The Dynamic Cerebral Laterality Effect:
Group Differences in Hostility, Cardiovascular Regulation, and Sensory Recognition

by

Brian Vincent Shenal

Thesis submitted to the Faculty of

Virginia Polytechnic Institute and State University

in partial fulfillment of the requirements for the degree of

MASTER OF SCIENCE

in

Psychology

David W. Harrison, Ph.D., Chair
Richard A. Winett, Ph.D.
Martha Ann Bell, Ph.D.

April, 1998

Blacksburg, Virginia

Key Words: Hostility, Lateralization, Stress, Asymmetry, Tachistoscope, Dichotic Listening, Cardiovascular Disease

THE DYNAMIC CEREBRAL LATERALITY EFFECT: GROUP DIFFERENCES IN HOSTILITY, CARDIOVASCCULAR REGULATION, AND SENSORY RECOGNITION

By Brian Vincent Shenal

David W. Harrison, Ph.D., Chairman
Psychology
(ABSTRACT)

This experiment tested two hypotheses linking the right cerebral regulation of hostility and cardiovascular arousal. First, replication of previous research supporting heightened cardiovascular (systolic blood pressure, diastolic blood pressure, and heart rate) reactivity among high hostile participants was attempted. Second, dynamic variations in functional cerebral asymmetry in response to pain (cold pressor) and emotional linguistic processing was measured.

Low- and high-hostile participants were identified using the Cook Medley Hostility Scale (CMHS). All participants completed either the negative affective verbal learning test (Experiment 1) or the cold pressor paradigm (Experiment 2). Cardiovascular measures (SBP, DBP, and HR) were recorded and either dichotic listening procedures (Experiment 1) or tachistoscopic lexical recognition procedures (Experiment 2) were administered before and after the stressor.

The primary finding of this research was greater left cerebral activation (decreased cardiovascular reactivity) following the dichotic phoneme listening and the tachistoscopic lexical recognition tasks and greater right cerebral activation following pain (cold pressor) and emotionally linguistic (affective verbal learning) stressors.

ACKNOWLEDGEMENTS

This project has culminated from many exceptional individual efforts. First, Dr. David W. Harrison offered continuous support and invaluable guidance throughout this project. This work reflects his commitment, expertise, and character. I greatly appreciate his constant encouragement and motivation. My committee members were also extremely helpful throughout this project. Dr. Bell and Dr. Winett offered supportive energies, theoretical insights, and encouraging suggestions and feedback throughout this research endeavor. I would also like to thank Heath Demaree for his ideas, influences, and support with this research project. With these individuals, this work remained an enjoyable and educational project.

I would also like to thank my parents, Johnny and Emily Shenal, for their continuous and lifelong support. Their commitment to me and my education is truly reflected in this project.

This work is dedicated to my wife, Cathy Shenal, whose understanding, tolerance, and love provided me with the confidence and support to work diligently on this project.

TABLE OF CONTENTS

THE DYNAMIC CEREBRAL LATERALITY EFFECT: GROUP DIFFERENCES IN HOSTILITY, CARDIOVASCCULAR REGULATION, AND SENSORY RECOGNITION

Heart disease is a major health threat to Americans. Each year, in the United States alone, about 750,000 people die of heart disease, 1.25 million people suffer heart attacks, and over 6 million people display heart disease symptoms (Wardlaw, Insel, & Seyler, 1992). The cost of cardiovascular disease to society including medications, health professional services, and lost productivity due to death and disability is over $80 billion per year (American Heart Association, 1988). 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.

Recent research has examined hostility and its correlates with cardiovascular reactivity to provide a better understanding of risk factors. As this literature has evolved, hostility has been implicated as a primary risk factor for cardiovascular disease. This paper will describe pertinent theories of hostility and emotion, recent developments within the literature on the neuropsychology of negative emotions, and a logical step which would integrate these literatures to investigate the neuropsychological underpinnings of hostility and cardiovascular reactivity or lability. It is proposed that such research will afford 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.

Neuropsychological research provides a useful framework to study emotions, such as hostility, and their correlates. Prominent neuropsychological theories of emotion and hostility will be reviewed. Functional neuro-anatomical systems involved in negative emotional processing, and potentially hostility, will be reviewed. As will be discussed, multiple theories exist describing cerebral lateralization for emotion and, potentially, hostility. The preponderance of research to date has implicated the right cerebrum in the reception, comprehension, expression, and regulation of negative emotions (Heilman, Bowers, & Valenstein, 1985). Alternate accounts and controversy over cerebral mechanisms 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).

In order to study hostility and cardiovascular reactivity, many researchers have utilized stress paradigms. Cardiovascular measures such as heart rate (HR), 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) and a predictor indicant for cardiovascular risks (e.g., Suls & Wan, 1993). It has been suggested that cardiovascular reactivity to stress may be differentially mediated by the right and left cerebrum (Demaree & Harrison, 1997a, 1997b). 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) and quantitative electroencephalography (Demaree & Harrison, 1997b) has been implicated in cardiovascular stability and in decreased blood pressure. The latter finding corresponds with positive emotional experiences and learning a positive verbal list in recent research from our laboratory (Snyder, Harrison, & Shenal, 1998).

Recently, we have investigated shifts in cerebral lateralization in response to the cold pressor pain and to cognitive stressors (Demaree & Harrison, 1997a; Snyder, Harrison, & Shenal, 1998). Findings indicate that hostile subjects evidence right cerebrum activation to the cold pressor stressor while low-hostile individuals evidence a diametrically opposite activation of the left cerebrum (Demaree & Harrison, 1997b). This paper will propose a method for the continued study of dynamic cerebral lateralization and potential applications for education and the prevention of cardiovascular risks.

Neuropsychology of emotion

Early research on the biological substrates of emotion focused on the autonomic changes that accompany affect. Subcortical limbic system circuits were described which mediate specific emotional behaviors (e.g., Heilman, et al., 1985). As a result of recent research, however, it is clear that the cerebral cortex plays an important role in emotional behavior (see Davidson, 1993, for review). As a result of increased focus on the cerebral cortex, researchers have proposed multiple neuropsychological theories of emotion.

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

Similarly, Heilman (1982) speculated that the right cerebrum may have greater control of the subcortical systems, which are largely responsible for arousal and emotion. Additionally, Heilman, Bowers, and Valenstein (1985) reported that emotions, in general, are processed in the right cerebrum and that negative emotions are often associated with increased arousal. These researchers used galvanic skin response (GSR) measures to demonstrate that subjects with right cerebrum dysfunction evidenced altered arousal and reactivity. Further, they demonstrated that arousal within posterior cerebral regions, particularly right parietal and right temporal, is likely to result in emotional responses (Heilman, 1993). The researchers interpreted these findings as support for the right cerebral mediation of emotion. Alternately, 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).

In contrast to Heilman et al. (1985), Tucker and Frederick (1989) described a balance model of emotion. It was argued that the left cerebrum primarily processes positive emotions and the right cerebrum primarily processes negative emotions. With the balance model, 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 results in an increase of negative emotion and a deactivation of the right cerebrum results in an increase of positive emotion.

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 through the use of electroencephalographic (EEG) measures, that disgust is associated with less alpha power (i.e., more activation) in the right frontal region and anterior temporal electrodes 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.

Additionally, Robinson, Kubos, Starr, Rao, and Price (1984) have reported that left frontal lobe damage resulted in depressive symptomatology and that patients who developed hostile, manic-like, symptomatology were more likely to have sustained right hemisphere damage, sparing the left hemisphere. Similarly, Davidson (1993) proposed that activation of the left anterior region is associated with approach-related emotions and that activation of the right anterior region is associated with withdrawal-related emotions. As evidenced in Robinson et al's (1984) findings, damage to the left cerebrum may result in depression, which can be viewed as a lack of approach-related emotions. Further, damage to the right cerebrum may result in increased hostility, which can be viewed as a lack of withdrawal-related emotions.

Hostility

Hostility and 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. Hostility has traditionally 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).

Neuropsychology of hostility

Recently, research has begun to focus on 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 activity (Herridge, Harrison, & Demaree, 1997; Demaree & Harrison, 1997a, 1997b). In a recent review of hostility and self-awareness, Demaree and Harrison (1997c) 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. 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, Bowers, & Valenstein, 1993).

Furthermore, if the construct of hostility holds 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.

Clinical 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 activity 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; Everhart, Demaree, & Harrison, 1997). 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 research

Studies of hostility and its effects on the bilateral processing and recognition of stimuli have led to research on emotional sensation and 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, Harrison, and Demaree (1997) found evidence that hostile participants were more accurate at assessing angry and happy affective faces and less accurate at assessing a neutral affective face in their left visual field than low-hostile participants. This experiment demonstrated that hostile individuals show improved 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 understanding of 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) (Harbin, 1989; Dembroski, MacDougall, Herd, & Shields, 1983; Glass & Contrada, 1982). Numerous studies have indicated that hostility may be the primary 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.

Role of stress

It is common to utilize cold pressor stress methods to study hostility and its correlates. Cardiovascular measures such as heart rate (HR), systolic blood pressure (SBP), and diastolic blood pressure (DBP), which are commonly used as dependent variables, generally suggest increased brain activity to stress for high-hostiles. A current issue within neuropsychology is the respective roles that the right and left cerebral regions play in mediating cardiovascular responsivity. It is hypothesized that as the right cerebrum is activated by painful cold pressor stress cardiovascular measures become more reactive as a result of a dual-task.

Hostility from a functional cerebral space model

Dual-tasks are traditionally viewed as tasks that activate adjacent regions of the cortex. Demanding tasks may overload a cerebrum and decrease processing capacity for other tasks, especially when tasks compete for similar cerebral areas (Kinsbourne & Hisock, 1983). Tasks represented in adjacent regions are thought to be affected via spreading activation through a connected cerebral network (Kinsbourne & Byrd, 1985). As Kinsbourne and Byrd (1985) demonstrated, the effort or load of a task is integral to explain priming or interference effects (Kinsbourne & Byrd, 1985). It is hypothesized that cold pressor stress for high-hostile individuals produces an interference effect with cardiovascular control, leading to increased cardiovascular reactivity and disregulation. The very essence of the relationship between this affect and the decompensation in the physiological control of blood pressure is suggestive of a dual task interference effect. Moreover, it is proposed that the functional cerebral-space that processes anger or hostility may mediate the ongoing dynamically responsive cardiovascular system as a background or secondary task. Under dual task demands (i.e., hostility and cardiovascular control) the secondary task of cardiovascular control may suffer.

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 increase cardiovascular activity (e.g., Demaree & Harrison, 1997a, 1997b; Glass, McKinney, Hofschire, & Fedorko, 1990). Further, it has been shown 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 thereby increasing cardiovascular reactivity.

Past research has supported the notion of right cerebrum involvement during stress and arousal inducing conditions (Heilman & Van Den Abell, 1979; Tucker, Roth, Arneson, & Buckingham, 1977). More recently, Demaree and Harrison (1997a, 1997b) suggested that because stressful cardiovascular measures may tap right cerebrum activity, the effects of stress and arousal on hostile individuals may best be accounted for by right cerebrum reactivity. Tasks, such as the painful cold pressor, which have been shown to induce painful stress, provide a method for testing these hypotheses.

Additional tasks thought to induce stress and arousal have recently been pursued and developed. Snyder and Harrison (1997) recently developed the Auditory Affective Verbal Learning Test 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). In an experiment subsequent to the Auditory Affective Verbal Learning Test development, the affective version was shown to affect cardiovascular function (Snyder, Harrison, & Shenal, 1998). As with the painful cold pressor task, 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. This reactivity may be accounted for by dual task processing of affect and cardiovascular reactivity with decrements of the regulation occurring on the latter.

Cerebral dynamics

Historically, cerebral functioning has been conceptionalized as the study of static, localized “centers.” Researchers have described these areas as fixed functional areas. Recently, however, research has 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 cerebral activation.

Cerebral dynamics with the dichotic listening method

The dichotic listening task has been shown to be effective for studying sensory perception and a cerebral lateralization shift. A study by Demaree and Harrison (1997a) utilized a cold pressor method and dichotic listening procedures to evaluate lateralization of cerebral activity in response to painful stress. This study found that high-hostile participants evidenced greater right cerebral activation to cold pressor stress as indicated by enhanced left ear intention. Diametrically opposite effects were found among the low-hostile participants with left cerebral activation to the painful cold pressor stressor. This phenomenon was described as a dynamic cerebral laterality effect with divergent cerebral processing mechanisms underlying high-hostile cardiovascular reactive participants compared to low-hostile, less cardiovascular reactive participants. Additionally, these data suggest a positive relationship between right cerebral activity and cardiovascular arousal. Further research will enhance our understanding of the dynamic lateralization and its applied implications.

Cerebral dynamics with the tachistoscopic method

As part of the systematic research of dynamic laterality, the tachistoscope may provide an effective methodology to further demonstrate a cerebral lateralization shift through the visual sensory modality. The tachistoscopic method has been demonstrated to be effective for studying sensory perception. It has been shown that there exists an asymmetry among the left and the right cerebrums during a word recognition task (McKeever & Huling, 1971). This study demonstrated that participants correctly identified words presented to their right visual field (RVF), which is processed by the left cerebrum, more often than words presented to their left visual field (LVF), which is processed by the right cerebrum. Additionally, Herridge, Harrison, and Demaree (1997) found left versus right cerebral asymmetry during a facial affect recognition task.

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 right cerebral activation for high-hostiles and left cerebral activation for low-hostiles. Similarly, Herridge, Harrison, and Demaree (1997) found that reactivity to cold pressor stress persists longer at the left hand. This left extremity persistence in GSR arousal was interpreted to result from persistence in right cerebral activation among high-hostiles. As Demaree and Harrison (1997a, 1997b) point out, the use of methods more sensitive to brain lateralization may produce additional furtive data about dynamic cerebral lateralization. Snyder, Harrison, and Shenal (1998) demonstrated that the negative affective learning task increased cardiovascular reactivity. Specifically, individuals' heart rate and blood pressure increased following the negative affective list learning task. It was suggested that the cognitive negative list learning task was an analogous stressor to the physical cold pressor stressor. An increased understanding of the negative affective list learning task's effect 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 more efficient prevention methods compared to invasive, painful physical tasks.

The purpose of this experiment was to learn more about the role of painful 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 study aimed to demonstrate dynamic cerebral lateralization to affective auditory verbal learning stress and physical cold pressor stress by comparing high- and low-hostile individuals' cardiovascular reactivity and bilateral sensory recognition preference following stress.

The proposed model describes a system where cognitive stress mediates the accuracy of an individual's perception of auditorily presented word fragments. In the first experiment, stress was induced via a cognitive task (AAVLT). In the second experiment, a physical task (cold pressor) was used to induce stress. The first experiment utilized a negative affective list learning condition, the dichotic listening presentation of dual-concurrent phonemes to the left and right ears, and cardiovascular measures to demonstrate dynamic cerebral laterality for low- and high-hostile participants. The second experiment utilized a physical stressor, the visual tachistoscopic method and cardiovascular measures, to demonstrate dynamic cerebral laterality for low- and high- hostiles. These experiments aimed to demonstrate that high-hostile participants show greater cardiovascular reactivity to cognitive and physical stress than low-hostiles across multiple sensory modalities. Further, it was hypothesized that participants would demonstrate dynamic cerebral lateralization. It was expected that the lateralization shift would be evidenced through high-hostile participants' relative increased left visual field accuracy following cold pressor stress compared to low-hostile participants.

EXPERIMENT 1

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 SBP, DBP, and HR. Second, lateralization of brain activity was measured by ear advantage observed using dual-phoneme auditory dichotic listening procedures.

HYPOTHESES

Hypothesis 1: High-hostile participants will show greater cardiovascular (Blood Pressure, Heart Rate) reactivity to stress on exposure to the cognitive list learning task than low-hostile participants.

Hypothesis 2: Low- and High- hostile participants will differ on the dichotic listening task following cognitive stress. This will be evidenced through relative increased left ear accuracy scores (right cerebral arousal) for high-hostiles and right ear accuracy scores (left cerebral arousal) for low-hostiles.

METHOD

Methods for this experiment have been adapted from procedures used by Demaree and Harrison (1997a), Herridge, Harrison, and Demaree (1997), and McKeever and Huling (1971).

Participants

Group testing at Virginia Tech, a large state university, yielded the experimental groups composed of high-hostile and low-hostile male participants. Volunteer participants were recruited from the Virginia Tech undergraduate pool. A sign-up folder, which explained the experiment and scheduled screening times, was made available to undergraduates. The participants were informed that this experiment was a two-phase experiment and that they would receive one credit point for each session that they attended. Further, they were informed that participation in the group screening did not guarantee inclusion in the experimental phase. It was expected that 100 participants would be screened to complete the final participant pool of 30 needed for the experiment.

Inventories and Questionnaires

The screening involved groups of 10 - 30 participants. An Informed Consent Form 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, 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 are self report, and are 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 more on this questionnaire.

Participants must have self-reported no history of hearing difficulties, major illness, or major head injury on a 18 question Medical History Questionnaire. 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 experiment.

The Cook-Medley Hostility Scale (CMHS) was then administered to the group (Cook & Medley, 1954). This scale consisted 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." 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." 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. Thirty participants (fifteen each) were classified as high-hostile or low-hostile based on the scores from this questionnaire (CMHS).

Participants also completed the validated self-report Beck Depression Inventory (Beck, 1972). This measure consisted of 21 questions which tap specific symptoms, attitudes, and behavioral manifestations of depression. Scores on this inventory, may range from 0 to 63, with 0 – 9 considered “nondepressed,” 10 – 15 indicating “mild depression,” 16 – 19 indicating “mild-moderate depression,” 20 – 29 indicating “moderate-severe depression,” and 30 – 63 indicating “severe depression.”

Participants also completed the validated self-report State-Trait Anxiety Inventory (Speilberger, 1968). This is a 40 item questionnaire which inventories state and trait anxiety. Twenty questions are provided to evaluate the way a person feels “right now” (state anxiety). In addition, 20 questions are provided to assess the way a person feels “in general” (trait anxiety). Scores on the STAI may range from 20 to 80 for the State Inventory and from 20 to 80 for the Trait Inventory.

The participants chosen for the experimental phase 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. Participants again completed all self-report questionnaires.

Apparatus

The participants were seated in a sound-attenuated room. The automated programming equipment and experimenter were located in an adjacent room. The participants were monitored through a one-way observation window and were prompted via an intercom.

Cardiovascular

Blood pressure and heart rate were assessed using the Industrial and Biomedical Sensors Corporation SD-700A Blood Pressure / Pulse Monitor. Systolic blood pressure and diastolic blood pressure was assessed using the Korotkoff method.

Dichotic Listening

A computer-synthesized audiotape, made by the Kresge Hearing Research Laboratory, of thirty pairs of concurrently voiced consonant vowels (CV) (ba, da, ga, ka, pa, ta) was played for each participant. This tape has been used as a dichotic listening device in numerous studies (e.g., Demaree & Harrison, 1997a). Stimuli were presented at about 75 dB by a Marantz PMD-340 dual channel tape player using Koss Pro/4X Plus headphones. The interstimulus interval was 6 seconds. The six CVs were printed as 2 cm black upper-case letters on a 96 X 144 cm index card displayed about .5 m in front of the participant.

Auditory Stimuli

Stimuli were two letter phonemes, and duplicated the phonemes used by Demaree and Harrison (1997a). The six phonemes were: *ba, da, ga, ka, pa,* and *ta*. Two sets of phoneme pairs were prepared and responses were recorded on the Participant Response Form. The first set of phonemes consisted of the following pairs: *pa-ka,da-ka,ka-ta,pa-ba,ka-da,ga-ka,ta-ka,ta-ga,ba-da,ga-ta,ta-ba,ka-ba,ga-pa,ga-ba,da-pa,da-ga,ka-pa,pa-ta,da-ta,ba-pa,ba-ga,da-ba,ga-da,ta-da,ka-ga,ba-ta,pa-ga,pa-da,ba-ka,ta-pa*. The second set of phonemes consisted of the following pairs: *pa-ba,ka-pa,ga-ta,da-pa,ka-da,ba-ta,ga-pa,da-ga,ba-ka,pa-da,ba-ga,pa-ga,ta-ba,ka-ga,ka-ta,ta-da,ga-ka,ta-pa,ba-pa,da-ka,ta-ga,ga-ba,da-ta,ka-ba,ga-da,da-ba,pa-ka,ta-ka,ba-da,pa-ta*. The order of the presentation of these phoneme sets was counterbalanced across participants. A stimulus recognition report card was presented to the participants listing these six phonemes.

During each stimulus presentation one phoneme was presented to the left ear and one phoneme was presented to the right ear. Scores for each participant were the number of correct phoneme recognitions originating at each ear. A total of 30 recognitions at each ear were possible. Participants were not informed of the accuracy of their responses.

Affective Auditory Verbal Learning Test

The affective auditory verbal learning procedure utilized a lists of 15 words consisting of a negatively valenced word list. Construction of the negative affective list of the AAVL consisted of selecting 15 words having the lowest mean pleasing (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. Snyder, Harrison, and Shenal (1998) demonstrated that the negative affective learning task increased cardiovascular reactivity. Specifically, individuals' heart rate and blood pressure increased following the negative affective list learning task.

Procedure

High- and low-hostile participants were asked to participate in the experiment following the group screening. Participants were requested to sign another Informed Consent Form. To continue in the experiment, participants were required to identify ten of twelve two letter phonemes presented individually to each ear.

Cardiovascular Phase (Baseline):

Participants were fitted for BP and HR readings. The blood pressure cuff was strapped to each participant's left upper-arm. The cuff was positioned on the left arm with the transducer centered over the brachial artery. Palpation was used to determine the position of the brachial artery. The cuff's transducer was positioned approximately 2.5 cm superior to the anticubital space. The bladder was inflated to 30 mmHg above the expected systolic blood pressure. Deflation did not exceed the rate of 3 mmHg per second. Exhaust was performed automatically following the reading.

The experimenter then left the room and gave the following instructions: "Please sit still in the chair and take about one minute to become accustomed to your surroundings." Heart rate (HR), systolic blood pressure (SBP), and diastolic blood pressure (DBP) data were collected twice at the end of the baseline period. A third reading was taken if the first two readings differed by 6 beats per minute (HR) or 20 mmHg for either SBP or DBP.

Dichotic Listening Phase

Each trial consisted of a brief and concurrent presentation of the stimuli at the left and right ears.

Participants were told:

> You are about to hear thirty trials of word fragments. You will hear a word fragment in the left ear and a word fragment in the right ear at the same time. Your job is to point to the word fragment on the chart that you hear most clearly. Please remember to report only one word fragment.

All responses were recorded.

Cardiovascular Phase (Post-Dichotic1):

Participant's HR, SBP, and DBP were then assessed and recorded following the procedure in the initial cardiovascular phase.

Negative List Learning Phase:

Participants then 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 negative list for a series of five trials. Words were read to the participants at a rate of approximately one word every 1.0 second. When the participant no longer recalled any more words, the next trial began. Participants' responses were recorded on the data sheet.

Cardiovascular Phase (Post-Cognitive Affective Stressor):

Participant's HR, SBP, and DBP were then assessed and recorded following the procedures in the initial cardiovascular phase.

Dichotic Listening Phase:

The auditory recognition task was then performed again according to the procedures outlined in the initial dichotic listening phase.

Cardiovascular Phase (Post-Dichotic2):

Participant's HR, SBP, and DBP were then assessed and recorded following the procedures in the initial cardiovascular phase.

Participants were thoroughly debriefed at the end of the experiment.

Analyses

T-tests were conducted to assess differences among high-hostile and low-hostile participants on the CMHS, BDI, STAI, and the handedness questionnaire.

For each participant, the Percentage of Correct responses (POC) index was calculated for auditory recognition accuracy using the following formula:

$$POC = (pR - pL) / (pR + pL)$$

pR = proportion of correctly identified right ear phonemes

pL = proportion of correctly identified left ear phonemes

The POC scores range from +1 (perfect right ear advantage) to -1 (perfect left ear advantage). Independent ANOVAs were conducted using auditory recognition variables – POC, and number of correctly identified stimuli presented to both the left (NOL) and right ear (NOR). A mixed design ANOVA with the fixed factor of Group(2) (high-hostile and low-hostile) and repeated measure of Trial(2) (pre-stress and post-stress) was used.

Multivariate analysis of variance (MANOVA) was performed on the cardiovascular variables (SBP, DBP, HR) to address reactivity to the cold pressor test: Group2 X (Trial2 X Participant).

Analysis of variance (ANOVA) with the fixed factor of Group(2) and the repeated measure of Trial(5) was performed on the cognitive affective learning task to address group differences in the list learning task.

A priori criterion for reliability was p ≤ .05.

Results

Descriptive Measures

T-tests were conducted on scores obtained on the Cook-Medley Hostility Scale, Coren, Porac and Duncan's Laterality Questionnaire, Beck Depression Inventory, and State – Trait Anxiety Inventory in order to compare groups (low- and high- 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 = 32.80, SD = 4.26) than did low-hostiles (M = 16.67, SD = 3.74), t(28) = 11.02, p ≤ .05.

On the Coren, Porac, and Duncan Laterality Questionnaire, high-hostiles (M = 9.93, SD = 2.81) did not reliably differ from the low-hostiles (M = 9.20, SD = 2.46), t(28) = 0.76, p ≥ .05.

On the BDI, high-hostiles (M = 12.13, SD = 8.43) scored significantly higher than low-hostiles (M = 4.00, SD = 4.88), t(22.5) = 3.23, p ≤ .05. On the STAI (State Form), high-hostiles (M = 43.20, SD = 11.33) scored significantly higher relative to their low-hostile counterparts (M = 31.13, SD = 5.69), t(20.6) = 3.68, p ≤ .05. Likewise, on the STAI (Trait Form), high-hostiles (M = 47.87, SD = 8.55) scored significantly higher than low-hostiles (M = 32.80, SD = 6.13), t(28) = 5.55 p ≤ .05.

Cardiovascular

Group means and standard deviations of cardiovascular measures (SBP, DBP, and HR) are displayed in Table 2. Multivariate analysis of variance (MANOVA) with the fixed factor of Group and repeated measures of Trial and Participants was performed on the three cardiovascular variables – SBP, DBP, and HR. MANOVA results are represented in Table 3. The main effect of Trial was statistically significant, F(1,9) = 4.12, p ≤ .05. No other main effect or interaction effects were reliable.

Independent analyses of variance (ANOVAs) were performed on the three cardiovascular variables – SBP, DBP, and HR. In order to conduct a more refined analysis of the data, a series of independent ANOVAs were conducted to compare the cardiovascular reactivity from Baseline to Post-Dichotic1; from Post-Dichotic1 to Post-Cognitive Affective Stressor; and from Post-Cognitive Affective Stressor to Post-Dichotic2. All pairwise comparisons were made using Tukey's Studentized Range Test (see Winer, 1971).

Systolic Blood Pressure

For SBP, three two-factor, mixed design analyses of variance (ANOVAs) were performed (see Table 4). Data are represented in Figure 1.

Baseline to Post-Dichotic1 SBP

A fixed factor of Group (low- and high-hostile) and repeated measure of Trial (pre- and post-dichotic1) ANOVA was performed. The main effect of Trial was statistically significant F(1,28) = 4.42, p ≤ .05. That is, SBP was reliably lower following the dichotic verbal recognition task.

Post-Dichotic1 to Post-Cognitive Affective Stressor SBP

A fixed factor of Group (low- and high-hostile) and repeated measure of Trial (pre- and post-stress) ANOVA was performed. There were no significant main or interaction effects. However, more specific analyses revealed that there was a main effect of Trial from the SBP measure immediately prior to the cognitive affective stressor to the

trial immediately following the cognitive stressor, F(1,28) = 4.75, p ≤ .05. That is, SBP was reliably higher for the first measure immediately following the AAVLT.

Post-Cognitive Affective Stressor to Post-Dichotic2 SBP

A fixed factor of Group (low- and high-hostile) and repeated measure of Trial (pre- and post-dichotic2) ANOVA was performed. The main effect of Trial was statistically significant, F(1,28) = 10.01, p ≤ .05. That is, SBP was reliably lower following the second dichotic verbal recognition task. These results replicate the results of the Baseline to Post-Dichotic1 SBP analysis.

Diastolic Blood Pressure

To analyze DBP, three two-factor, mixed design analyses of variance (ANOVAs) were performed (see Table 4).

Baseline to Post-Dichotic1 DBP

A fixed factor of Group (low- and high-hostile) and repeated measure of Trial (pre- and post-dichotic1) ANOVA was performed. No main or interaction effects were statistically significant.

Post-Dichotic1 to Post-Cognitive Affective Stressor DBP

A fixed factor of Group (low- and high-hostile) and repeated measure of Trial (pre- and post-stress) ANOVA was performed. The main effect of Trial was significant, F(1,28) = 6.50, p ≤ .05. Specifically, DBP was reliably higher following the cognitive affective stressor.

Post-Cognitive Affective Stressor to Post-Dichotic2 DBP

A fixed factor of Group (low- and high-hostile) and repeated measure of Trial (pre- and post-dichotic2) ANOVA was performed. No main or interaction effects were statistically significant.

Heart Rate

Three two-factor, mixed design analyses of variance (ANOVAs) were performed to analyze HR during the three phases (see Table 4).

Baseline to Post-Dichotic1 HR

A fixed factor of Group (low- and high-hostile) and repeated measure of Trial (pre- and post-dichotic1) ANOVA was performed. The main effect of trial was statistically significant, F(1,28) = 8.94, p ≤ .05. That is, HR was reliably higher following the dichotic verbal recognition task.

Post-Dichotic1 to Post-Cognitive Affective Stressor HR

A fixed factor of Group (low- and high-hostile) and repeated measure of Trial (pre- and post-stress) ANOVA was performed. No main or interaction effects were significant.

Post-Cognitive Affective Stressor to Post-Dichotic2 HR

A fixed factor of Group (low- and high-hostile) and repeated measure of Trial (pre- and post-dichotic2) ANOVA was performed. No main or interaction effects were significant.

Dichotic Listening Recognition

Independent analyses of variance (ANOVAs) were performed on three dependent variables obtained during the dichotic listening paradigm – percent correct responses (POC) score, number of correctly identified stimuli in the left ear, and number of correctly identified stimuli in the right ear. POC scores were analyzed first to assess general trends of ear dominance. The number of phonemes identified at each ear were then analyzed to more specifically test for altered cerebral hemispheric activation. Group means and standard deviations of POC scores are displayed on Table 5 and independent ANOVA results are depicted on Table 6. All pairwise comparisons were made using Tukey's Studentized Range Test (Winer, 1971) to control for experimentwise error rate.

Recall that POC scores were obtained by the calculation below:

$$POC = (pR - pL) / (pR + pL)$$

where:

pR = proportion of correctly identified right-ear stimuli
pL = proportion of correctly identified left-ear stimuli

The POC score ranges from +1 (perfect right ear advantage) to –1 (perfect left ear advantage).

For POC scores, a two-factor, mixed design analysis of variance (ANOVA) was performed with the fixed factor of Group (low- and high-hostile) and the repeated measure of Trial (pre- and post-stress). No reliable main effect or interaction effects were found.

To analyze the number of stimuli correctly identified by the left ear (indicative of right cerebral activation), a two-factor, mixed design analysis of variance (ANOVA) was performed with the fixed factor of Group and the repeated measure of Trial (see Table 6). Group means and standard deviations of left-ear phoneme identification are presented in Table 7. No reliable main effects or interaction effects were found.

To analyze the number of stimuli correctly identified by the right ear (indicative of heightened left cerebral activation), a two-factor, mixed design analysis of variance (ANOVA) was performed with the fixed factor of Group and the repeated measure of Trial (see Table 6). Group means and standard deviations of right-ear phoneme identification are presented in Table 8. No reliable main effect or interaction effects were found.

To analyze the number of stimuli correctly identified before the cognitive affective stressor (comparing left ear to right ear), a two-factor, mixed design analysis of variance (ANOVA) was performed with a fixed factor of Group and the repeated measure of Ear. Results are displayed in Table 9. The main effect of Ear was statistically significant, $\underline{F}(1,28) = 10.26$, $\underline{p} \leq .05$. That is, more phonemes were recognized at the right ear than the left ear before the cognitive affective stressor (see Figure 2). Group means and standard deviations of pre-stress phoneme identification are displayed in Table 10.

To analyze the number of stimuli correctly identified after the cognitive affective stressor (comparing left ear to right ear), a two-factor, mixed design analysis of variance (ANOVA) was performed with the fixed factor of Group and the repeated measure of Ear (see Table 9). The main effect of Ear was statistically significant, $\underline{F}(1,28) = 9.82$, $\underline{p} \leq .05$. That is, more phonemes were recognized at the right ear than the left ear after the cognitive affective stressor (see Figure 3). Group means and standard deviations of pre-stress phoneme identification are displayed in Table 11.

Affective Auditory Verbal Learning

To analyze group differences in the number of words learned during the cognitive affective stressor (AAVLT), an analysis of variance (ANOVA) was performed with the fixed factor of Group and the repeated measure of Trial. The main effect of Trial was statistically significant, $\underline{F}(1,4) = 84.73$, $\underline{p} \leq .05$. That is, both low- and high-hostiles learned more words during successive trials of recalling the list of 15 words.

DISCUSSION

The present experiment compared low- and high-hostile participants from an undergraduate population on self-report, cardiovascular, and brain laterality measures. Relative to low-hostiles, high-hostiles scored reliably higher on the CMHS. Likewise, relative to low-hostiles, high-hostiles scored reliably higher on the BDI. Moreover, high-hostiles scored reliably higher than low-hostiles on the STAI (State and Trait forms). These results suggest that high-hostiles may experience heightened depressive and anxious symptomatology.

Cardiovascular activation with heightened blood pressure and/or heart rate may indicate heightened right cerebrum activation (Wittling, 1990; Herridge & Harrison, 1997; Demaree & Harrison, 1997a). Accordingly, data collected during this experiment on three cardiovascular measures (HR, SBP, and DBP) may suggest decreased right-cerebrum activation (increased left-cerebrum activation) following the verbal listening task and greater right-cerebrum activation following the cognitive affective stressor.

As expected, significant main effects of Trial were found on HR, SBP, and DBP. Specifically, participants demonstrated decreased cardiovascular arousal after the verbal dichotic listening tasks. These results were replicated within the experiment, as participants showed decreased cardiovascular arousal following the initial dichotic verbal recognition task as well as the second dichotic verbal recognition task. These results may reflect heightened brain activation, particularly within the left cerebrum, after verbal processing tasks. Additionally, participants showed greater cardiovascular arousal after the cognitive affective stressor. These results may reflect heightened brain activation, particularly within the right cerebrum after the cognitive affective stressor (see Figure 1). These results replicate the cardiovascular reactivity to stress reported by Demaree and Harrison (1997a). These results also replicate the findings of heightened DBP following the negative list learning phase by Snyder, Harrison, and Shenal (1998).

Although the analyses did not reveal reliable Group effect differences or significant Group X Trial interactions, enhanced investigations of cardiovascular data may yield more detailed conclusions supporting cerebral asymmetry differences between low- and high-hostile participants. The nonsignificance may partially result from the law of initial values (LIV) (Wilder, 1931, 1957), with high-hostile participants demonstrating reduced reactivity due to higher initial values (Furedy & Scher, 1989). Using past research that has shown the relationship between right-cerebrum arousal and cardiovascular reactivity, these results may further suggest heightened right cerebral reactivity to the cognitive affective stressor.

This research shows a negative relationship between left-cerebrum activation and cardiovascular reactivity. Furthermore, Demaree and Harrison (1997a) suggest enhanced right cerebral reactivity to stress among high-hostiles. This may provide a partial explanation for the relationship between hostility and cardiovascular disease (Kubany et al., 1994; Treiber et al., 1989; Lee and Cameron, 1987). Accordingly, reactivity to stress with heightened right cerebrum arousal may induce greater cardiovascular lability and cardiovascular disease.

Results of the present study are consistent with effects that would be derived from the a priori hypothesis of right cerebrum activation and, thus, increased cardiovascular reactivity following the cognitive affective stressor. However, this research also adds important and promising findings that cardiovascular reactivity may be decreased following dichotic verbal recognition tasks. While these results were replicated within- and across experiments, a control group may more clearly establish this effect. These results have potential implications for treatment strategies of cardiovascular reactivity and hostility. Furthermore, these results demonstrate that cardiovascular reactivity is dynamic. That is, cardiovascular reactivity may increase following a cognitive affective stressor and it may decrease following the dichotic verbal recognition tasks. Additional research utilizing control groups may be useful to experimentally replicate the results of decreased cardiovascular reactivity following dichotic verbal recognition tasks.

The dichotic listening paradigm was used in this experiment to assess cerebral asymmetry as a function of hostility and cognitive affective stress. A significant main effect of Ear was found pre- and post-stress for both low- and high-hostiles (see Figures 2 and 3). That is, participants recognized more right ear phonemes (left cerebrum) than left ear phonemes (right cerebrum). This is not surprising given robust research findings that the left hemisphere (right ear) is typically superior at processing auditorally-presented verbal stimuli (Snyder, Harrison, & Gorman, 1994; Mondor & Bryden, 1992; Bryden, Free, Gagne, & Groff, 1992; Ley & Bryden, 1982).

Data collected from the dichotic listening procedure did not support the a priori hypothesis of greater right-cerebrum reactivity to the cognitive affective stressor among high-hostiles. The analysis of POC scores revealed that neither low- nor high-hostiles showed a heightened left ear advantage after the cognitive stressor. The main effects of Group and of Trial, as well as the Group X Trial interaction, may not have reached significance for two reasons. First, nonsignificant differences between groups following the cognitive affective stressor may have reduced changes in POC scores. Second, this experiment may have used too few subjects, thereby reducing power. However, similar experiments have found significant results with comparable group sizes (Demaree, & Harrison, 1997a; Herridge, Harrison, & Shenal, 1998).

No other significant effects were found when analyzing the number of correctly identified phonemes presented to the left or the right ears. However, both groups showed small, nonsignificant increases in the number of identified right-ear phonemes after the cognitive affective stressor. These results replicate the subtle findings of increased right ear accuracy following stress reported by Demaree and Harrison (1997a). This may suggest similar left cerebrum activation among the groups. This similarity was further supported by the results of the cardiovascular data which indicate that both groups evidenced decreased cardiovascular reactivity (left-cerebrum activation) following the dichotic listening task. Accordingly, nonsignificant left cerebral reactivity to stress may be cautiously inferred. Moreover, the similarity between groups on this right-ear measure potentially reduced the statistical significance of the Group X Trial POC effect. Evidence of heightened right cerebral arousal to the cognitive affective stressor was not obtained by the dichotic listening data.

These findings should not be generalized outside of this research population. Participants showed no signs of major illness, head trauma, or neurological impairment. One must not conclude that left-cerebrum activation will always reduce cardiovascular reactivity and that right-cerebrum activation will always increase cardiovascular reactivity. These findings, however, suggest that an individual's cardiovascular reactivity is dynamic and may change following a cognitive affective stressor and dichotic verbal recognition tasks.

As Demaree and Harrison (1997a) report, the use of paradigms more sensitive to brain lateralization may enhance the probability of obtaining more statistically significant data. Dichotic listening may only conservatively measure hemispheric differences due to the large proportion of ipsilateral bifurcations associated with audition. As the above researchers state, quantitative electroencephalography (QEEG) may be a better indicator for similar experiments.

More furtive data supporting Group X Trial interactions may help identify who is most likely to exhibit cardiovascular reactivity to cognitive stressors and who may benefit most from tasks that decrease cardiovascular reactivity. Accordingly, therapy that promotes increased left-cerebrum activation and decreased right-cerebrum activation may lower the cardiovascular risks of individuals with hostility problems.

Experiment 2

Rationale

The following experiment utilized a physical task (cold pressor), as opposed to the previous cognitive list learning task, to demonstrate a dynamic cerebral lateralization effect for low- and high- hostile participants. This experiment utilized a tachistoscopic method to test reactivity to physical stress through the visual sensory modality. As Demaree and Harrison (1997a) indicated, the use of methods sensitive to brain lateralization may provide additional furtive data about the dynamic cerebral laterality effect.

Tachistoscopic methods have been proven to be useful in the provision of evidence for hemispheric lateralization (McKeever & Huling 1971; Harrison &

Gorelczenko, 1990; Harrison, Gorelczenko, & Cook, 1990; Herridge, Harrison, & Demaree, 1997; Herridge, Harrison, & Shenal, 1998). It is expected that the tachistoscopic method will provide a logical extension for the continued study of the effects of stress and right cerebral activation. High-hostile individuals may activate the right cerebrum following a painful cold pressor stressor task or challenging cognitive stressful task, thus increasing left visual field recognition, while low-hostile individuals may demonstrate relative activation of the left cerebrum following painful stress or cognitive stress (i.e., increasing right visual field recognition).

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 SBP, DBP, and HR. Second, lateralization of brain activity was measured by visual field advantage observed using dual-phoneme visual tachistoscopic perception methods.

HYPOTHESES

Hypothesis 1: High-hostile participants will show greater cardiovascular (Blood Pressure, Heart Rate) reactivity to the physical stress through the painful cold pressor task than low-hostile participants.

Hypothesis 2: Low- and High- hostile participants will differ on the tachistoscopic task following physical stress. This will be evidenced through relative increased left visual field accuracy scores (right cerebral arousal) for high-hostiles and right visual field accuracy scores (left cerebral arousal) for low-hostiles.

METHOD

Participants

Group testing at Virginia Tech, a large state university, yielded the experimental groups composed of high-hostile and low-hostile male participants. Volunteer participants were recruited from the Virginia Tech undergraduate pool. A sign-up folder, which explained the experiment and scheduled screening times, was made available to undergraduates. The participants were informed that this experiment was a two-phase experiment and that they would receive one credit point for each session that they attended. Further, they were informed that participation in the group screening did not guarantee inclusion in the experimental phase. It was expected that 100 participants would be screened to complete the final participant pool of 30 needed for the experiment.

Inventories and Questionnaires

The screening involved groups of 10 - 30 participants. An Informed Consent Form 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, 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 are self report, and are 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 more on this questionnaire.

Participants must have self-reported no history of visual difficulties, major illness, or major head injury on a 18 question Medical History Questionnaire. Participants with vision corrected to normal were included in the experiment. 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 experiment.

The Cook-Medley Hostility Scale (CMHS) was then administered to the group (Cook & Medley, 1954). This scale consisted 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." 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." 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. Thirty participants (fifteen each) were classified as high-hostile or low-hostile based on the scores from this questionnaire (CMHS).

Participants also completed the validated self-report Beck Depression Inventory (Beck, 1972). This measure consisted of 21 questions which tap specific symptoms, attitudes, and behavioral manifestations of depression. Scores on this inventory, may range from 0 to 63, with 0 – 9 considered "nondepressed," 10 – 15 indicating "mild depression," 16 – 19 indicating "mild-moderate depression," 20 – 29 indicating "moderate-severe depression," and 30 – 63 indicating "severe depression."

Participants also completed the validated self-report State-Trait Anxiety Inventory (Speilberger, 1968). This is a 40 item questionnaire that inventories state and trait anxiety. Twenty questions are provided to evaluate the way a person feels "right now" (state anxiety). In addition, 20 questions are provided to assess the way a person feels "in general" (trait anxiety). Scores on the STAI may range from 20 to 80 for the State Inventory and from 20 to 80 for the Trait Inventory.

The participants chosen for the experimental phase 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. Participants again completed all self-report questionnaires.

Apparatus

The participants were seated in a sound-attenuated room. The automated programming equipment and experimenter were located in an adjacent room. The participants were monitored through a one-way observation window and were prompted via an intercom.

Cardiovascular

Blood pressure and heart rate were assessed using the Industrial and Biomedical Sensors Corporation SD-700 A Blood Pressure / Pulse Monitor which was used during the first experiment.

Tachistoscope

A constant illumination tachistoscope (Lafayette Instruments 42011) presented the stimuli onto a screen 2.38 m. in front of the participant. The center of the screen was marked with a black dot positioned 1.47 m. above the floor and at the participant's eye level. Tachistoscopic-trial onset was signaled by a 2000 Hz, 55 dB (A-scale) tone produced through the speaker located behind the participant. Each trial consisted of a .05 s. presentation of the stimuli.

Visual Stimuli

Stimuli were two letter visual phonemes developed to replicate the phonemes used during the first experiment but within the visual modality. The stimuli were dually and concurrently presented as in the procedure of the first experiment. Scores for each participant were the number of correct phoneme recognitions within each visual field.

During each stimulus presentation one phoneme was presented to the left visual field and one phoneme to the right visual field. Horizontal distances of the nearest letter from the fixation point corresponded to 3.0 degrees of visual angle for both the left and right field. Scores for each participant were the number of correct phoneme recognitions within each visual field. A total of 30 recognitions in each field were possible. Participants were not informed of the accuracy of their responses.

Cold Pressor

The cold pressor stress procedure utilized a small ice cooler (Gott Corporation, Model 1916/2) to maintain the water temperature at about 0 degrees Celsius. Water temperature was measured using a standard mercury thermometer (Fisher Scientific, Model 14-985E).

Procedure

This experiment used a painful cold pressor stressor.

Cardiovascular Phase (Baseline):

Participants were fitted for blood pressure (BP) and heart rate (HR) readings. The blood pressure cuff was strapped to each participants left upper-arm. The experimenter then left the room and gave the following instructions: "Please sit still in the chair and take about one minute to become accustomed to your surroundings." Heart rate (HR), systolic blood pressure (SBP), and diastolic blood pressure (DBP) data were collected twice at the end of the baseline period. A third reading was taken if the first two readings differed by 6 beats per minute (HR) or 20 mmHg for either SBP or DBP.

Tachistoscopic Phase:

Tachistoscopic-trial onset was signaled by a 2000 Hz, 55 dB (A-scale) tone presented through a speaker located behind the participant. Each trial consisted of a .05 second presentation of the stimuli.

Participants were told:

> You are about to see thirty trials of word fragments presented visually. You will see a word fragment to the left of the fixation point and a word fragment to the right of the fixation point at the same time. Your job is to focus on the fixation point very carefully and point to the word fragment on the chart that you see most clearly. Please remember to keep your eyes focused on the fixation point.

All responses were recorded.

Cardiovascular Phase (Post-Tscope1):

Participant's HR, SBP, and DBP were assessed and recorded following the procedure in the initial cardiovascular phase.

Cold Pressor Phase:

Participants were given the following instructions:

> When you are instructed, please place your left hand in the water to a point about one inch above your wrist. You will be asked to keep your hand in the water for 45 seconds. Although this may be difficult, please try your hardest to keep your hand in the water until instructed to take it out. Do you have any questions? O.K., begin.

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

Cardiovascular Phase (Post-Cold Pressor Stressor):

Participant's HR, SBP, and DBP were assessed and recorded following the procedure in the initial cardiovascular phase.

Tachistoscopic Phase:

The visual recognition task was then performed according to the procedures outlined in the initial tachistoscopic phase.

Cardiovascular Phase (Post-Tscope2):

Participant's HR, SBP, and DBP were assessed and recorded following the procedure in the initial cardiovascular phase.

Participants were thoroughly debriefed following completion of the experiment.

Analyses

For each participant, the Percentage of Correct responses (POC) index was calculated for visual recognition accuracy using the following formula:

$$POC = (pR - pL) / (pR + pL)$$

pR = proportion of correctly identified right visual field phonemes

pL = proportion of correctly identified left visual field phonemes

The POC scores range from +1 (perfect right visual field advantage) to -1 (perfect left visual field advantage). Independent ANOVAs were conducted using visual recognition variables – POC, and number of correctly identified stimuli presented to both the left (NOL) and right visual field (NOR). Mixed design ANOVAs with the fixed factor of Group(2) (high-hostile and low-hostile) and the repeated measure of Trial(2) (pre-stress and post-stress) were used.

Multivariate analysis of variance (MANOVA) was performed on the cardiovascular variables (SBP, DBP, HR) to address reactivity to the cold pressor stressor: Group(2) X (Trial X Participant).

A priori criterion for reliability was p ≤ .05.

RESULTS

Descriptive Measures

T-tests were conducted on scores obtained on the Cook-Medley Hostility Scale, Coren, Porac and Duncan's Laterality Questionnaire, Beck Depression Inventory, and State – Trait Anxiety Inventory in order to compare groups (low- and high- hostiles) on the descriptive measures. Table 12 provides a summary of the group means and the standard deviations for each measure.

High-hostiles scored significantly higher on the CMHS (M = 31.67, SD = 3.09) than did low-hostiles (M = 12.53, SD = 6.08), t(28) = 10.87, p ≤ .05.

On the Coren, Porec, and Duncan Laterality Questionnaire, high-hostiles (M = 9.40, SD = 2.85) did not statistically differ reliably from the low-hostiles (M = 10.73, SD = 2.12), t(28) = 1.4542, p ≥ .05.

On the BDI, high-hostiles (M = 10.60, SD = 6.70) scored significantly higher than low-hostiles (M = 5.33, SD = 6.09), t(28) = 2.25, p ≤ .05. On the STAI (State Form), high-hostiles (M = 43.20, SD = 11.33) scored higher relative to their low-hostile counterparts (M = 31.13, SD = 5.69). This comparison approached statistical significance, t(28) = 1.92, p = .0653. However, on the STAI (Trait Form), high-hostiles (M = 41.20, SD = 7.20) scored significantly higher than low-hostiles (M = 34.20, SD = 10.04), t(28) = 2.19, p ≤ .05.

Cardiovascular

Group means and standard deviations for the cardiovascular measures (SBP, DBP, and HR) are displayed in Table 13. Multivariate analysis of variance (MANOVA) with the fixed factor of Group and the repeated measures of Trial and Participants was performed on the three cardiovascular variables – SBP, DBP, and HR. MANOVA results are represented in Table 14. The main effect of Group was statistically significant, F(1,3) = 3.57, p ≤ .05. Additionally, the main effect of Trial was statistically significant, F (1,9) = 2.00, p ≤ .05. No interaction effects were reliable.

In order to conduct a more refined analysis of the data, a series of independent ANOVAs were performed to compare the cardiovascular measures from Baseline to Post-Tscope1; from Post-Tscope1 to Post-Cold Pressor Stressor; and from Post-Cold Pressor Stressor to Post-Tscope2. All pairwise comparisons were made using Tukey's Studentized Range Test (see Winer, 1971).

Systolic Blood Pressure

For SBP, three two-factor, mixed design analyses of variances (ANOVAs) were performed (see Table 15). The results are represented in Figure 4.

Baseline to Post-Tscope1 SBP

A fixed factor of Group (low- and high-hostile) and repeated measure of Trial (pre- and post-Tscope1) ANOVA was performed. The main effect of Group was statistically significant, F(1,28) = 5.54, p ≤ .05. That is, SBP was reliably higher for high-hostiles compared to their low-hostile counterparts. Additionally, the main effect of

Trial was statistically significant, F(1,28) = 8.23, $p \leq .05$. That is, SBP was reliably lower following the tachistoscopic linguistic recognition task.

Post-Tscope1 to Post-Cold Pressor Stressor SBP

A fixed factor of Group (low- and high-hostile) and repeated measure of Trial (pre- and post-stress) ANOVA was performed. The main effect of Group was statistically significant, F(1,28) = 6.99, $p \leq .05$. Accordingly, SBP was reliably higher for high-hostiles compared to their low-hostile counterparts. Additionally, the main effect of Trial was statistically significant, F(1,28) = 7.24, $p \leq .05$. That is, SBP was reliably higher following the painful cold pressor stressor.

Post-Cold Pressor Stressor to Post-Tscope2 SBP

A fixed factor of Group (low- and high-hostile) and repeated measure of Trial (pre- and post-Tscope2) ANOVA was performed. The main effect of Group was statistically significant, F(1,28) = 5.86, $p \leq .05$. Accordingly, SBP was reliably higher for high-hostiles compared to their low-hostile counterparts. Additionally, the main effect of Trial was statistically significant, F(1,28) = 8.04, $p \leq .05$. That is, SBP was reliably lower following the second tachistoscopic linguistic recognition task. These results replicate the results of the Baseline to post-Tscope1 analysis.

Diastolic Blood Pressure

To analyze DBP, three two-factor, mixed design analyses of variances (ANOVAs) were performed (see Table 15).

Baseline to Post-Tscope1 DBP

A fixed factor of Group (low- and high-hostile) and repeated measure of Trial (pre- and post-Tscope1) ANOVA was performed. No main or interaction effects were statistically significant.

Post-Tscope1 to Post-Cold Pressor Stressor DBP

A fixed factor of Group (low- and high-hostile) and repeated measure of Trial (pre- and post-stress) ANOVA was performed. The Group X Trial interaction was significant, $\underline{F}(1,28) = 4.26$, $\underline{p} \leq .05$. That is, high-hositles' DBP increased following the painful cold pressor stressor and low-hostiles' DBP decreased following the painful cold pressor stressor.

Post-Cold Pressor Stressor to Post-Tscope2 DBP

A fixed factor of Group (low- and high-hostile) and repeated measure of Trial (pre- and post-Tscope2) ANOVA was performed. The Group X Trial interaction was significant, $\underline{F}(1,28) = 4.42$, $\underline{p} \leq .05$. That is high-hostiles' DBP increased following the tachistoscopic linguistic recognition task and low-hostiles' DBP decreased following the tachistoscopic linguistic recognition task.

Heart Rate

Three two-factor, mixed design analyses of variance (ANOVAs) were performed to analyze HR during the three phases (see Table 15).

Baseline to Post-Tscope1 HR

A fixed factor of Group (low- and high-hostile) and repeated measure of Trial (pre- and post-Tscope1) ANOVA was performed. There were no significant main or interaction effects.

Post-Tscope1 to Post-Cold Pressor Stressor HR

A fixed factor of Group (low- and high-hostile) and repeated measure of Trial (pre- and post-stress) ANOVA was performed. The main effect of Trial was statistically significant, $\underline{F}(1,28) = 8.95$, $\underline{p} \leq .05$. That is, HR was reliably lower following the painful cold pressor stressor.

Post-Cold Pressor Stressor to Post-Tscope2 HR

A fixed factor of Group (low- and high-hostile) and repeated measure of Trial (pre- and post-Tscope2) ANOVA was performed. The main effect of Trial was significant, $\underline{F}(1,28) = 3.98$, $\underline{p} \leq .05$. Accordingly, HR reliably increased following the second tachistoscopic linguistic recognition task.

Tachistoscopic Recognition

Independent analyses of variance (ANOVAs) were performed on three dependent variables obtained during the tachistoscopic paradigm – percent correct responses (POC) score, number of correctly identified stimuli in the left visual field, and number of correctly identified stimuli in the right visual field. POC scores were analyzed first to assess general trends of visual field dominance. The number of phonemes identified at each visual field were then analyzed to more specifically test for altered cerebral hemispheric activation. Group means and standard deviations of POC scores are displayed in Table 16 and independent ANOVA results are depicted in Table 17. All pairwise comparisons were made using Tukey's Studentized Range Test (Winer, 1971) to control for experimentwise error rate.

Recall that POC scores were obtained by the calculation below:

$$POC = (pR - pL) / (pR + pL)$$

where:

pR = proportion of correctly identified right-visual field stimuli

pL = proportion of correctly identified left-visual field stimuli

The POC scores range from +1 (perfect right visual field advantage) to –1 (perfect left visual field advantage).

For POC scores, a two-factor, mixed design analysis of variance (ANOVA) was performed with a fixed factor of Group (low- and high-hostile) and the repeated measure of Trial (pre- and post-stress). No reliable main effect or interaction effects were found.

To analyze the number of stimuli correctly identified in the left visual field (indicative of right cerebral activation), a two-factor, mixed design analysis of variance (ANOVA) was performed with a fixed factor of Group and the repeated measure of Trial (see Table 17). Group means and standard deviations of left-visual field phoneme identification are presented in Table 18. No reliable main effect or interaction effects were found.

To analyze the number of stimuli correctly identified by the right visual field (indicative of heightened left cerebral activation), a two-factor, mixed design analysis of variance (ANOVA) was performed with a fixed factor of Group and the repeated measure of Trial (see Table 17). Group means and standard deviations of right-visual field phoneme identification are presented in Table 19. No reliable main effect or interaction effects were found.

To analyze the number of stimuli correctly identified before the painful cold pressor stressor (comparing left VF to right VF), a two-factor, mixed design analysis of variance (ANOVA) was performed with a fixed factor of Group and the repeated measure of VF. Results are displayed in Table 20. The main effect of VF was statistically significant, $\underline{F}(1,28) = 5.35$, $\underline{p} \leq .05$. That is, more phonemes were recognized at the right VF than the left VF before the cold pressor stressor (see Figure 5). Group means and standard deviations of pre-stress phoneme identification are displayed in Table 21.

To analyze the number of stimuli correctly identified after the painful cold pressor stressor (comparing left VF to right VF), a two-factor, mixed design analysis of variance (ANOVA) was performed with a fixed factor of Group and the repeated measure of VF (see Table 20). The main effect of VF was statistically significant, $\underline{F}(1,28) = 4.66$, $\underline{p} \leq .05$. That is, more phonemes were recognized at the right VF than the left VF after the cold pressor stressor (see Figure 6). Group means and standard deviations of pre-stress phoneme identification are displayed in Table 22.

Discussion

As with the previous experiment, Experiment 2 compared low- and high-hostile participants from an undergraduate population on self-report, cardiovascular, and brain laterality measures. Relative to low-hostiles, high-hostiles scored significantly higher on the CMHS. Likewise, relative to low-hostiles, high-hostiles scored significantly higher on the BDI. Moreover, high-hostiles scored significantly higher than low-hostiles on the STAI. These results suggest that high-hostiles may experience heightened depressive and anxious symptomatology.

As discussed earlier, cardiovascular arousal may indicate heightened right cerebral activation (Wittling, 1990; Herridge & Harrison, 1997, Demaree & Harrison, 1997a). Therefore, the results from this experiment on three cardiovascular measures (HR, SBP, and DBP) may indicate decreased right-cerebrum activation (increased left-cerebrum activation) following the tachistoscopic linguistic recognition task and greater right-cerebrum activation following the painful cold pressor stressor (see Figure 4).

As with the first experiment, significant main effects of Trial were found using cardiovascular measures. Specifically, participants demonstrated decreased cardiovascular arousal after the linguistic recognition T-scope tasks. These results were replicated within the experiment, as participants showed decreased cardiovascular arousal following the initial T-scope presentation as well as after the second T-scope presentation. These results also replicate the findings from Experiment 1 in which participants showed decreased cardiovascular arousal following the dichotic verbal recognition tasks. These results may reflect heightened brain activation, particularly within the left cerebrum, after linguistic tasks. Additionally, participants showed greater cardiovascular arousal after the cold pressor stressor. These results replicate Experiment 1, which showed greater cardiovascular arousal following the cognitive affective stressor. These results also replicate the cardiovascular reactivity to stress reported by Demaree and Harrison (1997a). The results may reflect heightened brain activation, particularly within the right cerebrum, after a painful cold pressor stressor.

As a point of interest, one participant's results were excluded from the final data pool due to his brief loss of consciousness following the cold pressor task. This low-hostile participant's blood pressure and heart rate decreased dramatically following the stressor and he slumped to the right of his chair. When he awoke, he was cold and clammy and appeared fatigued. This parasympathetic response is consistent with increased left-cerebrum activation, and may reflect the proposed pattern of low-hostiles having increased left-cerebrum activation (decreased right-cerebrum activation) as compared to high-hostiles with increased right-cerebrum activation.

These data also revealed a consistent main effect of Group for SBP throughout the experiment. Specifically, high-hostiles evidenced significantly higher SBP than low-hostiles. The high-hostiles' higher SBP measures may have produced a ceiling effect, decreasing the probability of finding Group X Trial interaction effects. It is interesting to note that results from Experiment 1 did not reveal significant Group effects. One possible explanation for the discrepant results may be seasonal effects. Experiment 1 was performed during the months of October, November, and early December, while Experiment 2 was performed during the months of January and February. Also, the 15 high-hostile participants were recruited faster during Experiment 2 than Experiment 1. Further research may be warranted to investigate potential seasonal effects of hostility and cardiovascular reactivity.

These analyses also revealed significant Group X Trial interactions for DBP. Specifically, low-hostiles' DBP increased following the cold pressor stressor while high-hostiles' DBP decreased following the cold pressor stressor. Additionally, low-hostiles' DBP decreased following the second tachistoscopic linguistic recognition task while high-hostiles' DBP increased following the second tachistoscopic recognition task. These analyses revealed no other significant Group X Trial interactions. The nonsignificant SBP and HR interactions may have partially resulted from the previously discussed law of initial values (LIV) (Wilder, 1931, 1957), with high-hostiles demonstrating reduced reactivity due to higher initial SBP values (Furedy & Scher, 1989). Considering high-hostiles' higher initial values and past research, these data may further suggest heightened right cerebral reactivity to the painful cold pressor stressor.

As with Experiment 1, these data show a negative relationship between left-cerebrum activation and cardiovascular reactivity. These results also demonstrate a positive relationship between the cold pressor stressor and cardiovascular reactivity. Combined with Demaree and Harrison's (1997a) evidence of enhanced right cerebral reactivity to stress among high-hostiles, these data may infer more evidence for the relationship between hostility and cardiovascular disease (Kubany et al., 1994; Treiber et al., 1989; Lee & Cameron, 1987). Accordingly, reactivity to the cold pressor stressor with heightened right cerebrum activation may induce greater cardiovascular lability and cardiovascular disease.

Results of the present study are consistent with effects that would be derived from the a priori hypothesis of right cerebrum activation and, thus, increased cardiovascular reactivity following the painful cold pressor stressor. However, as with Experiment 1, this research also adds important and promising findings that cardiovascular reactivity may be decreased following linguistic tasks. While these results were replicated within- and across experiments, a control group may more clearly establish this effect. These results have potential implications for treatment strategies of cardiovascular reactivity and hostility. Furthermore, these results demonstrate that cardiovascular reactivity is dynamic. That is, cardiovascular reactivity may increase following a painful cold pressor stressor and may decrease following tachistoscopic linguistic recognition tasks. Additional research utilizing control groups may be useful to experimentally replicate the results of decreased cardiovascular reactivity following tachistoscopic linguistic recognition tasks.

The T-scope paradigm was used in this experiment to assess cerebral asymmetry as a function of hostility and painful cold pressor stress. A significant main effect of visual field (VF) was found pre- and post-stress for both low- and high-hostiles (see Figures 5 and 6). That is, participants recognized more right VF phonemes (left cerebrum) than left VF phonemes (right cerebrum). These results replicate those of Experiment 1, which showed more right-ear phoneme recognitions that left-ear phoneme recognitions. These results are not surprising given the previously mentioned research findings that the left hemisphere (right VF) is typically superior at processing verbal stimuli (Snyder, Harrison, & Gorman, 1994; Mondor & Bryden, 1992; Bryden Free, Gagne, & Groff, 1992; Ley & Bryden, 1982).

Data collected from the T-scope procedure did not support the a priori hypothesis of greater right-cerebrum reactivity to the cold pressor stressor among high-hostiles. The analysis of POC scores revealed that neither low- nor high-hostiles showed a heightened left VF advantage after the painful stressor. The main effects of Group and Trial, as well as the Group X Trial interaction, may not have reached significance for two reasons. First, nonsignificant differences between groups following the painful cold pressor stressor may have reduced changes in POC scores. Second, this research may have used too few subjects, thereby reducing power. However, similar experiments have found significant results with comparable group sizes (Demaree & Harrison, 1997a; Herridge, Harrison, & Shenal, 1998).

No other significant effects were found when analyzing the number of correctly identified phonemes presented to the left or right visual fields. However, consistent with the a priori hypotheses, high-hostiles showed small, nonsignificant increased left VF recognitions pre- and post-stress as compared to their low-hostile counterparts. Similarly, high-hostiles showed small, nonsignificant decreased right VF recognitions pre- and post-stress as compared to low-hostiles (see Figures 5 and 6). Additionally, both groups showed small, nonsignificant increases in the number of identified right VF phonemes after the cold pressor stressor. These results replicate the subtle increased right ear accuracy following the cognitive affective stress reported in Experiment 1 and in the study by Demaree and Harrison (1997a). Again, this may further suggest similar left cerebrum activation between groups. This similarity was further supported by the results of the cardiovascular data which suggests that both groups evidenced decreased cardiovascular reactivity (left-cerebrum activation) following the tachistoscopic linguistic recognition task. Again, the similarity between groups on this right VF measure may have reduced the statistical significance of the Group X Trial POC effect. As with Experiment 1, reliable evidence of heightened right cerebral activation to stress was not obtained by the T-scope data.

As discussed with Experiment 1, these findings should not be generalized outside of this research population. Participants showed no signs of major illness, head trauma, or neurological impairment. One must not conclude that left-cerebrum activation will always reduce cardiovascular reactivity and that right-cerebrum activation will always increase cardiovascular reactivity. These findings, however, suggest that an individual’s cardiovascular reactivity is dynamic and may change following a painful cold pressor stressor and tachistoscopic linguistic recognition tasks.

As Demaree and Harrison (1997a) report, using paradigms more sensitive to brain lateralization may enhance the probability of obtaining more statistically significant data. Quantitative electroencephalography (QEEG), for example, may be a better indicator for similar experiments.

More furtive data supporting Group X Trial interactions may help identify who is most likely to exhibit cardiovascular reactivity to painful stress and who may benefit most from tasks that decrease cardiovascular reactivity. Accordingly, therapy that promotes increased left-cerebrum activation and decreased right-cerebrum activation may lower the cardiovascular risks of individuals with hostility problems.

Conclusions and Integration of Experiments 1 and 2

These results have demonstrated through both within-experiment replication and across-experiment replication that cardiovascular reactivity is dynamic. These data show that both dichotic verbal recognition tasks and visual linguistic recognition tasks may lower cardiovascular reactivity and may increase left-cerebrum activation. Additionally, right-cerebrum activation and increased cardiovascular reactivity may result from exposure to both cognitive affective stressors and painful physical stressors.

These results demonstrate that the cognitive negative affective learning task (Snyder & Harrison, 1997) is an analogous stressor to the more common physical cold pressor task. Accordingly, the negative list learning task offers a non-invasive, cognitive task that may be utilized in future experiments to induce stress. Again, these results replicate those of Snyder, Harrison, & Shenal (1998).

Previous research has suggested that positive affective learning tasks (left-cerebrum activation) may lower cardiovascular reactivity (Snyder, Harrison, & Shenal, 1998). Data from Experiment 1 and Experiment 2 also suggest that cardiovascular reactivity may be decreased following auditory and visual linguistic recognition. These data have significant implications for the prevention and treatment of cardiovascular reactivity. These cognitive tasks may provide an effective, noninvasive task that reduces cardiovascular reactivity.

Future research should aim to identify individuals who may benefit most by these cardiovascular reactivity reducing tasks. Paradigms such as the QEEG may afford additional data on brain lateralization and hemispheric activation following stress. Additional research should also investigate potential seasonal effects on hostility, as Experiment 2 yielded significant group effects for cardiovascular reactivity while Experiment 1 did not. Moreover, further research should aim to develop additional non-invasive tasks to reduce cardiovascular reactivity and to further develop neuropsychological theories of the brain-behavior effects of cognitive stressors and physical stressors and hostility on cardiovascular reactivity.

REFERENCES

American Heart Association (1988). *Heart Facts.* Dallas, Tex.: American Heart Association.

Beck, A. T. (1972). Depression: Causes and treatment. Philadelphia: University of Philadelphia Press, Chapter 12.

Borod, J. C., Koff, E., Lorch, M. P., & Nicholas, M. (1986). The expression and perception of facial emotion in brain damaged patients. Neuropsychologia, 24, 169-180.

Bryden, M. P., Free, T., Gagne, S., & Groff, P. (1991). Handedness effects in the detection of dichotically-presented words and emotions. Cortex, 229-235.

Butter, C. M., Snyder, D. R. & McDonald, J. A. (1970). Effects of orbital frontal lesions on aversive and aggressive behaviors in rhesus monkeys. Journal of Comparative Physiology and Psychology, 72, 132-144.

Contrada, R. J. & Jussim, L. (1992). What does the Cook-Medley hostility scale measure? In search of an adequate measurement model. Journal of Applied Social Psychology, 22, 615-627.

Cook, W. W. & Medley, D. M. (1954). Proposed hostility and pharisaic-virtue scales for the MMPI. Journal of Applied Psychology, 238, 414-418.

Coren, S. P., Porac, C. & Duncan, P. (1979). A behaviorally validated self-report inventory to assess 4 types of lateral preferences. Journal of Clinical Neuropsychology, 1, 55-64.

Cummings, J. L., & Mendez, M. P. (1984). Secondary mania with focal cerebrovascular lesions. American Journal of Psychiatry, 141, 1084-1087.

Davidson, R. J. (1992). Emotion and affective style: Hemispheric substrates. Psychological Science, 3, 29-43.

Davidson, R. J. (1993). The neuropsychology of emotion and affective style. In M. Lewis and J. M. Haviland (Eds.), Handbook of Emotions (pp.143-154). New York: The Guilford Press.

Davidson, R. J., Ekman, P., Saron, C. D., Senulis, J. A., & Friesen, W. V. (1990). Approach/withdrawal and cerebral asymmetry: Emotional expression and brain physiology. I. Journal of Personality and Social Psychology, 58, 330-341.

Demaree, H. A., & Harrison, D. W. (1996). Case study: Topographical brain mapping in hostility following mild closed head injury. International Journal of Neuroscience, 87, 97-101.

Demaree, & Harrison, (1996). Topographical brain mapping: Hostility following mild closed head injury. International Journal of Neuroscience, 87, 97-101.

Demaree, H. A., & Harrison, D. W. (1997a). Behavioral, Physiological, and Neuropsychological Correlates of Hostility. Neuropsychologia. 35(10), 1405-1411.

Demaree, H.A., & Harrison, D. W. (1997b). Quantitative electroencephalographic analyses of low- and high-hostiles to stress. *Manuscript submitted.*

Demaree, H. A, & Harrison, D. W. (1997c). A neuropsychological model relating self-awareness to hostility. Neuropsychology Review, 7(4), 171-185.

Dembroski, T. M., MacDougall, J. M., Herd, J. A. & Shields, J. L. (1979). Effect of level of challenge on pressor and heart rate responses in Type A and B participants. Journal of Applied Social Psychology, 9, 209-228.

Dembroski, T. M., MacDougall, J. M., Herd, J.A., & Shields, J. L. (1983). Perspectives on coronary-prone behavior. In D. S. Krantz, A. Baum, & J. E. Singer (Eds.), Handbook of psychology and medicine. Vol 1: Cardiovascular disease (pp.57-83). Hillsdale, NJ: Erlbaum.

Etcoff, N. L. (1989) Asymmetries in recognition of emotion. In F. Boller & J. Grafman (Eds.), Handbook of neuropsychology (Vol. 3, pp. 363-382). New York: Elsevier.

Furedy, J. J., & Scher, H. (1989). The law of initial values: Differentiated testing as an empirical generalization versus enshrinement as a methodological rule. Psychophysiology, 26, 120-121.

Glass, D. C., & Contrada, R. J. (1982). Type A behavior and catecholamines: A critical review. In C. R. Lake & M. Ziegler (Eds.), Norepinephrine: Clinical aspects (pp. 346-367). Baltimore: Williams & Wilkins.

Glass, J. C., McKinney, M. E., Hofschire, P. J., & Fedorko, S. (1990). Cardiovascular reactivity to stress: an examination of familial trends. International Journal of Psychophysiology, 9, 1-11.

Harbin, T. J. (1989). The relationship between the Type A behavior pattern and physiological responsivity: A quantitative review. Psychophysiology, 26(1), 110-119.

Harrison, D. W., & Gorelczenko, P. M. (1990). Functional asymmetry for facial affect perception in high and low hostile men and women. International Journal of Neuroscience, 55, 89-97.

Harrison, D. W., Gorelczenko, P. M. & Cook, J. (1990). Functional asymmetry for facial affect perception. International Journal of Neuroscience, 52, 11-16.

Heilman, K. M. (1982). Amnestic disturbance following the left dorsomedial nucleus of the thalamus. Neuropsychologia, 20, 597-604.

Heilman, K. M., Bowers, D. & Valenstein, E. (1985). Emotional disorders associated with neurological diseases. In K. M. Heilman, & E. Valenstein (Eds.), Clinical; Neuropsychology. (2nd ed.) Oxford University Press: New York.

Heilman, K. M., Bowers, D. & Valenstein, E. (1993). Emotional disorders associated with neurological diseases. In K. M. Heilman, E. Valenstein (Eds.), Clinical Neuropsychology. Oxford University Press: New York.

Heilman, K. M., & Van Den Abell, T. (1979). Right hemisphere dominance for mediating cerebral activation. Neuropsychologia, 17, 315-321.

Herridge, M. L., Harrison, D. W., & Demaree, H. A. (1997). Hostility, facial configuration, and bilateral asymmetry on galvanic skin response. Psychobiology, 25, 71-76.

Herridge, M. L., Harrison, D. W., & Shenal, B. V. (1998). The effects of Hostility and arousal on facial affect perception: A test of a neuropsychological model of hostility. *Manuscript submitted*.

Houston, B. K. (1992). Personality characteristics, reactivity, and cardiovascular disease. In J. R. Turner, A. Sherwood, & K. C. Light (Eds.) Individual Differences in Cardiovascular Response to Stress, New York: Plenum Press.

Kinsbourne, M. & Bemporad, B. (1984). Lateralization of emotion: A model and the evidence. In N. A. Fox & R. J. Davidson (Eds.), *The psychobiology of affective development* (pp. 259-291). Hillsdale, NJ: Erlbaum.

Kinsbourne, M. & Byrd, M. (1985) Word list and visual hemifield shape recognition: Priming and interference effects. In M. J. Posner and O. S. M. Marin (Eds.), *Mechanisms of attention: Attention and performance IX*, Hillsdale, NJ: Erlbaum.

Kinsbourne, M. & Hisock, H. (1983). Asymmetries of dual-task performance. In J. B. Hellige (Ed.) *Cerebral hemisphere asymmetries: Method, theory and application.* New York: Praeger Press.

Kolb, B., & Taylor, L. (1990). Neocortical substrates of emotional behavior. In N. L. Stein, B. Leventhal, & T. Trabasso (Eds.), Psychological and biological approaches to emotion)pp. 115-144). Hillsdale, NJ: Erlbaum.

Krantz, D. S., Manuck, S. B., & Wing, R. R. (1986). Psychological stressors and task variables as elicitors of reactivity. In K. A. Matthews (Ed.) Handbook of Stress, Reactivity, and Cardiovascular Disease, pp. 85-108, New York: Wiley.

Kubany, E. S., Gino, A., Denny, N. R. & Torigoe, R. Y. (1994). Relationship of cynical hostility and PTSD among Vietnam veterans. Journal of Traumatic Stress, 7 21-31.

Lee, M. A. & Cameron, O. G. (1986-87). Anxiety, Type A behavior, and cardiovascular disease. International Journal of Psychiatry in Medicine, 16, 123-129.

Ley, R. G., & Bryden, M. P. (1982). A dissociation of right and left hemispheric effects for recognizing emotional tone and verbal content. Brain and Cognition, 1(1), 3-9.

Matthews, K. A., & Haynes, S. G. (1986). Type A behavior pattern and coronary risk: Update and critical evaluation. American Journal of Epidemiology, 123, 923-960.

McKeever, W. F., & Huling, M. D. (1971). Lateral dominance in tachistoscopic word recognition performances obtained with simultaneous bilateral input. Neuropsychologia, 9, 15-20.

Mondor, T. A., & Bryden, M. P. (1992). On the relation between auditory spatial attention and auditory perceptual asymmetries. Perception and Psychophysics, 52, 393-402.

Rey, A. (1964). L'Examin clinique en psychologie. Paris: Universitaire de France.

Robinson, R. G., Kubos, K. L., Starr, L. B., Rao, K., & Price, T. R. (1984). Mood disorders in stroke patients: Importance of location of lesion. Brain, 107, 81-93.

Schneider, R. H., Julius, S. & Karunas, R. (1989). Ambulatory blood pressure monitoring and laboratory reactivity in Type A behavior and components. Psychosomatic Medicine, 51, 290-305.

Smith, T. W. & Allred, K. D. (1989). Blood-pressure during social interaction in high and low cynically hostile males. Journal of Behavioral Medicine, 12, 135-143.

Snyder, K. A., & Harrison (1997) An affective auditory verbal learning test. The Archives of Clinical Neuropsychology, 12, 477-482.

Snyder, K. A., Harrison, D. W., & Gorman, W. (1994). Cerebral asymmetry correlates as a function of illumination level. *Unpublished manuscript*.

Snyder, K. A., Harrison, D. W. & Shenal, B. V. (1998). An affective auditory verbal learning test: Psychophysiological correlates. The Archives of Clinical Neuropsychology. *In press.*

Speilberger, C. D. (1968). Self-evaluation questionnaire. STAI Form X-2. Palo Alto, California: Consulting Psychologists Press.

Suls, J. & Wan, C. K. (1993). The relationship between trait hostility and cardiovascular reactivity: A quantitative review and analysis. Psychophysiology, 30, 615-626.

Toglia, M. P., & Battig, W. F. (1978). Handbook of word norms. Hillsdale, NJ: Lawrence Erlbaum.

Tomarken, A. J., Davidson, R. J., Wheeler, R. W., & Doss, R. (1992). Individual differences in anterior brain asymmetry and fundamental dimensions of emotion. Journal of Personality and Social Psychology, 62, 676-687.

Treiber, F. A., Musante, L., Riley, W. T., & Mabe, P. A. (1989). The relationship between hostility and blood pressure in children. Behavioral Medicine, 15, 173-178.

Tucker, D. M., and Frederick, S. L. (1989). Emotion and brain lateralization. In Wagner, H. and Manstead, A. (Eds.) Handbook of Social Psychophysiology, pp.27-70. New York: Wiley.

Tucker, D. M., Roth, R. S., Arneson, B. A., & Buckingham, V. (1977). Right hemisphere activation during stress. Neuropsychologia, 15, 697-700.

Wardlaw, G. M., Insel, P. M., and Seyler, M. F. (Eds.). (1992). Contemporary Nutrition. St. Louis, MO: Mosby.

Wilder, J. (1931/1957). The “law of initial values”, a neglected biological law and its significance for research and practice. In S. W. Porges and M. G. H. Coles (Eds.), Psychophysiology, (pp. 38-46). Stroudsburg, PA: Dowden, Hutchingson and Ross.

Williams, R. B., & Williams, V. W. (1993). Anger Kills, New York: Times Books.

Winer, B. J. (1971). Statistical principles in experimental design (2nd ed.). New York: McGrawHill.

Wittling, W. (1990). Psychophysiological correlates of human brain asymmetry: Blood pressure changes during lateralized presentation of an emotionally laden film. Neuropsychologia, 28, 457-470.

Table 1. Means and Standard Deviations for CMHS, CPD, BDI, STAI (State and Trait). Experiment 1

	Low-Hostile		High-Hostile	
Questionnaire	MEAN	SD	MEAN	SD
CMHS	16.67	3.74	32.80	4.26
CPD	9.20	2.46	9.93	2.81
BDI	4.00	4.88	12.13	8.43
STAI (State)	31.13	5.69	43.20	11.33
STAI (Trait)	32.80	6.13	47.87	8.55

Table 2. Means and Standard Deviations of Cardiovascular Variables by Group and Trial.

Experiment 1

Baseline to Post-Dichotic1

	Low-Hostile		High-Hostile	
	Mean	SD	MEAN	SD
Pre-Dichotic 1				
SBP	127.53	8.81	127.57	9.84
DBP	78.80	12.13	75.87	7.87
HR	72.43	13.47	72.37	13.80
Post-Dichotic 1				
SBP	125.00	9.80	125.03	8.02
DBP	77.33	12.59	75.37	8.96
HR	75.33	12.41	75.17	13.51

Post-Dichotic1 to Post-Cognitive Affective Stressor

	Low-Hostile		High-Hostile	
	Mean	SD	MEAN	SD
Pre-Stess*				
SBP	125.00	9.80	125.03	8.02
DBP	77.33	12.59	75.37	8.96
HR	75.33	12.41	75.17	13.51
Post-Stress				
SBP	125.47	9.53	126.47	8.06
DBP	80.27	10.78	78.83	9.19
HR	74.53	12.74	75.27	12.76

Post-Cognitive Affective Stressor to Post-Dichotic2

	Low-Hostile		High-Hostile	
	Mean	SD	MEAN	SD
Pre-Dichotic 2**				
SBP	125.47	9.53	126.47	8.06
DBP	80.27	10.78	78.83	9.19
HR	74.53	12.74	75.27	12.76
Post-Dichotic 2				
SBP	123.20	8.19	123.23	5.82
DBP	80.50	9.93	76.83	8.46
HR	76.50	14.33	75.40	12.55

* Phase 1 Post-Dichotic1 measures become Phase 2 Pre-Stress measures

** Phase 2 Post-Stress measures become Phase 3 Pre-Dichotic 2 measures

Table 3. MANOVA Results for the Cardiovascular Measures.

Experiment 1

Source	df	F	p
Group	(1,3)	.29	<.8312
Trial	(1,9)	4.12	<.0001**
GroupXTrial	(1,9)	.19	<.9951

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

Table 4. Independent ANOVA Results for the Cardiovascular Measures.
Experiment 1

Baseline to Post-Dichotic1

Source	df	SS	MS	F	p
SBP					
Group	(1,28)	.02	.02	.00	<.9915
Trial	(1,27)	96.27	96.27	4.42	<.0447*
GroupXTrial	(1,27)	.00	.00	.00	<1.0000
DBP					
Group	(1,28)	90.04	90.04	.44	<.5146
Trial	(1,27)	14.50	14.50	.84	<.3675
GroupXTrial	(1,27)	3.50	3.50	.20	<.6560
HR					
Group	(1,28)	.20	.20	.00	<.9806
Trial	(1,27)	121.84	121.84	8.94	<.0058**
GroupXTrial	(1,27)	0.04	0.04	.00	<.9585

Post-Dichotic1 to Post-Cognitive Affective Stressor

Source	df	SS	MS	F	p
SBP					
Group	(1,28)	4.00	4.00	.03	<.8703
Trial	(1,27)	13.54	13.54	1.28	<.2682
GroupXTrial	(1,27)	3.50	3.50	.33	<.5701
DBP					
Group	(1,28)	43.35	43.35	.22	<.6419
Trial	(1,27)	153.60	153.60	6.50	<.0166*
GroupXTrial	(1,27)	1.07	1.07	.05	<.8333
HR					
Group	(1,28)	1.20	1.20	.00	<.9518
Trial	(1,27)	1.84	1.84	.27	<.6101
GroupXTrial	(1,27)	3.04	3.04	.44	<.5128

Post-Cognitive Affective Stressor to Post-Dichotic2

Source	df	SS	MS	F	p
SBP					
Group	(1,28)	4.00	4.00	0.03	<.8546
Trial	(1,27)	113.44	113.44	10.01	<.0037**
GroupXTrial	(1,27)	3.50	3.50	0.31	<.5827
DBP					
Group	(1,28)	97.54	97.54	0.59	<.4507
Trial	(1,27)	11.70	11.70	0.63	<.4354
GroupXTrial	(1,27)	18.70	18.70	1.00	<.3256
HR					
Group	(1,28)	.50	.50	.00	<.9690
Trial	(1,27)	16.54	16.54	1.10	<.3034
GroupXTrial	(1,27)	12.60	12.60	.84	<.3678

** p ≤ .01 * p ≤ .05

Table 5. Means and Standard Deviations of POC Scores by Group and Trial. Experiment 1

	Low-Hostile		High-Hostile	
Trial	Mean	SD	Mean	SD
Pre-Stress	.13	.22	.20	.34
Post-Stress	.15	.25	.22	.40

Table 6. Independent ANOVA Results for POC Scores and Number of Correctly Identified Phonemes Presented to the Left and Right Ears.

Experiment 1

Source	df	SS	MS	F	p
POC Scores					
Group	(1,28)	.07	.07	.40	<.5299
Trial	(1,28)	.01	.01	.46	<.5039
GroupXTrial	(1,28)	.00	.00	.00	<.9449
Left Ear					
Group	(1,28)	15.00	15.00	.49	<.4875
Trial	(1,28)	.00	.00	.00	<1.000
GroupXTrial	(1,28)	.60	.60	.37	<.5479
Right Ear					
Group	(1,28)	18.15	18.15	.48	<.4963
Trial	(1,28)	8.82	8.82	2.65	<.1150
GroupXTrial	(1,28)	.42	.42	.13	<.7262

Table 7. Means and Standard Deviations of Phonemes Identified at the Left Ear by Group and Trial
Experiment 1

	Low-Hostile		High-Hostile	
Trial	Mean	SD	Mean	SD
Pre-Stress	11.20	2.91	10.40	4.34
Post-Stress	11.40	3.38	10.20	5.02

Table 8. Means and Standard Deviations of Phonemes Identified at the Right Ear by Group and Trial.

Experiment 1

	Low-Hostile		High-Hostile	
Trial	Mean	SD	Mean	SD
Pre-Stress	14.60	3.07	15.87	4.85
Post-Stress	15.53	3.60	16.47	6.09

Table 9. Independent ANOVA Results for Number of Correctly Identified Phonemes Presented Pre- and Post- Stress (comparing left and right ears).

Experiment 1

Source	df	SS	MS	F	p
Pre-Stress					
Group	(1,28)	.82	.82	.59	<.4483
Ear	(1,28)	294.82	294.82	10.26	<.0034**
GroupXEar	(1,28)	16.02	16.02	.56	<.4616
Post-Stress					
Group	(1,28)	.27	.27	.13	<.7204
Ear	(1,28)	405.60	405.60	9.82	<.0040**
GroupXEar	(1,28)	17.07	17.07	.41	<.5255

** p ≤ .01   * p ≤ .05

Table 10. Means and Standard Deviations of Phonemes Identified Pre-Stress by Group and Ear.
Experiment 1

	Low-Hostile		High-Hostile	
Trial	Mean	SD	Mean	SD
Left Ear	11.20	2.91	10.40	4.34
Right Ear	14.60	3.07	15.87	4.85

Table 11. Means and Standard Deviations of Phonemes Identified Post-Stress by Group and Ear.

Experiment 1

	Low-Hostile		High-Hostile	
Trial	Mean	SD	Mean	SD
Left Ear	11.40	3.38	10.20	5.02
Right Ear	15.53	3.60	16.47	6.09

Table 12. Means and Standard Deviations for CMHS, CPD, BDI, STAI (State and Trait). Experiment 2

	Low-Hostile		High-Hostile	
Questionnaire	MEAN	SD	MEAN	SD
CMHS	12.53	6.08	31.67	3.09
CPD	10.73	2.12	9.40	2.85
BDI	5.33	6.09	10.60	6.70
STAI (State)	34.47	10.21	40.40	6.27
STAI (Trait)	34.20	10.04	41.20	7.20

Table 13. Means and Standard Deviations of Cardiovascular Variables by Group and Trial.

Experiment 2

Baseline to Post-Tscope1

	Low-Hostile		High-Hostile	
	Mean	SD	MEAN	SD
Pre-Tscope 1				
SBP	122.90	10.70	129.77	10.62
DBP	78.43	6.75	78.53	8.37
HR	74.23	9.88	70.30	7.45
Post-Tscope 1				
SBP	119.47	8.51	128.80	8.55
DBP	79.17	6.63	80.47	10.27
HR	75.77	11.16	73.17	7.81

Post-Tscope1 to Post-Cold Pressor Stressor

	Low-Hostile		High-Hostile	
	Mean	SD	MEAN	SD
Pre-Stess*				
SBP	119.47	8.51	128.80	8.55
DBP	79.17	6.63	80.47	10.27
HR	75.77	11.16	73.17	7.81
Post-Stress				
SBP	123.57	9.63	130.70	9.49
DBP	81.20	9.40	77.67	7.89
HR	74.17	13.73	69.10	7.56

Post-Cold Pressor Stressor to Post-Tscope2

	Low-Hostile		High-Hostile	
	Mean	SD	MEAN	SD
Pre-Tscope2**				
SBP	123.57	9.63	130.70	9.49
DBP	81.20	9.40	77.67	7.89
HR	74.17	13.73	69.10	7.56
Post-Tscope 2				
SBP	119.87	8.74	127.50	8.03
DBP	77.87	7.86	79.80	8.62
HR	76.10	9.94	71.63	9.27

* Phase 1 Post-Tscope1 measures become Phase 2 Pre-Stress measures

** Phase 2 Post-Stress measures become Phase 3 Pre-Tscope2 measures

Table 14. MANOVA Results for the Cardiovascular Measures.
Experiment 2

Source	df	F	p
Group	(1,3)	3.57	<.0282*
Trial	(1,9)	2.00	<.0406*
GroupXTrial	(1,9)	1.06	<.3914

** p ≤ .01   * p ≤ .05

Table 15. Independent ANOVA Results for the Cardiovascular Measures.
Experiment 2

Baseline to Post-Tscope1

Source	df	SS	MS	F	p
SBP					
Group	(1,28)	984.15	984.15	5.54	<.0258*
Trial	(1,27)	72.60	72.60	8.23	<.0078**
GroupXTrial	(1,27)	22.82	22.82	2.59	<.1191
DBP					
Group	(1,28)	7.35	7.35	.06	<.8062
Trial	(1,27)	26.67	26.67	2.09	<.1593
GroupXTrial	(1,27)	5.40	5.40	.42	<.5206
HR					
Group	(1,28)	160.07	160.07	1.14	<.2941
Trial	(1,27)	72.60	72.60	2.47	<.1274
GroupXTrial	(1,27)	6.67	6.67	.23	<.6377

Post-Tscope1 to Post-Cold Pressor Stressor

Source	df	SS	MS	F	p
SBP					
Group	(1,28)	1016.82	1016.82	6.99	<.0133**
Trial	(1,27)	135.00	135.00	7.24	<.0119**
GroupXTrial	(1,27)	18.15	18.15	.97	<.3324
DBP					
Group	(1,28)	18.70	18.70	.14	<.7068
Trial	(1,27)	2.20	2.20	.11	<.7459
GroupXTrial	(1,27)	87.60	87.60	4.26	<.0484*
HR					
Group	(1,28)	220.42	220.42	1.09	<.3054
Trial	(1,27)	120.42	120.42	8.95	<.0057**
GroupXTrial	(1,27)	22.82	22.82	1.70	<.2033

Post-Cold Pressor Stressor to Post-Tscope2

Source	df	SS	MS	F	p
SBP					
Group	(1,28)	817.70	817.70	5.86	<.0223*
Trial	(1,27)	178.54	178.54	8.04	<.0084**
GroupXTrial	(1,27)	.94	.94	.04	<.8386
DBP					
Group	(1,28)	9.60	9.60	.08	<.7776
Trial	(1,27)	5.40	5.40	.21	<.6479
GroupXTrial	(1,27)	112.07	112.07	4.42	<.0445*
HR					
Group	(1,28)	340.82	340.82	1.73	<.1985
Trial	(1,27)	74.82	74.82	3.98	<.0559
GroupXTrial	(1,27)	1.35	1.35	.07	<.7907

** $p \le .01$ * $p \le .05$

Table 16. Means and SD of POC Scores by Group and Trial.
Experiment 2

	Low-Hostile		High-Hostile	
Trial	Mean	SD	Mean	SD
Pre-Stress	.29	.45	.10	.47
Post-Stress	.28	.45	.12	.55

Table 17. Independent ANOVA Results for POC Scores and Number of Correctly Identified Phonemes Presented to the Left and Right Visual Fields.

Experiment 2

Source	df	SS	MS	F	p
POC Scores					
Group	(1,28)	.46	.46	1.07	<.3094
Trial	(1,28)	.00	.00	.00	<.9444
GroupXTrial	(1,28)	.00	.00	.06	<.8155
Left VF					
Group	(1,28)	112.07	112.07	1.17	<.2885
Trial	(1,28)	.07	.07	.01	<.9277
GroupXTrial	(1,28)	1.07	1.07	.13	<.7171
Right VF					
Group	(1,28)	98.82	98.82	1.01	<.3242
Trial	(1,28)	.42	.42	.06	<.8140
GroupXTrial	(1,28)	.15	.15	.02	<.8877

Table 18. Means and Standard Deviations of Phonemes Identified at the Left Visual Field by Group and Trial.

Experiment 2

	Low-Hostile		High-Hostile	
Trial	Mean	SD	Mean	SD
Pre-Stress	10.47	6.59	13.47	7.12
Post-Stress	10.80	6.74	13.27	8.23

Table 19. Means and Standard Deviations of Phonemes Identified at the Right Visual Field by Group and Trial.

Experiment 2

	Low-Hostile		High-Hostile	
Trial	Mean	SD	Mean	SD
Pre-Stress	19.13	6.93	16.47	7.06
Post-Stress	19.20	6.74	16.73	8.23

Table 20. Independent ANOVA Results for Number of Correctly Identified Phonemes Presented Pre- and Post- Stress (comparing left and right visual fields).

Experiment 2

Source	df	SS	MS	F	p
Pre-Stress					
Group	(1,28)	.42	.42	.68	<.4180
VF	(1,28)	510.42	510.42	5.35	<.0282*
GroupXVF	(1,28)	120.42	120.42	1.26	<.2706
Post-Stress					
Group	(1,28)	.00	.00	.00	<.9999
VF	(1,28)	528.07	528.07	4.66	<.0395*
GroupXVF	(1,28)	91.27	91.27	.81	<.3770

** p ≤ .01   * p ≤ .05

Table 21. Means and Standard Deviations of Phonemes Identified Pre-Stress by Group and VF.

Experiment 2

	Low-Hostile		High-Hostile	
Trial	Mean	SD	Mean	SD
Left VF	10.47	6.59	13.47	7.12
Right VF	19.13	6.93	16.47	7.06

Table 22. Means and Standard Deviations of Phonemes Identified Post-Stress by Group and Ear.
Experiment 2

	Low-Hostile		High-Hostile	
Trial	Mean	SD	Mean	SD
Left VF	10.80	6.74	13.27	8.23
Right VF	19.20	6.74	16.73	8.23

Figure Caption

<u>Figure 1.</u> Mean SBP measures as a function of Trial for Experiment 1.

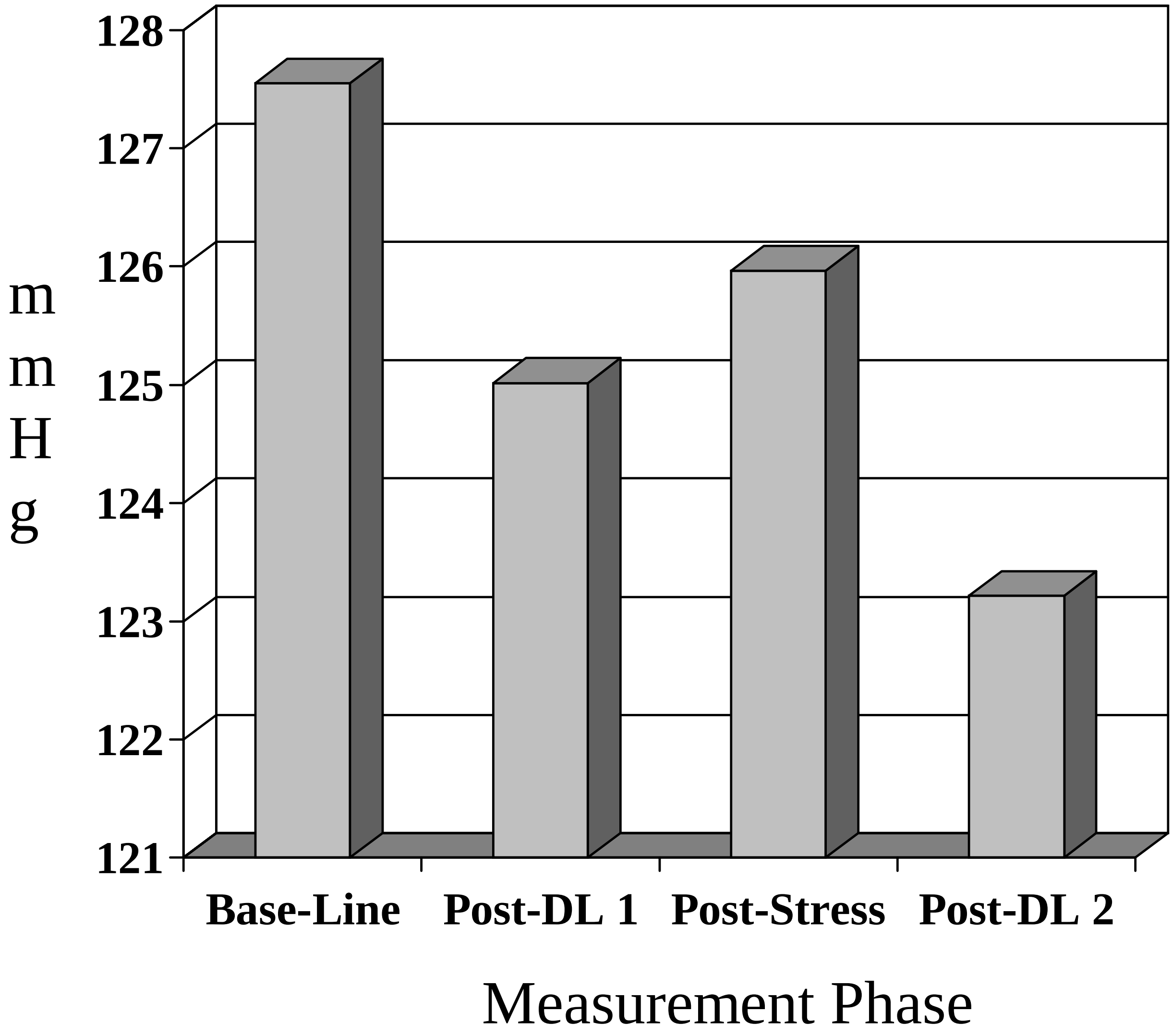

Figure 1. Mean SBP

Figure Caption

Figure 2. Mean number of dichotic verbal recognitions for low- and high-hostiles by ear during the Pre-Cognitive Affective Stressor for Experiment 1.

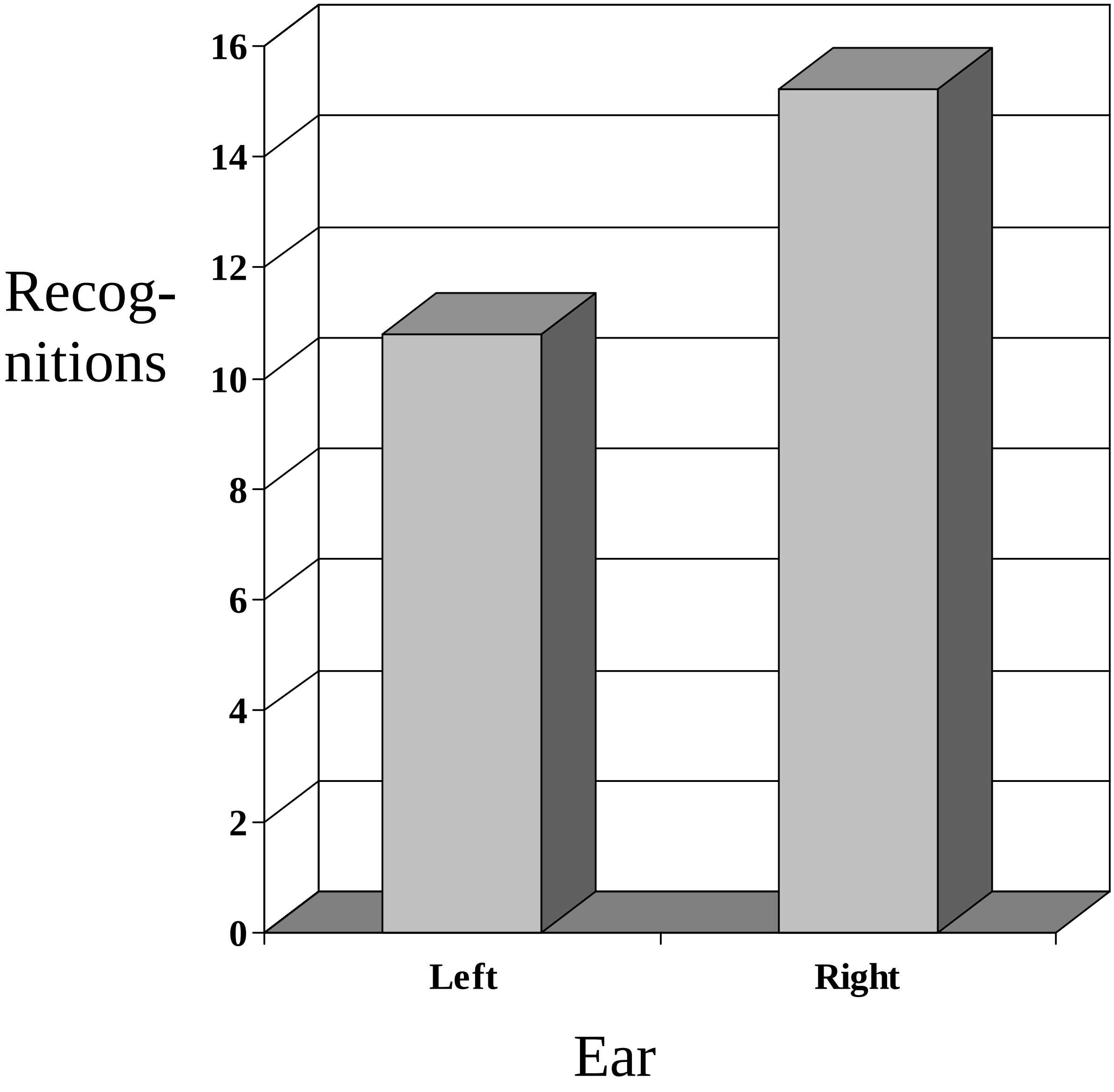

Figure 2. Pre-Stress Recognitions

Figure Caption

Figure 3. Mean number of dichotic verbal recognitions for low- and high-hostiles by ear during the Post-Cognitive Affective Stressor for Experiment 1.

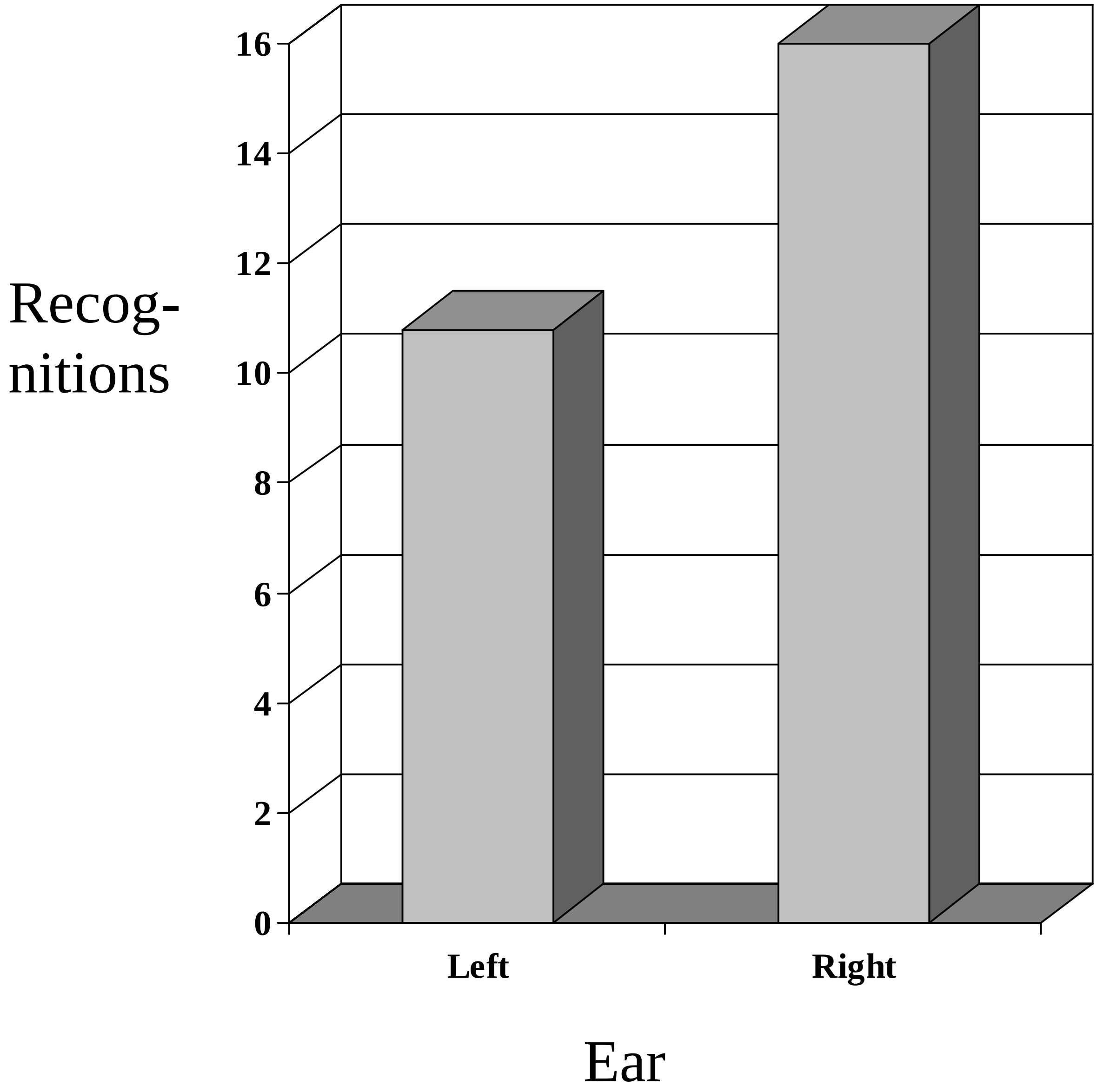

Figure 3. Post-Stress Recognitions

Figure Caption

Figure 4. Mean SBP measures as a function of Trial for Experiment 2.

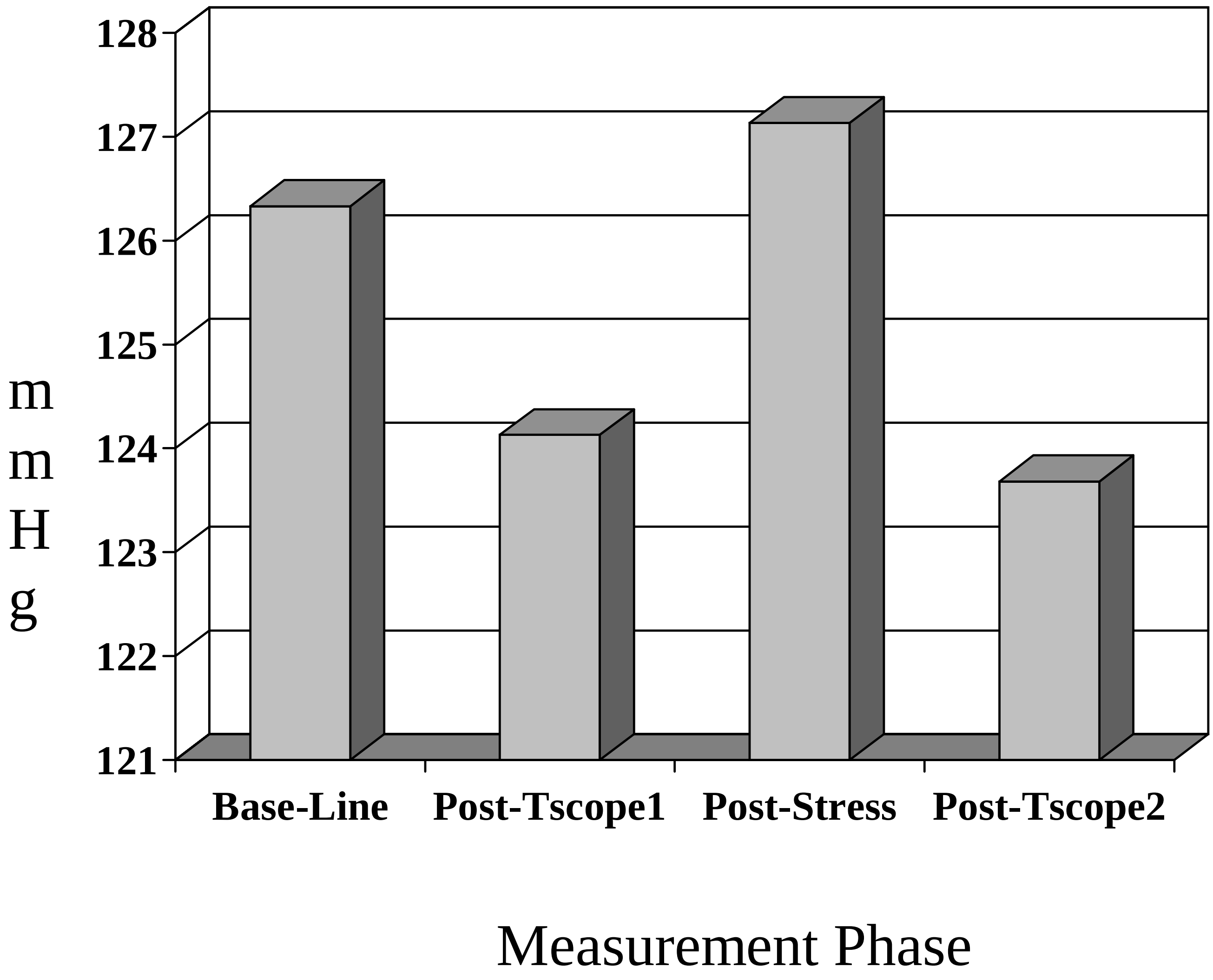

Figure 4. Mean SBP

Figure Caption

Figure 5. Mean number of tachistoscopic linguistic recognitions for low- and high-hostiles by visual field during the Pre-Cold Pressor Stressor for Experiment 2.

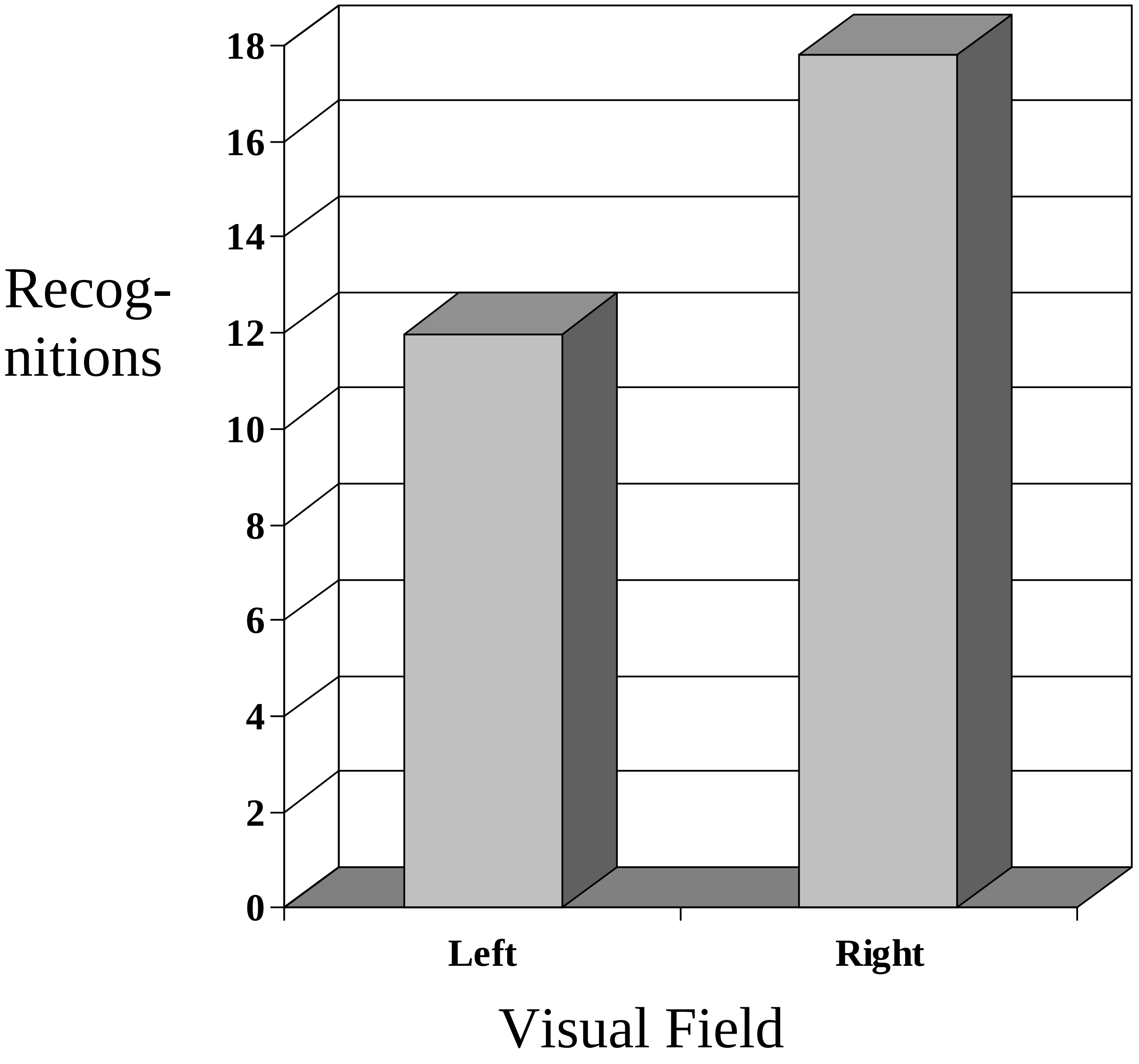

Figure 5. Pre-Stress Recognitions

Figure Caption

Figure 6. Mean number of tachistoscopic linguistic recognitions for low- and high-hostiles by visual field during the Post-Cold Pressor Stressor for Experiment 2.

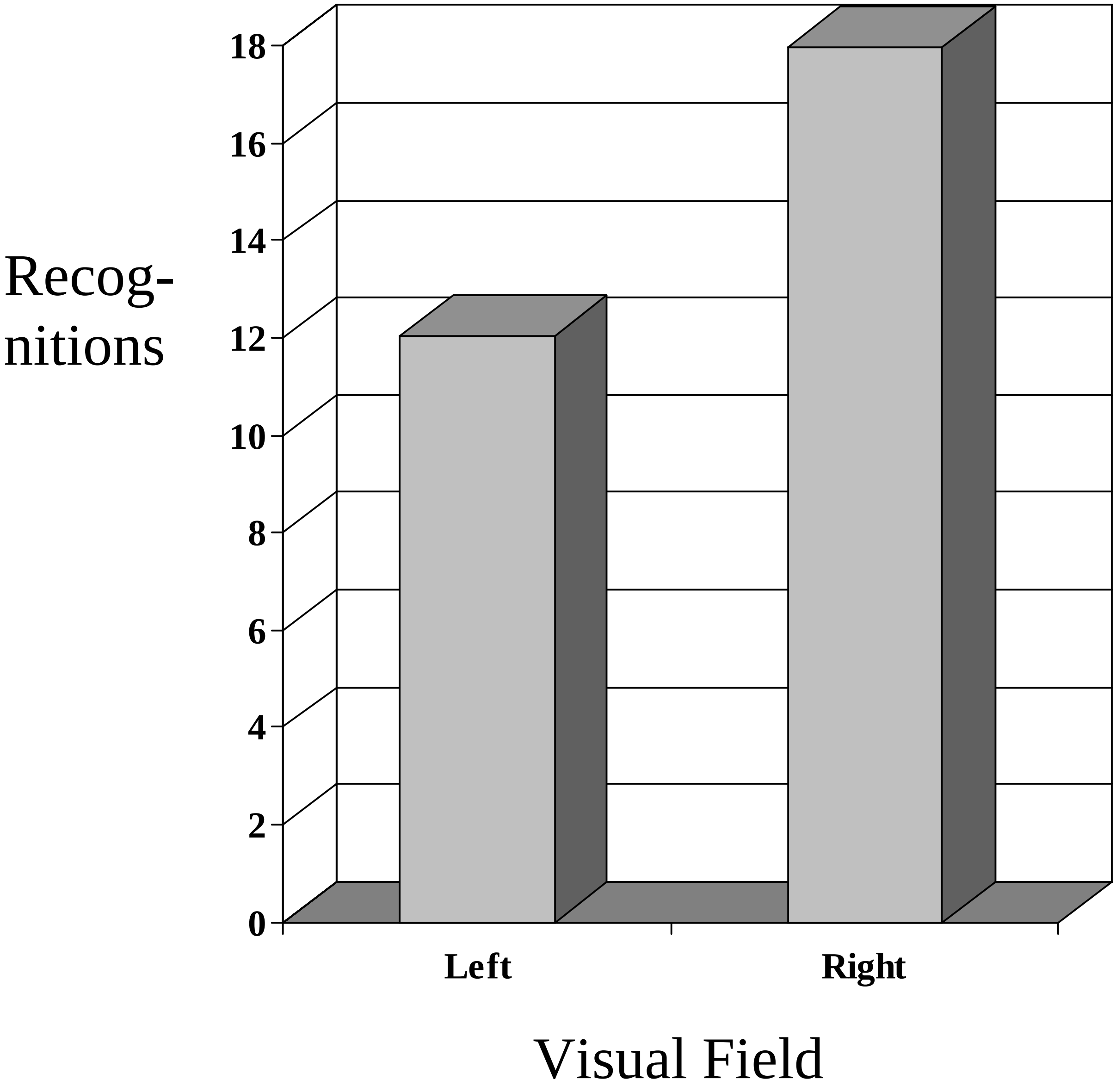

Figure 6. Post-Stress Recognitions

April, 1998

CURRICULUM VITAE

Brian V. Shenal

PERSONAL INFORMATION

Born: March 1, 1973, Richmond, VA

Marital Status: Married

Business Address:	Department of Psychology Virginia Polytechnic Institute and State University Blacksburg, Virginia 24061-0436
Business Phone:	(540) 231-6914
Home Phone:	(540) 961-9339

EDUCATION

M.S. Master of Science candidate, 1996-present
Degree expected, May, 1998
Virginia Polytechnic Institute and State University

Title of thesis: The Dynamic Cerebral Laterality Effect: Group Differences in Hostility, Cardiovascular Regulation, and Sensory Recognition

Major advisor: Dr. David W. Harrison

B.S. Virginia Polytechnic Institute and State University, 1995
Major field of study: Psychology

Major advisor: Dr. Joseph Germana

HONORS AND AWARDS

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

CLINICAL TRAINING

1996 - present
Neuropsychological Practicum Team – Graduate Clinician
Psychological Services Center
Virginia Tech, Blacksburg, Virginia
Graduate level practicum team specializing in the assessment and treatment of a variety of neuropsychological difficulties, including head injury, stroke, traumatic brain injury, and other neuropsychological problems.
Supervisor:
January 1998 – Present David W. Harrison, Ph.D.

Clinical Practicum Team - Graduate Clinician
Psychological Services Center
Virginia Tech, Blacksburg, Virginia
Graduate level practicum team specializing in the assessment and treatment of a variety of psychological disorders, including depression, anxiety, learning disability (LD), attention deficit disorder (ADD), and relationship problems.
Supervisor:
August 1997 – December 1997 Russell Jones, Ph.D.
Approximate number of hours: 125

Supervisor:
August 1996 - May 1997 Robert S. Stephens, Ph.D.
Approximate number of hours: 250

1997
Paid therapist - Graduate Clinician
Psychological Services Center and Child Study Center, Va. Tech
Graduate level team specializing in the assessment and treatment of a variety of psychological disorders, including depression, anxiety, learning disability (LD), attention deficit disorder (ADD), and relationship problems.
Supervisor:
May 1997 - August 1997 Thomas H. Ollendick, Ph.D.
Approximate number of hours: 250

1994-1997 Paid therapist, full time position 1/96-8/96, part-time position 11/94 - 1/96;
8/96-12/96, - Therapist/ Certified Rehabilitation Provider (CRP)
Hollins Head Injury Program, Hollins, Virginia
Responsibilities included designing and implementing individual and group therapeutic protocols based on neuropsychological assessments, recording on-going assessments of treatment efficacy, developing overall rehabilitation goals and activities, aiding in activities of daily living and health maintenance, and administering physical therapy and work-out routines.
Approximate number of hours: 1,700
Supervisor: Bill James, MA, LPC, CRP

EMPLOYMENT

1998 - present Assessment Supervisor/Clinician
Virginia Polytechnic Institute and State University, Blacksburg, Virginia
Assessment Clinician/Supervisor for Child Study Center and Psychological Services Center
Duties included administering comprehensive intellectual, psychoeducational, and academic assessments for children, supervising graduate students in the administration of intellectual, academic, and attentional testing and subsequent feedback sessions, and providing individual supervision and assistance for graduate clinicians.

1997 Teaching Assistant
Virginia Polytechnic Institute and State University, Blacksburg, Virginia
Teaching assistant for Intellectual Assessment graduate course.
Duties included supervising graduate students in the administration of intellectual and psychoeducational assessments and subsequent feedback sessions for children and adults, grading intellectual assessment reports and exams, and providing individual supervision and assistance for graduate clinicians.

1997 - present Employment Assessment Administrator
Affiliates in Psychology and Therapy, Inc., Huntington, WV
Duties included administering and proctoring job relevant assessments (i.e., personality inventories, achievement tests) for job applicants.

1997 Graduate Clinician
Psychological Services Center and Child Study Center, Va. Tech

1997	Teaching Assistant Virginia Polytechnic Institute and State University, Blacksburg, Virginia Taught two (2) Introduction to Psychology laboratories. Duties involved teaching a laboratory section associated with an undergraduate introductory psychology class, writing and administering exams, grading essays, and providing individual assistance to students.
1994-1997	Therapist Hollins Head Injury Program, Hollins, Virginia (previously discussed)

TEACHING EXPERIENCE

1997	Teaching Assistant - Intellectual Assessment Teaching Assistant (previously discussed)
1997	Teaching Assistant - Introductory Psychology Teaching Assistant (previously discussed)

PROFESSIONAL ACTIVITIES

Membership

American Psychological Association (APA) - student affiliate
American Psychological Society (APS) – student affiliate
Graduate Student Assembly - Department of Psychology Representative
    Commission on Graduate Studies and Policies
    Committee on Graduate Relations
National Academy of Neuropsychology (NAN) - student affiliate
Virginia Psychological Association (VPA) - student affiliate

CURRENT RESEARCH AND SCHOLARLY INTERESTS

Cortical, subcortical, and autonomic correlates of emotion and cardiovascular reactivity, especially in relation to hostility; methods in neuropsychology, psychophysiology, and psychopharmacology.

REFEREED PUBLICATIONS

Refereed Articles

Shenal, B. V., & Harrison, D. W. (1998). The dynamic cerebral laterality effect: Group differences in hostility, cardiovascular regulation, and tachistoscopic and dichotic listening recognition. Manuscript in preparation for submission.

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. Manuscript in preparation for submission.

Everhart, D. E., Harrison, D. W. & Shenal, B. V. (1998) Neuropsychological effects of anxiety without depression on grip strength. Manuscript in preparation for submission.

Shenal, B. V., Crews, Jr., W. D., & Harrison, D. W. (1998). Assessment techniques of neuropsychological sequelae following electrical shock: QEEG and syndrome analysis. Manuscript in preparation for submission.

Demaree, H. A., Harrison, D. W., Everhart, D. E., & Shenal, B. V. (1997) QEEG-assisted neuropsychological evaluation of autism. International Journal of Neuropsychology.

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

Refereed Abstracts and Conference Proceedings

(one-page publications)

Everhart, D. E., Shenal, B. V., & Harrison, D. W. (1997). Neuropsychological effects of anxiety without depression on functional motor asymmetry. Archives of Clinical Neuropsychology, 13(1), 55.

Shenal, B. V., Demaree, H. A., & Harrison, D. W. (1997). Analysis of QEEG and cardiovascular responses to stress. Archives of Clinical Neuropsychology, 13(1), 127.

PRESENTATIONS AND PAPERS

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. Submitted to the annual meeting of the Virginia Psychological Association, Wintergreen, VA. April, 1998.

Shenal, B. V., Demaree, H. A., & 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.

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.

REFERENCES

David W. Harrison, Ph.D., Associate Professor of Psychology and Director of Laboratory of Neuropsychology, Virginia Polytechnic Institute and State University, Department of Psychology, Blacksburg, Virginia 24061-0436.

Russell T. Jones, Ph.D., Professor of Psychology, Virginia Polytechnic Institute and State University, Department of Psychology, Blacksburg, Virginia 24061-0436.

Thomas H. Ollendick, Ph.D., Professor of Psychology and Director of Child Study Center

and Psychological Services Center, Virginia Polytechnic Institute and State University, Department of Psychology, Blacksburg, Virginia 24061-0436.

Richard A. Winett, Ph.D., Professor of Psychology and Director of Clinical Psychology, Virginia Polytechnic Institute and State University, Department of Psychology, Blacksburg, Virginia 24061-0436.