValenceXTrial	(4, 240)	1.28	.32	.74	.5679	
GroupXConditionXValenceXTrial	(4, 240)	1.29	.32	.74	.5663	
Location	(2, 120)	380.13	190.07	73.52	<.0001	**
GroupXLocation	(2, 120)	1.01	.51	.20	.8222	
ConditionXLocation	(2, 120)	.16	.08	.03	.9689	
GroupXConditionXLocation	(2, 120)	2.28	1.14	.44	.6442	
ValenceXLocation	(2, 120)	52.54	26.27	12.68	<.0001	**
ConditionXValenceXLocation	(2, 120)	.72	.36	.17	.8408	
GroupXValenceXLocation	(2, 120)	4.21	2.10	1.02	.3654	
GroupXConditionXValenceXLocation	(2, 120)	1.68	.84	.40	.6683	
TrialXLocation	(8, 480)	37.64	4.70	6.20	<.0001	**
ConditionXTrialXLocation	(8, 480)	4.92	.61	.81	.5938	
GroupXTrialXLocation	(8, 480)	8.35	1.04	1.38	.2048	
GroupXCondtionXTrialXLocation	(8, 480)	6.96	.87	1.15	.3309	
ValenceXTrialXLocation	(8, 480)	28.21	3.53	3.95	.0002	**
ConditionXValenceXTrialXLocation	(8, 480)	10.76	1.34	1.51	.1527	
GroupXValenceXTrialXLocation	(8, 480)	9.64	1.20	1.35	.2173	
GroupXConditionXValenceXTrialXLocation	(8, 480)	10.04	1.26	1.40	.1917	

** $p \leq .01$ * $p \leq .05$

Figure Caption

Figure 1. A comparison of mean arterial pressure across trials.

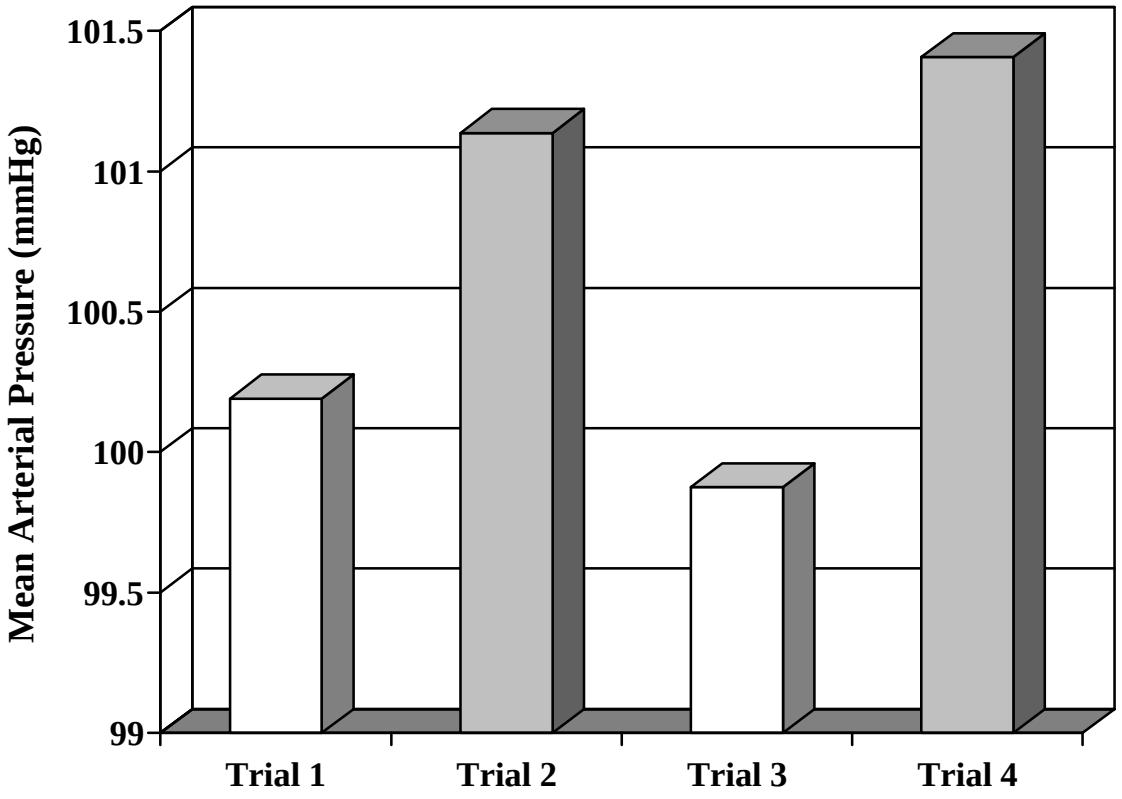


Figure Caption

Figure 2. A comparison of mean arterial pressure for the stress and no-stress conditions across trials.

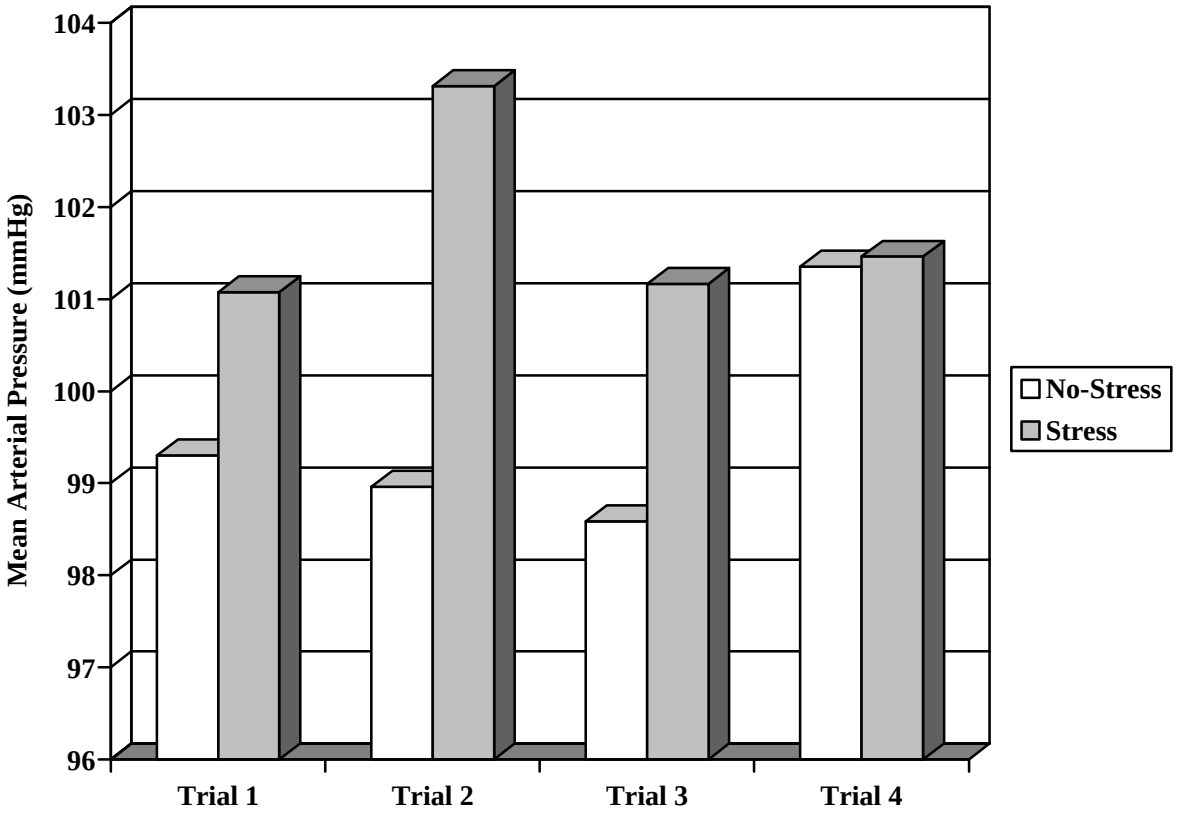


Figure Caption

Figure 3. A comparison of mean arterial pressure for the stress and no-stress conditions with the positive and negative affective list learning tasks across trials.

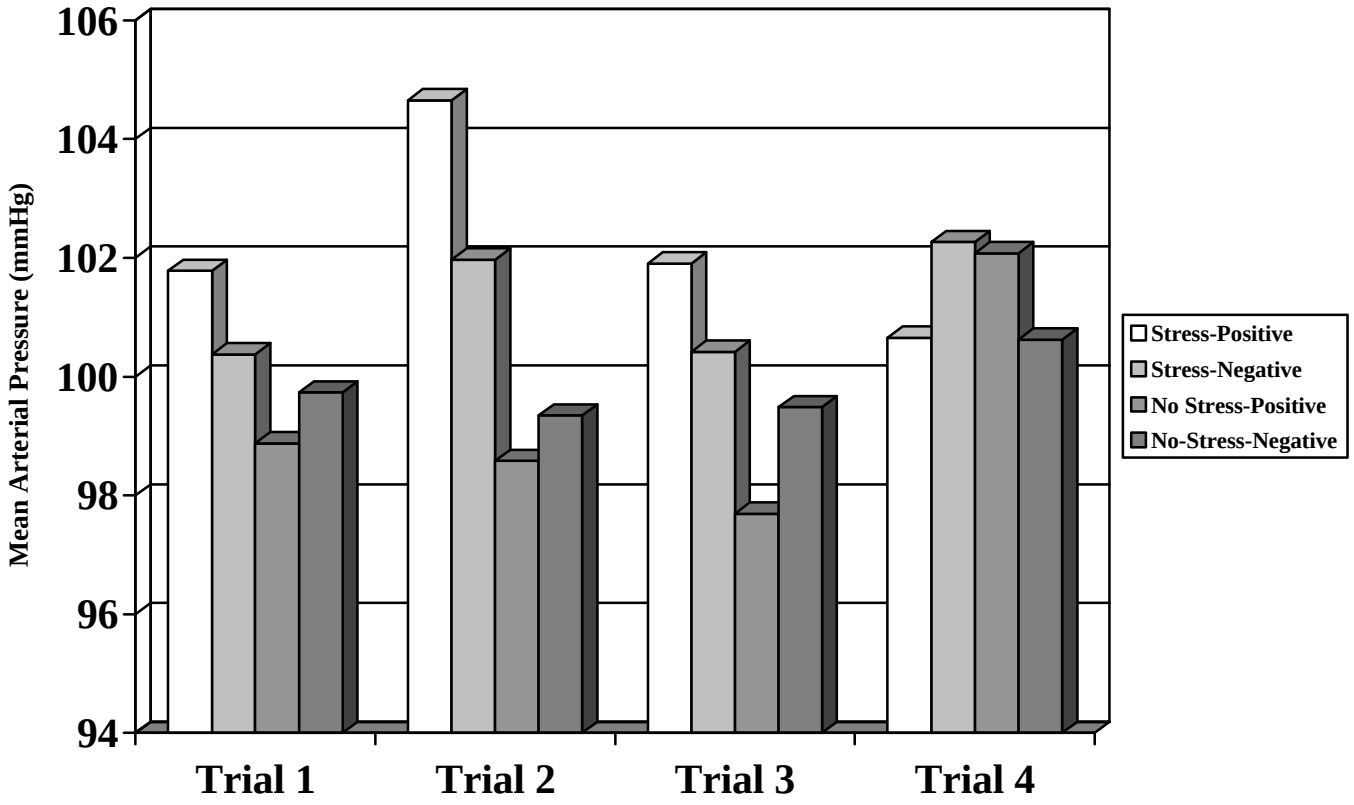


Figure Caption

Figure 4. A comparison of mean arterial pressure for the high- and low-hostile groups with the stress and no-stress conditions for the positive and negative affective list learning tasks.

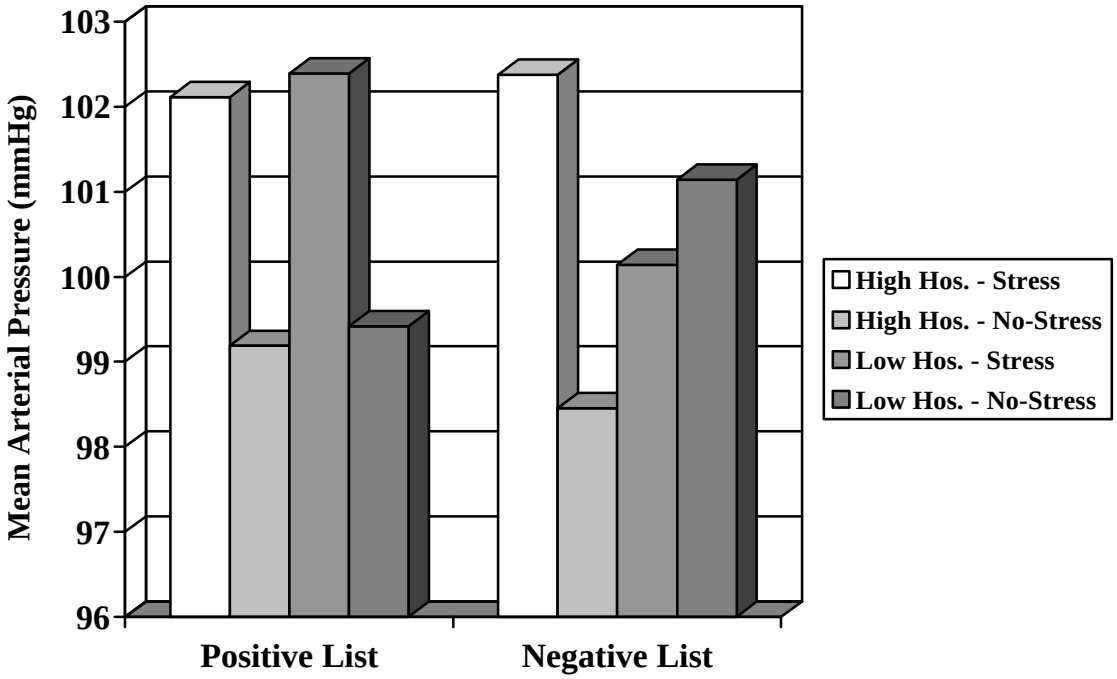


Figure Caption

Figure 5. A comparison of systolic blood pressure for the positive and negative affective list learning tasks.

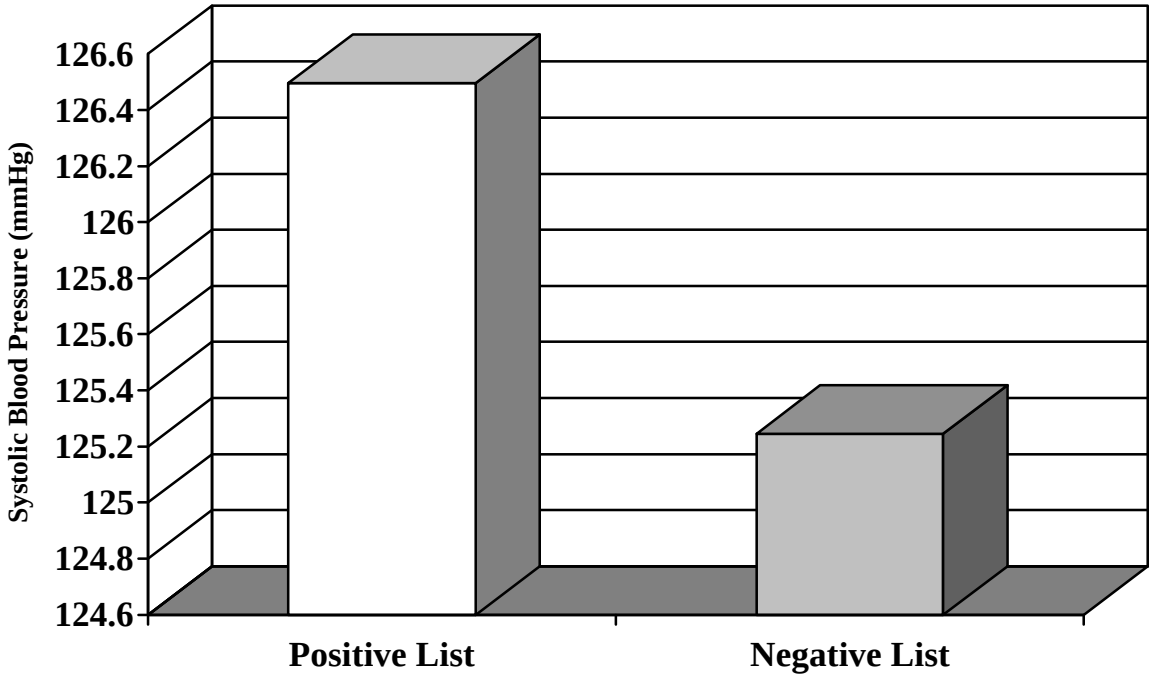


Figure Caption

Figure 6. A comparison of systolic blood pressure across trials.

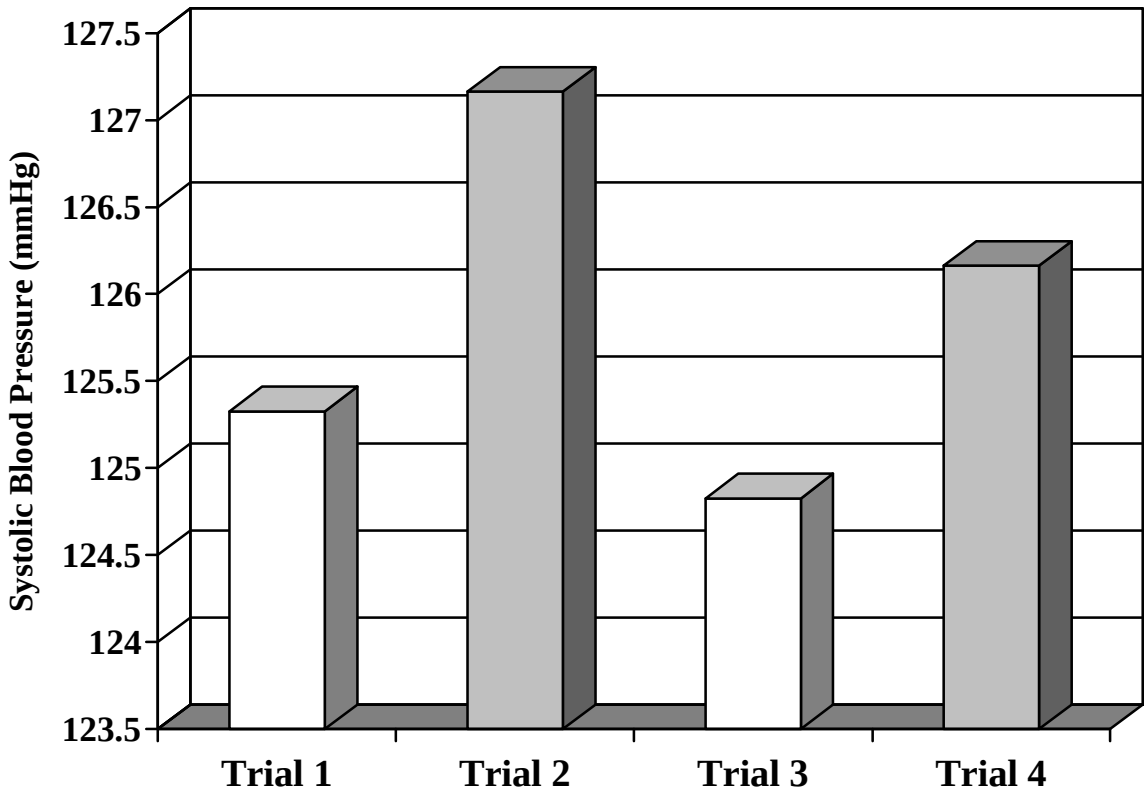


Figure Caption

Figure 7. A comparison of systolic blood pressure for the stress and no-stress conditions across trials.

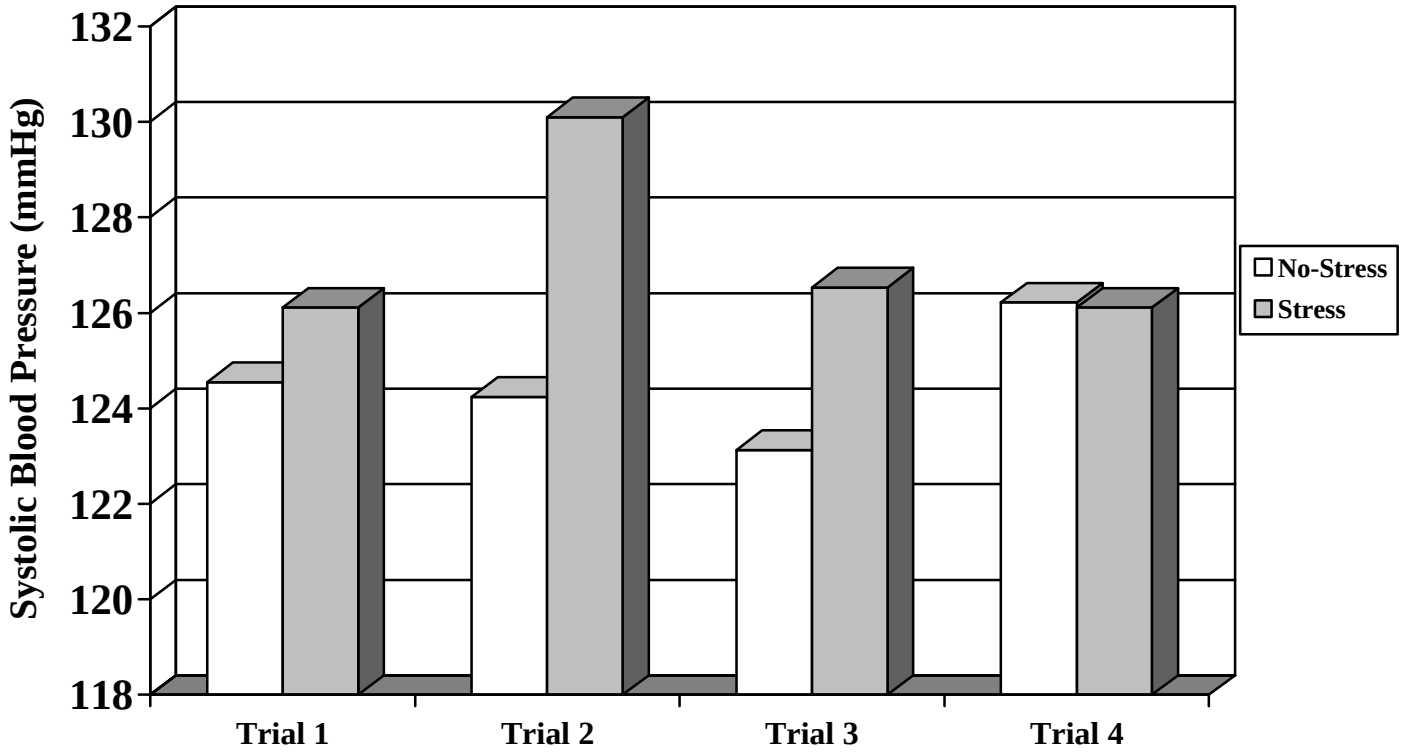


Figure Caption

Figure 8. A comparison of systolic blood pressure for the stress and no-stress conditions across the positive and negative affective list learning tasks.

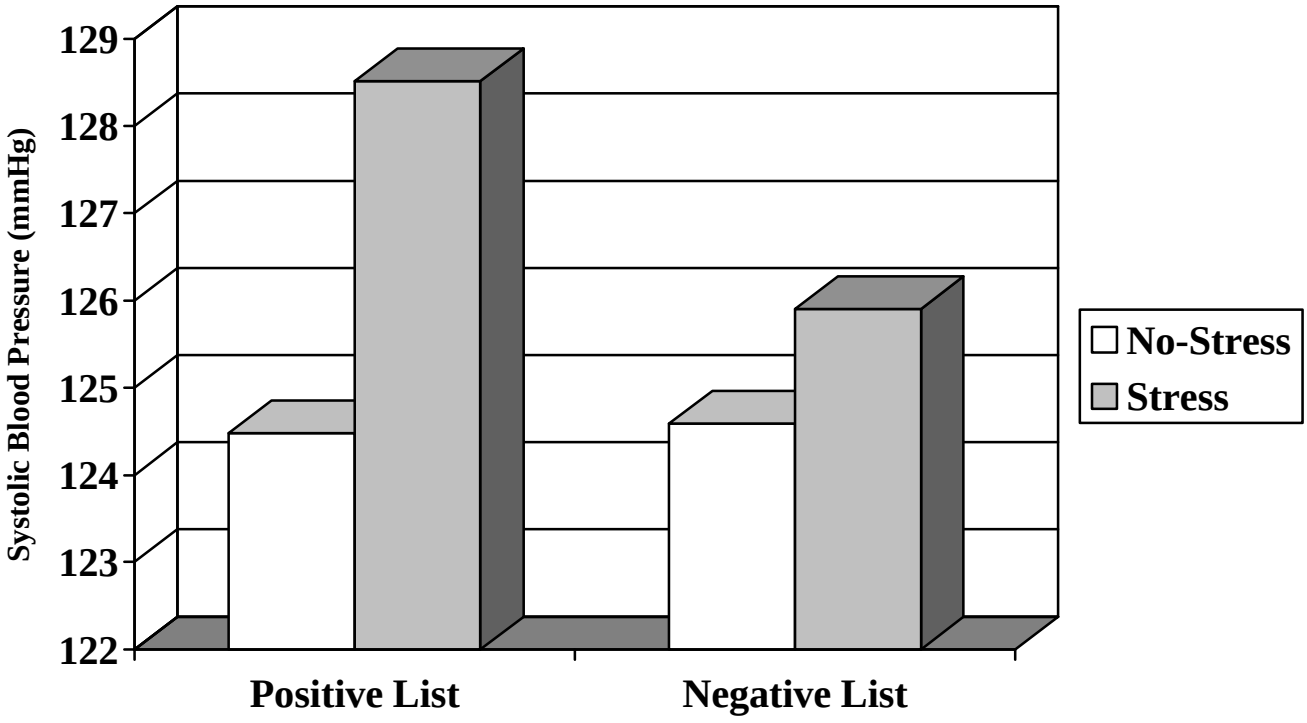


Figure Caption

Figure 9. A comparison of systolic blood pressure for high- and low-hostile groups with the stress and no-stress conditions for the positive and negative affective list learning tasks.

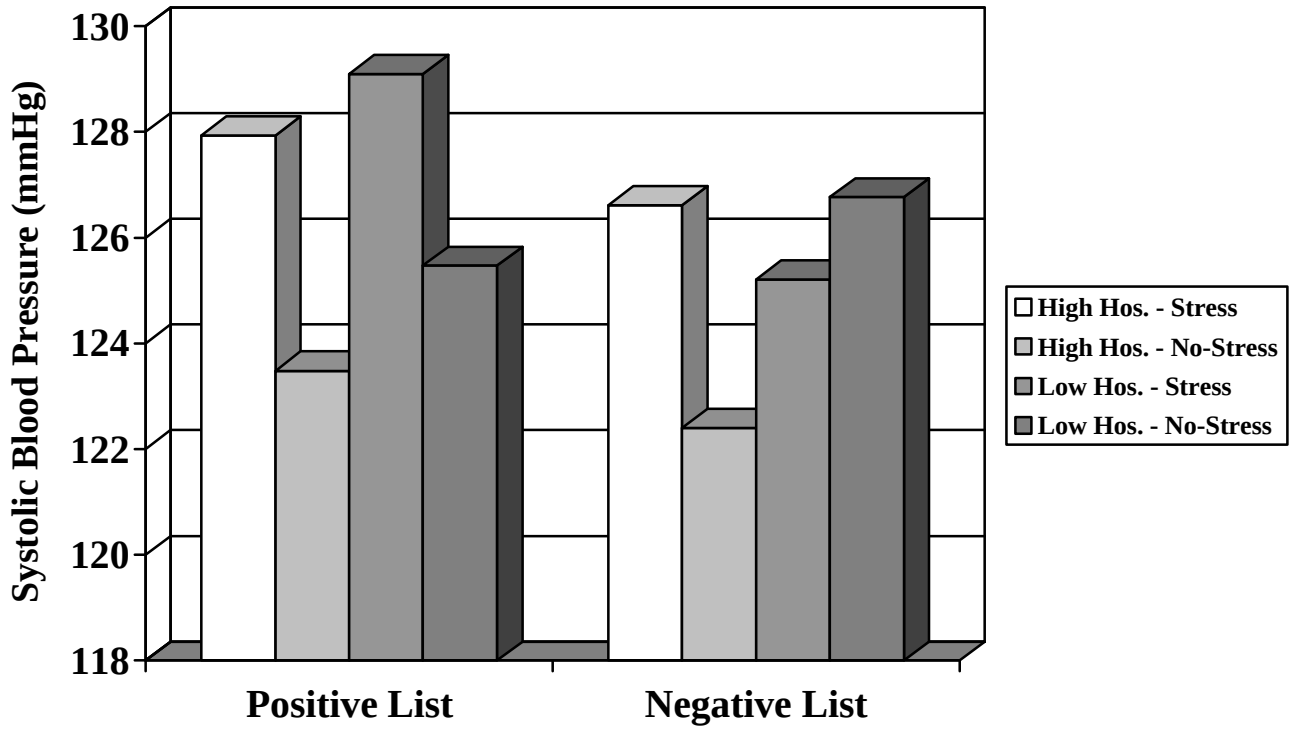


Figure Caption

Figure 10. A comparison of diastolic blood pressure for the stress and no-stress conditions with the positive and negative affective list learning tasks across trials.

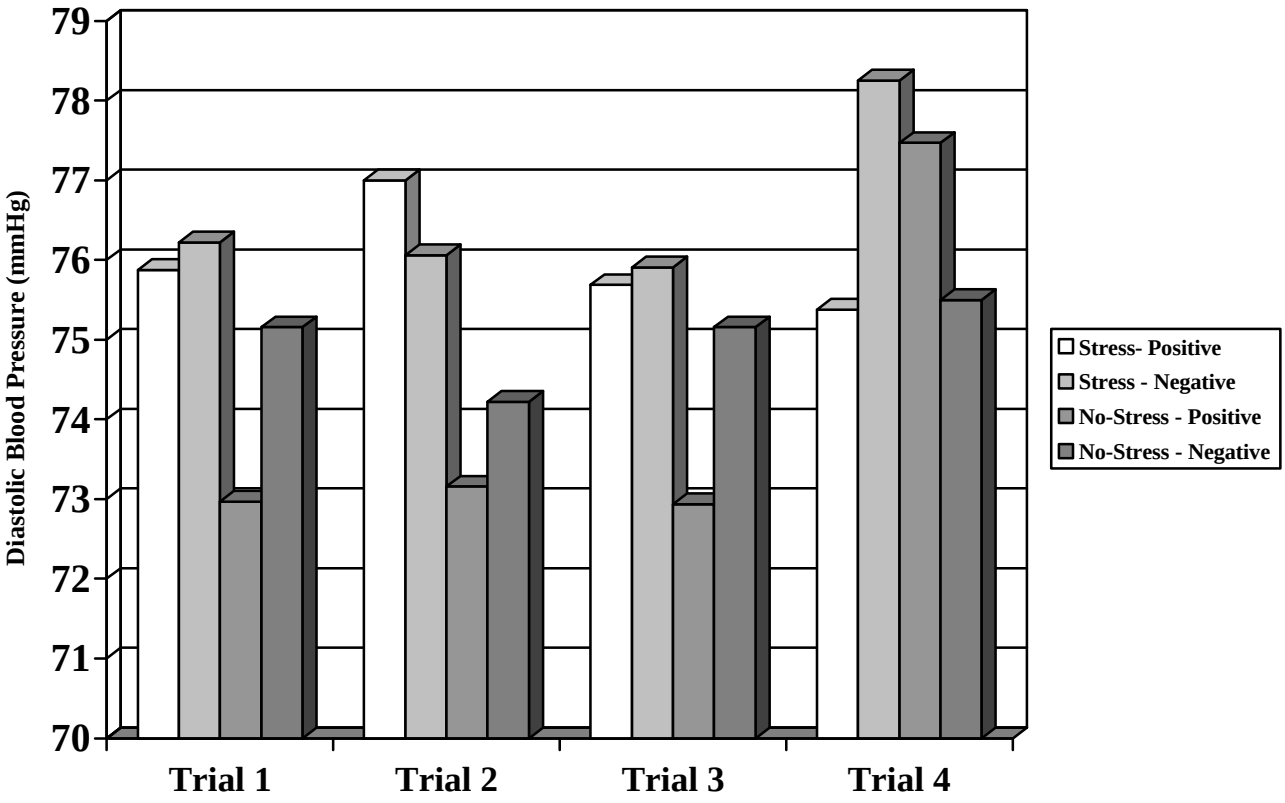


Figure Caption

Figure 11. A comparison of heart rate for the stress and no-stress conditions across trials.

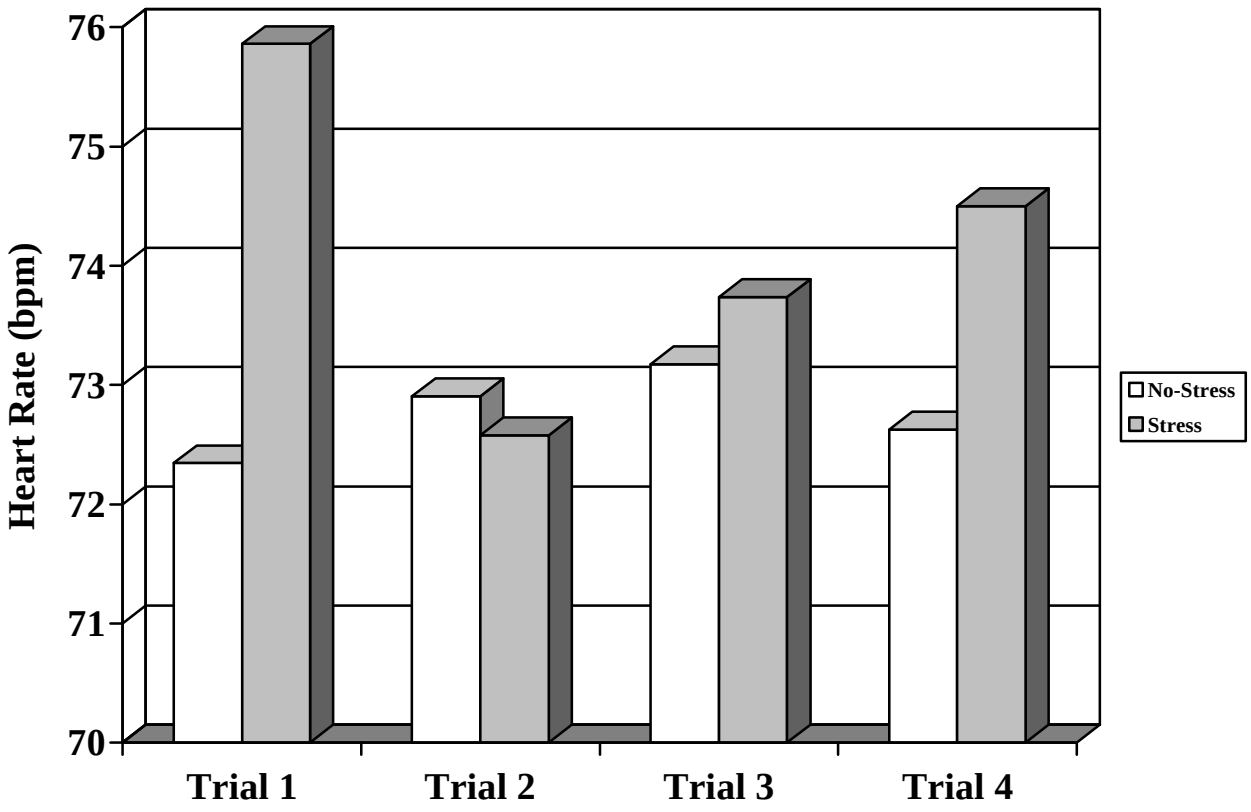


Figure Caption

Figure 12. A comparison of heart rate for the high- and low-hostile groups for the positive and negative affective list learning tasks across trials.

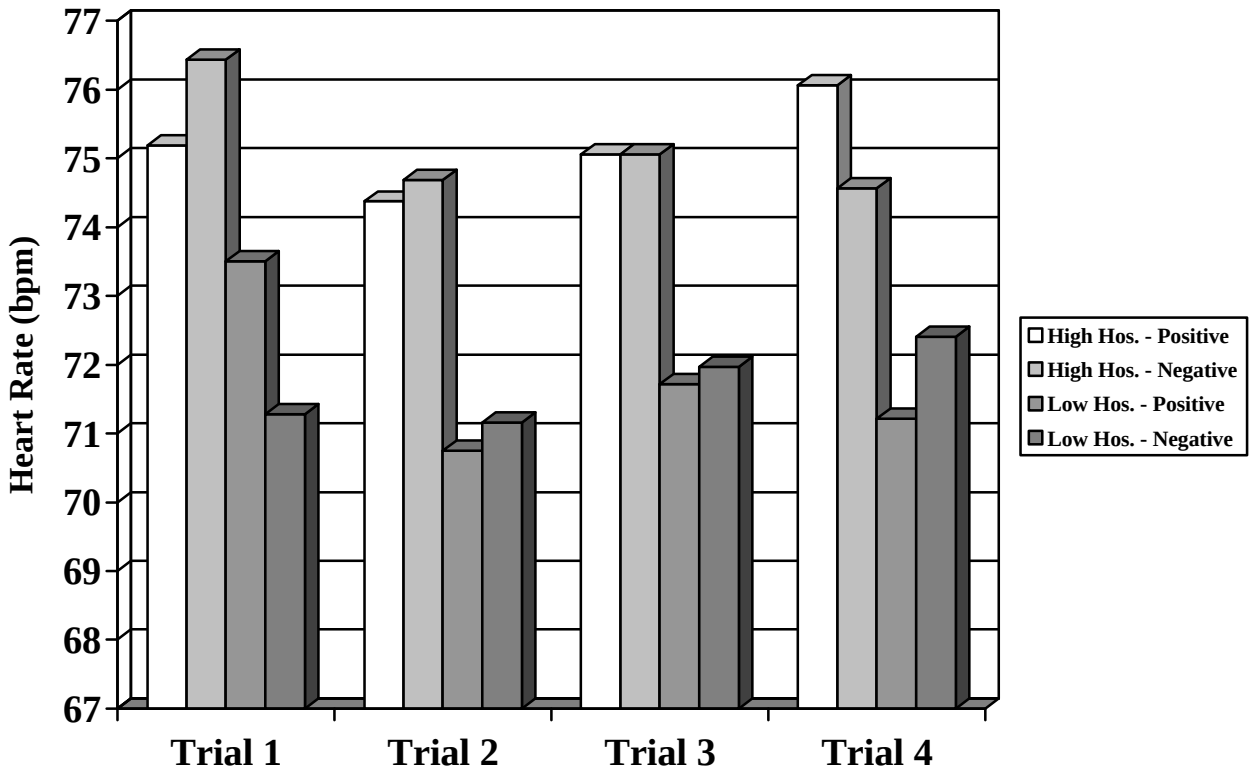


Figure Caption

Figure 13. Average words recalled per location for the high- and low-hostile groups.

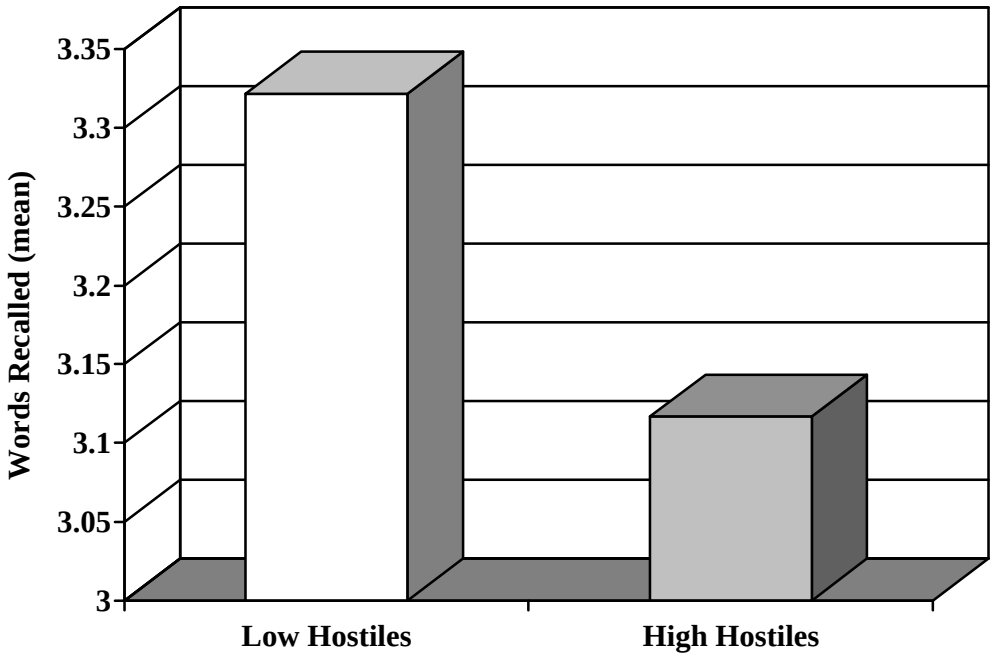


Figure Caption

Figure 14. Average words recalled per location for the positive affective list and the negative affective list.

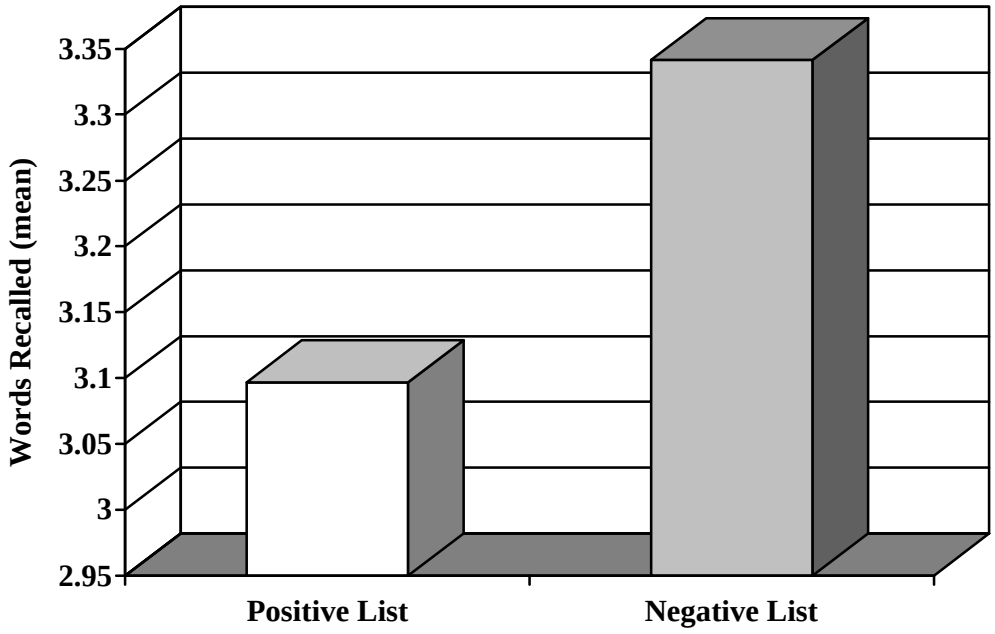


Figure Caption

Figure 15. Average words recalled per location for each trial.

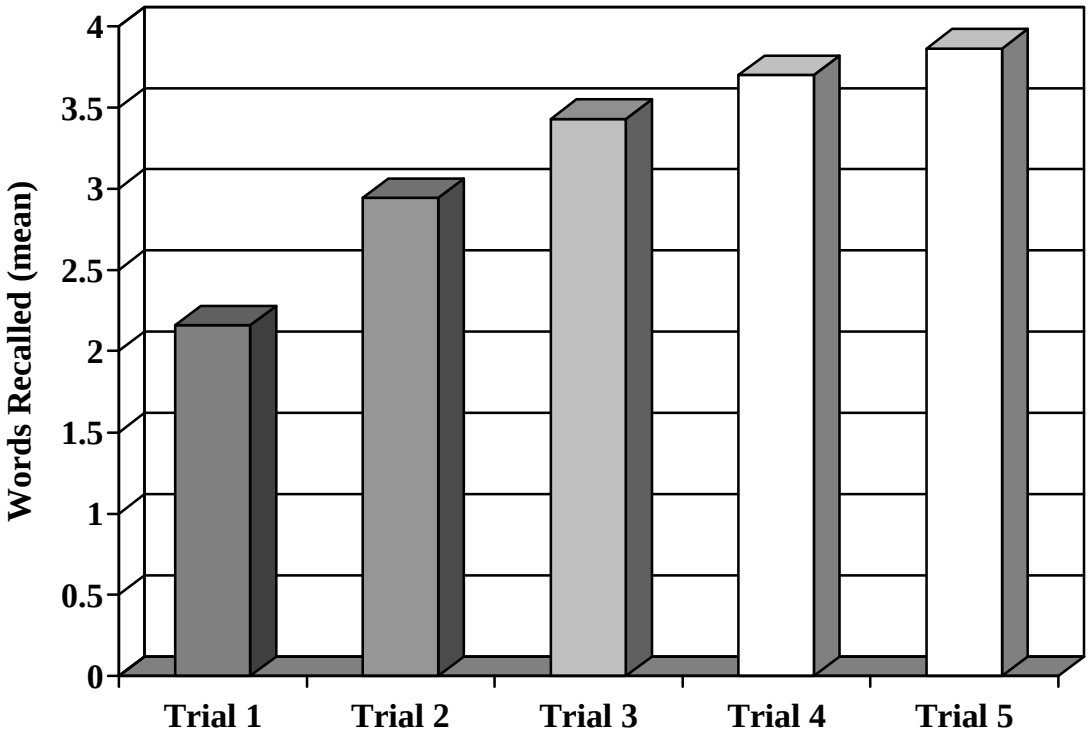


Figure Caption

Figure 16. Average words recalled per location for the first five words (first third), the second five words (second third), and the last five words (last third) in the affective lists.

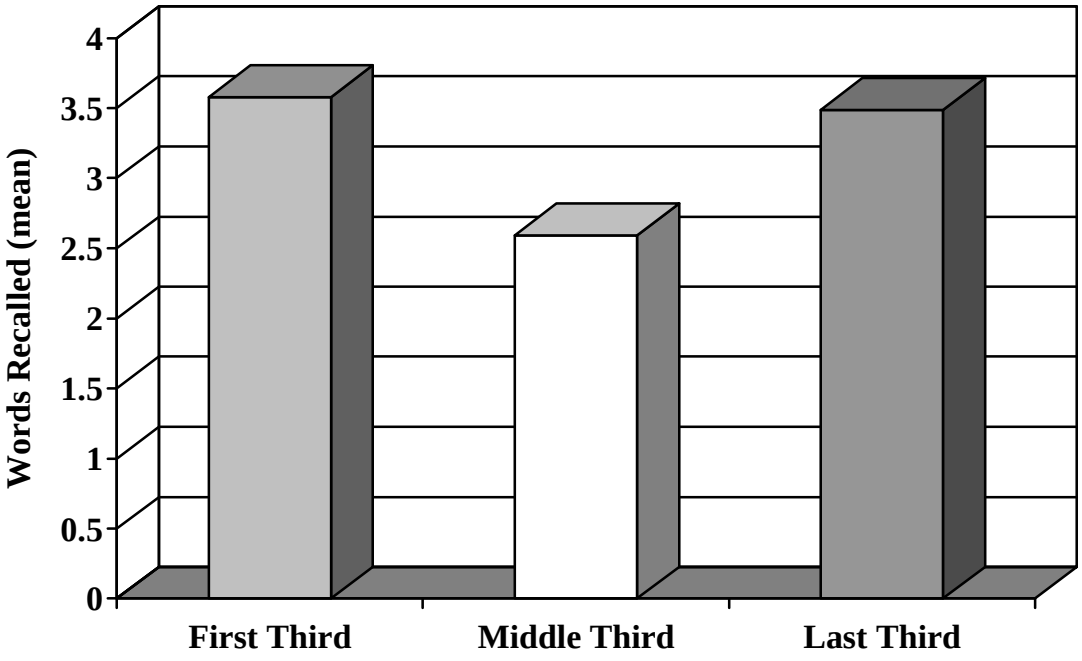


Figure Caption

Figure 17. Average words recalled per location for the high- and low-hostile groups across trials.

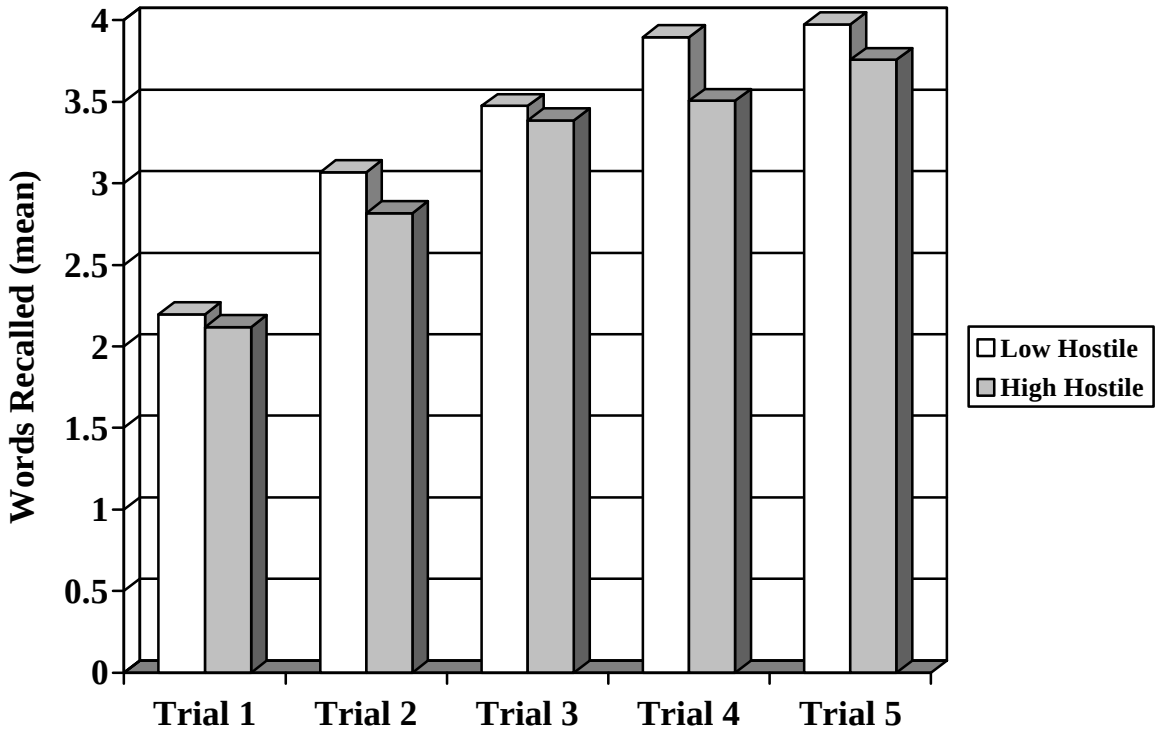


Figure Caption

Figure 18. Average words recalled per location for the positive and negative affective verbal learning tasks across trials.

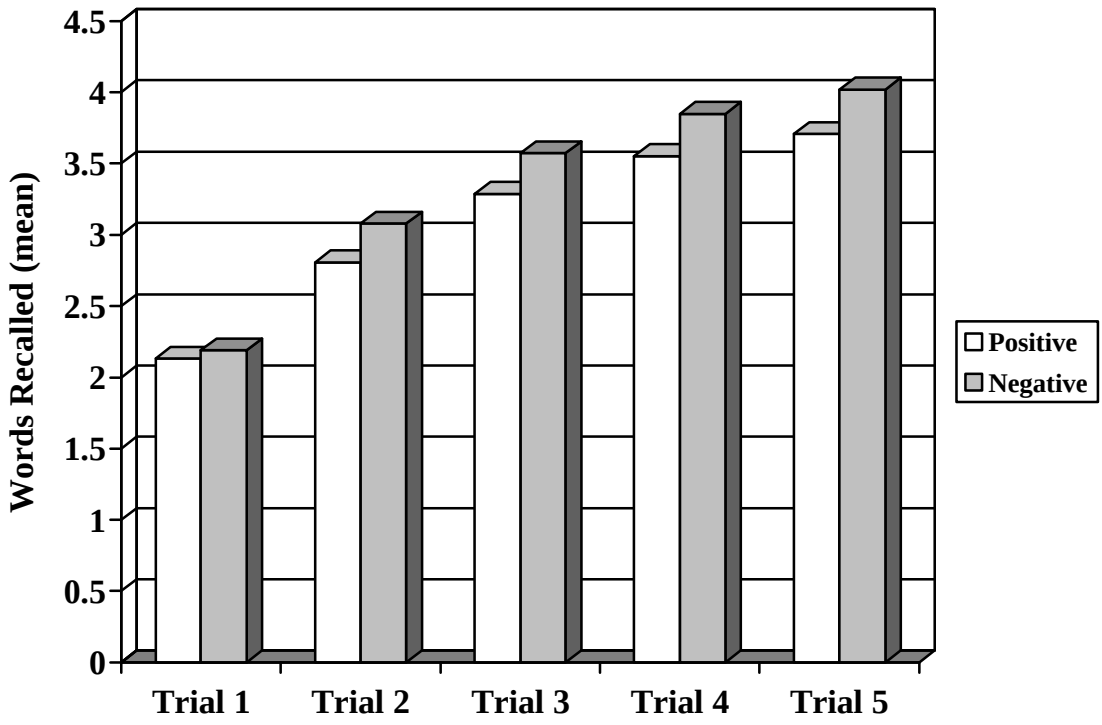


Figure Caption

Figure 19. Average words recalled per location for the positive and negative affective verbal learning tasks across the first five words (first third), the second five words (second third), and the last five words (last third) in the affective lists.

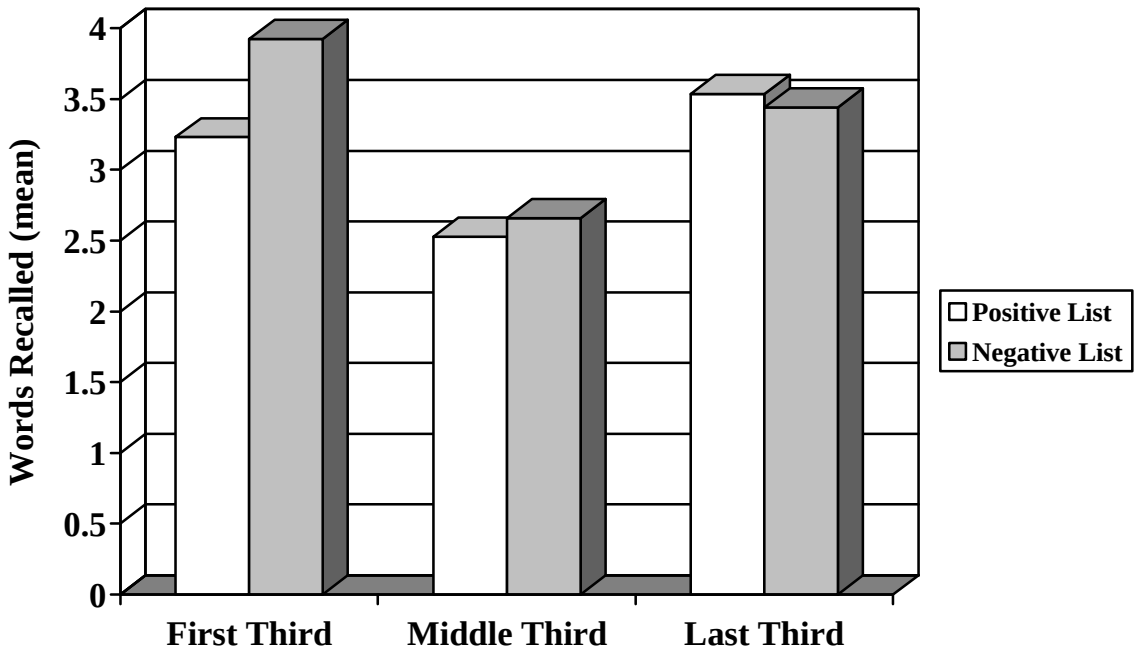


Figure Caption

Figure 20. Average words recalled per location for the first five words (first third), the second five words (second third), and the last five words (last third) in the affective lists across trials.

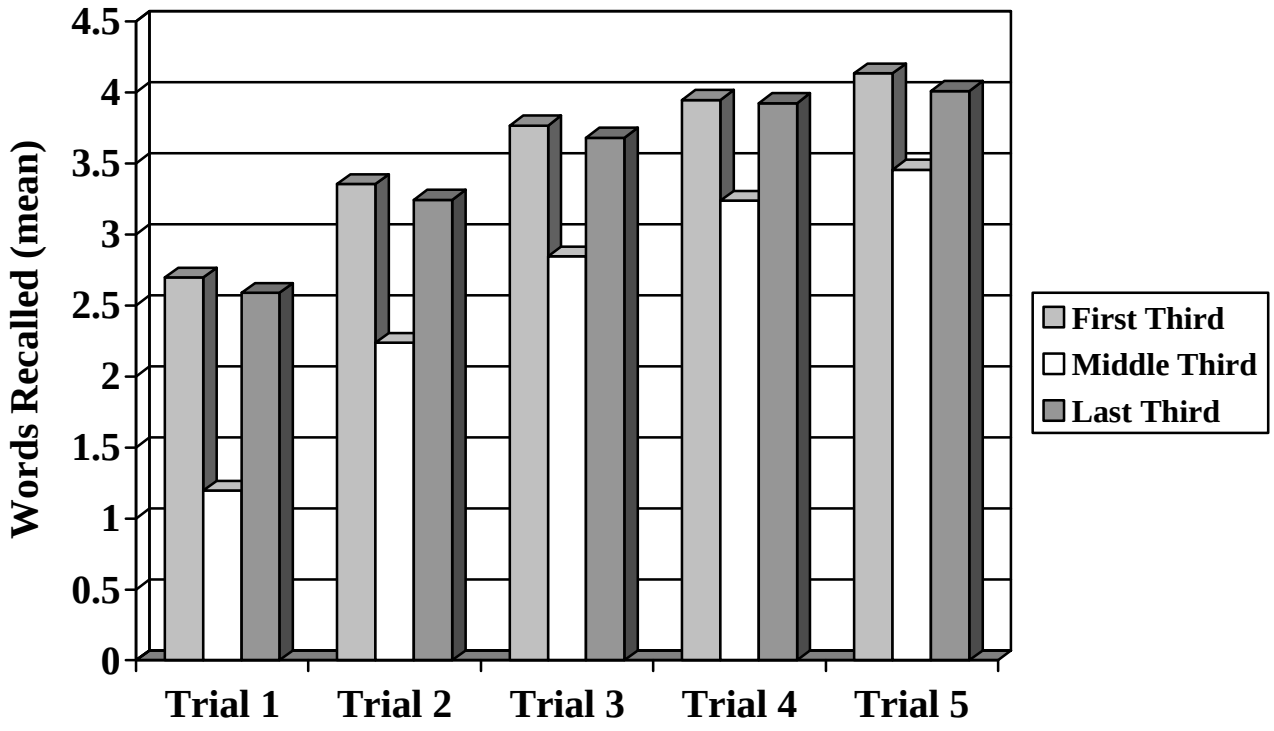
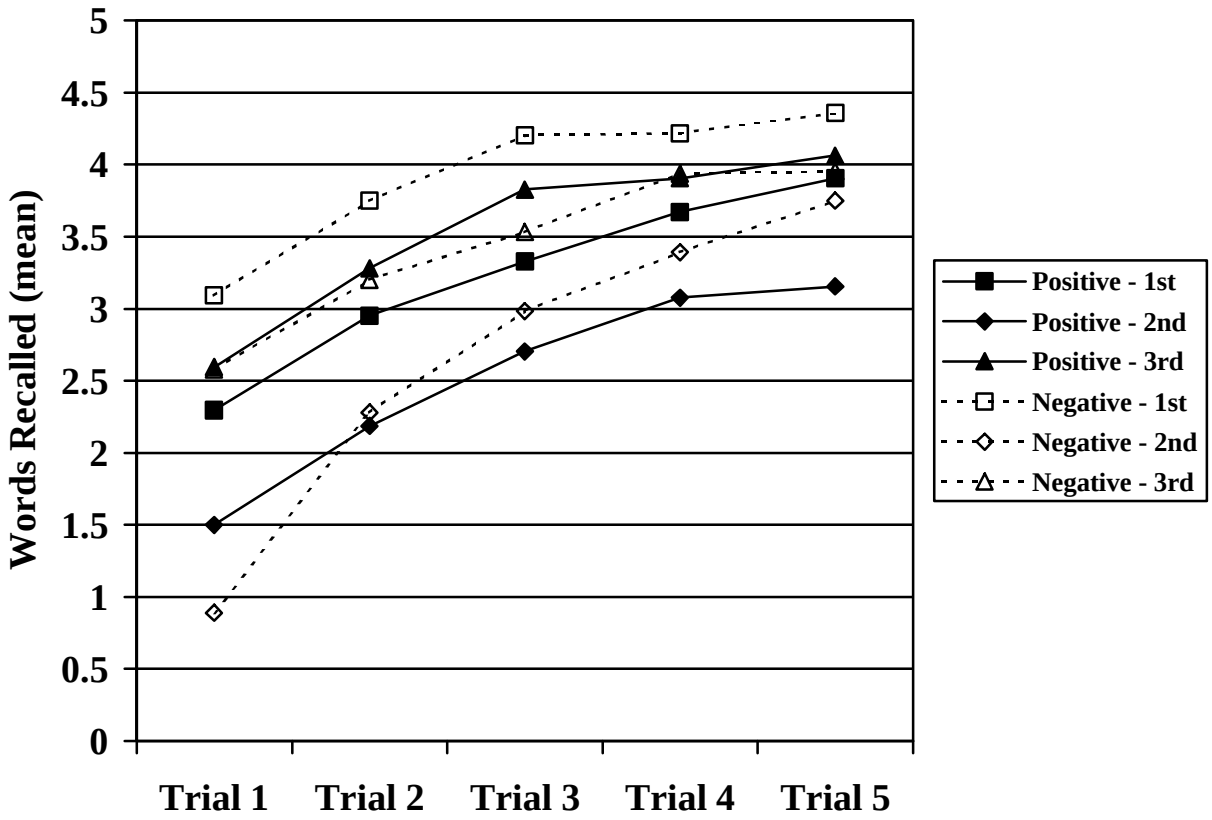


Figure Caption

Figure 21. Average words recalled per location for the positive and negative affective lists for the first five words (first third), the middle five words (middle third), and the last five words (last third) across each trial.



CURRICULUM VITAE

Brian V. Shenal, M.S.

Home address:
P. O. Box 11604
Blacksburg, Virginia 24062
(540) 961-9339
bshenal@vt.edu

Work address:
Department of Psychology
Virginia Tech
Blacksburg, Virginia 24061-0436
(540) 231-6914

PERSONAL INFORMATION

Born: March 1, 1973; Richmond, VA
Marital Status: Married

EDUCATION

Doctoral Candidate, May 1998 - present

Clinical Psychology
Emphasis in neuropsychological assessment, treatment, and research
Virginia Polytechnic Institute and State University, Blacksburg, Virginia

Master of Science, May 1998

Clinical Psychology
Emphasis in neuropsychological assessment, treatment, and research
Virginia Polytechnic Institute and State University, Blacksburg, Virginia

Bachelor of Science, December 1995

Psychology Major
Virginia Polytechnic Institute and State University, Blacksburg, Virginia

EXPERIENCE

Clinical

Graduate Clinician/Supervisor, August 1996 - present

Neuropsychological Practicum Team
Psychological Services Center and Child Study Center
Virginia Polytechnic Institute and State University
3110 Prices Fork Road; Blacksburg, Virginia 24060
Supervisor: David W. Harrison, Ph.D.

Duties included the assessment and treatment of a variety of neuropsychological difficulties, including head injury, stroke, traumatic brain injury, and other neuropsychological problems through syndrome analysis, standardized testing, and quantitative electroencephalography (QEEG) techniques. Provided individual and group supervision of graduate clinicians from January 1999 to present.

Graduate Clinician/Graduate Supervisor, May 1999 - August 1999

Summer Practicum Team
Psychological Services Center and Child Study Center
Virginia Polytechnic Institute and State University
3110 Prices Fork Road; Blacksburg, Virginia 24060
Supervisor: Lee Cooper, Ph.D.

Duties included the assessment and treatment of a variety of psychological disorders, including depression, anxiety, learning disability (LD), attention deficit disorder (ADD), and relationship problems through individual, couples, and family therapies. Provided individual and group supervision of graduate clinicians.

Psychology Trainee, May 1998 - August 1998

Clinical Externship
Mental Health Service, Veteran Affairs Medical Center
Salem, Virginia 24153
Supervisor: M. K. Johnson, Ph.D.

Completed rotations in Outpatient Psychological Services, Neuropsychology, Behavioral Medicine/Primary Care, and the Psychiatric Emergency Room. Duties included supervised psychological assessments/evaluations, neuropsychological assessments, individual and group treatments (including depression, PTSD, anxiety, psychoses, and personality disorders), and psychiatric hospitalization admission evaluations (voluntary and involuntary).

Assessment Clinician/Supervisor, January 1998 - May 1998

Child Assessment Team
Psychological Services Center and Child Study Center
Virginia Polytechnic Institute and State University
3110 Prices Fork Road; Blacksburg, Virginia 24060
Supervisor: Thomas H. Ollendick, Ph.D.

Duties included the administration of comprehensive intellectual, psycho-educational, and academic assessments for children and the provision of subsequent feedback sessions. Also provided individual supervision and assistance for graduate students in the administration of intellectual, academic, and attentional testing.

Graduate Clinician, May 1997 - August 1997

Summer Practicum Team
Psychological Services Center and Child Study Center
Virginia Polytechnic Institute and State University
3110 Prices Fork Road; Blacksburg, Virginia 24060
Supervisor: Thomas H. Ollendick, Ph.D.

Duties included the assessment and treatment of a variety of psychological disorders, including depression, anxiety, learning disability (LD), attention deficit disorder (ADD), and relationship problems through individual, couples, and family therapies.

Graduate Clinician, August 1996 - December 1997

Clinical Practicum Team
Psychological Services Center and Child Study Center
Virginia Polytechnic Institute and State University
3110 Prices Fork Road; Blacksburg, Virginia 24060
Supervisor: Russell Jones, Ph.D. (August 1997 – December 1997)
Robert S. Stephens, Ph.D. (August 1996 - May 1997)

Duties included the assessment and treatment of a variety of psychological disorders, including depression, anxiety, learning disability (LD), attention deficit disorder (ADD), and relationship problems through individual, couples, and family therapies.

Teaching

Graduate Instructor, January 2000 - present

Department of Psychology
Virginia Polytechnic Institute and State University
Blacksburg, Virginia 24061-0436

Instructor of Senior Seminar: Introduction to Clinical Psychology course for 25 students. Duties involved teaching three-credit undergraduate course, developing syllabus and course content, selecting appropriate reading, facilitation of class discussion, writing and administering exams, grading essays, and providing individual assistance to students.

Graduate Instructor, January 1999 - December 1999

Department of Psychology
Virginia Polytechnic Institute and State University
Blacksburg, Virginia 24061-0436
Student evaluations (4.0 scale): Spring 1999– 3.8;
Summer 1999 – 3.7;
Fall 1999 - 3.8.

Instructor of three (3) Abnormal Psychology courses for 75 students per course. Duties involved teaching three-credit undergraduate course, writing and administering exams, grading essays, and providing individual assistance to students.

Graduate Instructor, August 1999 - December 1999

Department of Psychology

Virginia Polytechnic Institute and State University
Blacksburg, Virginia 24061-0436
Student evaluations (4.0 scale): Spring 1999 – 3.8;

Instructor of Principles of Psychological Research course for 53 students. Duties involved teaching three-credit undergraduate course, writing and administering exams, grading essays, and providing individual assistance to students.

Graduate Instructor, August 1998 - May 1999

Department of Psychology
Virginia Polytechnic Institute and State University
Blacksburg, Virginia 24061-0436
Student evaluations (4.0 scale): Fall 1998 – 3.8;
Spring 1999 - 3.9.

Instructor of two (2) Psychological Theories of Personality courses for 75 students per course. Duties involved teaching three-credit undergraduate course, writing and administering exams, grading essays, and providing individual assistance to students.

Graduate Teaching Assistant, August 1997 - December 1997

Department of Psychology
Virginia Polytechnic Institute and State University
Blacksburg, Virginia 24061-0436
Supervisor: Thomas H. Ollendick, Ph.D.

Teaching assistant of Intellectual Assessment graduate course. Duties included supervising graduate students in the administration of intellectual and psycho-educational assessments and subsequent feedback sessions for children and adults, grading intellectual assessment reports and exams, and providing individual supervision and assistance for graduate clinicians.

Graduate Teaching Assistant, January 1997 - May 1997

Department of Psychology
Virginia Polytechnic Institute and State University
Blacksburg, Virginia 24061-0436
Supervisor: David W. Harrison, Ph.D.

Teaching assistant of two (2) Introduction to Psychology laboratories for 35 students per course. Duties involved teaching a laboratory section associated with an undergraduate introductory psychology class, writing and administering quizzes, grading essays, and providing individual assistance to students.

Research

Dissertation Research, April 1999 - present

Title: Dynamic effects of stress and hostility: Group differences in cardiovascular regulation and learning.
Virginia Polytechnic Institute and State University
Blacksburg, Virginia 24061-0436
Chairperson: David W. Harrison, Ph.D.

Designed and conducted an experiment that demonstrated the dynamic cerebral lateralization of cognitive tasks and a physical task. This project will provide useful information of how high- and low-hostile participants differ on affective learning and cardiovascular reactivity following stress induction via the cold-pressor stressor.

Research Assistant, August 1996 - present

Virginia Polytechnic Institute and State University
Blacksburg, Virginia 24061-0436
Advisor: David W. Harrison, Ph.D.

Participated in a research program designed to investigate cortical, subcortical, and autonomic correlates of emotion and cardiovascular reactivity. Utilized scientific methods in neuropsychology, psychophysiology, and neuroimaging. Duties included data analysis, project design and conceptualization, quantitative electroencephalograph (QEEG) administration and interpretation, and the supervision of undergraduate research assistants.

Preliminary Exam Research, September 1998 - December 1998

Title: The effects of light on depression: A neuropsychological analysis.
Virginia Polytechnic Institute and State University
Blacksburg, Virginia 24061-0436
Chairperson: David W. Harrison, Ph.D.

Conducted a comprehensive review of relevant literature and developed a testable neuropsychological model of the effects of light on emotion. The proposed model is comprised of four functional neuroanatomical divisions and corresponding neuropsychological emotional sequelae associated with dysfunction of each interactive quadrant.

Master's Thesis Research, January 1997 - April 1998

Title: The dynamic cerebral laterality effect: Group differences in hostility, cardiovascular regulation, and sensory recognition.
Virginia Polytechnic Institute and State University
Blacksburg, Virginia 24061-0436
Chairperson: David W. Harrison, Ph.D.

Designed and conducted an experiment that linked the right cerebral

regulation of hostility with cardiovascular regulation. Results were indicative of greater left cerebral activation following linguistic tasks and greater right cerebral activation following cognitive and physical stressors.

Undergraduate Research Assistant, January 1994 - December 1995

Virginia Polytechnic Institute and State University
Blacksburg, Virginia 24061-0436
Advisor: David W. Harrison, Ph.D.

Participated in a research program designed to investigate the neuropsychological correlates of emotion. Participated in projects of auditory affective learning and covert recognition. Conducted relevant literature reviews and participated in data collection.

Undergraduate Research Assistant, January 1995 - December 1995

Virginia Polytechnic Institute and State University
Blacksburg, Virginia 24061-0436
Advisor: Russell T. Jones, Ph.D.

Participated in a research program designed to investigate post-traumatic stress disorders in children. Conducted relevant literature reviews and participated in data collection.

Employment

Assessment Specialist, March 1999 - March 2000

Columbia/HCA – Montgomery Regional Hospital/Lewis-Gale Pavilion
Respond Assessment Service
1900 Electric Road
Salem, Virginia 24153

Duties included psychiatric hospitalization admission evaluations (voluntary and involuntary), outpatient referrals, brief bereavement counseling, and the provision of crisis hotline services.

Employment Assessment Administrator, May 1997 - October 1999

Affiliates in Psychology and Therapy, Inc.
845 Fourth Avenue, Suite 301
Huntington, WV 25701

Duties included administering and proctoring job relevant assessments (i.e., personality inventories, achievement tests, writing samples) for prospective employees.

Therapist/ Certified Rehabilitation Provider, August 1994 - January 1997

Hollins Head Injury Program

7916 Williamson Road
Roanoke, Virginia 24020
Supervisor: Bill James, MA, LPC, CRP

Responsibilities included designing and implementing individual and group therapeutic protocols based on neuropsychological assessments, recording ongoing assessments of treatment efficacy, developing overall rehabilitation goals and activities, and providing strategies for activities of daily living and health maintenance.

HONORS AND AWARDS

Who's Who in American Colleges and Universities, 1995 - 1996
President's List of Notable Academic Achievement, 1995
Dean's List of Academic Achievement, 1994 - 1995
Psi Chi, National Honor Society in Psychology, 1994 - 1995

PROFESSIONAL ORGANIZATIONS AND ACTIVITIES

Service Activities

Graduate Student Assembly –Psychology Representative, 1997 - present
Commission on Graduate Studies and Policies, 1997
Committee on Graduate Relations, 1997
Earlysville Fire Department, Emergency Medical Technician (EMT), 1991 - 1995

Invited Addresses

Presentation of "Issues of Cognition and Aging" to four (4) staff training sessions and one (1) residential family meeting at Richfield Retirement Community and Alzheimer Residential Center, Salem, Virginia. August 1999

Journal Reviews

Guest Reviewer – Journal of Gender, Culture, and Health, October 1998

Membership

American Psychological Association (APA) - student affiliate
Division 40 (Neuropsychology) – student affiliate
National Academy of Neuropsychology (NAN) - student affiliate
Southeastern Psychological Association (SEPA) – student affiliate

PUBLICATIONS

Refereed Articles

Moore, T. M., Shenal, B. V., Rhodes, R. D., & Harrison, D. W. (1999). Forensic neuropsychological evaluations and quantitative electroencephalography (QEEG). Forensic Examiner, March/April, 12-15.

Harrison, D. W., Demaree, H. A., Shenal, B. V., & Everhart, D. E. (1998). QEEG-assisted neuropsychological evaluation of autism. International Journal of Neuroscience, 93(1-2), 133-140.

Snyder, K. A., Harrison, D. W., & Shenal, B. V. (1997). The affective auditory verbal learning test (AAVLT): Peripheral arousal correlates. Archives of Clinical Neuropsychology, 13(3), 251-258.

Manuscripts in Submission

Shenal, B. V., & Harrison, D. W. (2000). The neuropsychology of depression: A critical review and comprehensive model. Manuscript submitted for publication.

Shenal, B. V., & Harrison, D. W. (2000). The dynamic cerebral laterality effect: Group differences in hostility, cardiovascular regulation, and sensory recognition. Manuscript submitted for publication.

Shenal, B. V., Rhodes, R. D., Moore, T. M., Higgins, D. A., & Harrison, D. W. (2000). Quantitative electroencephalography (QEEG) facilitates neuropsychological syndrome analysis: An alternative to the nomothetic approach. Manuscript submitted for publication.

Manuscripts in Preparation

Shenal, B. V., Crews, Jr., W. D., & Harrison, D. W. (1999). Assessment techniques of neuropsychological sequelae following electrical shock: QEEG and syndrome analysis. Manuscript in preparation for submission.

Williamson, J., Rhodes, R. D., Shenal, B. V., & Harrison, D. W. (1999). Case study: Topographical brain mapping in an adolescent diagnosed expressive aprosodic. Manuscript in preparation for publication.

Everhart, D. E., Harrison, D. W., & Shenal, B. V. (1999). Neuropsychological effects of anxiety without depression on grip strength. Manuscript in preparation for submission.

REFEREED ABSTRACTS

Shenal, B. V., Rhodes, R. D., & Harrison, D. W. (1999). Cardiovascular reactivity and hostility: The dynamic cerebral laterality effect. Archives of Clinical Neuropsychology, 14(8), 671-672.

Rhodes, R. D., Shenal, B. V., & Harrison, D. W. (1999). A functional system approach to the study of hemispheric asymmetries in the processing of emotion. Archives of Clinical Neuropsychology, 14(8), 771-772.

Rhodes, R. D., Shenal, B. V., & Harrison, D. W. (1999). Cardiovascular dynamics and cerebral asymmetry as a function of hostility and stress. Archives of Clinical Neuropsychology, 14(8), 772.

Williamson, J., Everhart, E., Shenal, B., & Emerson, C. (in press). Hand fatigue asymmetry in the motor performance of depressed boys. Clinical Neuropsychologist.

Everhart, D. E., Shenal, B. V., & Harrison, D. W. (1999). Asymmetrical regulation of cardiovascular functioning and fluency among anxious and nonanxious men. Archives of Clinical Neuropsychology, 14(1), 113.

Everhart, D. E., Shenal, B. V., & Harrison, D. W. (1998). Neuropsychological effects of anxiety without depression on functional motor asymmetry. Archives of Clinical Neuropsychology, 13(1), 55.

Demaree, H. A., Shenal, B. V., & Harrison, D. W. (1998). Analysis of QEEG and cardiovascular responses to stress. Archives of Clinical Neuropsychology, 13(1), 127.

CONFERENCE PRESENTATIONS AND PAPERS

Primary Author/Presenter

Shenal, B. V., Rhodes, R. D., & Harrison, D.W. (1999). Cardiovascular reactivity: The dynamic cerebral laterality effect. Accepted for the annual meeting of the National Association of Neuropsychology, San Antonio, TX. November, 1999.

Shenal, B. V., & Harrison, D. W. (1999). Cerebral laterality dynamics: Cardiovascular reactivity and hostility. Annual meeting of the Virginia Psychological Association, Virginia Beach, VA. April, 1999.

Shenal, B. V., Rhodes, R. D., & Harrison, D. W. (1999). Dynamic cerebral lateralization: Hostility and cardiovascular reactivity to stress. Annual meeting of the Southeastern Psychological Association, Savannah, GA. March, 1999.

Shenal, B. V., & Harrison, D. W. (1998). Hostility, cardiovascular regulation, and linguistic recognition. The Annual Virginia Collegiate Psychology Conference, Blacksburg, VA. April, 1998.

Shenal, B. V., Rhodes, R. D., Moore, T. M., Higgins, D. A., & Harrison, D. W. (1998). Quantitative electroencephalography (QEEG) facilitates neuropsychological syndrome analysis: An alternative to the nomothetic approach. Annual meeting of the Virginia Psychological Association, Wintergreen, VA. April, 1998.

Shenal, B. V., Crews,, Jr., W. D., Barth, J. T., & Harrison, D. W. (1997). Neuropsychological and topographical brain mapping dysfunction following severe accidental electrical shock: A case study. Annual meeting of the Virginia Psychological Association, Roanoke, VA. February, 1997.

Significant Contributions – Presentations and Papers

Rhodes, R. D., Shenal, B. V., & Harrison, D. W. (1999). Cardiovascular dynamics and cerebral asymmetry as a function of hostility and stress. Accepted for the annual meeting of the National Association of Neuropsychology, San Antonio, TX. November, 1999.

Rhodes, R. D., Shenal, B. V., & Harrison, D. W. (1999). A functional system approach to the study of hemispheric asymmetries in the processing of emotion. Accepted for the annual meeting of the National Association of Neuropsychology, San Antonio, TX. November, 1999.

Williamson, J., Everhart, E., Shenal, B., & Emerson, C. (1999). Hand fatigue asymmetry in the motor performance of depressed boys. Annual meeting of the American Psychological Association, Boston, MA. August, 1999.

Rhodes, R. D., Shenal, B. V., & Harrison, D. W. (1999). Cerebral asymmetry in cardiovascular regulation: Hostility and stress. Annual meeting of the Southeastern Psychological Association Conference, Savannah, GA. March, 1999.

Rhodes, R. D., Shenal, B. V., & Harrison, D. W. (1999). Cerebral mediation of emotion: Hostility and the autonomic nervous system. Annual meeting of the Southeastern Psychological Association Conference, Savannah, GA. March, 1999.

Everhart, D. E., Shenal, B. V., & Harrison, D. W. (1998). Asymmetrical regulation of cardiovascular functioning and fluency among anxious and nonanxious men. Annual meeting of the National Academy of Neuropsychology. Washington, DC. November, 1998.

Demaree, H. A., Shenal, B. V., & Harrison, D. W. (1997). Analysis of QEEG and cardiovascular responses to stress. Annual meeting of the National Academy of Neuropsychology. Las Vegas, NV. November, 1997.

Everhart, D. E., Harrison, D. W., & Shenal, B. V. (1997). Neuropsychological effects of anxiety without depression on functional motor asymmetry. Annual meeting of the National Academy of Neuropsychology. Las Vegas, NV. November, 1997.