Frontal Lobe Correlates in Hostile Men: Analysis of Facial Motor Tone and Cardiovascular Regulation

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Dissertation submitted to the faculty of the Virginia Polytechnic Institute and State University in partial fulfillment of the requirements for the degree of

Doctor of Philosophy
In
Psychology

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April 28, 2010
Blacksburg, VA

Keywords: Corrugator, EMG, Cardiovascular Regulation, Hostility, Masseter

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ABSTRACT

This experimented proposed to test the relationships between self-reported hostility and both facial muscle tone and cardiovascular functioning. Based on previous research, it was proposed that individuals high in self-reported hostility would show increased cardiovascular reactivity in response to a physical stressor (the cold-pressor task). Additionally, based on the integration of multiple lines of research, it was proposed that individuals with high levels of self-reported hostility would show asymmetric facial tone, with greater muscle activation at the left-hemiface. Results showed increased cardiovascular responding in the high-hostile participants following exposure to the cold-pressor task. Additionally, the individuals with high levels of self-reported hostility did show asymmetric facial tone, with increased left-hemifacial EMG values. These differences were present prior to exposure to the cold-pressor task, and were increased following the stressor. Results supported the literature showing increased cardiovascular responding to stress in high-hostile individuals, and also supported the proposed relationship between right orbitofrontal functioning and hostility.

Table of Contents

List of Figures

List of Tables

Introduction

Neuropsychological models for the processing of emotion developed initially out of case studies with individuals suffering unilateral cerebral damage. From these studies, theories of hemispheric asymmetries in the processing and production of affect developed, with most theories positing a left hemisphere superiority for positive affect, and a right hemisphere superiority for negative affect (e.g. Reuter-Lorenz & Davidson, 1981; Wheeler, Davidson, & Tomarken, 1993). In contrast to these hemispheric specialization theories, stands the theory of unilateral (right hemisphere) processing of all emotion, regardless of valence (e.g. Borod, Kent, Koff, Martin, & Alpert, 1988; Bryden, Ley, & Sugarman, 1982; Spence, Shapiro, & Zaidel, 1996). Following the review of the primary neuropsychological theories of emotion, a model of cerebral regulation of hostility will be presented.

Following the review of the neuropsychology of emotion, a review of the relevant literature on facial motor asymmetries is presented, as well as research on hemispheric asymmetries for the control and regulation of the autonomic nervous system. Following the review of these three literatures, an experiment attempting to link these three different processes (emotion, facial motor tone, and cardiovascular regulation) in terms of functional cerebral regions is presented.

Neuropsychology of Emotion

There exists today an extensive literature on the neuropsychology of emotion. The majority of the research falls within two primary domains: Specialization of the right hemisphere for the processing of all emotion and hemispheric asymmetries depending upon emotional valence. Research results supportive of the proposed right hemisphere superiority in the processing of emotion include: increased emotional expression at the left hemiface (spontaneous and posed expressions; Borod et al., 1988; Braun, Raribeau, Ethier, Guerette, & Proulx, 1988; Schmitt, Hartje, & Willmes, 1997), left ear advantage in the identification of emotional tone (Bryden, Ley, & Sugarman,

1982; Schmitt et al., 1997), and significantly greater proportion of left lateral eye movements during generation of emotional images (Borod, Vingiano, & Cytryn, 1988).

Theories of hemispheric asymmetries in emotional processing evolved largely from case-studies on individuals with unilateral brain damage. Results from 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 emotional laterality construct through 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 high proportion of individuals who obtained elevated depression scores in the left hemisphere lesion group was in sharp contrast to the right hemisphere lesion group, in which no patients evidenced a heightened score for depression (Gasparrini, et al., 1977).

While patients participating in research have long been grouped according to interhemispheric differences in cerebral dysfunction, similar results have been obtained based upon intrahemispheric criteria. Robinson, Kubos, Starr, Rao, and Price (1983) reported on 48 patients with unilateral cerebral infarctions, in which the presence of depressive symptomatology 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 the interhemispheric differences, it was possible to further divide the groups based on the lesion location within each hemisphere. For patients with left hemisphere dysfunction, depression rating scores were significantly higher for an anterior, as compared to posterior, lesion. Patients 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.

The findings of emotional regulation deficits relative to lateralized cerebral dysfunction led to the development of emotional lateralization hypotheses. Specifically, it has been hypothesized that the left hemisphere is responsible for positive emotions and that the right hemisphere is responsible for negative emotions (Ahern & Schwartz, 1985; Rueter-Lorenz & Davidson, 1981). The relationships between the left hemisphere and positive affect, and the right hemisphere and negative affect have been well documented. This asymmetry in the processing of positive and negative emotions has been shown for facial motor asymmetries (Schwartz, Ahern, & Brown, 1979), perception of positive or negatively valenced faces presented tachistoscopically (Reuter-Lorenz & Davidson, 1981), and through numerous studies looking at EEG patterns across the two hemispheres (e.g.. Ahern & Schwartz, 1985; Ekman, Davidson, & Friesen, 1990; Sobotka, Davidson, & Senulis, 1992).

With respect to EEG patterns associated with emotional experience, Davidson and colleagues have taken a developmental view of the relationship between hemispheric asymmetries and affective style. Starting with research participants less than one year old, Davidson and Fox (1982) reported significantly different results for each anterior hemisphere based upon presentation of happy or sad videotaped facial expressions. Fox and Davidson (1987) extended this research to include live interactions between infants and their mothers. Results showed reliable differences between left and right frontal regions during maternal approach. Specifically, infants showed increased left frontal activation in response to the maternal approach condition. Also consistent with the hypothesized asymmetries in emotional processing were the results from the maternal separation condition. During this phase of the experiment, children who cried in response to their mother's departure showed an increase in right frontal activation.

Henriques and Davidson (1991) extended the findings of increased right frontal activation during negative affect in infants to a study using a clinically depressed subject pool. Results showed that depressed subjects had less left-sided frontal activation in comparison to the control group. These results were interpreted to suggest a diathesis-stress model, with decreased left frontal activation resulting in increased depression, in response to a negative affect inducing situation. The authors did not say that left frontal hypoactivation automatically resulted in depression, but that it would predispose one to

depression given the appropriate circumstances (Davidson, 1992a; 1992b). In order to test this stress-diathesis model, Wheeler, Davidson, and Tomarken (1993) reported on a study using a non-clinical pool of participants. Results showed that for those participants who evidenced good stability for EEG measures across a three week baseline period, responses to positive or negative film clips were reliably predicted by frontal asymmetries. Specifically, subjects who showed relative left hemisphere activation across baseline measures reported higher levels of positive affect in response to films selected to elicit this emotion. Similarly, subjects who showed stability for relative right hemisphere activation reported more negative affect in response to a film segment selected to elicit this emotion. Thus, subjects who showed stability for the relative left hemisphere activation across baseline measurements were predicted to report increased levels of positive affect in response to this type of film. Similarly, participants who showed a reliable right hemisphere activation bias were predicted to be more responsive to a negative affect film. Consistent with the diathesis-stress model presented above (Henriques and Davidson, 1991), relative activation of one hemisphere over the other does not guarantee the corresponding affect will be shown, but it does make the expression more likely, given the appropriate stimuli.

Heller and colleagues (Heller, 1993; Heller, Etienne, & Miller, 1995) have proposed a similar theory of emotional asymmetries. Just as with the previous theories, Heller's theory places positive emotional processing within the left hemisphere, and negative emotional processing within the right hemisphere. Where this theory differs from the proceeding is in the inclusion of posterior brain regions. Specifically, Heller (1993) proposes that emotional valence (i.e. positive or negative) is a function of relative activation within the frontal lobes, while arousal is a function of the right posterior portion of the brain. According to the theory, a ‘happy’ emotional state results from relative activation of the left frontal lobe with concurrent activation within the right posterior region to increase physiological arousal. Similarly, a ‘calm’ or pleasant emotional state results from relative left frontal activation, concurrent with decreased activation within the right posterior region (i.e. reduced physiological arousal). In contrast, negative emotional states (anxiety and depression) result from relative right frontal activation, with differentiation of mood based on activation within the right

posterior region. Specifically, 'anxiety' results from relative right frontal activation concurrent with right posterior cerebral activation. 'Depression' on the other hand, results from relative right frontal activation concurrent with deactivation within the posterior portions of the right cerebrum.

There are abundant research findings to support both theories of emotional lateralization (i.e. valence theories or right-hemisphere superiority), with neither theory garnering the lion's share of support. As can be seen from the preceding review, however, regardless of which theory of emotional lateralization one supports, negative affect is universally viewed as a right-hemisphere mediated process.

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 of 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 region. 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. For example, 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 region. Clinically, this was reported in a woman with 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.

Demaree and Harrison (1997) also reported findings consistent with increased physiological reactivity concurrent with increased anterior temporal lobe activation within the right hemisphere. In this study, high, as compared to low, hostile men were shown to have increased physiological arousal (HR and BP) following a stressor. Additionally, they reported increased perceptual abilities at the left ear following the stressor. Taken together, these findings were interpreted as supporting increased right hemisphere activation in the high-hostile men following exposure to the physiological stressor. The increased cardiovascular reactivity was replicated by Rhodes and Harrison (1998), with high-hostile men evidencing significantly greater reactivity to the cold-pressor task when compared to their low-hostile counter-parts.

More recently, Foster, Drago, Ferguson, and Harrison (2008) proposed a quadrant model for the cerebral regulation of cardiovascular functioning. This quadrant model allows for a differentiation of functional space, with different influences from the four cortical areas. Thus, the frontal lobes assume an inhibitory role over the posterior regions, while the left and right posterior cortical regions have differential access to the parasympathetic and sympathetic branches of the autonomic nervous system. This model allows for predictions based on the relative activation/arousal within any single quadrant, and the resulting distal effects on other brain areas. For example, the frontal lobes perform an inhibitory function over the posterior regions, with increased right frontal activation resulting in decreased right posterior activation. This would result in decreased heart rate and blood pressure, as sympathetic tone would be reduced resulting in disinhibition of the left posterior regions yielding increased parasympathetic tone. The same type of relationship would occur within the left hemisphere, with increased left frontal activation resulting in decreased left posterior arousal. This inhibition of the left posterior region would then result in increased blood pressure and heart rate, as there would now be a relative activation within the right posterior regions which would result in increased sympathetic nervous system arousal.

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.

Cerebral Asymmetry in Autonomic Nervous System Regulation

Just as there are theories of unilateral and bilateral cerebral emotional processing, so too are there theories for unilateral and bilateral cerebral regulation of the autonomic nervous system. Review of published articles has suggested a gradual transition from theories of primarily unilateral control of the autonomic nervous system to the current theory postulating bilateral cerebral regulation of the autonomic nervous system, with unique abilities ascribed to each hemisphere. Published results from non-human animal studies will be discussed first, followed by studies and experiments focused on regulation of the human autonomic nervous system.

Smith, Hachinski, Gibson, and Ciriello (1986) reported research with left middle cerebral artery occlusion in cats designed to mimic stroke conditions in humans. The authors were attempting to replicate evidence from previous studies that reported increased levels of catecholamines (epinephrine and norepinephrine) in hospital patients following cerebrovascular accidents (Myers, Norris, Hachinski, & Sole, 1981). Procedures for this experiment involved surgical access to the left middle cerebral artery in a group of 32 cats, with implantation of an occlusion device to be used following successful recovery from surgery. After the subjects had recovered from surgery, blood

assays were conducted and the artery was occluded. Following the artificially induced stroke, blood assays revealed significantly increased levels of epinephrine, norepinephrine, and dopamine. Additionally, the findings appeared to apply only to those subjects who showed infarction of the insular cortex on post-mortem examination. These findings were interpreted as showing that the insular cortex maintains an inhibitory influence on 'sympathoexcitatory' structures, and that removal of this inhibitory influence results in increased activity within the sympathetic nervous system (Smith, et al., 1986).

Hachinski, Oppenheimer, Wilson, Guiraudon, and Cechetto (1992) extended the experimental procedures of Smith, et al. (1986) to include occlusions of the left and right middle cerebral arteries in two separate experimental groups of rats. Analyses of blood samples following experimental stroke revealed significant effects based on site of infarction. Specifically, right hemisphere infarcts showed increases in mean arterial pressure, and plasma norepinephrine levels as compared to both the left infarct group and the control (sham operation) group. These results were interpreted to show that right hemisphere infarcts produce greater sympathetic consequences than left hemisphere infarcts.

Robinson (1979) reported biochemical changes in rats similar to Hachinski et al. (1992), but with exactly opposite lateralization. Where Hachinski et al. (1992) reported increased norepinephrine levels following right middle cerebral artery occlusion, Robinson (1979) reported decreased norepinephrine levels following right middle cerebral artery occlusion. Thus, while both studies reported biochemical changes following right hemisphere infarction in rats, supporting the notion of lateralized functions, the effects were diametrically opposite across the two experiments.

Guided by animal research showing asymmetrical consequences of experimental stroke, researchers began looking at human patients following cerebrovascular accident. Early studies (Kolin & Norris, 1984; Myers, Norris, Hachinski, Weingert, & Sole, 1982; Silver, Norris, Lewis, & Hachinski, 1984) reported acute cardiac abnormalities (structure or rhythm) following infarction of the cerebral hemispheres. For example, Myers et al. (1982) reported significantly more cardiac arrhythmias in hospital patients admitted to an acute stroke clinic as compared to non-stroke patients admitted to the same hospital. Stroke patients also evidenced increased levels of norepinephrine in comparison to the

control patients. Kolin and Norris (1984) reported damage to myocardial tissue following cortical infarction or hemorrhage. The changes in myocardial tissue were only present in patients who had suffered sudden increases in intracranial pressure associated with extensive areas of infarction or hemorrhage. The change in myocardial tissue was suspected to result from increased levels of circulating catecholamines following the stroke, but this hypothesis remained unconfirmed. However, postulating increased levels of catecholamines as being responsible for changes in myocardial tissue would be consistent with evidence from the non-human animal literature. Hachinski, Smith, Silver, Gibson, and Ciriello (1986) reported that nearly half of their feline subjects who underwent experimental occlusion of the left middle cerebral artery showed evidence of myocardial damage. Of the subjects who showed changes in myocardial tissue, all were found to have elevated levels of plasma epinephrine and norepinephrine.

Further evidence supporting the lateralization of autonomic processes has been supplied by Heilman and colleagues (Heilman, Schwartz, & Watson, 1978; Heilman & Van Den Abell, 1979). Heilman et al. (1978) reported hypoarousal, as measured by Galvanic Skin Response and a left hemi-neglect in patients following right hemisphere strokes. These results were interpreted as supporting an intimate relationship between right hemisphere functioning and autonomic arousal. In order to test the theory that the right hemisphere is preferentially involved in autonomic arousal, Heilman and Van Den Abell (1979) presented lateralized warning stimuli and observed the effect on the participant's subsequent reaction time. Warning stimuli presented in the left visual field (right hemisphere) reduced reaction times at the right hand significantly more than warning stimuli presented in the right visual field (left hemisphere) reduced reaction times at the left hand. Moreover, presentation of the warning stimulus to left visual field (right hemisphere) resulted in faster reaction times at the right hand than did direct presentation to the left hemisphere. These results were cited as evidence that the right hemisphere is intimately involved in arousal, as the left hemisphere was unable to evidence any effect on right hemisphere performance, whereas stimuli presented to the right hemisphere produced global effects. In other words, the right hemisphere was seen to have greater access to systems responsible for general arousal, as a warning stimulus

presented to the right hemisphere was capable of reducing the amount of time necessary for responding, regardless of which hand was being monitored for reaction time.

In addition to the lesion studies sampled above, there also exists a large literature relating right hemisphere functioning and awareness of autonomic processes. Montgomery and Jones (1984) reported that participants who were most accurate at perceiving their own heart-rate made significantly more lateral eye movements (LEMs) to the left hemispace. This leftward movement of the eyes was viewed as an index of right hemisphere activation, thereby providing a link between increased activation within the right hemisphere and the ability to detect internal autonomic processes. Usage of LEMs to indicate contralateral cerebral activation is not without criticism (Flynn and Clemens, 1988), but is well supported within the literature (Katkin, 1985; Violani, Lombardo, De Gennaro, & Devoto, 1996).

Katkin (1985) reported a thorough history of research associated with his laboratory. Additionally, results of previously unpublished work were presented, which were primarily supportive of the hypothesized relationship between the right hemisphere and autonomic processes. Support for this relationship was provided by research that showed a significant relationship between direction of lateral eye movements during interview and the subsequent ability to detect one's own heartbeat during a discrimination task. Specifically, participants identified as left movers were significantly better at the heartbeat discrimination task without benefit of performance feedback. While both groups (left and right movers) improved with provision of feedback, the left movers were significantly better during the baseline recording phase. Also, while performance improved in both groups, the right movers never reached the initial level of proficiency displayed by the left movers during the baseline session. The review by Katkin (1984) reported that heartbeat discrimination ability was significantly related to the sex of the subject, with men performing significantly better than women. Violani et al. (1996) replicated the finding of increased heartbeat detection abilities for individuals classified as left lateral eye movers, but also extended the findings by showing that women classified as left movers are also able to reliably detect their own heartbeat.

In addition to research indicating a link between physiological functioning and the right hemisphere in humans, findings of physiological changes as the direct result of

manipulations performed at the level of the cerebral cortex have also been reported. Zamrini et al. (1990) reported on 25 epileptic patients who underwent intracarotid amobarbital procedures (Wada test) prior to surgical evaluation. Their results showed that amobarbital introduction in the left internal carotid artery was associated with an increase in heart rate. Conversely, amobarbital injection into the right internal carotid artery was associated with a decrease in heart rate. These findings were interpreted to suggest that the two hemispheres have differential access to sympathetic and parasympathetic control of heart rate. Accordingly, amobarbital injection in the right carotid resulted in decreased heart rate, presumably reflecting a loss of right hemisphere mediated sympathetic arousal. Similarly, anesthetizing the left hemisphere resulted in increased heart rate, presumably indicating loss of parasympathetic control from the left hemisphere.

Further support for the hypothesis of lateralized control of the autonomic nervous system was reported by Oppenheimer, Gelb, Girvin, and Hachinski (1992). Like Zamrini et al. (1990), this study supported the finding of hemispheric asymmetries in the regulation of the autonomic nervous system. In contrast to the use of anesthesia by Zamrini et al., Oppenheimer et al. (1992) obtained their results through direct cortical stimulation. Electrical stimulation of the left insular cortex was reported to reliably elicit bradycardia and decreased diastolic blood pressure. In contrast, the most frequent result reported for electrical stimulation of the right insular cortex was tachycardia and increased diastolic blood pressure. Combined results from these two studies (Oppenheimer et al., 1992; Zamrini et al., 1990) support hemispheric asymmetries in the regulation of the autonomic nervous system. Specifically, the left hemisphere in humans appears to have relatively greater access to the parasympathetic branch of the autonomic nervous system, while the right hemisphere appears to have relatively greater access to the sympathetic branch of the autonomic nervous system. This interpretation for left hemisphere superiority in access to the parasympathetic nervous system is supported by findings of decreased autonomic arousal (decreased heart rate and diastolic blood pressure) following direct cortical stimulation of the left insular region (Oppenheimer et al., 1992), and by findings of decreased autonomic arousal (decreased heart rate) following unilateral right cerebral inactivation with subsequent autonomic tone being a

function of the left hemisphere (Zamrini et al., 1990). Similarly, interpretation of greater access to the sympathetic branch of the autonomic nervous system by the right hemisphere would be supported by findings of increased autonomic arousal following electrical stimulation of the right insular region (Oppenheimer et al., 1992), and by findings of increased autonomic arousal following unilateral left cerebral inactivation with subsequent increase in autonomic tone being a function of the right hemisphere (Zamrini et al., 1990).

While the results reported above are consistent across studies, some authors (e.g. Berntson, Cacioppo, & Quigley, 1993) have argued that simple measures of end-organ state (i.e. heart rate or blood pressure) are not sufficiently precise to allow for discussion of effects as being attributable to sympathetic or parasympathetic nervous system activity. In response to this argument researchers have become increasingly focused on indicators of autonomic nervous system functioning. Yoon, Morillo, Cechetto, and Hachinski (1997) reported on eight patients undergoing intracarotid amobarbital injections prior to surgery for intractable epilepsy. Measurement of autonomic nervous system activity was by power spectral analysis of heart rate variability, using a ratio of low-frequency (LF; sympathetic) to high-frequency (HF; parasympathetic) activity as an index of sympathovagal balance. Results showed that inactivation of the left hemisphere produced a significant increase in the LF/HF ratio, suggestive of a shift toward primarily sympathetic tone. In contrast, inactivation of the right hemisphere resulted in essentially stable performance (very slight, non-significant decrease in the LF/HF ratio). The lack of findings for right hemisphere inactivation were attributed as being due to either small sample size or less prominent lateralized cortical representation for the parasympathetic nervous system.

Based on findings of cerebral asymmetries in the regulation of the autonomic nervous system, Wittling (1995; 1997) proposed a model of autonomic control of the heart based on asymmetrical cerebral access to the sympathetic and parasympathetic branches of the autonomic nervous system. According to the proposed model, regulation of the parasympathetic nervous system is primarily a function of the left hemisphere. Similarly, regulation of the sympathetic nervous system is primarily under the control of the right hemisphere. As support for this model of asymmetrical regulation of the

autonomic nervous system, Wittling, Block, Genzel, and Schweiger (1998) reported evidence indicating left hemisphere predominance in the parasympathetic control of cardiac activity. The experiment consisted of lateralized emotional (neutral or negative) film presentation to either the left or right hemisphere with concurrent measurement of the electrocardiogram for the subsequent analysis of heart rate variability. Results showed a significant increase in high frequency power (HF indicative of parasympathetic activity) as a function of visual hemifield. Specifically, presentation of the visual stimuli to the right visual hemifield produced significant increases in HF power, indicative of increased parasympathetic activation. Minimal support for increased LF power was found for film presentation to the left visual hemifield. No interaction between visual hemifield presentation and emotional valence of the film was found. Thus, the results showed that lateralized presentation of visual information was sufficient to produce changes in cardiovascular performance, and that these changes were primarily parasympathetic in nature and secondary to sensory stimulation of the left hemisphere.

In order to extend the previously mentioned findings to the right hemisphere and to the sympathetic nervous system, Wittling, Block, Schweiger, and Genzel (1998) used the same lateralized display of an emotional film while recording numerous measures of cardiovascular performance including heart rate, stroke volume, cardiac output, peak ejection velocity, left-ventricular ejection time and other physiological measures. The results were interpreted as showing greater cardiac contractility during lateralized film presentation to the right hemisphere. Specifically, presentation of the film to the left visual field resulted in increased stroke volume, cardiac output, rapid ejection period, peak ejection velocity, and other physiological measures, all purportedly indicative of increased contractility and generally more efficient cardiac output. As with Wittling, Block, Genzel, and Schweiger (1998), no significant interaction was seen between emotional valence of the film and the visual hemifield of presentation. Thus, cardiac performance measures seemed to indicate that lateralized presentation of visual material to the right hemisphere was sufficient to change sympathetic nervous system regulation of the heart.

Cerebral Regulation of Facial Movement

Motor control of the hemibodies is lateralized to the contralateral cerebral hemispheres, with the primary motor cortex lying just anterior to the central sulcus. In addition to each motor strip innervating the contralateral hemibody, the layout of the motor strip is arranged in an inverted fashion (Carlson, 1994). Thus, the topographical map of the body represented in the motor strip starts with the lower extremities represented along the midline between the hemispheres, progressing to the hips and trunk, with the hands and face comprising the majority of the lateral convexity. In contrast to the rest of the motor strip, however, cortical representation of the face is not inverted, but is arranged in the appropriate anatomical orientation, progressing from the upper face, to the eyes, and eventually to the muscles of the mouth as motor representation is tracked from the dorsolateral cortex down toward the temporal pole (Rinn, 1984). When comparing cortical representation devoted to the various parts of the body, the hands and face comprise the majority of the motor strip, allowing for greater fine motor control for these areas (Ghez, 1991). In addition to the disproportionate representation for the hands and face in comparison to the rest of the body, there is also a disproportionate representation of the lower face in comparison to the upper facial regions. It has been theorized that this greater cortical representation of the lower face is indicative of the greater fine motor control necessary for communication (Rinn, 1984).

In general, the more distal the bodily region, the more complete the contralateral cerebral control of movement (Gardner, 1963). For example, motor control of the hands is a function of the contralateral motor cortex, whereas control of the shoulder muscles is primarily contralateral, but with some input from the ipsalateral motor cortex. In contrast, muscular control of the face does not show the same contralateral control. Control of the distal extremities is a function of the contralateral motor cortex, regardless of whether one is discussing the upper or lower extremity. For the face, however, there are both lateral differences in cerebral control of motor abilities, as well as differences in control of the upper, versus the lower, face. Exclusive contralateral muscle control is present only within the lower facial regions, with the right motor strip controlling the left lower face and the left motor strip controlling the right lower face. In contrast, control of

the upper facial regions is under bilateral cerebral control with each hemisphere's motor cortex contributing to movements of the upper facial muscles (Rinn, 1984).

The preceding discussion of motor control of the face however, is limited to the movement of muscles related to facial expression. While the lower half of the face is contralaterally enervated for facial expression, the large muscles of mastication (temporalis, masseter, and the internal and external pterygoid muscles) are controlled by Cranial Nerve V (Trigeminal Nerve) and are bilaterally controlled (Rinn, 1984).

Thus, facial expression is a function of Cranial Nerve VII, the Facial Nerve, with bilateral control of the upper face, but with contralateral control of the lower face. In contrast to the control of facial expression, however, the large muscles of mastication are bilaterally controlled by Cranial Nerve V, the Trigeminal Nerve. (Borod, 1993; Richardson, Bowers, Bauer, Heilman, & Leonard, 2000; Rinn, 1984)

The preceding depiction of facial motor control, however, is not without controversy. For example, several studies have reported findings which seem to indicate more contralateral, than bilateral, control in the muscles of the upper face (Asthana, 2001; Asthana & Manas, 1997; Richardson, et al., 2000). In general, though, the consensus agreement seems to be that the upper facial regions are subject to bilateral neural innervation.

Just as different muscle groups are under the control of different cortical areas, selected lesions within the motor pathways result in altered muscle tone, with different effects based on lesion location. Dysfunction at the level of the spinal cord are known as lower motor neuron lesions, and are associated with decreased muscle tone, decreased muscle stretch reflexes (hyporeflexia), and muscle atrophy (Loring, 1999). Lesions above the level of the spinal cord (supraspinal) result in a condition known as the upper motor neuron syndrome (Gardner, 1963; Ghez, 1991). The upper motor neuron syndrome is identified by increased tone (hypertonicity or spasticity), increased muscle stretch reflexes (hypertonicity), and a lack of muscle atrophy (Gardner, 1963; Ghez, 1991; Loring, 1999).

Rationale

As described during the initial literature review the right frontal cortical regions are intimately involved in the regulation of hostility and cardiac functioning. If the right orbitofrontal region is responsible for the regulation of hostility and cardiovascular responding, then based on the neuroanatomy of the cortex, the motor asymmetries should be most evident when investigated within the facial musculature.

In addition to extending the research on facial asymmetry into a relatively under-researched clinical group, this experiment also looked at a neglected facial muscle region: the masseter. There are few references to the masseter in studies of emotion (Brown & Schwartz, 1980; Schwartz, Brown & Ahern, 1980; Rinn, 1984), and when it is mentioned, it is done so somewhat dismissively, suggesting that the masseter may play some role in emotional expression. This is in spite of the fact that one of the primary indicators of anger is a clenched jaw. Thus, the focus for this project was on two muscle groups (corrugator and masseter) involved in the display of anger, without the attention often devoted toward the zygomatic muscle, which is often measured to detect 'smiling.'

Variables

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

Two dependent variable categories were used in this experiment. First, physiological indicators were SBP, DBP, and HR. The second dependent variable category was EMG measured at the bilateral corrugator and masseter muscles.

Hypotheses

Hypothesis 1: High-hostile participants were predicted to show greater cardiovascular reactivity to the cold-pressor stressor.

Hypothesis 2: High hostile participants were predicted to demonstrate increased muscle tone at the face in comparison to low hostile participants.

Hypothesis 3: High hostile participants were predicted to show increased muscle tone at the left, versus the right, hemiface.

Hypothesis 4: Following exposure to the cold-pressor, high hostile participants were predicted to show increased facial muscle tone, as compared to low hostile participants.

Hypothesis 5: Following exposure to the cold-pressor stressor, high hostile participants were predicted to show a differential increase in hemi-facial muscle tone, with a relatively greater increase at the left face.

Method

Participants

Subjects consisted of a total of 48 college-age men evenly divided between two groups, twenty-four low-hostiles and twenty-four high-hostiles. All participants were acquired from the undergraduate Psychology pool. In order to continue in the experiment, participants had to have scored within either the low (0-19) or high (29-50) range of self-reported hostility on the Cook-Medley Hostility Scale. Due to their relatively heightened cerebral lateralization, only men were used to ensure as much homogeneity as possible within the experiment, so as to draw conclusions based solely on independent variable differences. All participants received course credit for their participation. Confidentiality was maintained throughout data collection and analysis.

In addition to the Cook-Medley self-report questionnaire, participants were required to complete surveys related to medical history and hemibody preference. In order to continue in the experiment, participants must have reported an unremarkable medical history as pertaining to head injury, learning disability, neurological dysfunction

or cardiovascular abnormalities. Further, participants had to report sufficient right hemibody preference based on the Coren, Porac, & Duncan laterality test (+7 or above).

Self-Report

All potential participants were initially seen during group screening sessions, at which time they were required to read and to sign an informed consent form (Appendix A). A questionnaire assessing medical history (Appendix B) was also given. Participants then completed the Coren, Porac, and Duncan laterality test (CPD; Coren, Porac, & Duncan, 1979) (Appendix C) to determine hemibody preference. This self-report measure assessed right (+1) and left (-1) hemibody preference based on reported preferred use of eye, ear, arm, and leg. Test scores range from a -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) (Appendix D). The Cook-Medley is the most often used measure of hostility and shows construct validity as a predictor of interpersonal, medical and psychological outcomes (Contrada & Jussim, 1992). Participants had to report hostility levels corresponding to well-documented data within this research setting. Specifically, participants scoring less than 19 were classified as low-hostile, while those scoring 29 and above were classified as high-hostile.

Participants also completed the Beck Depression Inventory (BDI; Beck, 1972) (Appendix E) and the Self-Evaluation Questionnaire (STAI; Spielberger, 1983) (Appendix F). Neither the BDI nor the STAI were used as exclusionary criteria for this experiment.

Apparatus

The laboratory chamber consisted of a chair facing into a flat-white curtain enclosure. The cold pressor equipment was located in this room. Physiological recording equipment was located in an adjacent room.

Physiological

SBP, DBP, and HR were assessed using the Industrial and Biomedics Sensors Corporation pulse/pressure machine (Model SD700A). SBP and DBP were measured by obtaining Korotkoff sounds at the left arm. Accuracy of BP was estimated to be $\pm$ 3 mm Hg.

EMG activity was recorded from electrodes placed over the bilateral corrugator and masseter muscles, with signal amplification and recording by the Grass Polygraph (Model 7P1).

Cold Pressor

The ice water for the CPT was maintained in a small ice cooler at 0 ($\pm$3) degrees Celsius. Water temperature was measured using a standard mercury thermometer.

Procedure

High and low hostility participants were scheduled for further testing in the experiment within one week of their group screening session. Participants were required to read and to sign a second informed consent form upon entering the laboratory chamber (Appendix G).

The experiment consisted of three parts: Prestress, Stress, and Poststress.

Pre-Stress

Once participants had read and signed the second informed consent form, they were seated in the experimental chamber and fitted with physiological recording equipment at the left upper arm for BP and HR measures. Additionally, they were fitted with electrodes at the bilateral corrugator and masseter muscles for recording of muscle tone at the bilateral hemi-face.

Once all physiological recording apparati were in place, EMG readings for facial muscle tone were collected for nine concurrent five-second samples. Following collection of EMG data, HR and BP were measured. 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) 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 by 20 mm Hg (BP).

Stress

Participants were then given the following instructions for completion of the cold pressor task:

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, participants were asked to remove their hand from the water.

EMG data from the face was recorded during the cold-pressor task.

Post-Stress

The participants were then asked to sit still in their chair during the recording of BP and HR. Once the cardiovascular measures had been taken, EMG was again recorded from the bilateral hemi-face for nine five-second periods.

Results

Descriptive Measures

To compare groups (low- and high-hostiles) on descriptive measures taken during the 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 age and the Coren, Porac, and Duncan Laterality Questionnaire. High-hostiles scored significantly higher on the CMHS ($M=32.04$, $SD=3.96$) than did low-hostiles ($M=14.46$, $SD=5.42$), $t(46)=12.82$, $p<.05$. On the BDI, high-hostiles ($M=8.63$, $SD=4.64$) evidenced significantly higher depression scores than the low-hostiles ($M=3.13$, $SD=2.67$), $t(46)=5.03$, $p<.05$. 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 ($M=40.54$, $SD=10.75$) than low-hostiles ($M=28.04$, $SD=7.79$), $t(45)=4.54$, $p<.05$. Similarly, high-hostiles ($M=45.21$, $SD=10.28$) evidenced significantly higher STAI-Trait scores as compared to low-hostiles ($M=29.96$, $SD=8.88$), $t(45)=5.43$, $p<.05$.

In contrast to the other descriptive measures, and in accordance with the inclusionary criteria, only the participants' age and scores on the Coren, Porac, and Duncan Laterality Questionnaire failed to show significant differences between the groups. There was no significant difference in age between the high-hostile group ($M=21.46$, $SD=3.63$) and the low-hostile group ($M=20.83$, $SD=3.21$), $t(46)=.631$, $p=.531$.

For the handedness questionnaire, high-hostiles (M=9.25, SD=3.04) were not found to be significantly different from low-hostiles (M=8.13, SD=2.45), $t(46)=1.411$, $p=.165$.

Cardiovascular Measures

For SBP, there was a significant Group effect, with the high-hostile group having significantly higher SBP, $F(1, 138)=5.98$, $p<.05$. The effect of Trial was also significant, $F(2, 138)=9.25$, $p<.05$. See Figure 1. The group by trial interaction was not significant.

For DBP, there was a significant Group effect, with the high-hostile group having significantly higher DBP, $F(1, 138)=5.24$, $p<.05$. Neither Trial, nor the Group by Trial interaction, was significant. See Figure 2.

For HR, there was no significant Group, Trial, or Group by Trial interaction. See Figure 3.

Electromyographic Measures

For the EMG data, there were several significant main effects. The effects listed here remained significant when corrected with the Greenhouse-Geiser correction factor. The main effect of Group (High-hostile, Low-Hostile) was significant, $F(1, 552)=32.829$, $p<.05$. Similarly, the main effect of Muscle (Left Corrugator, Right Corrugator, Left Masseter, Right Masseter) was also significant, $F(3, 552)=25.09$, $p<.05$. Finally, the main effect of Time (Pre-stress, Cold Pressor, Post-stress) was significant, $F(2, 552)=11.88$, $p<.05$.

In addition to the previously listed main effects, there were also several significant interaction effects. The Group X Muscle interaction was significant, $F(3, 552)=3.074$, $p<.05$. Similarly, there was a statistically significant interaction between Time and Muscle, $F(6, 552)=4.368$, $p<.05$. There was no significant interaction for either Group X Time, $F(2, 552)=.514$, $p>.05$, or Group X Muscle X Time, $F(6, 552)=.509$, $p>.05$.

Figure 4 shows the significant effect of Group on facial EMG levels. As stated above, the high-hostile participants displayed significantly higher levels of facial EMG, and this is readily apparent in the graphical representation.

The significant effect for Time on the EMG data can be seen in Figure 5. There was a significant effect of the cold pressor on facial muscle tone levels, with a significant increase in facial EMG following exposure to the cold pressor.

Finally, the main effect of Muscle was also significant, and this data is presented in Figure 6. Subsequent analysis of this data revealed that the bilateral corrugator muscles (upper face) displayed essentially the same amount of muscle tone, and were significantly greater than the EMG levels of the masseter muscles (lower face). Additionally, EMG levels from both corrugator muscles were significantly higher than the EMG level of the left masseter, which was also significantly higher than the right masseter.

The Group X Muscle interaction was significant. The data are presented in Figure 7. Post-hoc analyses using Tukey's HSD showed that there was a significant difference between the groups at the left corrugator region, with the high-hostile participants showing significantly higher levels of muscle tone. There were no group differences at the right corrugator region. EMG levels at the masseter regions revealed significantly higher left hemifacial masseter muscle tone, in comparison to the right masseter region.

The interaction for Time X Muscle was also significant, and the data are presented in Figure 8. As noted previously, there was a significant main effect for Time, with increased facial muscle tone following exposure to the cold pressor. There were also significant differences between the upper and lower facial regions, with significantly higher EMG levels at the corrugator regions in comparison to the masseter regions.

There were no significant effects for the analyses of Group X Time, or for Group X Time X Muscle. These data are presented graphically in Figures 9 and 10, respectively.

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

	Low-Hostile		High-Hostile	
Questionnaire	Mean	SD	Mean	SD
CMHO	14.46	5.42	32.04	3.96
BDI	3.13	2.67	8.63	4.64
STAI-State	28.04	7.79	40.54	10.75
STAI-Trait	29.96	8.88	45.21	10.28
Laterality	8.13	2.45	9.25	3.04
Age	20.83	3.21	21.46	3.63

Figure 1. Systolic Blood Pressure X Group X Time

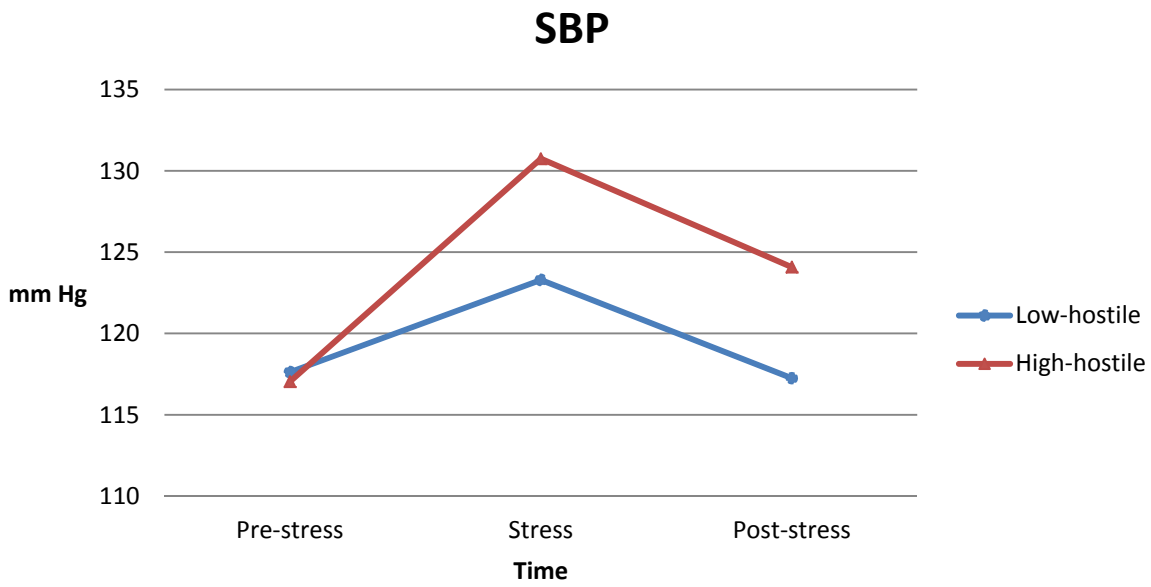


Figure 2. Diastolic Blood Pressure X Group X Time

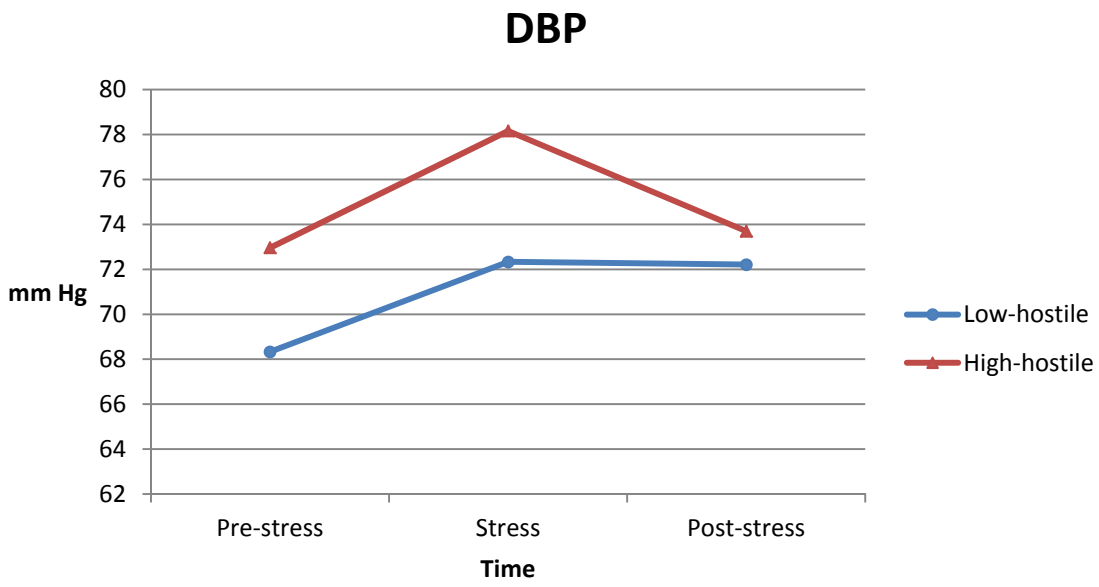


Figure 3. Heart Rate X Group X Time

Figure 4. EMG: Group

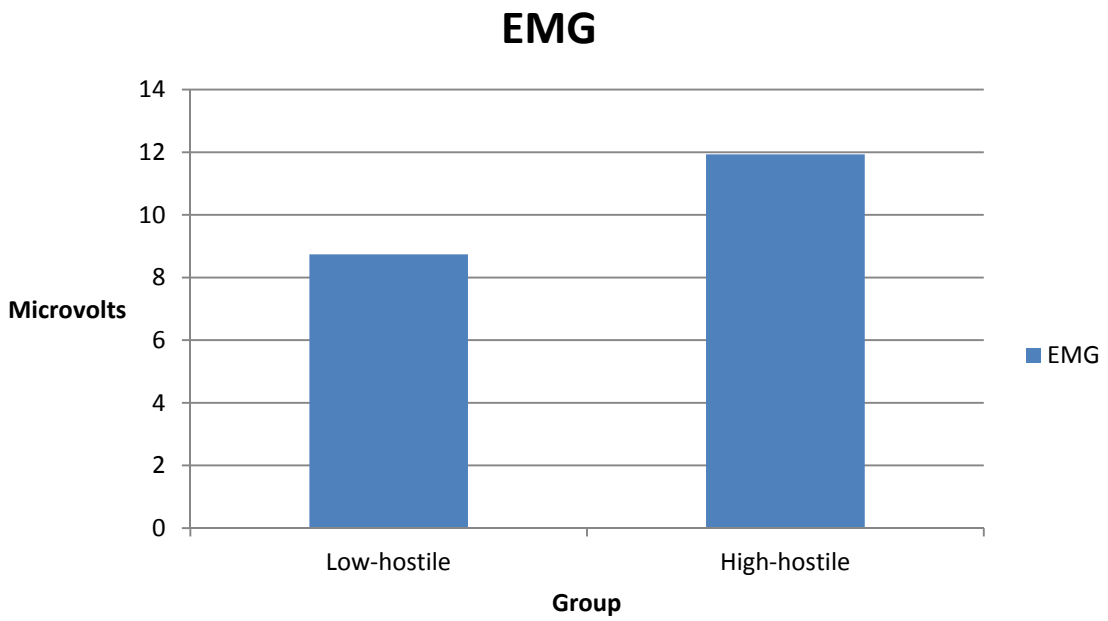


Figure 5. EMG: Time

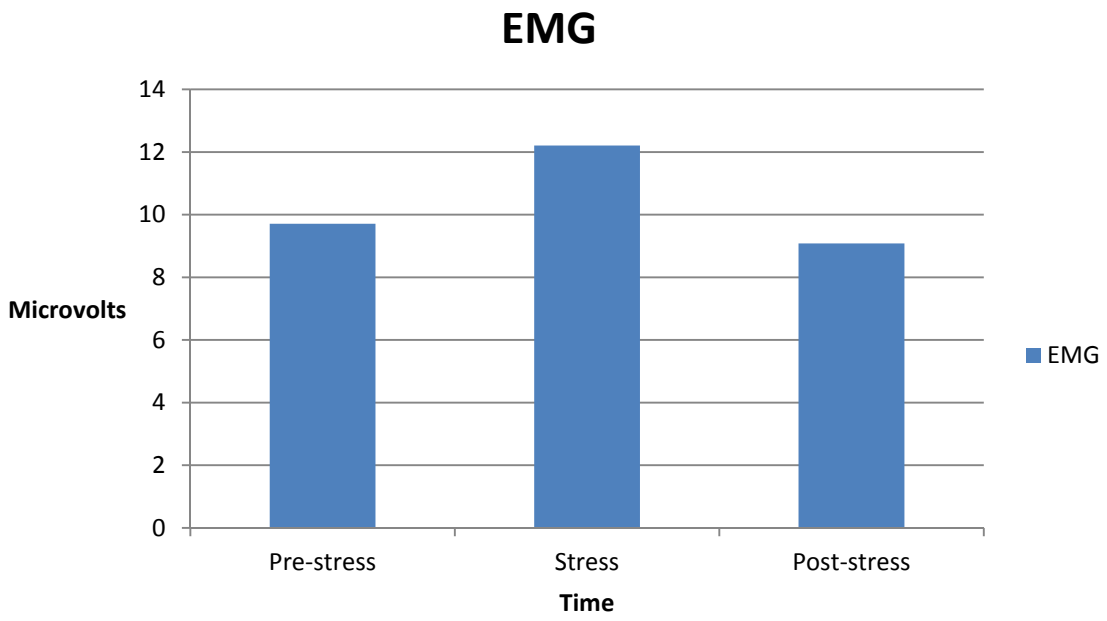


Figure 6. EMG: Muscle

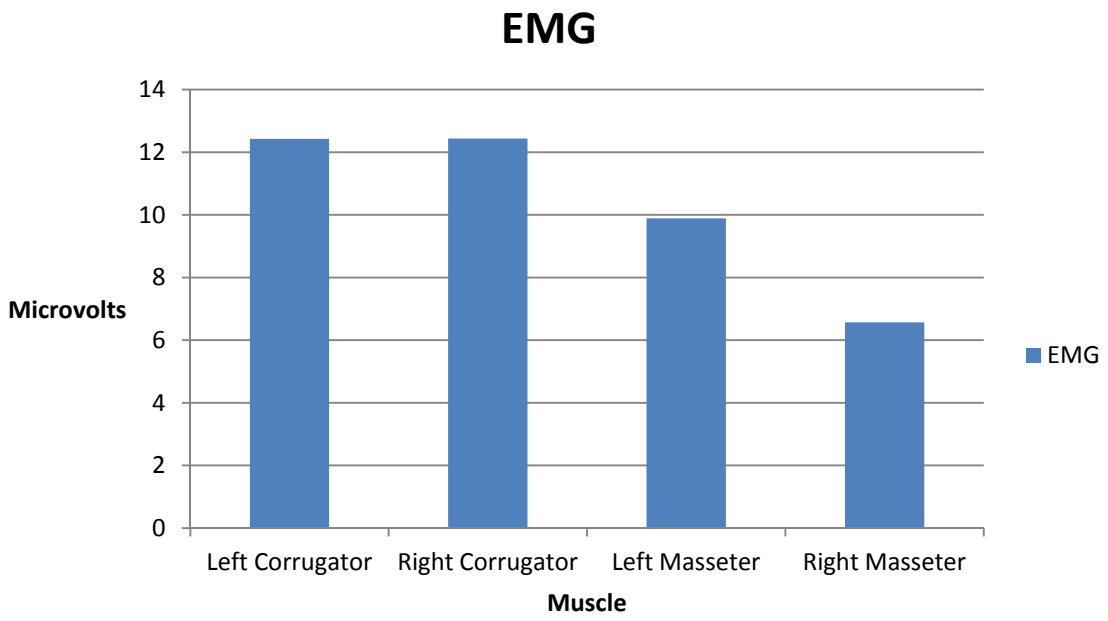


Figure 7. EMG: Group X Muscle

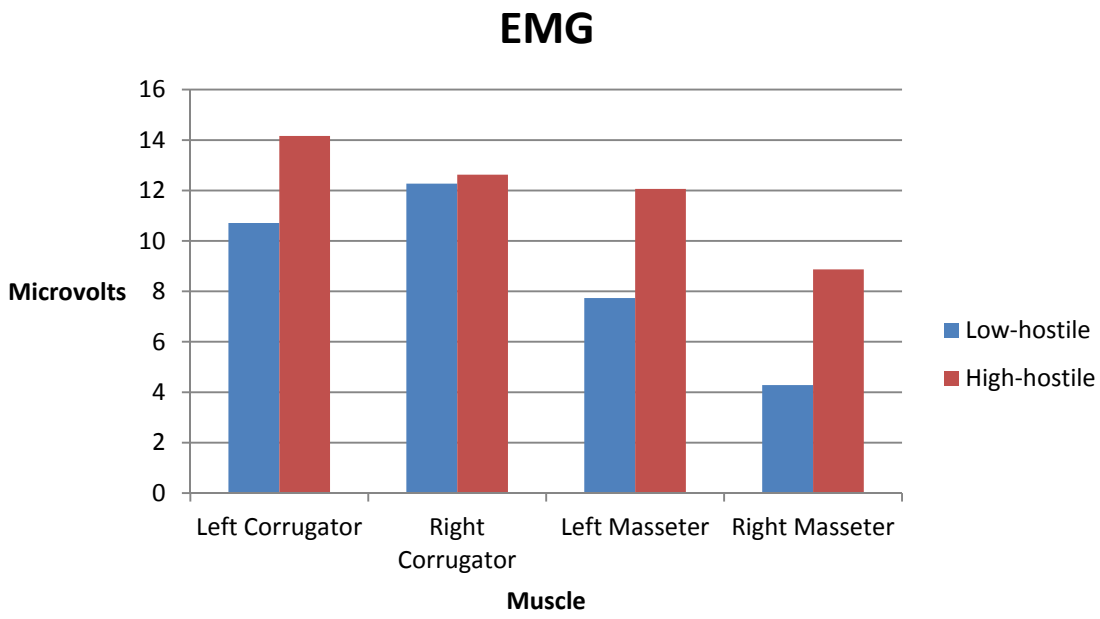


Figure 8. EMG: Time X Muscle

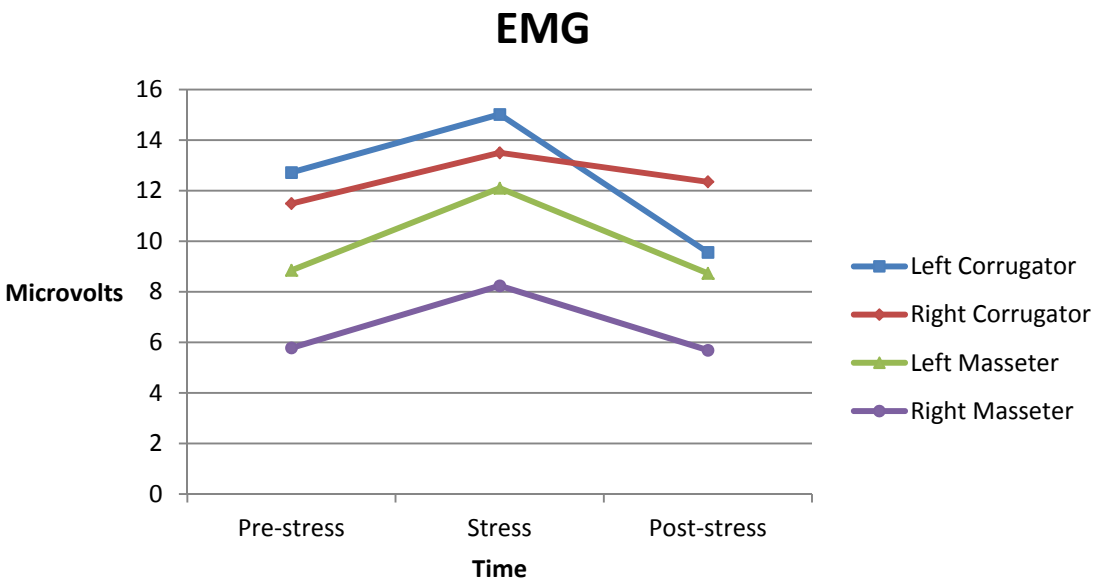


Figure 9. EMG: Time X Group

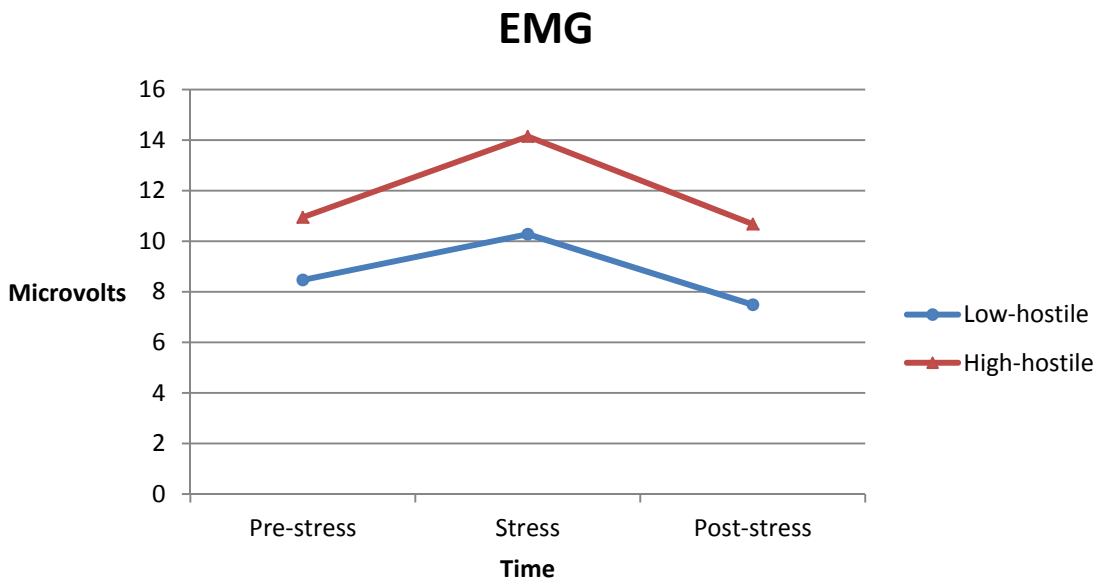
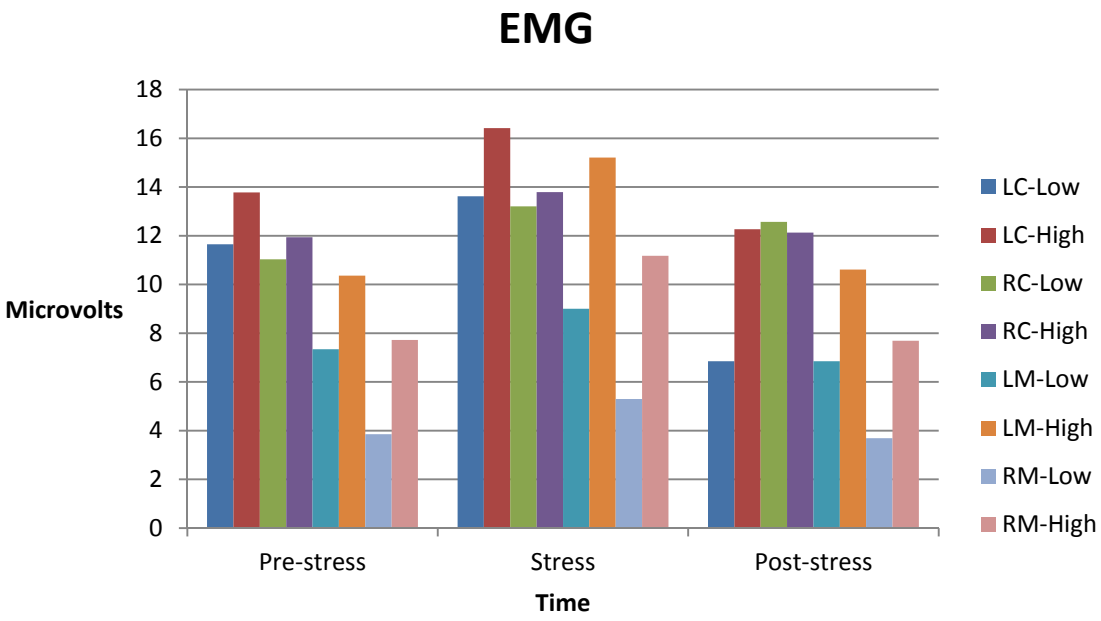


Figure 10. EMG: Group X Muscle X Time



Discussion

There exists an extensive literature devoted to facial affect perception and expression. Much of this work, though, has been targeted toward identifying possible facial asymmetries related to positive and negative affect in normal individuals. The results from these studies have generally fallen into two camps, with many studies supporting a role for the right hemisphere in the production of affective faces, regardless of emotional tone, and the other studies generally supporting the valence model of affective processing, with positive emotions being processed by the left hemisphere and negative emotions being processed by the right hemisphere. This project was also looking at facial asymmetries, but instead of assessing any neurologically-intact person, the participants for this study were males who were either high, or low, in self-reported hostility. Moreover, most facial affect research has focused on the zygomatic and corrugator muscles as indices of positive and negative emotions, respectively, whereas the purpose of this study was to look at facial motor asymmetries as they related specifically to hostility level. As such, it was important to use muscle groups that are associated with negative emotion, specifically the corrugator muscles (frowning, furrowed brow) and the masseter muscles (clenched jaw).

Hypothesis 1, which predicted that the high-hostile participants would be more reactive in terms of cardiovascular activity following the stressor, was partially supported. For SBP, the high-hostile participants displayed significantly higher pressures. While the interaction between group and stress condition was not significant, there were significant differences between the groups across the three different stress periods. The groups started out equivalent during the Pre-stress measurement, but both groups increased significantly from their baseline conditions, and also differed significantly from each other during the Stress measurement. Finally, both groups showed significant declines from the Stress to the Post-stress measurement, with the difference between the two groups continuing to be significant. Additionally, the low-hostile participants had returned to their initial systolic pressure level, whereas the high-hostiles continued to show a significant elevation in SBP relative to their Pre-stress level.

For the DBP data, there was only one significant difference between the two groups, and that was for the Stress measurement, with the high-hostile participants showing significantly higher DBP than the low-hostile participants. While both groups showed increases in DBP, none of the values were significantly different between groups or across trials. The HR data was similar to the DBP data, except that there were absolutely no significant differences found in the HR results.

Hypothesis 2 was a prediction that high-hostile participants would show increased facial EMG, in comparison to the low-hostile group. This hypothesis was supported. High-hostile individuals showed significantly greater facial EMG levels. This finding is consistent with other research showing that there is increased facial muscle tone at the left hemiface (for review, see Borod, 1993). However, those previous research studies have been conducted without regard to hostility, so these results are the first to be reported for individuals scoring high in self-reported hostility.

Hypothesis 3 was a prediction that the high-hostile group would show asymmetrical EMG levels, with greater muscle tone at the left, as compared to right, hemiface. This hypothesis was supported, as there was a significant difference in facial motor tone between the left and right hemifaces in the high-hostile group. Consistent with the a priori expectations, the high-hostile participants showed increased muscle tone at the left hemiface. This was predicted based on the proposed model of hostility, which necessitates right orbitofrontal deficits in the high-hostile group. The same area of relative right-frontal dysfunction which is believed to result in high-hostility would also affect other functions within the same cortical region. This was supported, as the proposed right orbital dysfunction that resulted in increased hostility also resulted in increased facial motor tone at the contralateral hemiface.

The next hypothesis, #4, was a prediction that following exposure to the cold-pressor stressor, high-hostile participants would show greater facial muscle tone, in comparison to the low-hostile group. This hypothesis was supported, as the high-hostile group showed significantly higher facial EMG levels following exposure to the cold-pressor.

Finally, the fifth hypothesis was a prediction that exposure to the cold-pressor would differentially increase the facial EMG of the high-hostile participants, with a

relatively greater increase in muscle tone at the left hemiface. This hypothesis was partially supported. While the high-hostile group did show an increase in facial EMG following the cold-pressor, the increases were not the same across all muscle regions, but were primarily seen at the left corrugator and the bilateral masseter regions.

These findings match the research of others (Zhou & Hu, 2006) on hemifacial motor asymmetries, but also extend this literature into a new population, those with high self-reported hostility. Much of the previous research in this area has focused on positive and negative emotions as indexed by zygomatic and corrugator activity, respectively. These results are consistent with previous research looking at hemifacial asymmetries, with increased muscle tone at the left hemiface, as well as extending the research literature to a new muscle group (masseter) and a new clinical group (high-hostiles).

While previous research projects have described facial motor asymmetries in normal individuals, and those with depression, there has not been any attempt to extend this to the population of individuals with high levels of self-reported hostility. This is an obvious match, though, since emotions are expressed through the facial musculature, and because of the unique association suspected between the neuroanatomy of hostility and the neuroanatomical layout of the face. Schwartz, Brown and Ahern (1980) reported on masseter tone in the early 1980s, but it is an area that has gone largely unnoticed since that time. The masseter, though, is the ideal muscle group for evaluating facial EMG in hostile participants since it is in close proximity to the area believed to be dysfunctional in hostile individuals, and because it provides one of the most telling signs of hostility, a tightly clenched jaw.

This proposed relationship was supported in the current study, as high-hostile participants were proposed to show difficulties in cardiovascular regulation and asymmetrical facial-motor tone as a function of their supposed right-orbitofrontal dysfunction. This relationship was well-supported by the data collected in this projected. High-hostile individuals were expected to show increased left facial muscle tone, should have been more reactive to a physiological stressor, and should have had their hemifacial motor asymmetries exaggerated in response to the cold-pressor. These expectations were confirmed.

Smith (1994) described multiple possible routes of how hostility may develop, and one of his theories was a transactional model allowing for the person and the environment to interact with one another. According to this theory, it would be quite likely that those individuals with right orbitofrontal dysfunction present a face that the rest of the world perceives as angry. The individual does not do this purposely, it is simply the result of brain functioning. Over time, though, the fact that their facial display is being interpreted by others as negative or hostile, would lead other people to shy away from them or treat them in an offensive manner. This would then predispose the individual to expect negative reactions from other people, and therefore to be automatically on the defensive when interacting with others. Whether this is the case or not remains to be seen, but the next step could be to have independent raters judge pictures of low and high-hostile individuals and see if they can replicate the EMG findings of this study. Additionally, those faces could be used to produce split-half composites, along the lines of Sackheim and Gur (1978), and then see if the raters would select the left-face composites as being even higher in hostility.

The proposed study on chimeric faces suggested above is only one possible project that would logically flow from this data. As suggested by the Quadrant Model of Foster et al. (2008), disruption in any cerebral quadrant should have a measurable effect on functioning within other parts of the brain. In the current study, the focus was on taxing right hemisphere functioning in a group already believed to be at-risk for right hemisphere deficits, and looking at subsequent regulation of facial motor tone and cardiovascular regulation. However, it should be possible to effect a change within any quadrant and look for effects on functioning within other areas.

References

Ahern, G. L. & Schwartz, G. E. (1985). Differential lateralization for positive and negative emotion in the human brain: EEG spectral analysis. Neuropsychologia, 23, 745-755.

Berntson, G. G., Cacioppo, J. T., & Quigley, K. S. (1993). Cardiac psychophysiology and autonomic space in humans: Empirical perspectives and conceptual implications. Psychological Bulletin, 114. 296-322.

Borod, J. C., Kent, J., Koff, E., Martin, C., & Alpert, M. (1988). Facial asymmetry while posing positive and negative emotions: Support for the right hemisphere hypothesis. Neuropsychologia, 26, 759-764.

Borod, J. C., Vingiano, W., & Cytryn, F. (1988). The effects of emotion and ocular dominance on lateral eye movement. Neuropsychologia, 26, 213-220.

Braun, C. M. J., Raribeau, J. M. C., Ethier, M., Guerette, R., & Proulx, R. (1988). Emotional facial expressive and discriminative performance and lateralization in normal young adults. Cortex, 24, 77-90.

Bryden, M. P., Ley, R. G., & Sugarman, J. H. (1982). A left-ear advantage for identifying the emotional quality of tonal sequences. Neuropsychologia, 20, 83-87.

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

Carlson, N. R. (1998). Structure of the nervous system. Physiology of behavior (6th ed.), pp. 56-85. Allyn and Bacon: Boston.

Davidson, R. J. (1992a). Anterior cerebral asymmetry and the nature of emotion. Brain and Cognition, 20, 125-151.

Davidson, R. J. (1992b). Emotion and affective style: Hemispheric substrates. Psychological Science, 3, 39-43.

Davidson, R. J., & Fox, N. A. (1982). Asymmetrical brain activity discriminates between positive versus negative affective stimuli in ten month old infants. Science, 218, 1235-1237.

Demaree, H., & Harrison, D. W. (1997). Physiological and neuropsychological correlates of hostility. Neuropsychologia, 35, 1405-1411.

Demaree, H. A., Harrison, D. W., & Rhodes, R. D. (in press). Quantitative electroencephalographic analyses of cardiovascular regulation in low- and high-hostile men. Psychobiology.

Demaree, H. A., Higgins, D. A., Williamson, J., & Harrison, D. W. (2000). Asymmetry in hand grip strength and fatigue in low- and high-hostile men. (Manuscript submitted to Neuropsychiatry, Neuropsychology, and Behavioral Neurology for publication.)

Ekman, P., Davidson, R. J., & Friesen, W. V. (1990). The Duchenne smile: Emotional expression and brain physiology II. Journal of Personality and Social Psychology, 58, 342-353.

Flynn, D. M. & Clemens, W. J. (1988). On the validity of heartbeat tracking tasks. Psychophysiology, 25, 92-96.

Foster, P. S., Drago, V., Ferguson, B. J., & Harrison, D. W. (2008). Cerebral moderation of cardiovascular functioning: A functional cerebral systems perspective. Clinical Neurophysiology, 119, 2846-2854.

Fox, N. A., & Davidson, R. J. (1987). Electroencephalogram asymmetry in response to the approach of a stranger and maternal separation in 10-month-old infants. Developmental Psychology, 23, 233-240.

Gainotti, G. (1972). Emotional behavior and hemispheric side of lesion. Cortex, 8, 41-55.

Gardner, E. (1963). Fundamentals of neurology. Philadelphia: W. B. Saunders Co.

Gasparrini, W. G., Satz, P., Heilman, K. M., & Coolidge, F. L. (1977). Hemispheric asymmetries of affective processing as determined by the Minnesota Multiphasic Personality Inventory. Journal of Neurology, Neurosurgery, and Psychiatry, 41, 470-473.

Ghez, C. (1991). The control of movement. In E. Kandel, J. Schwartz, and T. Jessell (Eds.), Principles of Neural Science. Norwalk, Connecticut: Appleton & Lange, pp. 533-547.

Hachinski, V. C., Oppenheimer, S. M., Wilson, J. X., Guiraudon, C., & Cechetto, D. F. (1992). Asymmetry of sympathetic consequences of experimental stroke. Archives of Neurology, 49, 697-702.

Hachinski, V. C., Smith, K. E., Silver, M. D., Gibson, C. J., & Ciriello, J. (1986). Acute myocardial and plasma catecholamine changes in experimental stroke. Stroke, 17, 387-390.

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

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

Heilman, K. M., Schwartz, H., & Watson, R. T. (1978). Hypoarousal in patients with neglect syndrome and emotional indifference. Neurology, 28, 229-232.

Heilman, K. M. & Van Den Abell, T. (1980). Right hemisphere dominance for attention: The mechanism underlying hemispheric asymmetry of inattention. Neurology, 30, 327-330.

Heller, W. (1993). Neuropsychological mechanisms of individual differences in emotion, personality, and arousal. Neuropsychology, 7, 476-489.

Heller, W., Etienne, M. A., & Miller, G. A. (1995). Patterns of perceptual asymmetry in depression and anxiety: Implications for neuropsychological models of emotion and psychopathology. Journal of Abnormal Psychology, 104, 327-333.

Henriques, J. B., & Davidson, R. J. (1991). Left frontal hypoactivation in depression. Journal of Abnormal Psychology, 100, 535-545.

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

Katkin, E. S. (1985). Blood, sweat, and tears: Individual differences in autonomic self-perception. Psychophysiology, 22, 125-137.

Kolin, A. & Norris, J. W. (1984). Myocardial damage from acute cerebral lesions. Stroke, 15, 990-993.

Loring, D. W. (1999). INS dictionary of neuropsychology. New York: Oxford University Press.

Montgomery, W. A. & Jones, G. E. (1984). Laterality, emotionality, and heartbeat perception. Psychophysiology, 21, 459-465.

Myers, M. G., Norris, J. W., Hachinski, V. C., & Sole, M. J. (1981). Plasma norepinephrine in stroke. Stroke, 12, 200-204.

Myers, M. G., Norris, J. W., Hachinski, V. C., Weingert, M. E., & Sole, M. J. (1982). Cardiac sequelae of acute stroke. Stroke, 13, 838-842.

Oppenheimer, S. M., Gelb, A., Girvin, J. P., & Hachinski, V. C. (1992). Cardiovascular effects of human insular cortex stimulation. Neurology, 42, 1727-1732.

Reuter-Lorenz, P., & Davidson, R. J. (1981). Differential contributions of the two cerebral hemispheres to the perception of happy and sad faces. Neuropsychologia, 19, 609-913.

Rhodes, R.D, Everhart, D.E, & Harrison, D.W. (2000). Anger, fear, and the autonomic nervous system: A case study using quantitative EEG. (Paper submitted for publication.)

Rhodes, R. D. & Harrison, D. W. (1998). Reactivity and recovery of physiological and neuropsychological variables as a function of hostility. Unpublished manuscript.

Rinn, W. E. (1984). The neuropsychology of facial expression: A review of the neurological and psychological mechanisms for producing facial expressions. Psychological Bulletin, 95, 52-77.

Robinson, R. G. (1979). Differential behavioral and biochemical effects of right and left hemispheric cerebral infarction in the rat. Science, 205, 707-710.

Robinson, R. G., Kubos, K. L., Starr, L. B., Rao, K., & Price, T. R. (1983). Mood changes in stroke patients: relationship to lesion location. Comprehensive Psychiatry, 24, 555-566.

Schmitt, J. J., Hartje, W., & Willmes, K. (1997). Hemispheric asymmetry in the recognition of emotional attitude conveyed by facial expression, prosody, and propositional speech. Cortex, 33, 65-81.

Schwartz, G. E., Ahern, G. L., & Brown, S. (1979). Lateralized facial muscle response to positive and negative emotional stimuli. Psychophysiology, 16, 561-571.

Silver, F. L., Norris, J. W., Lewis, A. J., & Hachinski, V. C. (1984). Early mortality following stroke: A prospective review. Stroke, 15, 492-496.

Smith, K. E., Hachinski, V. C., Gibson, C. J., & Ciriello, J. (1986). Changes in plasma catecholamine levels after insula damage in experimental stroke. Brain Research, 375, 182-185.

Sobotka, S., Davidson, R. J., & Senulis, J. (1992). Anterior brain electrical asymmetries in response to reward and punishment. Electroencephalography and Clinical Neurophysiology, 83, 236-247.

Spence, S., Shapiro, D., & Zaidel, E. (1996). The role of the right hemisphere in the physiological and cognitive components of emotional processing. Psychophysiology, 33, 112-122.

Ursin, (1960). The temporal lobe substrate of fear and anger. Acta of Psychiatria Scandinavia, 35, 378-396.

Violani, C., Lombardo, C., De Gennaro, L., & Devoto, A. (1996). Left movers' advantage in heartbeat discrimination: A replication and extension. Psychophysiology, 33, 234-238.

Wheeler, R. E., Davidson, R. J., & Tomarken, A. J. (1993). Frontal brain asymmetry and emotional reactivity: A biological substrate of affective style. Psychophysiology, 30, 82-89.

Williamson, J. & Harrison, D. W. (2000). Cerebral asymmetry in the dual-task performance of high-hostile males. Manuscript in preparation.

Wittling, W. (1995). Brain asymmetry in the control of autonomic-physiologic activity. In R. J. Davidson & K. Hugdahl (Eds.) Brain asymmetry, pp. 305-357. Cambridge, MA: MIT Press.

Wittling, W. (1997). Brain asymmetry and autonomic control of the heart. European Psychologist, 2, 313-327.

Wittling, W., Block, A., Genzel, S., & Schweiger, E. (1998). Hemisphere asymmetry in parasympathetic control of the heart. Neuropsychologia, 36, 461-468.

Wittling, W., Block, A., Schweiger, E., & Genzel, S. (1998). Hemisphere asymmetry in sympathetic control of the human myocardium. Brain and Cognition, 38, 17-35.

Woods, J. W. (1956). Taming of the wild in Norway rat by rhinocephalic lesions. Nature, 170, 869.

Yoon, B., Morillo, C.A., Cechetto, D.F., & Hachinski, V. (1997). Cerebral hemispheric lateralization in cardiac autonomic control. Archives of Neurology, 54, 741-744.

Zamrini, E. Y., Meador, K. J., Loring, D. W., Nichols, F. T., Lee, G. P., Figueroa, R. E., & Thompson, W. O. (1990). Unilateral cerebral inactivation produces differential left/right heart rate responses. Neurology, 40, 1408-1411.

Appendix A

INFORMED CONSENT FOR PARTICIPANTS OF INVESTIGATIVE PROJECTS

Title of Project: Frontal Lobe Correlates in Hostile Men: Analysis of Motor Tone and Cardiovascular Regulation.

Experiment Number:

1 **PURPOSE OF EXPERIMENT**

You are invited to participate in a study to obtain data on measures purportedly associated with stress.

1 **PROCEDURE TO BE FOLLOWED IN THE STUDY**

To accomplish the goals of this study, you will be asked to complete four questionnaires. You may then be selected for further participation in this research.

1 **ANONYMITY OF SUBJECTS AND CONFIDENTIALITY OF RESULTS**

Identifying information will be kept strictly confidential. At no time will the researchers release your personal information from the study to anyone other than individuals working on the project without your written consent. The information you provide will have your name removed and only a subject number will identify you during analyses and any written reports of the research.

1 **DISCOMFORTS AND RISKS FROM PARTICIPATING IN THE STUDY**

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

1 **EXPECTED BENEFITS**

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

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

1 **FREEDOM TO WITHDRAW**

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

7. EXTRA CREDIT COMPENSATION

For participation in this study, you will receive one point extra credit for each hour (or portion thereof) of time spent participating in this research. The extra credit point(s) accumulated will be applicable for your grade in Psychology 2004.

1 **USE OF RESEARCH DATA**

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

1 **APPROVAL OF RESEARCH**

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

1 **PARTICIPANT'S PERMISSION**

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

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

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

Robert D. Rhodes	231-6914
Primary Researcher	Phone
David W. Harrison, Ph.D.	231-4422
Faculty Advisor	Phone
David W. Harrison, Ph.D.	231-4422
Chair, HSC	Phone
David M. Moore, DVM	231-4991
Chair, IRB	Phone

Participant's Signature: _____ Date: _____
Participant's ID: _____ Participant's Telephone: _____

Appendix B

MEDICAL HISTORY QUESTIONNAIRE

NAME:_____ ID #: _____

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

1	Severe head trauma/injury……	Yes	No
2	Stroke ……..	Yes	No
3	Learning disabilities (problems with reading, writing, or comprehension)……..	Yes	No
1	Epilepsy or seizures…….	Yes	No
2	Paralysis……	Yes	No
3	Neurological surgery……	Yes	No
4	Other neurological/nervous system problems……..	Yes	No
5	Alcohol or drug problems……	Yes	No
6	Using alcohol or drugs (other than for prescribed purposes) at present……	Yes	No
1	Past psychological/psychiatric problems……..	Yes	No
2	Are you currently taking any prescription medications/drugs……	Yes	No
1	Are you currently suffering from any medical conditions or illnesses……..	Yes	No
1	Arthritis……	Yes	No
2	Any head or lung problems……..	Yes	No
3	Reynaud's Syndrome……	Yes	No
4	Any cardiac (heart) problems……	Yes	No
5	Hearing problems……..	Yes	No

Please explain "Yes" responses:

_____
_____
_____
_____
_____
_____
_____
_____
_____

Appendix C

Handedness Questionnaire

Subject #: _____

Circle the appropriate number after each item.

	Right	Left	Both
With which hand would you throw a ball to hit a target?	1	-1	0
With which hand do you draw?	1	-1	0
With which hand do you use an eraser on paper?	1	-1	0
With which hand do you remove the top card when dealing?	1	-1	0
With which foot do you kick a ball?	1	-1	0
If you wanted to pick up a pebble with your toes, which foot would you use?	1	-1	0
If you had to step up onto a chair, which foot would you place on the chair first?	1	-1	0
Which eye would you use to peep through a keyhole?	1	-1	0
If you had to look into a dark bottle to see how full it was, which eye would you use?	1	-1	0
Which eye would you use to sight down a rifle?	1	-1	0
If you wanted to listen to a conversation going on behind a closed door, which ear would you place against the door?	1	-1	0
If you wanted to listen to someone's heartbeat, which ear would you place against their chest?	1	-1	0
Into which ear would you place the earphone of a transistor radio?	1	-1	0

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

Is mother left or right hand dominant? _____

Is father left or right hand dominant? _____

Appendix D

Cook-Medley Hostility Scale. Derived from the Minnesota Multiphasic Personality Inventory – 2, published by the University of Minnesota Press. See publisher for details.

Appendix E

Beck Depression Inventory. Published by PsychCorp. See publisher for details.

Appendix F

State-Trait Anxiety Inventory. Published by Mind Garden. See publisher for details.

Appendix G

INFORMED CONSENT FOR PARTICIPANTS OF INVESTIGATIVE PROJECTS

Title of Project: Frontal Lobe Correlates in Hostile Men: Analysis of Motor Tone and Cardiovascular Regulation.

Experiment Number:

1 **PURPOSE OF EXPERIMENT**

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

1 **PROCEDURE TO BE FOLLOWED IN THE STUDY**

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

You will be hooked up to heart rate and blood pressure equipment to be monitored and recorded before and after the cold pressor test. This will help determine physiological reactivity to the cold pressor test. Additionally, electrodes will be applied to regions of your face to measure the amount of muscle activity present within certain muscle groups.

1 **ANONYMITY OF SUBJECTS AND CONFIDENTIALITY OF RESULTS**

Identifying information will be kept strictly confidential. At no time will the researchers release your personal information from the study to anyone other than individuals working on the project without your written consent. The information you provide will have your name removed and only a subject number will identify you during analyses and any written reports of the research.

1 **DISCOMFORTS AND RISKS FROM PARTICIPATING IN THE STUDY**

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

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

1 **EXPECTED BENEFITS**

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

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

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

1 **FREEDOM TO WITHDRAW**

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

7. EXTRA CREDIT COMPENSATION

For participation in this study, you will receive one point extra credit for each hour (or portion thereof) of time spent participating in this research. The extra credit point(s) accumulated will be applicable for your grade in Psychology 2004.

1 **USE OF RESEARCH DATA**

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

1 **APPROVAL OF RESEARCH**

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1 **PARTICIPANT'S PERMISSION**

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

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

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

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Chair, HSC	Phone
David M. Moore, DVM	231-4991
Chair, IRB	Phone

Participant's Signature: _____ Date: _____

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