

REACTIVITY AND RECOVERY OF PHYSIOLOGICAL AND
NEUROPSYCHOLOGICAL VARIABLES AS A FUNCTION OF HOSTILITY

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(ABSTRACT)

This experiment tested three hypotheses regarding right cerebral involvement in hostility and physiological arousal. First, replication of previous research indicating heightened physiological responding to stress among high hostile individuals was attempted. Second, high hostile individuals were predicted to an increased tendency toward right hemisphere dominance following exposure to a stressor. Third, high hostile individuals were expected to maintain their physiological arousal and shift in cerebral laterality longer than the low-hostile comparison group.

Low- and high-hostile participants (25 males per group, drawn from the undergraduate Psychology pool) were identified using the Cook-Medley Hostility Scale (CMHS). Physiological measures (SBP, DBP, HR) were recorded at baseline, as were results from a dichotic listening procedure. Participants were then administered the cold-pressor procedure, and physiological recordings were taken again. Dichotic listening procedures were then administered 3 consecutive times to monitor for shifts in cerebral laterality. After the final dichotic listening procedure, physiological measures were taken once again to determine recovery rates.

Results did not support the a priori hypotheses. Specifically, low-hostile individuals were consistently higher on the physiological measures when compared to the high-hostiles. The predicted maintenance of increased physiological arousal was also not supported by the data. Neuropsychological measures also failed to differentiate between the groups, and failed to demonstrate the predicted shift in cerebral laterality.

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Finally, I would like to remind everyone of the popular expression, "Life is a journey, not a destination." Just think of this thesis as having been the scenic route.

Table of Contents

Abstract.....	ii
Acknowledgments.....	iii
List of Figures.....	v
List of Tables.....	vi
Introduction.....	1
Fundamental Neuropsychological Concepts.....	1
Cerebral Lateralization of Emotion.....	1
Right Hemisphere and Autonomic Arousal.....	5
Hostility.....	7
Neuropsychological Model of Hostility.....	10
Rationale.....	10
Variables.....	11
Hypotheses.....	11
Method.....	11
Participants.....	11
Self-Report Measures.....	12
Apparatus.....	12
Procedure.....	13
Analyses.....	14
Results.....	14
Discussion.....	29
References.....	33
Vita.....	37

List of Figures

Figure 1. Systolic Blood Pressure by Group by Trial.....25
Figure 2. Heart Rate by Group by Trial.....27

List of Tables

Table 1. Means and Standard Deviations for the Cook-Medley Hostility Scale, Beck Depression Inventory, State-Trait Anxiety Inventory, and Coren, Porac, and Duncan Laterality Questionnaire.....	16
Table 2. Means and Standard Deviations of Percentage of Correct Scores by Group and Trial.....	17
Table 3. Independent ANOVA Results for POC Scores, and Number of Correctly Identified Stimuli at the Left and Right Ears.....	18
Table 4. Means and Standard Deviations of Stimuli Correctly Identified by Left Ear by Group and Trial.....	20
Table 5. Means and Standard Deviations of Stimuli Correctly Identified by Right Ear by Group and Trial.....	21
Table 6. Means and Standard Deviations of Physiological Variables by Group and Trial.....	22
Table 7. Summary of the Independent ANOVA Results for Physiological Variables.....	24

Introduction

There is a long history, dating back to the ancient Greeks, relating psychological phenomena and personal health (Stern, Ray, & Davis, 1980). Ancient physicians recorded numerous instances where a person's emotions had a negative impact on their physiological well-being. There are reports of speech deficits, increased perspiration, cardiac arrhythmias, and near death experiences, all as a result of being 'love-sick.' Though not nearly as far-reaching chronologically, but much more rigorous in its methodology has been the study of affective style and its relationship to cardiovascular disease (Friedman & Rosenman, 1974). The relationship between affect and coronary heart disease was first described as resulting from the Type A Behavior Pattern (TABP; Friedman & Rosenman, 1974). The global TABP was then narrowed to a more specific subcomponent, namely hostility (Rosenman, 1986). The search for the key link between behavior and increased cardiovascular risk has continued to evolve, with the current focus being on expressive hostility (Smith & Christensen, 1992). This aspect of hostility emphasizes the necessity of negative interpersonal interactions with others. Thus, it is required that a person not simply feel angry or hostile, but that they actually express these feelings in their interactions with others.

Fundamental Neuropsychological Concepts

Prior to further discussions on hostility and its effect on physiology, two key neuropsychological assumptions will be introduced. One of the primary assumptions of neuropsychological theory is that all behavior is the result of physical changes in the brain (Heilman & Valenstein, 1993). Thus, every experience that one perceives is the result of physical changes within the brain. Likewise, every cognition, expressed or not, and every behavior exhibited is the direct result of changes within the physical structure or functional integrity of at least one brain system.

A second underlying principle is the concept of laterality. This refers to the structural and functional differences between the two cerebral hemispheres (Kolb & Whishaw, 1990). One of the most clearly delineated examples of lateralized functioning is the projection of sensory and motor nerves from one hemisphere to the contralateral hemibody (Kandel, 1991). For example, afferent and efferent information arising at the right leg, right arm, and right visual field are under the dominion of the left cerebral hemisphere.

Cerebral Lateralization of Emotion

There is an extensive literature regarding the lateralization of emotion within the cerebral hemispheres. Most findings, however, can be placed under two general models of emotional processing, the right-hemisphere (RH) model or the valence model. The RH model proposes that emotions are primarily processed within the RH. The valence model proposes that emotion is split in a bivalent manner (positive/negative, happy/sad, approach/avoidance) with each hemisphere being specialized for one valence. Within this model, the left hemisphere is seen as dominant for positive affect, while the right hemisphere is seen as dominant for negative affect.

Support for the right-hemisphere model of emotion is reported by Bryden, Ley, and Sugarman (1982). In this study, participants were asked to judge the affective valence of dichotically presented tonal sequences. Results show a left ear advantage for accuracy ratings of tones presented to the left ear. Specifically, participants showed an enhanced ability to identify emotional tonal sequences presented to the left ear, regardless of affective valence. The left ear advantage was consistent across both positive and negative tonal sequences.

While the preceding study showed a right-hemisphere superiority in the perception of affect, other studies provide support for right-hemisphere dominance in the expression of affect, as well. Borod, Kent, Koff, Martin, and Alpert (1988) report motor asymmetries in the production of posed, as opposed to spontaneous, affective facial expressions. More specifically, participants were rated as expressing more intense emotion at the left hemi-face as compared to the right hemi-face. These results support the right-hemisphere model of emotion, as there was greater perceived left hemi-facial affective intensity regardless of emotional valence. Given the contralateral cerebral control of voluntary muscle, this increased affect intensity of the left hemi-face points to greater right-hemisphere involvement in the production of facial affect, regardless of the emotion that is being portrayed.

Borod (1992) provides a review of the lateralization literature, and reports relative support for the right-hemisphere dominance for emotion. This review indicates that for the perception of affect (facial and prosodic), neurologically-healthy participants show a right-hemisphere dominance for the perception of emotion. Stroke patients show a similar pattern, with right-brain stroke patients being more impaired on facial affect perception than either left-brain stroke patients or controls. This dominance of the right-hemisphere for the perception of emotion is independent of emotional valence. Borod (1992) also summarizes results from studies focused on the expression of affective information. This review suggests greater involvement of the right-hemisphere in the expression of facial affect (i.e. significantly more movement at the left hemi-face, and significantly higher ratings for emotional expression at the left hemi-face). The data reviewed for facial expression in stroke patients is not as easily interpreted, with contradictory findings within this area. However, there is general agreement reported for greater impairments in the right-hemisphere stroke patients for the production of emotional prosody as compared to either the left-hemisphere stroke samples or controls. Once again, this deficit in the production of emotional prosody is independent of emotional valence, with right-hemisphere stroke patients being impaired for the production of positive or negative intoned prosody.

While much of the evidence supporting the right-hemisphere model of emotion is based on empirical studies, the literature regarding the lateralization of positive and negative emotion to separate hemispheres is largely the result of early case studies focused on patients with unilateral brain lesions. Results from those early studies reported that left hemisphere lesions were found to produce a depressive-catastrophic reaction, while lesions of the right hemisphere were associated with inappropriate optimism or denial of problems (Gainotti, 1972). This apparent discrepancy between the

effects of left or right hemisphere damage was investigated by Gasparrini, Satz, Heilman, and Coolidge (1977). They examined the laterality construct through the administration of the Minnesota Multiphasic Personality Inventory (MMPI) to patients with unilateral cerebral dysfunction. Nearly 50% of their patients with left hemisphere damage reported an elevated score for depression, supporting the depressive/catastrophic reaction seen with left hemisphere damage (Gainotti, 1972). The elevated depression scores in the left hemisphere lesion group are starkly contrasted with the right hemisphere lesion group, in which no patients evidenced a heightened score for depression.

While research participants have long been grouped according to interhemispheric differences in cerebral dysfunction, it is also possible to differentiate participants based on intrahemispheric criteria. Robinson, Kubos, Starr, Rao, and Price (1983) report on a study of 48 patients with unilateral cerebral infarctions, in which the appearance of depressive symptoms was significantly higher for participants with left hemisphere damage. Conversely, the appearance of inappropriate cheerfulness was significantly higher for the right hemisphere-damaged group. In addition to these interhemispheric differences, it was possible to further divide the groups based on the lesion site within each hemisphere. For participants with left hemisphere dysfunction, depression rating scores were significantly higher for an anterior, as compared to posterior, lesion. Participants with right hemisphere damage showed a similar relationship between the location of right hemisphere damage and expressions of undue cheerfulness. Specifically, those participants with damage to the anterior portion of the right hemisphere were significantly more likely to show undue cheerfulness when compared with a more posterior stroke location.

Robinson, Kubos, Starr, Rao, and Price (1984) replicated these findings of inter- and intrahemispheric differences associated with lesion location, but also extended the results by demonstrating a significant correlation between depression scores and proximity of the lesion to the left frontal pole. Based on these findings, we might expect that not only would a left anterior lesion be more likely to result in depression than any other lesion location, but within that grouping, the closer the lesion was to the frontal pole, the more severe the depression would likely be. Starkstein, Robinson, and Price (1987) supported and extended these findings, showing that the relationships held for subcortical, as well as cortical lesions. Left subcortical lesions were associated with depression, whereas right subcortical lesions were associated with undue cheerfulness.

Studies such as these have led to the development of emotional lateralization hypotheses. Specifically, it has been hypothesized that the left hemisphere is responsible for positive emotions and the right hemisphere is responsible for negative emotions (Ahern & Schwartz, 1985; Davidson, 1984; Reuter-Lorenz & Davidson, 1981). The depression or cheerfulness reactions have been hypothesized to result from the loss of reciprocal inhibition between the two hemispheres. More simply stated, cerebral damage resulting from a lesion in one hemisphere is presumed to result in the disinhibition of the uninjured hemisphere (Flor-Henry, 1979; Swartzburg, 1983). This has been referred to as the contralateral release hypothesis, wherein damage to one hemisphere allows the other hemisphere to express its emotional tone unimpeded.

While most lesion studies have dealt with either depression or undue cheerfulness/mania (Coffey, 1987; Cummings & Mendez, 1984; Otto, Yeo, & Dougher, 1987), research with normal participants has addressed emotions in the normal range as well. Davidson and Fox (1982) assessed laterality of emotion by measuring reaction times for the perception of happy and sad faces. Using a tachistoscopic presentation, results show that happy faces presented to the right visual field (left hemisphere) are more readily identified than sad faces. Likewise, sad faces are more quickly perceived than happy faces upon presentation to the left visual field (right hemisphere). These differences support the hypothesized left-positive, right-negative differentiation of emotion. Further evidence for the split between hemispheric emotional valences in these studies was provided by accuracy ratings for the correct identification of either happy or sad faces. Although not reaching the level of statistical significance, accuracy rating results did suggest the same pattern of responding as reaction time. Presentation to the right visual field (left hemisphere) resulted in higher accuracy ratings for the identification of happy faces as compared to sad. Likewise, left visual field (right hemisphere) presentation was associated with higher accuracy ratings for sad faces as compared to happy. Thus, reaction times were lower, and accuracy ratings were higher, for happy faces presented to the right visual field (left hemisphere). Similarly, reaction times were lower, and accuracy ratings were higher, for sad faces presented to the left visual field (right hemisphere).

Reuter-Lorenz and Davidson (1981) reported similar results from another study of tachistoscopically-presented stimuli. Results once again showed that reaction times were faster for the identification of happy faces when they were presented in the right visual field (left hemisphere). Additionally, reaction times were fastest for the identification of sad faces when they were presented in the left visual field (right hemisphere).

Emotional lateralization studies have been carried out using EEG analysis as well. Ahern and Schwartz (1985) reported on a study in which EEG data were collected while participants were asked to respond to emotionally valenced questions. Results indicate that increased left hemisphere activation resulted from positive emotion questions, while right hemisphere activation resulted from negative emotion questions.

The laterality results are not limited solely to adults. Davidson and Fox (1982) used videotape presentations to ten-month-old infants to determine the extent of emotional lateralization at such a young age. As with adults, the infants showed increased left hemisphere activity (as measured by EEG asymmetry) in response to happy faces. While this study supported the left-right dichotomy for positive and negative emotions, it also emphasized the importance of the frontal lobes. EEG recordings were taken from the frontal and parietal regions of the infants' skulls, yet it was only the frontal measures which reliably differentiated between the perception of happy and sad expressions. Therefore, while the left and right hemispheres appear responsible for the perception and expression of positive and negative emotions, respectively, it appears that the frontal lobes, in particular, assess incoming emotional stimuli and produce outgoing emotional responses.

Heller and colleagues (Heller, 1993; Heller, Etienne, & Miller, 1995) propose a theory of emotional lateralization similar to those just discussed. However, while previous researchers have postulated that it is the left hemisphere that processes positive emotion and the right hemisphere that processes negative emotion, Heller's (1993) theory proposes that emotional processing is the result of a relative activation of one hemisphere over the other. Thus, negative affect is not the result of right hemisphere disinhibition, but it is the result of a relatively greater amount of activation for the right hemisphere as compared to the left hemisphere. Similarly, positive affect does not result from unrestricted expression of the left hemisphere, but results from a relative activation of the left hemisphere as compared to the right hemisphere. While previous theories proposed that affective valence is produced in one hemisphere or the other, this model proposes that affective valence is the result of a relative activation of one hemisphere over the other.

Right Hemisphere and Autonomic Arousal

In addition to potential differences in emotional valence, the two cerebrums also seem to differ in their ability to regulate physiological arousal and reactivity to stress. Tucker, Ruth, Arneson, and Buckingham (1977) report greater right than left hemisphere activation during a stressful condition. In this study, participants were asked questions under neutral and stress-inducing conditions. In the neutral condition, participants were merely asked to answer the questions, while under the stressful condition they were told that their responses would be indicative of intellectual ability and personality stability. Results showed an increased frequency of left-lateral eye movements during the stress condition, suggesting greater right hemisphere activation under conditions of arousal.

Flor-Henry (1979) summarized several different lines of research on the relationship between physiological arousal and cerebral lateralization. One example of right hemisphere dominance over physiological arousal is the finding that sleep is strongly related to right hemisphere activation. Heilman and Van Den Abell (1979) have proposed a link between right hemisphere activation and physiological arousal. The evidence, in their view, indicates that the right hemisphere mediates the activation process. Heilman, Schwartz, and Watson (1978), reported that right hemisphere lesions resulted in hypoarousal, as measured by Galvanic Skin Response (GSR), and a left hemi-neglect syndrome. These findings lend support to the dominant role of the right hemisphere in both arousal and attention. Heilman and Van Den Abell (1979) tested the supposition that the right hemisphere dominates activation by presenting a lateralized warning stimulus and observing its effect on the participant's behavior. Warning stimuli presented in the left visual field (right hemisphere) reduced reaction times significantly more than warning stimuli presented in the right visual field (left hemisphere). The hypothesis which emerged from this study was that each hemisphere is capable of controlling its own activation, but that with regard to bilateral activation, the right hemisphere is better able to activate the left hemisphere than vice-versa.

According to this theory, a left-sided lesion would not result in a unilateral neglect, since the right hemisphere appears to be capable of monitoring left and right hemi-space

and activating both cerebrums when necessary. A right hemisphere lesion, on the other hand, could well result in a left-neglect; the left hemisphere appears incapable of monitoring activity in the left hemi-space, as its orientation is directed primarily into the right hemi-space. This theory fits well with clinical data, in which reports of a left-sided neglect are fairly common, whereas reports of a right-sided neglect are rare. Heilman and Van Den Abell (1980) tested this hypothesis empirically, using an EEG activity measure to determine laterality. Results showed that the left parietal lobe desynchronized (increased activation) more to a right-sided stimulus than to a left-sided stimulus, indicating that the left hemisphere is primarily attending to the right hemi-space. In contrast, the right parietal lobe desynchronized equally for both a right or left-sided stimulus. Therefore, the right hemisphere would seem specialized not only for perception and expression of negative emotion but also for the regulation of attention and autonomic arousal.

Davidson (1984) suggested that this right hemisphere specialization for negative emotions, attention, and autonomic arousal fits well with theories of adaptive evolution. According to Davidson, stimuli associated with negative emotions are generally harmful or dangerous and therefore need to be noticed and attended to in order to insure survival. Having both functions localized in the same hemisphere, then, would allow for faster responses to environmental threats.

Along with noticing and classifying potentially threatening stimuli, it is necessary to be able to rapidly prepare for activity once dangerous stimuli are perceived. As cited earlier, there is a relationship between right hemisphere activity and autonomic arousal which allows for this rapid mobilization of resources (Flor-Henry, 1979; Tucker et al., 1977). Further evidence of a relationship is provided by Newlin (1981), who found that right hemisphere-oriented participants (as assessed by lateral eye movements) were found to have significantly higher heart rates compared to their left hemisphere-oriented counterparts. Further links between heart rate and right hemisphere arousal are provided by DePascalis, Alberti, and Pandolfo (1984), as well as by Hatfield, Landers, and Ray (1987). DePascalis et al. (1984) reported a strong correlation between right hemisphere arousal (as measured by EEG) and the ability to perceive heart activity. Participants who scored high on a test of heart activity perception showed significantly greater amounts of right hemisphere EEG activity, as compared to those who scored low on heart activity perception. Hatfield et al. (1987) reported similar heart rate and right hemisphere EEG relationship in a participant pool consisting of elite marksmen. In this study, there was a significant effect of heart rate on right hemisphere EEG activity during the final phase of the marksman's preparatory state.

In addition to results indicating a link between physiological arousal and the right hemisphere, there are also reports of physiological changes as the direct result of manipulations performed at the level of the cerebral cortex. Zamrini et al. (1990) reported on 25 epileptic subjects who underwent intracarotid amobarbital procedures (Wada test) prior to surgical evaluation. Results show that amobarbital introduction in the left internal carotid artery was associated with an increase in heart rate (HR). Conversely, amobarbital injection in the right internal carotid artery was associated with

a decrease in HR. These findings suggest that the two hemispheres have differential access to sympathetic and parasympathetic control of HR. According to this study, amobarbital injection in the right carotid results in a decrease for HR, presumably reflecting a loss of right-hemisphere mediated sympathetic arousal. Similarly, anesthetizing the left-hemisphere results in a cardiac increase, possibly indicating the loss of parasympathetic activation from the left-hemisphere.

Further support for this hypothesized left-parasympathetic, right-sympathetic differentiation for cardiac control is presented by Oppenheimer, et al. (1992). Whereas Zamrini et al. (1990) showed cardiac effects through anesthesia, Oppenheimer et al. (1992) obtained their results through cortical stimulation. Electrical stimulation of the left insular cortex reliably elicited bradycardia and depressor (diastolic blood pressure) effects, whereas stimulation of the right insular cortex was more likely to result in tachycardia and pressor (diastolic blood pressure) responses. Similar to Zamrini et al. (1990), this study supports a pronounced sympathetic nervous system effect associated with right hemisphere activation, and a parasympathetic effect from the left-hemisphere.

Hostility

The valence and right hemisphere theories of emotional lateralization discussed previously propose quite different mechanisms for the perception and production of affect. However, they overlap in the realm of negative affect, with both theories positing a dominant role for the right hemisphere in the processing and production of negative affect. Therefore, regardless of which theory of emotional lateralization that is proposed to be in effect, negative affect will be seen as a right hemisphere event.

Originally, hostility was studied as a subcomponent of the TABP (Friedman & Rosenman, 1974). It was seen as one aspect of the TABP that contributed to the development of coronary heart disease. It has now become the main focus of research seeking to determine the affective determinants of cardiovascular disease. Smith (1994) discusses five possible routes by which hostility could lead to coronary heart disease. The five proposed models are psychophysiological reactivity, psychosocial vulnerability, transactional, biological vulnerability, and correlates of health behavior.

The first model linking hostility and cardiovascular disease is the psychophysiological reactivity model (Smith, 1994). According to this model, hostile persons are more likely to be vigilant for possible conflicts in their environment, and are more likely to respond in a physiologically exaggerated style to these stressors. Cardiovascular disease is thought to appear early in these individuals, as they 'burn out' from their chronic and exaggerated response style. Suarez and Williams (1990) reported that high hostile men (as determined by the Cook-Medley Hostility Scale) experienced greater physiological reactivity (as measured by heart rate, blood pressure and blood flow) during an anagram task accompanied by harassment than did low hostile men. Additionally, high hostile men exhibited the slowest recovery for blood flow following the harassment procedure. These results are indicative of both increased reactivity in general as well as increased resistance to recovery (perseveration).

Ganster, Schaubroeck, Sime, and Mayes (1991) report the same patterns of physiological responding. Specifically, individuals rated as high in hostility (as measured by the hostility subcomponent of the Structured Interview) were found to more physiologically reactive (blood pressure, heart rate, skin temperature, and electrodermal responding) to the Structured Interview and the Stroop Color-Word Conflict Task than were participants rated as lower in hostility. Additionally, the high hostile individuals were also found to persist in their physiological responding for longer periods of time than did the low hostile individuals. Similar findings of persistence on physiological measures have been reported from our own lab (Herridge, Harrison & Demaree, 1997). In this study, bilateral habituation for GSR was seen in low hostile men after participants posed angry faces. In contrast, high hostile men showed habituation only at the right hand, while the left hand continued to show the higher GSR rates associated with posing an angry face. This perseveration of negative affect and continued physiological responding is indicative of right cerebral dysfunction.

Another proposed mechanism linking hostility and coronary heart disease is the psychosocial vulnerability model (Smith, 1994). According to this model, individuals high in hostility would lack adequate social support while at the same time encountering increased levels of interpersonal conflicts. For this model to work, though, it must have some mechanism similar to that proposed in the psychophysiological reactivity model (i.e. exaggerated response style to stressors). Smith and Frohm (1985) report that individuals scoring high in hostility (as measured by the Cook-Medley Hostility Scale) displayed more anger, endorsed more frequent and more severe hassles, and reported fewer and less satisfactory social supports, as compared to individuals scoring lower in hostility.

The transactional model (Smith, 1994) linking hostility and cardiovascular disease is actually an integration of the psychophysiological reactivity and psychosocial vulnerability models. According to the transactional model, hostile individuals are more reactive to stressful situations and they are also more likely to encounter them based on their specific style of responding. High hostile individuals would be predicted to be more reactive physiologically to a stressful event, and they would be more likely to encounter such an event due to their reported increase in frequency and severity of hassles. Along these lines of eliciting negative interactions within their environments, Harrison, Gorelczenko, and Cook (1990) report that high hostile participants are more likely to rate neutral faces as angry than are low hostiles. These results are suggestive of a negative attributional style, which will result in increased exposure to negative interactions based on the hostile person's assumptions regarding the affective tone of other people.

The transactional model can be further clarified by looking at specific subtypes of hostility. Felsten & Leitten (1993) report that individuals scoring high in hostility (as measured by the Buss-Durkee Hostility Inventory) showed greater physiological reactivity (blood pressure and heart rate) in response to harassment than did individuals scoring low in hostility. However, this effect only held for those subjects rated as high in expressive hostility. Participants who scored high in neurotic hostility did not show the

same pattern of elevated physiological responding in the presence of an interpersonal stressor. Thus, some individuals seem to be predisposed to evidence enhanced physiological responding, but the appropriate environmental stimuli must be present to elicit the response.

The fourth mechanism proposed to result in cardiovascular disease is the constitutional vulnerability model (Smith, 1994). According to this model, hostility and eventual cardiovascular disease are not causally related, but they are both the result of a common biological factor (i.e. overly responsive sympathetic nervous system). It is important to remember that these models are not mutually exclusive of one another. For example, within the constitutional vulnerability model, there would need to be some mismatch between the individual's biological make-up and their environment. There must be the environmental stressor and the biological diathesis necessary for dysfunction to result from the combination of the two.

The final mechanism linking hostility and cardiovascular disease is the health behavior model (Smith, 1994). The important elements in this model are the health behavior correlates of hostility. Leiker & Hailey (1988) report that in comparison to low hostile individuals, participants scoring high in hostility had worse health habits overall. Additionally, high hostile individuals scored significantly lower on three of the four subscales used to measure health related behaviors (physical fitness, self-care, and drugs and driving). Thus, high hostile individuals may be at greater risk for developing cardiovascular disease simply as a result of their health related behaviors. Cardiovascular disease would not necessarily result from an exaggerated physiological response style, but could result from inadequacies in health-care demonstrated by high hostile individuals.

The preceding review presents a fairly cohesive picture of the hostility literature, yet there are reports that point out flaws with the construct. Previously, it was reported that there was a significant relationship between hostility and CVR, but only for individuals scoring high in expressive hostility as opposed to neurotic hostility (Felsten & Leitten, 1993). This finding is repeated in a meta-analysis of 28 articles focused on hostility and CVR (Suls & Wan, 1993). The meta-analysis supports the finding of greater physiological reactivity (increased SBP) to provocative stressors for individuals scoring high in antagonistic hostility as compared to individuals who score high on measures of neurotic hostility. This analysis also indicates little evidence for group (high-hostile versus low-hostile) differences in CVR when the groups are formed from conventional hostility inventories.

Myrtek (1995) provides another meta-analysis of the literature. This analysis reports a significant relationship between the TABP personality construct and increased CVR. However, this relationship is not as clear as it might seem. The results of previous studies that made up the sample for the meta-analysis reveal that the research studies published prior to 1983 show a very robust relationship between TABP and increased SBP. This relationship is still observed in studies published after 1983, but the relationship is much weaker. Myrtek (1995) proposes that this decline in the relationship between TABP and CVR may be the result of publication bias, with failures to replicate

and negative findings being more readily accepted for publication. Along with the review of the relationship between TABP and CVR, Myrtek (1995) also includes a brief analysis of hostility and CVR. This analysis reveals a significant effect of hostility on systolic blood pressure reactivity, but shows no relationship between hostility and either heart rate or diastolic blood pressure.

The lack of consistent findings, including contradictory ones, could be due to the nature of the hostility construct. In fact, this has already been alluded to, with a differentiation of neurotic versus expressive hostility (Felsten & Leitten, 1993). Perhaps, then, clarification within the field could come from better operationalizing the construct of hostility. Both Thoresen and Powell (1992) and Steinberg and Jorgensen (1996) point out the multidimensional nature of hostility, and the need for multimodal measures to be able to better define the construct under investigation.

Neuropsychological Model of Hostility

Neuropsychological theories point to very specific neuroanatomical sites as the basis for hostility. Heilman, Bowers & Valenstein (1993) discuss the extensive interconnections between the right frontal and right temporal regions, with the understanding that the frontal region is thought to be inhibitory over the temporal. Additionally, the temporal region is proposed to be the origin of hostile/aggressive behavior. This model is supported by work done with non-human primates. Butters (1970) reports that stimulation of the right frontal region produces a placid animal. Conversely, (Ursin, 1960) reports that stimulation of the right temporal lobe results in an aggressive, rage response from the animal. Similar to Butters (1970) who produced a placid animal through stimulation of the right frontal region, Woods (1956) reports that a placid animal results from deactivation (ablation) of the right temporal region. Therefore, aggressive behavior can be increased by either deactivation of the right frontal region or by activation of the right temporal. Clinically, this was reported in a woman with self-described 'panic attacks' (Rhodes, Everhart, & Harrison, 1997). This woman showed relative symmetry across cortical regions during the baseline recording period of a quantitative EEG evaluation. However, following a negative emotional mood induction there was a marked increase for Delta activity (indicative of deactivation) at the right frontal (F8) region with a substantial increase for Beta activity (indicative of activation) at the right temporal (T4) region.

This neuropsychological model of hostility, when combined with the evidence linking the right hemisphere to increased physiological arousal, is compatible with the five mechanisms linking hostility and CHD previously discussed (Smith, 1994). Specifically, it can account for the psychophysiological reactivity, transactional, and constitutional vulnerability models which all require an underlying physiological mechanism. The psychosocial vulnerability model would fit with the preceding three, in that it requires an increase in sympathetic nervous system activity. Finally, the health behavior theory linking hostility and CHD could be accounted for by the neuropsychological model, if it is assumed that high hostile individuals engage in smoking or drinking as a means of reducing their sympathetic nervous system activity.

Thus, these individuals are chronically aroused and are seeking ways to reduce their sympathetic nervous system arousal.

Rationale

As suggested by previous research, hostility and autonomic arousal appear to be mediated by the right hemisphere. Therefore, exposure of high hostile individuals to a cold pressor stressor would be expected to result in increased physiological reactivity, as measured using cardiovascular indices (BP and HR), and increased right hemisphere activation as measured by a dichotic listening task. However, while we know that physiological arousal is maintained across time in high hostile individuals in which negative emotional states have been induced (e.g. Herridge, Harrison & Demaree, 1997), it remains unclear what effect these emotions have on other measures of laterality across time. In addition, the magnitude of reactivity and the resistance to recovery in reaction to stress in these individuals has not been explored across time. Therefore, the current study will build on existing knowledge by examining the magnitude and persistence of laterality effects in high hostile individuals exposed to stressful situations.

Variables

High and low hostile groups will be established using self-reported hostility level as reported on the Cook-Medley Hostility Scale (Cook & Medley, 1954).

Two dependent variable categories will be used in this experiment. First, physiological indicators will be SBP, DBP, and HR. The second dependent variable category will be a measure of lateralized brain activity as assessed by a dichotic listening paradigm using concurrently voiced consonant vowels (CV) (ba, da, ga, ka, pa, ta).

Hypotheses

Hypothesis 1: High hostile participants will show greater physiological (SBP, DBP, HR) reactivity to stress as compared to low hostiles.

Hypothesis 2: Following exposure to the stressor, high hostile individuals, as compared to low hostiles, will show increased right hemisphere arousal, indicated by an increased left ear advantage.

Hypothesis 3: Following confirmation of Hypothesis 2, high hostile participants, as compared to low hostiles, are expected to require extended time periods to return to prestress levels as evidenced by persistence of the left ear advantage and increased physiological arousal during the recovery period.

Method

Participants

Participants consisted of 25 right-handed, undergraduate men scoring low in self-reported hostility, and 25 right-handed, undergraduate men scoring high in self-reported hostility. All participants were acquired from the undergraduate Psychology pool. Participants had to have self-reported no history of hearing aids, hearing problems (i.e. infections or tubes in ears), major illness or head injury. Due to the relatively heightened cerebral lateralization among men, only males were used to ensure as much homogeneity

as possible within the experiment to draw conclusions based solely on independent variable differences. All participants received course credit for their participation. All identifying materials collected from participants were destroyed after data collection.

Participants had to have reported no remarkable medical history to be eligible for inclusion in the experiment. Participants had to report sufficient right hemibody preference based on the Coren, Porac, & Duncan laterality test (+7 or above). Additionally, for inclusion in the study, participants had to report scores falling in the top/bottom third of the Cook-Medley Hostility Scale distribution for all possible participants screened for this experiment.

Self-Report

During group testing, participants were first required to read and sign an informed-consent form. A questionnaire assessing medical history was also given. Participants were then administered the Coren, Porac, and Duncan laterality test (CPD; Coren, Porac, & Duncan, 1979) to determine hemibody preference. This self-report assesses right (+1) and left (-1) hemibody preference based on reported preferred use of either eye, ear, arm, and leg. Scores on the test range from a possible -13 to +13, indicating extreme left and right "handedness", respectively. A score of +7 was required for further participation in the experiment.

Participants were then administered the Cook-Medley Hostility Scale (CMHS) (Cook & Medley, 1954). The Cook-Medley is the most often used measure of hostility and shows construct validity as a predictor of interpersonal, medical, and psychological outcomes (Conrada & Jussim, 1992). Participants had to report hostility levels in the either the top or bottom third of this specific sample's distribution for inclusion in the experiment. For this sample, the bottom and top thirds of the Cook-Medley Hostility Scale distribution were 0-19 and 29-50, respectively.

Participants also completed the Beck Depression Inventory (BDI; Beck, 1972) and the Self-Evaluation Questionnaire (STAI; Spielberger, 1983). Neither the BDI nor the STAI were used as a criterion measure for inclusion in this study.

Apparatus

The laboratory chamber was comprised of a chair facing into a flat white curtain enclosure. Located in this chamber was the cold pressor equipment. Dichotic listening and physiological recording equipment were located in an adjacent room.

Physiological

SBP, DBP, and HR were assessed using the Industrial and Biomedical Sensors Corporation pulse/pressure machine (model SD 700A) with automatic print-out. SBP and DBP will be measured by obtaining Korotkoff sounds at the right arm. Accuracy of BP is estimated to be ± 3 mm Hg.

Hearing

Auditory acuity was assessed using the Qualitone Acoustic Appraiser (Model WR-C) and lightweight portable Qualitone TD-39 headphones.

Perceptual

The tape containing the dichotic stimuli was 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). Stimuli were presented at about 75 dB by a Marantz dual channel tape player using Koss Pro/4x Plus headphones. The interstimulus interval was 6 seconds. The six CVs were presented as 3.5 cm, black, upper-case letters on a 96 x 144 mm index card displayed approximately .5 m in front of the participant.

Cold Pressor

The ice water for the CPT was maintained in a small ice cooler at 4 (\pm 1) degrees Celsius. Water temperature was measured using a standard mercury thermometer.

Procedure

High and low hostility participants were scheduled for further participation in the experiment within one week of their group screening session. Participants were requested to read and sign another informed consent form upon entering the laboratory chamber. Participants also completed another CMHS, to be scored afterward, to ensure stability of hostility scores. Auditory acuity was then assessed by a pure-tone test using the Qualitone Acoustic Appraiser and lightweight portable Qualitone TD-39 headphones. To continue in the study, participants had to correctly identify ten of twelve two-syllable words presented individually to each ear at 20 decibels.

The experiment consisted of five parts -- Prestress, Stress, Recovery 1, Recovery 2, and Recovery 3 phases.

Prestress Phase. Participants were fitted for BP and HR readings. The blood pressure monitor was strapped to each participant's right upper-arm. Headphones were also placed appropriately, after explaining that any questions the participant might have would be answered via a two-way intercom. All procedures and instructions for the experiment were standardized on audio-tape and presented over the headphones. All data were recorded.

A brief training phase introduced the dichotic listening procedures. The participant was presented all six phonemes over the headphones, with the requirement that they repeat the phoneme after it has been presented. The experimenter provided corrective feedback for any phonemes incorrectly identified. Next, the participant was presented with five of the phonemes. The participant was instructed to point to the phoneme that they heard. Participants had to identify four of the five phonemes correctly in order to continue in the experiment.

Following the dichotic listening training procedures, participants were given the following instructions: "Your blood pressure will now be measured. Please sit still in the chair with your feet flat on the floor." Heart rate (HR), systolic blood pressure (SBP),

and diastolic blood pressure (DBP) data were then collected twice in succession. To determine the accuracy of the reading, a third reading was taken if the first two readings differed by 6 beats per minute (HR) or 20 mm Hg.

Participants were then told:

You are about to hear thirty trials of syllables. You will hear a syllable in one ear and another syllable in the opposite ear, and it will sound like two people talking to you at the same time. Your job is to listen very carefully and point to the syllable on the chart that you hear most clearly.

All responses were recorded.

Stress Phase: Participants were then 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. You have the option to withdraw your hand at any time, but we prefer that you do not. 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? Ready, begin.

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

Poststress Phase: The participants were then given the following instructions: “Your blood pressure will now be measured. Please sit still in the chair, and keep your feet flat on the floor.” HR, SBP, and DBP were then assessed and recorded following the procedures in the Prestress Phase.

The dichotic listening procedure was then performed three consecutive times according to the procedures outlined in the Prestress Phase. Total time for the three consecutive dichotic listening trials was nine minutes. At the end of the consecutive dichotic trials, the participant’s blood pressure and heart rate were once again recorded.

Analyses

T-tests were conducted to assess differences between high- and low-hostile participants on the descriptive measures -- CMHS, BDI, STAI, and the CPD laterality questionnaire.

Multivariate analysis of variance (MANOVA) was performed on the physiological variables (HR, SBP, DBP) to assess reactivity and recovery in response to the cold pressor test: GROUP2 X (CONDITION3).

For each participant, the Percentage of Correct responses (POC) index was calculated for hearing accuracy during the dichotic listening paradigm using the following formula:

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

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). Independent ANOVAs were conducted on dichotic listening variables -- POC, and number of correctly identified stimuli presented to both the left and right ear: GROUP2 X (TRIAL4).

RESULTS

Descriptive Measures

To compare groups (low- and high-hostiles) on descriptive measures taken during initial screenings, t-tests were conducted on scores from the Cook-Medley Hostility Scale (CMHS), Beck Depression Inventory (BDI), State-Trait Anxiety Inventory (STAI), and the Coren, Porac, and Duncan Laterality Questionnaire. Table 1 provides a summary of group means and standard deviations for each measure.

High-hostiles scored significantly higher than low-hostiles on all descriptive measures except for the Coren, Porac, and Duncan Laterality Questionnaire. High-hostiles scored significantly higher on the CMHS ($M=33.28$, $SD=3.48$) than did low-hostiles ($M=13.28$, $SD=4.67$), $t(48)=17.15$, $p<.05$. On the BDI, high-hostiles ($M=9.0$, $SD=4.52$) evidenced significantly higher depression scores than the low-hostiles ($M=2.64$, $SD=2.75$), $t(48)=6.00$, $p<.05$. Likewise, anxiety scores from the STAI for both State and Trait measures were significantly higher for high-hostiles as compared to low-hostiles. STAI-State scores were significantly higher for high-hostiles ($M=40.00$, $SD=12.50$) than low-hostiles ($M=29.88$, $SD=5.28$), $t(48)=3.72$, $p<.05$. Similarly, high-hostiles ($M=44.20$, $SD=8.66$) evidenced significantly higher STAI-Trait scores as compared to low-hostiles ($M=30.20$, $SD=6.02$), $t(48)=6.63$, $p<.05$.

In contrast to all other descriptive measures, and in accordance with the inclusionary criteria, only the Coren, Porac, and Duncan Laterality Questionnaire failed to show a significant difference between the groups. On this questionnaire, high-hostiles ($M=9.96$, $SD=2.44$) were not found to be significantly different from low-hostiles ($M=10.40$, $SD=2.51$), $t(48)=.6275$, $p>.05$.

Dichotic Listening Data

Independent analyses of variance (ANOVAs) were performed on the three dependent variable measures obtained during the dichotic listening procedure. Specifically, ANOVAs were performed for the percent of correct responses (POC), the number of correctly identified stimuli at the left ear, and the number of correctly identified stimuli at the right ear. POC scores were analyzed to assess for general trends in ear dominance, with analyses for stimuli detected at each ear providing a more specific measure of altered cerebral activation. Group means and standard deviations of POC scores are presented in Table 2, and the independent ANOVA results are depicted in Table 3.

Table 1. Means and Standard Deviations for the Cook-Medley Hostility Scale, Beck Depression Inventory, State-Trait Anxiety Inventory, and Coren, Porac, and Duncan Laterality Questionnaire.

Questionnaire	High-Hostile		Low-Hostile	
	MEAN	SD	MEAN	SD
CMHS	13.28	4.67	33.28	3.48
BDI	2.64	2.75	9.00	4.52
STAI - State	29.88	5.29	40.00	12.50
STAI - Trait	30.20	6.03	44.20	8.66
CPD	10.40	2.51	9.96	2.44

Table 2. Means and SD of POC scores by Group and Trial.

Trial		Mean	Low-Hostile SD	High-Hostile Mean
Baseline	.231	.227	.170	.261
Recovery 1	.212	.289	.231	.237
Recovery 2	.244	.303	.206	.218
Recovery 3	.247	.308	.235	.273

Table 3. Independent ANOVA Results for POC Scores, and Number of Correctly Identified Stimuli at the Left and Right ears.

Source	df	SS	MS	F	p
POC Scores					
Group	(1,48)	0.0267	0.0267	0.11	0.7399
Trial	(3,144)	0.0426	0.0142	0.94	0.4226
Group X Trial	(3,144)	0.0441	0.0147	0.97	0.4078
Left Ear					
Group	(1,48)	8.8200	8.8200	0.19	0.6615
Trial	(3,144)	6.3000	2.1000	0.64	0.5915
Group X Trial	(3,144)	10.9400	3.6467	1.11	0.3478
Right Ear					
Group	(1,48)	5.4450	5.4450	0.10	0.7517
Trial	(3,144)	18.4950	6.1650	1.75	0.1587
Group X Trial	(3,144)	7.0150	2.3383	0.67	0.5748

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 (baseline, recovery 1, recovery 2, recovery3). Neither main effect, nor the interaction between the two approached significance.

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 3). Group means and standard deviations of left-ear syllable identification are presented in Table 4. Neither main effect, nor the interaction between the two approached significance.

To analyze the number of stimuli correctly identified by the right ear (indicative of heightened left cerebral arousal), a two-factor, mixed design analysis of variance (ANOVA) was performed with a fixed factor of group and a repeated measure of trial (see Table 3). Group means and standard deviations of right-ear syllable identification are presented in Table 5. Neither main effect, nor the interaction between the two approached significance.

Physiological Data

Group means and standard deviations of physiological measures (SBP, DBP, and HR) are displayed in Table 6. Multivariate analysis of variance (MANOVA) was performed on

Table 4. Means and SD of Stimuli Correctly Identified by Left Ear by Group and Trial.

Trial		Low-Hostile		High-Hostile	
		Mean	SD	Mean	SD
Baseline	10.56	3.21	11.48	3.85	
Recovery 1	10.80	3.96	10.52	3.17	
Recovery 2	10.32	3.92	11.08	3.08	
Recovery 3	10.40	4.34	10.68	3.97	

Table 5. Means and SD of Stimuli Correctly Identified by Right Ear by Group and Trial.

Trial	Mean	Low-Hostile		High-Hostile		
		Mean	SD	Mean	SD	
Baseline	16.92	3.32	16.08	3.55		
Recovery 1	16.76	4.41	16.96	3.61		
Recovery 2	17.32	4.77	16.88	3.29		
Recovery 3	17.44	4.84	17.20	3.89		

Table 6. Means and SD of Physiological Variables by Group and Trial.

	SD	Low-Hostile		High-Hostile
		Mean	SD	Mean
Prestress				
SBP	121.40	9.88	115.14	10.05
DBP	72.14	7.15	71.52	8.87
HR	73.04	14.10	72.82	10.51
Poststress				
SBP	125.46	10.20	116.48	9.94
DBP	73.04	8.88	73.26	10.16
HR	70.84	10.49	75.08	11.06
Recovery				
SBP	118.76	10.19	112.70	9.89
DBP	72.34	8.22	71.24	8.18
HR	73.18	11.44	74.30	10.70

the three physiological variables (SBP, DBP, HR) for reactivity to the cold pressor. All pairwise comparisons were made using Tukey's Studentized Range Test. For this MANOVA, the overall effect of group was not significant (Hotellings $F(3,46)=2.413$, $p<.1383$).

To increase reliability of the MANOVA findings and to examine any significant physiological variable differences, independent analyses of variance (ANOVAs) were performed for each physiological measure. All post hoc, pairwise comparisons of the means were made using Tukey's Studentized Range Test. Table 7 provides an overview of the independent ANOVA results for the physiological measures.

For SBP, there was a significant effect for group, $F(1, 48) = 7.13$, $p < .05$. Specifically, the low-hostile individuals had higher SBP as compared to the high-hostile individuals. The effect of Trial was also significant, $F(2, 96) = 18.61$, $p < .05$. Systolic blood pressure was higher following exposure to the cold pressor (see Figure 1). The group x trial interaction was not significant. For DBP, neither main effect (Group or Trial), nor the interaction between the two was significant. For HR, neither main effect (Group or Trial) was significant. However, the interaction of Group x Trial was significant, $F(2, 96) = 3.32$, $p < .05$. Specifically, HR differed significantly following exposure to the cold pressor (see Figure 2).

As it was discovered after data collection ended that the groups differed on more than just hostility (i.e. depression and anxiety), the results were reanalyzed using the depression and anxiety scores as covariates. This re-analysis of the data did not reveal any differences between the groups as a function of either depression or anxiety.

Table 7. Summary of the Independent ANOVA Results for Physiological Variables.

Source	df	SS	MS	F	p
SBP					
Group	(1,48)	1890.375	1890.375	7.13	0.0103
Trial	(2,96)	686.653	343.327	18.61	0.0001
Group X Trial	(2,96)	66.520	33.260	1.80	0.1704
DBP					
Group	(1,48)	9.375	9.375	0.05	0.8283
Trial	(2,96)	59.893	29.947	2.28	0.1082
Group X Trial	(2,96)	11.160	5.580	0.42	0.6555
HR					
Group	(1,48)	110.082	110.082	0.31	0.5799
Trial	(2,96)	21.090	10.545	0.54	0.5874
Group X Trial	(2,96)	130.923	65.462	3.32	0.0403

Figure Caption

Figure 1. Systolic Blood Pressure X Group X Trial.

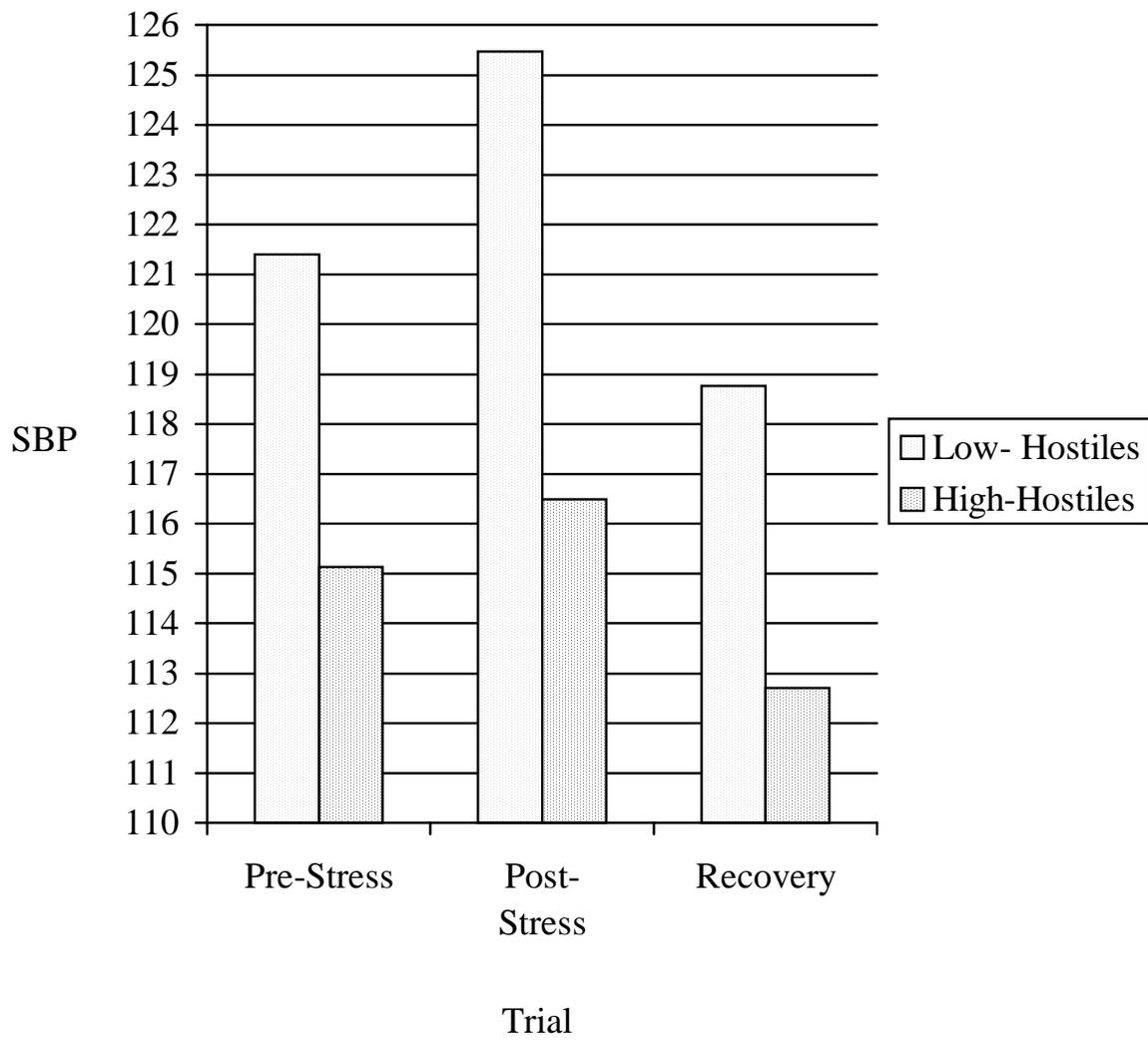
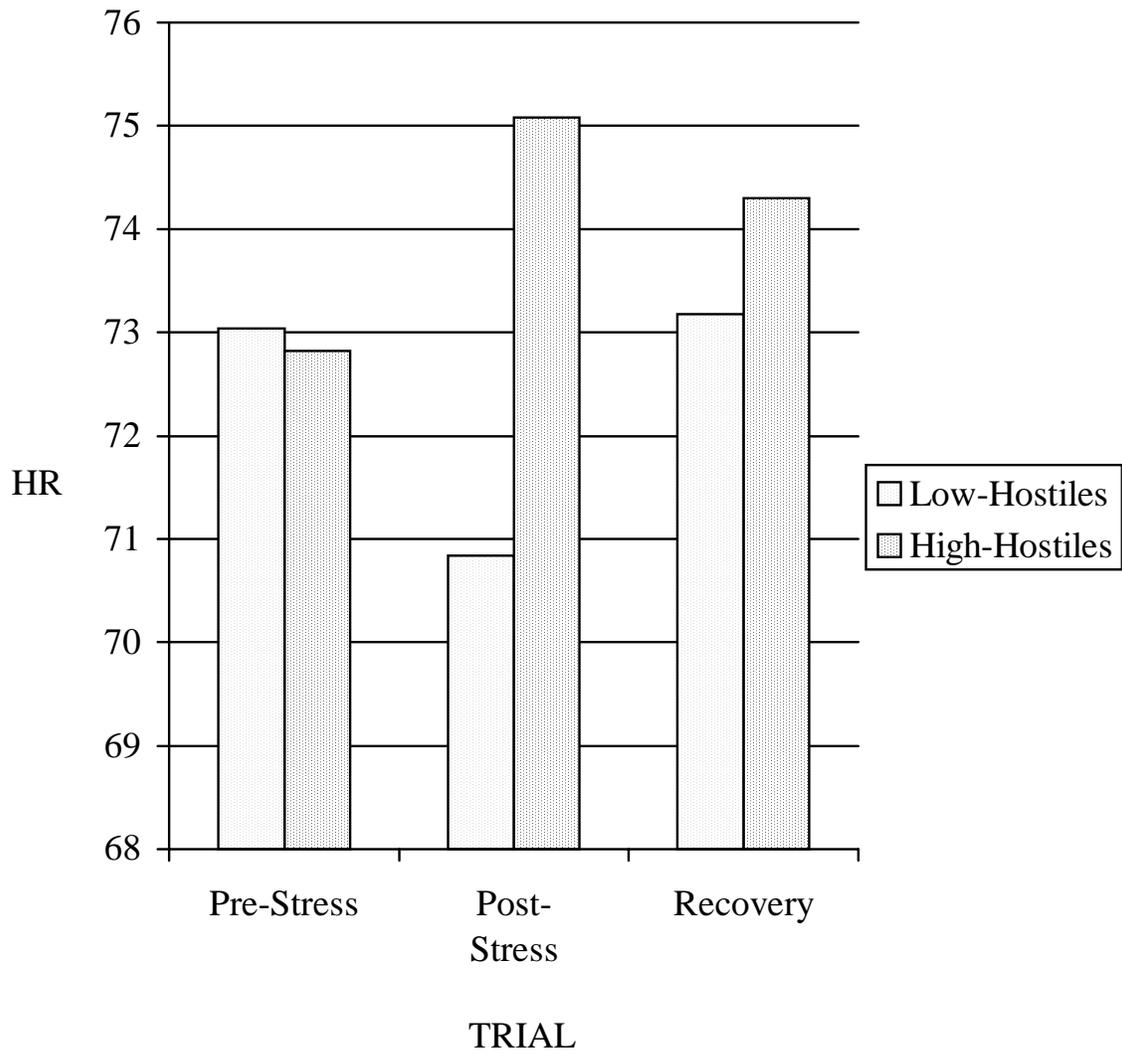


Figure Caption

Figure 2. Heart Rate X Group X Trial.



Discussion

This experiment was an attempt to clarify the relationships between hostility, physiological reactivity, and cerebral laterality. To accomplish this, a specific population of participants (high-hostiles) were chosen due to proposed cerebral activation pattern (Heilman, Bowers, & Valenstein, 1993). Specifically, high-hostiles are proposed to show decreased right frontal activation, with subsequent increased right temporal activation. The disinhibition of the right anterior temporal region would then allow for increased negative affect expression reported in the literature following cortical stimulation of the right temporal region (Ursin, 1960).

If the hostility reported by participants is due to increased right temporal activation, then it should be possible to detect pre-stress differences between groups on the physiological variables. This would support Oppenheimer, et al. (1992) who reported increased HR and BP following stimulation of the right insular cortex. If hostility is the result of right temporal activation, then these individuals would be displaying patterns of cerebral activation similar to that induced by Oppenheimer, et al. (1992). Not only is it possible that there would be baseline physiological differences between the groups, but these differences should become even more pronounced following exposure to the cold pressor at the left hand.

Along with the physiological measures, increases in right temporal activation should also be evident on the dichotic listening task. Once again, if hostility does result from increased activation of the right temporal region, then it would be theoretically possible to detect an increase in auditory perception at the left ear for the high-hostiles. Normally, individuals show a right ear (left hemisphere) advantage for processing linguistic stimuli (Geffen, 1978), but if the right temporal lobe is overly activated in hostile individuals, then it may be possible that they would show an increased ability to attend to their left ear (right hemisphere). Just as with the physiological measures, this effect should become more pronounced following left hand (right brain) exposure to the cold pressor. This relative increase in left ear perception has been demonstrated by Demaree and Harrison (1997). They report an increased ability to perceive word-sounds at the left ear following left arm exposure to the cold pressor.

The first hypothesis was that high-hostile individuals would show greater physiological reactivity to stress than would low-hostiles. This hypothesis was partially confirmed. For HR, high-hostiles did show a significant reaction to the cold pressor. Specifically, following exposure of the left arm to the cold pressor, their HR increased significantly from their pre-stress levels. Low-hostiles also showed a significant response to the cold pressor. However, the low-hostiles showed a significant decrease in HR following left arm exposure to the cold pressor. High-hostiles, then, do support the hypothesized link between the right hemisphere and the sympathetic nervous system (Oppenheimer et al., 1992; Zamrini et al., 1990). Interestingly, low-hostiles do not support this relationship, as they show decreased physiological arousal in response to the cold pressor.

Hypothesis 2 was related to the dichotic listening paradigm. This hypothesis predicted an increase in right hemisphere arousal, indicated by a relative increase in left

ear advantage (LEA), for high-hostiles as compared to low-hostile individuals. The POC score was analyzed for confirmation of this hypothesis. POC scores are purported to be a general measure of ear dominance, and would be hypothesized to reflect a shift toward the left ear following the stressor. Following exposure to the stressor, however, the POC scores for the two groups were not significantly different.

The third hypothesis was related to both physiological and neuropsychological variables. Physiologically, it was predicted that high-hostiles would show persistence of their arousal by taking longer to return to baseline levels than would the low-hostiles. For the SBP data, this hypothesis was not confirmed, as both groups had fallen below baseline levels for SBP at the final measurement. HR data also failed to support this hypothesis, as the groups were not significantly different from each other during the final measurement, nor were they significantly different from their own pre-stress levels.

Neuropsychologically, the third hypothesis predicted that the proposed relative increase in left ear perception for the high-hostiles following a stressor would persist over time. This would be indicated by an enduring decrease in the POC score over time for the high-hostiles as compared to the low-hostiles. Alternatively, it could be indicated by an increase in the number of stimuli detected at the left ear which persisted across dichotic listening trials for the high-hostiles when compared to the low-hostiles. POC scores were not supportive of this hypothesis, as there were no differences between groups across the consecutive dichotic listening procedures.

In summary, it was predicted that there would be increased right hemisphere activation in high-hostile individuals, as compared to low-hostile individuals, following exposure to a stressor. Moreover, this hypothesized increase in right-hemisphere activation was predicted to persist over time, and was predicted to be quantifiable through both physiological and neuropsychological measures. The only supportive evidence for the a priori predictions is related to the interaction effect of group and trial for the HR data. Specifically, high-hostile individuals showed a significant increase in HR following left arm exposure to the cold pressor.

Given the limited support for the a priori predictions, the focus now turns to possible reasons why these results occurred. One explanation could be that the groups differed on more than just levels of self-reported hostility (i.e. anxiety and depression). If orbitofrontal regions are truly inhibitory over temporal regions (Heilman, Bowers & Valenstein, 1993), and if left temporal is associated with parasympathetic nervous system arousal (Oppenheimer et al., 1992; Zamrini et al., 1990), then left frontal deactivation proposed to occur with depression (Henriques & Davidson, 1991) would result in increased parasympathetic arousal. This increased parasympathetic nervous system activation would result in decreased physiological responding and could account for the aberrant physiological differences between groups (low-hostiles with consistently higher SBP than high-hostiles). However nicely this explanation fits with current models of emotion and autonomic nervous system activation, it is not supported by the data. When differences in levels of depression between groups is controlled for in the analyses, there are no additional statistically significant differences between groups. Similar arguments apply for the differences between groups in levels of anxiety. If these

differences are controlled for statistically, there are no further significant differences between groups.

According to Heller (1993), the increased levels of depression and anxiety seen in the high-hostile group should serve to negate one another. Heller's (1993) proposes a relative increase in right frontal activation with both anxiety and depression. However, the posterior systems that she proposes for the regulation of autonomic arousal are diametrically opposite for these two affective states. Cerebral patterns for depression should include a relative right frontal increase in activation, coupled with decreased activation of the right parietotemporal areas. Anxiety, on the other hand, would be predicted to show the same relative right frontal activation, but this time there would be increased right parietotemporal activation. Regardless of whether or not the two states do negate one another, if they are controlled for statistically there is no effect attributable to them.

If the current findings are looked at in terms of the literature reviewed previously, then they are less surprising. Two meta-analyses (Myrtek, 1995; Suls & Wan, 1993) discussed earlier showed limited support for the physiological reactivity proposed to have occurred in the current group of high-hostile individuals. They report weak, null, and even contradictory findings for studies investigating the links between personal attributes and physiological responding. Suls and Wan (1993) show some support for a relationship between hostility and physiological responding. However, this link only applies for expressive hostility, not neurotic. As the Cook-Medley Hostility Scale has been described as a measure of neurotic or cynical hostility (Pope, Smith & Rhodewalt, 1990; Smith & Frohm, 1985; Smith, Sanders & Alexander, 1990), then it may not be ideally suited as a predictor of increased physiological responding due to hostility.

While limited support for increased physiological reactivity as a function of self-reported hostility levels on the Cook-Medley Hostility Scale would be predicted from the Suls and Wan (1993) meta-analysis, Myrtek (1995) would support similar outcomes. However, where Suls and Wan (1993) would predict a lack of findings based on the measurement tool used, Myrtek (1995) would predict a lack of findings based on the current state of the field. Myrtek (1995) reports that results supporting the link between personality and cardiovascular reactivity have been declining since approximately 1983. His interpretation of this decline is that it is the result of a publication bias on the part of authors and journals, with null or contradictory findings only making their way into the literature since the mid-1980s.

Based on the literature reviewed and the current findings, some recommendations are made for future research. First, given that the focus has narrowed to the deleterious effects of hostility on health, it is recommended that hostility is better operationalized. Hostility has emerged from the original Type-A Behavior Pattern (Friedman & Rosenman, 1974) research has the key element leading to coronary heart disease. However, even with this seemingly straightforward construct, it is possible to further specify what is meant by hostility. It is by no means a unitary construct (Pope, Smith & Rhodewalt, 1990; Smith & Frohm, 1985; Smith, Sanders & Alexander, 1990). Second, once the definition of hostility has been clearly delineated, it will be necessary to employ

the correct stressor. There are many types of manipulations reported in the literature, but interpersonal conflict seems to be a good predictor of increased physiological arousal in hostile individuals (Felsten & Leitten, 1993; Siegman, Anderson, Herbst, Boyle & Wilkinson, 1992). Thus, hostility assessment should be multimodal (Steinberg & Jorgensen, 1996; Thoresen & Powell, 1992) with a focus on expressive hostility (Pope, Smith & Rhodewalt, 1990; Smith & Frohm, 1985; Smith, Sanders & Alexander, 1990), and should involve an interpersonal challenge (Felsten & Leitten, 1993; Siegman, Anderson, Herbst, Boyle & Wilkinson, 1992).

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EDUCATION

B.S. Virginia Polytechnic Institute and State
University, Blacksburg, Virginia (1992)
Major field of study: Psychology

M.A. Hollins College, Hollins, Virginia (1993)
Major field of study: Psychology (General)

Thesis Title: Relationship between body
temperature and cognitive ability in performance
and verbal tasks in a brain injured adult male.
Major advisor: George W. Ledger, Ph.D.

M.S. Virginia Polytechnic Institute and State
University, Blacksburg, Virginia (1998)
Major field of study: Clinical Psychology/
Neuropsychology

Thesis Title: Reactivity and recovery of
physiologic and neuropsychological variables as
a function of hostility.
Major advisor: David W. Harrison, Ph.D.

APPOINTMENTS AND POSITIONS

8/1997 - present Graduate Clinician/Supervisor,
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Psychological Services Center, Virginia
Polytechnic Institute and State University,
Blacksburg, Virginia

8/1996 - 5/1997 Graduate Clinician, Neuropsychological
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5/1996 - 8/1996 Psychology Assistant/Researcher, Central
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8/1995 - 5/1996 Graduate Clinician, Neuropsychological
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5/1995 - 8/1995 Graduate Clinical Psychology Externship,
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8/1994 - 5/1995 Graduate Clinician, Psychological Services
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9/1993 - 8/1994 Psychology Assistant/Researcher, Central
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5/1993 - 9/1993 Volunteer Research Assistant, Central
Virginia Training Center, Lynchburg,
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8/1992 - 6/1993 Graduate Teaching Assistant, Hollins College,
Hollins, Virginia

12/1991 - 5/1992 Research Assistant, EEG Correlates of
Cognitive Processing, Virginia Polytechnic
Institute and State University, Blacksburg,
Virginia

TEACHING EXPERIENCE

- 8/1997 - present Graduate Teaching Assistant, Undergraduate Research/Field Study, Virginia Polytechnic Institute and State University, Blacksburg, Virginia
- 1/1997 - 5/1997 Course Instructor, Laboratory for Physiological Psychology, Virginia Polytechnic Institute and State University, Blacksburg, Virginia
- 1/1997 - 5/1997 Graduate Teaching Assistant, Undergraduate Research/Field Study, Virginia Polytechnic Institute and State University, Blacksburg, Virginia
- 8/1996 - 12/1996 Course Instructor, Laboratory for Sensation and Perception, Virginia Polytechnic Institute and State University, Blacksburg, Virginia
- 8/1995 - 5/1996 Graduate Teaching Assistant, Lecture Assistant for Introduction to Psychology, Virginia Polytechnic Institute and State University, Blacksburg, Virginia
- 1/1995 - 5/1995 Graduate Teaching Assistant, Lecture Assistant for Introduction to Psychology, Virginia Polytechnic Institute and State University, Blacksburg, Virginia
- 8/1994 - 12/1994 Graduate Teaching Assistant, Discussion Group for Introduction to Psychology, Virginia Polytechnic Institute and State University, Blacksburg, Virginia

PROFESSIONAL PUBLICATIONS

- Crews, Jr., W. D., Harrison, D. W., & Rhodes, R. D. (1997). Neuropsychological test performances of young depressed outpatient women: An examination of executive functions. (Manuscript submitted to Archives of Clinical Neuropsychology for publication.)
- Shenal, B. V., Rhodes, R. D., Moore, T. M., Higgins, D., & Harrison, D. W. (1997). Quantitative electroencephalography (QEEG) facilitates neuropsychological syndrome analysis: An alternative to the nomothetic approach. (Manuscript submitted to Neuropsychology Review for publication.)

Rhodes, R. D., Everhart, D. E., Arey, A. W., Harrison, D. W. (1997). Anger, fear, and the autonomic nervous system: A case study using quantitative EEG. (Manuscript being readied for submission to The Journal of Neurotherapy for publication.)

Crews, Jr., W. D., Rhodes, R. D., Bonaventura, S. H., Rowe, F. B., & Goering, A. M. (1997). Cessation of long-term Naltrexone administration: Longitudinal follow-ups. (Manuscript submitted to Research in Developmental Disabilities for publication.)

Gentry, W. D., Crews, Jr., W. D., Tatum, A., & Rhodes, R. D. (1997). Relationship of health attitudes to employee risk for injury and rehabilitation outcome. (Manuscript being readied for submission for publication.)

Crews, Jr., W. D., Sanders, E. C., Hensley, L. G., Johnson, Y. M., Bonaventura, S., Rhodes, R. D., & Garren, M. P. (1995). An evaluation of facilitated communication in a group of nonverbal individuals with mental retardation. Journal of Autism and Developmental Disorders, 25(2), 205-213.

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RESEARCH IN PROGRESS

Rhodes, R. D., & Harrison, D. W., Reactivity and recovery of function for physiological and neuropsychological measures following the cold pressor test.

Rhodes, R. D., & Harrison, D. W., The effect of stress on the perception of affect in high and low-hostile men.

Steele, S., Rhodes, R. D., & Harrison, D. W., Physiological variable differences as a function of hemibody exposure to stress.

Steele, S., Rhodes, R. D., & Harrison, D. W., Facial EMG asymmetries as a function of hostility.

PUBLISHED REFEREED ABSTRACTS AND CONFERENCE PROCEEDINGS

Rhodes, R. D., Everhart, D. E., Arey, A. W., Harrison, D. W. (1997). Anger, fear, and the autonomic nervous system: A

case study using quantitative EEG. Archives of Clinical Neuropsychology, 13 (1), 124.

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Rhodes, R. D., Harrison, D. W., & Everhart, D. E. (1996). Neuropsychological deficits in a child with Pervasive Developmental Disorder: A case study. In C. R. Ellis & N. N. Singh (Eds.), Children and Adolescents with Emotional and Behavioral Disorders: Proceedings of the Sixth Annual Virginia Beach Conference (p. 85). Richmond, VA: Commonwealth Institute for Child and Family Studies, Medical College of Virginia.

Crews, Jr., W. D., Rhodes, R. D., Bonaventura, S., & Rowe, F. B. (1995). Cessation of long-term Naltrexone administration: A longitudinal case report. In C. R. Ellis & N. N. Singh (Eds.), Children and Adolescents with Emotional and Behavioral Disorders: Proceedings of the Fifth Annual Virginia Beach Conference (p. 37). Richmond, VA: Commonwealth Institute for Child and Family Studies, Medical College of Virginia.

PROFESSIONAL PRESENTATIONS

Rhodes, R. D., Shenal, B. M., & Harrison, D. W., Cerebral Mediation of Emotion: Hostility and the Autonomic Nervous system. (Submitted as a Special Topic Presentation at 18th Annual National Academy of Neuropsychology Conference, November, 1998).

Rhodes, R. D., Steele, S., & Harrison, D. W., The dynamometer hand grip, perseveration, and fatigue test: Preliminary report of data across multiple age ranges and diagnostic categories. (Submitted for presentation at 10th Annual American Psychological Society Conference, 1998.)

Steele, S., Rhodes, R. D., & Harrison, D. W., Autonomic correlates of hemibody exposure to stress. (Submitted for presentation at 10th Annual American Psychological Society Conference, 1998.)

Rhodes, R. D., Steele, S., & Harrison, D. W., The dynamometer hand grip, perseveration, and fatigue test: Preliminary report of data across multiple age ranges and diagnostic categories. (Submitted for presentation at the Virginia Psychological Association Spring Conference, 1998.)

- Steele, S., Rhodes, R. D., & Harrison, D. W., Autonomic correlates of hemibody exposure to stress. (Submitted for presentation at the Virginia Psychological Association Spring Conference, 1998.)
- Crews, w. D., Ferro, N. M., Hart, A., Bolduc, T. A., Cary, H., Barth, J. T., & Rhodes, R. D., Prevalence of Cognitively Intact Individuals Residing in Extended Care Facilities. (Submitted for presentation at the Virginia Psychological Association Spring Conference, 1998.)
- Crews, Jr., W. D., Harrison, D. W., & Rhodes, R. D., Neuropsychological test performances of young depressed outpatient women: An examination of executive functions. (Paper presented at the 17th Annual National Academy of Neuropsychology Conference, 1997.)
- Rhodes, R. D., Everhart, D. E., Arey, A. W., Harrison, D. W., Anger, fear, and the autonomic nervous system: A case study using quantitative EEG. (Paper presented at the 17th Annual National Academy of Neuropsychology Conference, 1997.)
- Rhodes, R. D., Harrison, D. W., & Everhart, D. E., Neuropsychological deficits in a child with Pervasive Developmental Disorder: A case study. (Paper presented at the Sixth Annual Virginia Beach Conference; Children and Adolescents with Emotional and Behavioral Disorders, 1996.)
- Crews, Jr., W. D., Barth, J. T., Bonaventura, S., Rhodes, R. D., Goering, A. M., & Adams, C. M., upper extremity motor performances in individuals with mild, moderate, and severe mental retardation. (Paper presented at the Virginia Psychological Association, Spring Convention, April, 1996.)
- Crews, Jr., W. D., Harrison, D. W., Rhodes, R. D., & Demaree, H. A., Hand fatigue asymmetry in the motor performances of women with depressed mood. (Paper presented at the Virginia Psychological Association, Spring Convention, April, 1995)
- Crews, Jr., W. D., Rhodes, R. D., Bonaventura, S., & Rowe, F.B., Cessation of long-term Naltrexone administration: A longitudinal case report. (Paper presented at the Fifth Annual Virginia Beach Conference; Children and Adolescents with Emotional and Behavioral Disorders, 1995.)
- Crews, Jr., W. D., Bonaventura, S., Rhodes, R. D., & Rowe, F. B., Cessation of long-term Naltrexone administration: A two-year follow-up. Paper presented at the Virginia Psychological Association, Fall Convention, 1993.

Crews, Jr., W. D., Rhodes, R. D., & Hensley, L. G., A preliminary report of facilitated communication research conducted across two state facilities. Invited paper presented at the Virginia Foundation for the Exceptional Child and Adolescent Conference, November, 1993.

GRANT EXPERIENCES

Mix, J. A., Crews, W. D., Barth, J. T., Harrison, D. W., & Rhodes, R. D. An examination of the efficacy of Ginkgo Biloba extract Egb 761 in patients suffering from mild to moderate cognitive impairment: Neuropsychological findings. Partial funding obtained from Dr Willmar Schwabe Arzneimittel GmbH.

Mix, J. A., Crews, W. D., Linstra, R. F., Albers, G. R., Barth, J. T., Harrison, D. W., & Rhodes, R. D. An examination of the efficacy of Ginkgo Biloba extract Egb 761 in patients suffering from mild to moderate cognitive impairment: Neuropsychological findings. Grant proposal being readied for submission to Boehringer-Ingelheim Pharmaceuticals for consideration of funding.

PROFESSIONAL AFFILIATIONS

American Psychological Association (Student Affiliate)
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