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Exploring the Cardiovascular Response to Anger Imagery and Speech in Vietnam Veterans With and Without Posttraumatic Stress Disorder

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A thesis submitted in partial fulfillment of the requirements for the degree of Master of Science at Virginia Commonwealth University.

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Abstract

EXPLORING THE CARDIOVASCULAR RESPONSE TO ANGER IMAGERY AND SPEECH IN VIETNAME VETERANS WITH AND WITHOUT POSTTRAUMATIC STRESS DISORDER.

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Few studies directly compare the physiological consequences of anger under a variety of induction methods. The current study explored the patterns of cardiovascular responding associated with varying anger induction methods, specifically personal anger memory recall through verbalization versus imagery in a sample of Vietnam combat veterans with and without Posttraumatic Stress Disorder (PTSD). Spoken anger produced greater elevations in blood pressure than anger that was recalled through imagery but not spoken. This was true even after controlling for the metabolic activity associated with speech. However, for veterans with PTSD, anger imagery was also sufficient to produce an elevated response in cardiovascular activity.

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Introduction

Researchers exploring the cardiovascular response (CVR) to anger have approached their work with a variety of purposes, from pursuing the physiological and health related consequences of anger (Beckham, Taft, Vrana, Feldman, Barefoot, Moore, Mozley, Butterfield & Calhoun, 2003; Brosschot & Thayer, 2003; Davidson, MacGregor, Stuhr, Dixon, & MacLean, 2000; Finney, Stoney, Engebretson, 2002; Lovallo & Gerin, 2003; Powch & Houston, 1996; Siegman, 1993), to seeking physiological markers that differentiate anger from other emotions (Labouvie-Vief, Lumley, Jain, & Heinze, 2003; Prkachin, Williams-Avery, Zwaal, & Mills, 1999; Sinha, Lovallo, & Parsons, 1992; Waldstein, Kop, Schmidt, Haufler, Krantz, & Fox, 2000).

A variety of methods exist for inducing and studying the cardiovascular effects of anger. While most anger and physiology research, despite disparate modalities, suggests a reliable relationship between anger and blood pressure elevations, little can be said about how varying the methods or the population may (or may not) differentially affect the physiological consequences of anger induction.

Methods by which anger is induced range from "real-life" or actual anger created in the laboratory (Böddeker & Stemmler, 2000; Lavoie, Miller, Conway, & Fleet, 2001; Stemmler, Heldmann, Pauls & Scherer, 2001) to autobiographical recall of anger memory via imagery (Beckham, Vrana, Barefoot, Feldman, Fairbank, & Moore, 2002; Prkachin et al, 1999; Sinha et al, 1993) to verbalization of a personal anger memory (Friedman, Thomas, Kulick-Ciuffo, Lynch, & Suginohara, 1982; Siegman, Anderson, & Berger, 1990; Siegman, Dembroski, & Crump, 1992) to verbalization under hostile

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conditions such as being told to work faster or make fewer mistakes (Finney, Stoney, & Engebretson, 2002).

Few studies directly compare the physiological consequences of anger under a variety of induction methods (such as real-life, imagined anger recall, or verbalized anger recall); those that have, report incongruent results that are seemingly a function of anger modality. In work comparing a spoken anger memory with thinking about anger, only verbalized anger elicited significant increases in blood pressure, prompting the authors to claim verbalization of anger is not only sufficient but also necessary if cardiovascular increases are to be produced (Siegman & Snow, 1997). Similarly, a comparison of "real-life" anger produced via a laboratory manipulation (the anger was not verbalized) with later recall of the event during imagery found both conditions elicited significant increases in blood pressure, however the imagery condition responses were attenuated compared to the actual anger (Stemmler et al., 2001).

What explanation can be proffered for the discordance in cardiovascular responding among varying anger induction methods? Lang's bio-informational theory of emotion (1977; 1979; 1983) provides a basis for reconciling these inconsistent findings, or lack of consistent cardiovascular efferents across different techniques for anger induction. According to bio-informational theory, emotion memory structures are composed of information relating to stimuli, behavior, and meaning. Sufficient cues or cue intensity recalling the stimuli, behavior, and/or meaning are required for activation of the emotion memory. Insufficient cues or cue intensity may result in incomplete activation of the memory network and produce attenuated physiological responses. Bio-

informational theory suggests an explanation for why different methods of inducing anger (verbalization versus imagery for example) may result in different levels of activation of the anger memory networks.

The majority of aforementioned anger induction studies were conducted with non-clinical samples even though inferences are often drawn to treating anger in a therapeutic setting. Although anger is not a diagnostic category in the DSM-IV (American Psychiatric Association, 1994) there are disorders for which anger is a component, such as Posttraumatic Stress Disorder (PTSD). A recent review of the literature (Beckham, Moore & Reynolds, 2000) suggests both anger and violence are integral components of *combat*-related PTSD. Though the relationship between anger and PTSD is unclear, one study suggests pre-existing anger arousal may exist in combat veterans who later develop PTSD (McFall, Wright, Donovan, & Raskind, 1999).

Conducting anger research utilizing a population with known anger problems may help illuminate, and add to the understanding of, cardiovascular responding during anger induction using varying methods. A prior publication, using the same participants as this current study, found PTSD veterans experience higher diastolic blood pressure during anger imagery, and take longer to recover from anger imagery than veterans without PTSD (Beckham et al., 2002). This suggests that the associative strength of the anger memory networks of a PTSD population is stronger or more easily accessed than the associated memory networks of a sample without PTSD, during imagery. This is analogous to results obtained when investigating the *fear* memory networks of phobic and non-phobic individuals (Lang, Levin, Miller, & Kozak, 1983) and strengthens the

rational for applying bio-informational theory in the explication of disparate findings in anger induction research.

The current study is being undertaken to explore the patterns of cardiovascular responding associated with varying anger induction methods, specifically personal anger memory recall through verbalization versus imagery. In addition, this investigation is intended to compare the cardiovascular effects of anger in a clinical (PTSD) versus a non-clinical (non-PTSD) population. In order to provide background information relevant to this research, a review follows that covers the cardiovascular response to anger, a bio-informational approach to anger retrieval, and anger in a Posttraumatic Stress Disorder population.

Literature Review

The Cardiovascular Response to Anger

It is fairly well established that an angry person will exhibit some physiological change, most often manifested in blood pressure increases, when compared to a non-angry baseline (Stemmler et al., 2001; Suarez, Harlan, Peoples, & Williams, 1993; Suarez & Williams, 1989). This reactivity in the laboratory is most consistently observed in diastolic blood pressure (Böddeker & Stemmler, 2000; Siegman, 1993; Sinha et al., 1992; Stemmler et al., 2001) and can be successfully produced when using an anger provocation paradigm (Böddeker & Stemmler, 2000; Lavoie et al., 2001; Stemmler et al., 2001) or emotion activation methods using autobiographical anger memories (Friedman et al., 1982; Siegman, Anderson, & Berger, 1990; Siegman, Dembroski, & Crump, 1992; Finney t al., 2002; Prkachin et al., 1999; Sinha et al., 1993). Cardiovascular response to anger in laboratory settings has also been observed, though with less consistency, in heart rate and systolic blood pressure (Lavoie et al., 2001).

Anger provocation methods are intriguing not only due to the manner in which researchers attempt to anger participants (primarily via threats to self-esteem), but also in that they provide a snapshot of anger in action, often referred to as "real-life" anger. Studying anger in action should (theoretically) result in the most accurate picture of the responses associated with this emotion.
Anger has the potential of manifesting in affect, behavior, and physiology. Researchers often report focusing on physiological responses since they are less susceptible to volition, and affect (self-report) to insure the manipulation was successful.

Recording both physiology and affect also allows exploration of the congruency between response categories. Less usual in CVR and anger research is the calculated observance of non-prescribed behavior such as external, physical manifestations of anger, though there are examples of this in the literature (Böddeker & Stemmler, 2000). In the Böddeker and Stemmler study, video observations of spontaneous angry behavior did not detect group differences between angered and non-angered participants. The authors suggest the instructions to restrict movements (for the purpose of more accurate physiological recording) may have inadvertently encouraged participants to suppress facial or physical displays of anger. These instructions could impede accurate behavioral ratings in many physiology of anger studies in which unnecessary movement is usually discouraged.

As cited above, explorations of anger and cardiovascular responding often involve the recall of an autobiographical anger memory. In accordance with Lang's bio-informational theory of emotion (Lang, 1977, 1979, 1983) activation of an emotion memory structure, if the activation is sufficient, should produce the efferent activity associated with that memory (Cuthbert, Vrana, & Bradley, 1991) albeit at an attenuated level. As anger provocation research has established the likelihood of observing reactivity in diastolic blood pressure it is sensible that anger recall studies would (and do) observe the same.

Verbal Expression of Anger

Earlier anger verbalization work (Lynch, 1985) established a general association between anger speech behavior and elevated systolic blood pressure (SBP), diastolic

blood pressure (DBP), and heart rate (HR). Subsequent investigations have varied the circumstances, such as the topic of discussion, mode of speech, or manner of anger speech induction under which speech behavior affects CVR (Finney et al., 2002; Friedman et al., 1982; Siegman et al., 1990, 1992).

In a particular line of speech research investigating the role of verbalized anger in eliciting CVR (Siegman et al., 1990; Siegman & Boyle, 1993) the researchers conceptualize the role of vocal behavior as a feedback mechanism that incites greater physiological responding and increased anger affect. Siegman and Snow (1997, p.31) expound this idea, stating "…loud and rapid speech is critical for the cardiovascular manifestation of anger, and that its (speech's) attenuation or elimination will attenuate or eliminate the normal cardiovascular manifestations of anger". To support this assertion the researchers compared anger out (speaking in a loud and rapid speech style) versus anger in (reliving or thinking about anger) using recalled personal anger memories. As hypothesized, the verbalizing condition produced significantly greater blood pressure and heart rate elevations when compared to the reliving condition. Moreover, during the reliving condition, HR and BP levels were not significantly different from baseline levels (Siegman & Snow, 1997).

It is logical to suggest the elevations of CVR during the spoken anger condition are a function of increased physiological activity associated with speech. To refute this proposition the authors cite prior literature (Siegman et al., 1990; Siegman & Boyle, 1993; Siegman, Dembroski, & Crump, 1992) showing CVR levels increase minimally when using an angry speech style (fast and loud) in the discussion of *neutral* topics.

Siegman and Snow (1997) conclude angry speech behavior is integral in the relationship of anger and cardiovascular responding, and implicate angry speech style (fast and loud) as the pathogenic link between anger and poor health. It is implied the inner experience of anger is simply insufficient to elicit substantive increases in cardiovascular responding.

Using this same paradigm, Drummond and Quah (2001) manipulated speech style in an experiment comparing spoken anger in Chinese and Caucasian men. Although the results partially support the work of Siegman's group, a critical disparity emerged: anger had a greater impact on blood pressure during the *soft and slow* speech condition than the loud and fast condition. Furthermore, the effect on blood pressure of using a loud and fast speech style to describe an anger event versus reading neutral material, was negligible. Drummond and Quah (2001) conclude that the impact of a loud and fast speech style on blood pressure is primarily a metabolic one.

Sargent, Flora, and Williams (1999) varied the circumstances of anger verbalization by manipulating speech style during a dyadic encounter. This study yielded no significant differences in the physiological effect of anger during the varying speech conditions although participants did rate their affect as angriest during loud and fast anger verbalization. These latter two findings call into question the necessity of manipulating speech style in pursuit of anger verbalization's effect on blood pressure. Taking into account these results, and in an effort to more closely approximate a natural style of verbalizing an anger memory, the present study is conducted without the manipulation of speech style.

A de facto assumption in the anger speech research seems to be that if people with cardiovascular disease evidence more anger and hostility, and anger evokes increases in cardiovascular responding, then "angrier" (those with higher hostility and greater incidence of anger) people must experience greater CVR during anger, or at least more frequent bouts with anger elevated CVR (which then contributes to the development of cardiovascular disease). Contrary to that expectation, Finney et al., (2002) reported that African-American men *low* in hostility displayed significantly *greater* reactivity than high hostility African-American men and Caucasian men of any level of hostility, though on the whole, all participants experienced significant increases in blood pressure and heart rate during a stressful speech task. Similarly, Anderson, Linden, and Habra (2005) found that systolic blood pressure and heart rate during anger provocation were greatest in the participants lowest in hostility.

While Finney et al., (2002) suggest that low hostility is a liability that results in increased stress reactivity (in African-American men), it could be argued that high hostility is accompanied by a restriction in cardiovascular responding during spoken anger that is a sign of emotion dysregulation. The latter interpretation is consistent with results from alexithymia research that shows higher levels of alexithymia (which is associated with difficulty regulating emotion) are associated with lower levels of physiological responding during emotional stressors, including recalled verbalized anger (Linden, Lenz, & Stossel, 1996; Neumann, Sollers, Thayer, & Waldstein, 2004; Newton & Contrada, 1994; Wehmer, Brejnak, Lumley, & Stettner, 1995). Thus attenuated heart rate and blood pressure during spoken anger (in alexithymics) is seen as evidence of

autonomic nervous system dysregulation (Neumann et al., 2004), and heightened heart rate and blood pressure during anger may be viewed as appropriate mobilization of physiological resources that may facilitate coping with the stressor.

Imagining Anger

In contrast to anger studies conducted by investigators specializing in speech research, emotion researchers who focus on anger *imagery* have been successful in provoking a change in cardiovascular response even though anger is not vocalized. Stemmler and colleagues (2001) compared cardiovascular response to anger provocation in the laboratory (real-life anger) with later recall (imagery) of the anger incident finding both the real-life anger and the imagined anger produced significant increases in SBP and DBP. However the imagined anger, while manifesting the same physiological pattern present during real-life anger, showed a restricted cardiovascular effect compared to real-life anger. Interestingly, the anger control group, who received the same directions as the treatment group but who were told the anger provocation was not earnest, also experienced significant increases in blood pressure during the imagery condition even though their reactivity was minimal during the real-life condition.

Much anger imagery research has been conducted in an effort to use physiological correlates and levels of responding to differentiate emotion states. Sinha, Lovallo, and Parsons (1992), analyzed changes in blood pressure during recalled (autobiographical) emotional states and found that anger imagery elicited the largest cardiovascular change as compared to fear, joy, and sadness. Significant reactivity was present in systolic blood pressure changes, however, the increases in heart rate and systolic blood pressure were

not significantly different when comparing the emotion states. Measures of diastolic blood pressure indicated anger imagery elicited significant increases as compared with fear, joy, sadness, action, and neutral imagery. Overall, the greatest cardiovascular increases (HR, DBP, and SBP) were observed during anger imagery. Similarly, a study exploring the physiological correlates of emotions, including anger during imagery conditions, found that blood pressure increased significantly during recall of a personalized anger memory (Prkachin et al., 1999).

Though the results of these imagery/emotion studies are contrary to conclusions drawn in anger verbalization studies, which suggests that verbalization is necessary to produce increases in CVR (Siegman & Snow's 1997), there are salient differences in methodology that could account for the disparity. In studies restricted to imagery conditions, participants were either screened for greater imagery ability (Sinha et al., 1993) or screened for above average emotional intensity (Prkachin et al., 1999). Additionally, imagery studies frequently make use of prepared scripts that, even when autobiographical (Prkachin et al, 1999; Sinha et al, 1993), are often manipulated by the experimenters to include physiological response propositions in accord with Lang's bio-informational theory of emotional imagery (Lang, 1979; Lang, Kozak, Miller, Levin, & McLean, 1980). Furthermore, imagery/emotion studies use continuous heart rate and blood pressure monitoring (Beckham et al., 2002; Lang et al., 1980; Prkachin et al., 1999; Sinha et al., 1993; Vrana & Rollock, 2002) whereas the lone example of a study directly comparing anger verbalization and imagery (Siegman & Snow, 1997) manually monitored heart rate and blood pressure at two minute intervals and then averaged the

values. This infrequent monitoring in comparison to continuous monitoring may allow significant changes in cardiovascular responding to go unnoticed.

Foster, Webster, and Smith (1997) did find that imagery alone was sufficient to provoke a significant physiological reaction during recalled emotion, and they did not select participants based on imagery ability, or use prepared scripts. However, blood pressure was not one of the physiological correlates measured. Though participants recalled a variety of emotions (fear, anger, joy, sadness, and embarrassment) a limited number of physiological variables were recorded, namely skin resistance and heart rate. While each emotion evoked a significant physiological response, the level of response did not differentiate the emotions. This is similar to earlier findings by Vrana (1994) where both anger and disgust imagery, but not neutral imagery, brought about increases in heart rate.

Vrana and Rollock (2002) explored imagery and emotion using prepared anger scripts and found that while participants experienced significant increases in heart rate when comparing anger imagery to neutral imagery, significant increases were not observed in blood pressure. However, the lack of significant blood pressure reactivity during anger could be due to lack of power; blood pressure was only collected for 22 participants as opposed to the full sample (112 participants) used in the heart rate analysis.

In a study exploring CVR during anger versus neutral imagery in a population (primarily undergraduates) screened for high trait anger (Lineberger, 2004), non-significant increases in blood pressure during anger imagery were found even though

anger scripts were personalized and participants underwent response training (verbal reinforcement of physiological or behavioral descriptions: see Miller, Levin, Kozak, Cook, Mclean, & Lang [1987] for details).

The results of these two studies (Finney et al., 2002; Lineberger, 2004) with regard to anger imagery and blood pressure changes (or lack thereof), are inconsistent with prior work relating anger imagery to significant diastolic and systolic blood pressure increases (Prkachin et al., 1999; Sinha et al., 1993). One plausible explanation for this inconsistency is that the Finney and Lineberger studies did not rely upon screening participants based upon greater imagery ability (Sinha et al., 1993) or higher levels of emotional intensity (Prkachin et al., 1999) which may have, in a sense, stacked the deck in the latter two studies.

In an anger imagery study using the same participants as this proposed project (Beckham et al., 2002), Vietnam veterans with Posttraumatic Stress Disorder (PTSD) had increased levels of covert hostility and exhibited greater heart rate and blood pressure during anger imagery than veterans without PTSD. This study suggests the importance of conducting population-specific studies and the significance of measuring existing levels of hostility when investigating anger.

Bio-informational Approach to Anger Memory

The bio-informational model of emotional imagery comes from Lang's blending of information processing theory with psychophysiology (Lang, 1977, 1979). While much of the research supporting the bio-informational model concerns fear, Lang's model is theorized to apply to other emotion as well (Lang, 1993).

According to Lang, an emotion is arranged in abstract data structures in long-term memory, and these structures are organized into a network of related information. The structures contain stimulus (descriptions), semantic (meanings), and response (behaviors that can be executed, with relevant physiological correlates) information (Lang, 1983). When incoming information matches information stored in the structures, and enough cues or cue intensity is present, the network is activated.

The emotion memory network acts as the "blue print" for behavior associated with that emotion (Foa and Kozak, 1986; 1998). For example, in a fear of dogs stimulus input (e.g. the snarling dog) is directly connected to response output (e.g. fleeing from the dog) and its accompanying physiological support (e.g. changes in heart rate or other physiological variables associated with the fleeing behavior; Bradley et al., 2001; Lang, 1994). While the physiological responses accompanying a fear memory are attenuated during imagery (vs. real-life), they mirror what is present during the actual situation (Lang, 1993) and, when recorded with electrophysiological equipment, represent a real-time index of processing of the fear memory.

Much of the foundational work in imagery (Anderson & Borkovec, 1980; Bauer & Craighead 1979; Lang et al., 1980) centered on the use of fear versus neutral imagery in phobic populations. Interestingly, fear imagery, while eliciting efferent activity in phobics, does not elicit the same level of physiological responding in non-phobic participants (Lang, Levin, Miller, & Kozak, 1983). This suggests the associative fear memory network is stronger or more easily accessed by phobics than by non-phobic controls. If anger memory networks function similarly, the implication is that anger

imagery could be sufficient to evoke significant efferent activity in an anger population but then fail to do so in non-anger controls. Consequently a comparison of the cardiovascular effects of verbalization of anger versus imagery of anger may produce different results when contrasting participants with and without elevated levels of anger or hostility.

Thus, based upon bio-informational theory, two alternative explanations can be generated for inconsistent anger imagery findings, or lack of significant cardiovascular efferents: there were insufficient cues or cue intensity to activate the anger memory network and (or) the associative strength of the anger memory networks of a *general* sample may not be strong enough to elicit efferent activity without concomitant verbal behavior.

The idea of insufficient cues for activation of an anger memory network during re-lived (imaginal) anger is not inconsistent with Siegman and Snow's (1997) conclusion that loud and fast anger speech during anger recall is necessary to provoke efferent output. It is plausible that without the cue of speaking quickly and loudly, in conjunction with anger content, increases in efferent output cannot be obtained or may be substantially diminished in magnitude so as to be statistically irrelevant. Given that the activation of fear networks differs between phobic and non-phobic participants (Lang, Levin, Miller, & Kozak, 1983), it is conceivable that anger networks would similarly differ between a population with known anger problems and non-anger participants. Although research has focused on the structure of fear memories, regarding anger memory as a similar structure is consistent with a bio-informational model (Lang, 1993).

Anger in a Posttraumatic Stress Disorder Population

Much of the anger research, in particular that which investigates anger speech

behavior and anger imagery, is limited to non-clinical samples (Siegman et al., 1990,

1992; Siegman & Snow, 1997; Prkachin et al., 1999; Sinha et al., 1992; Velasco & Bond,

1998; Stemmler et al., 2001). As useful as non-clinical samples may be in preventing

obfuscation of the variables under consideration, inferences are often made regarding

populations for whom anger is a salient characteristic.

It has been suggested that, "modifying a person's vocal behavior can short-circuit

the escalating nature of these emotions and neutralize their physiological consequences."

(Siegman & Boyle, 1993, pp. 436). Prior to vocalized behavior interventions, a useful

step is to investigate anger speech and imagery under more naturalistic conditions with a

more relevant (i.e., clinical) population to determine whether findings are replicated in a

population for whom anger is a particularly prominent feature, such as individuals with

Posttraumatic Stress Disorder (PTSD).

According to the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM*

IV-TR; American Psychiatric Association, 1994), anger is not a necessary component in

the diagnosis of PTSD, though it is present in criterion D as a symptom of hyperarousal

(Novaco & Chemtob, 1998). It has been suggested that the exclusion of anger and

aggression as a feature of PTSD was a conscious attempt to avoid re-victimization of the

diagnosed (Yehuda, 1999). Notwithstanding, a review of the literature suggests anger

and hostility may be integral in the development and maintenance of PTSD, or at least is

likely a co-morbid factor influencing the course and prognosis of PTSD (Beckham, Moore, & Reynolds, 2000).

In a prospective study of the development of PTSD after a motor vehicle accident, (participants completed assessment between one and seven days after the incident) anger cognition was among the variables predicting the development of PTSD even after controlling for such factors as magnitude of the injury and threat level experienced during the accident (Ehlers, Mayou, & Bryant, 1998). Anger cognition was also associated with PTSD chronicity.

Using a population of crime victims, assessed at one and six months post event, Andrews, Brewin, Rose, and Kirk (2000) found anger toward others a significant predictor of PTSD symptomology at the one month mark and that presence of PTSD at month one predicted presence of PTSD symptomology at month six. Additionally, higher levels of both subjective and expressed anger in participants with PTSD have been linked to lower levels of functioning and greater levels of depression (Franklin, Posternak, & Zimmerman, 2002). Further work exploring the role of anger in anxiety disorders found participants with PTSD report significantly greater anger compared with non-PTSD anxiety patients (Kotler, Iancu, Efroni, & Amir, 2001).

Research investigating the role of anger and PTSD in combat veterans is limited to correlational rather than prospective studies and was recognized in the literature as a feature of combat PTSD as early as WW II (see Novaco & Chemtob, 1998 for a review). Both population- and laboratory-based investigations have found combat veterans with PTSD are significantly angrier than veterans without PTSD (Beckham, Feldman, Kirby,

Hertzberg, & Moore, 1997; Beckham, Roodman, Barefoot, Haney, Helms, Fairbank, Hertzberg, & Kudler, 1996; Chemtob, Harnada, Roitblat, & Muraoka, 1994; Kulka, Shlenger, Fairbank, Hough, Jordan, Marmar, & Weiss, 1990; Lasko, Gurvits, Kuhne, Orr, & Pittman, 1994; Novaco & Chemtob, 2002).

In work looking at motivation to change in a chronic combat PTSD population, screening of patients revealed trait anger well above the norm for adult males (Rosen, Chow, Murphy, Drescher, Ramirez, Ruddy, & Gusman, 2001) and self-report indicated 75% of PTSD sufferers felt they "definitely have" anger problems. In a comparison of veterans with PTSD to veterans with "other" psychiatric conditions, those with PTSD scored significantly higher on the Assault, Irritiability, Negativism, and Verbal Hostility subscales of the Buss-Durkee Hostility Inventory (BDHI; Castillo, C'De Baca, Conforti, Qualls, & Fallon, 2002) suggesting that elevated anger is not an artifact of either veteran or psychiatric status alone.

Though combat experience provides the basis for trauma exposure contributing to the development of PTSD in this population, research suggests diagnostic status and anger are not mediated by the level or duration of this exposure (McFall, Wright, Donovan & Raskind, 1999; Novaco & Chemtob, 2002). This echoes the Lasko and colleagues (1994) work, which found greater levels of aggression across a variety of measures for combat veterans *with* PTSD. Lasko and colleagues suggest these veterans' hostile outlook colors their interpretations of events, thus increasing the likelihood of aggressive acts. Furthermore, McFall and colleagues noted veterans *with combat* exposure who did not develop PTSD were significantly less angry than PTSD combat

veterans. The authors propose pre-existing anger arousal may exist in combat veterans who later develop PTSD and suggest the inclusion of this risk factor in prospective studies.

An exploration of the course and chronicity of PTSD in combat veterans over a fourteen-year period found greater anger was associated with the presence of PTSD symptomology at baseline, and predicted continued symptomology at the fourteen-year mark (Koenen, Stellman, Stellman, & Sommer, 2003). In addition to identifying anger as a risk factor for chronic PTSD (Koenen et al., 2003) its presence is associated with symptom severity as well (Beckham et al., 1997).

This view of anger as affecting the course, chronicity, and severity of combat-related PTSD is consistent with research suggesting treatment as usual (cognitive-behavioral therapy alone) is not sufficient in treating patients, but rather, treatment specifically addressing anger should be supplemented (Chemtob, Novaco, Harnada, & Gross, 1997). Forbes, Creamer, Hawthorne, Allen, and McHugh (2003) found anger contributed significantly to the variance in PTSD symptom reduction, furthermore, participants highest in anger did not benefit from a cognitive-behavioral intervention, confirming an earlier finding that high levels of anger impede the treatment of PTSD in victims of sexual assault (Foa, Riggs, Massie, & Yarczower, 1995).

There is a question as to whether anger operates as an impediment to the processing of fear (Foa, Steketee, & Rothbaum, 1989; Foa, Riggs, Massie, Yarczower, 1995; Riggs, Dancu. Gershuny, Greenberg, & Foa, 1997), or is a concomitant factor associated with cognitive dysregulation (Chemtob et al., 1997; Novaco & Chemtob,

2002) either of which could account for anger's association with chronicity and symptom severity. Agreement exists mainly in the preponderance of evidence suggesting an association of anger with combat (and non-combat) related PTSD.

In addition to the relationship of self-reported anger to PTSD, investigators have also compared physiological manifestations of anger in combat veterans with and without PTSD. Combat veterans diagnosed with PTSD display more anger during non-verbally relived anger (imagery) than combat veterans without PTSD, as manifest in measures such as heart rate and blood pressure as well as self -report (Beckham, Vrana, Barefoot, Feldman, Fairbank, & Moore, 2002). Furthermore, veterans with PTSD become angry faster, experience higher diastolic blood pressure during anger imagery, and take longer to recover from anger imagery than veterans without PTSD (Beckham et al., 2002).

Statement of the Problem

In summary, this review addresses the discrepancy between the anger speech literature and anger imagery literature in investigations of the cardiovascular response to anger. While imagery research suggests imagining anger is sufficient to generate a change in heart rate and blood pressure, speech research suggests verbalization of anger is necessary to provoke significant increases in cardiovascular responding. Few studies exist that directly compare the within-subject cardiovascular effect of anger imagery to anger speech (Siegman & Snow, 1997). The exploration of these anger modalities has been organized around Lang's Bio-Informational Model of Emotion (1977, 1979, 1993) in an effort to provide a framework against which two seemingly disparate literatures may be reconciled.

In addition, most anger speech and imagery research involves non-clinical samples. The current study is intended to extend anger speech findings to a population for whom anger is a relevant characteristic. Extrapolation of bio-informational studies of fear to anger, imply anger memory networks may be more easily accessed by participants with known anger problems. If so, cardiovascular responding would likely be greater in PTSD than non-PTSD veterans. However, the few studies that have investigated the relationship of hostility and spoken anger (though still using non-clinical samples) show that greater hostility is associated with attenuated cardiovascular responding and may be a symptom or result of emotion dysregulation. Thus it is unclear whether veterans with PTSD will show more or less cardiovascular responding during spoken anger than veterans without PTSD.

Hypotheses

I. Comparing the Cardiovascular Response to Anger during Imagery and Speech

As mentioned above, some research suggests there is a synergistic relationship between anger and vocalization (Siegman, 1994; Siegman & Boyle, 1993; Siegman & Snow, 1997). Thus it is predicted that after controlling for the metabolic activity associated with speech, the anger verbalization condition will evoke greater cardiovascular responding than the anger imagery condition in both PTSD and non-PTSD veterans. Although verbalized anger may elicit greater cardiovascular responding, it is also predicted that the anger imagery condition will still be sufficient to elicit a significant increase from baseline in the full sample of veterans.

II. Cardiovascular Response to Spoken Anger in PTSD and non-PTSD Veterans

Increases in systolic and diastolic blood pressure are seen when participants discuss an autobiographical or laboratory induced anger memory. It was hypothesized that both PTSD and non-PTSD veterans would experience significant increases in cardiovascular responding from neutral speech to anger verbalization.

It is unknown what the cardiovascular effect of verbalized anger is on participants with PTSD, who have elevated levels of anger and hostility in comparison to controls. Whereas a bio-informational approach to anger memory would suggest that veterans with PTSD would display greater cardiovascular response to spoken anger, an emotion dysregulation approach would suggest that veterans with PTSD would display an attenuated cardiovascular response to vocalized anger when compared to veterans without PTSD. Therefore it was predicted that if the bio-information approach to spoken

anger is accurate then veterans with PTSD will show greater CVR during anger speech than veterans without PTSD; on the other hand, if the emotion dysregulation approach to spoken anger is accurate, then veterans with PTSD will show attenuated CVR during anger speech compared to veterans without PTSD.

III. Cardiovascular Recovery from Spoken Anger in PTSD and non-PTSD Veterans

It is expected that elevated cardiovascular responding from the anger verbalization procedure will begin to diminish during the recovery from the anger period. It was predicted that if the bio-information approach to spoken anger is accurate then veterans with PTSD will show greater CVR during anger speech recovery than veterans without PTSD; on the other hand, if the emotion dysregulation approach to spoken anger is accurate, then veterans with PTSD will show attenuated CVR during anger speech recovery compared to veterans without PTSD.

Method

Participants

Ninety-nine male Vietnam combat veterans, 48 with PTSD and 51 without PTSD, participated in the study. Participants were solicited for a study concerning health and hostility at the Durham Veterans Affairs Medical Center in Durham, North Carolina (Beckham et al., 2001 & 2002) and were administered a demographic questionnaire to obtain age, race, current medications, and socioeconomic status (SES) (Hollingshead & Redlich, 1958), as well as completing the Combat Exposure Scale (CES; Keane et al., 1989), the Mississippi PTSD Scale (Kean, Caddell, & Taylor, 1988), and the Structured Clinical Interview for DSM-III-R criteria (SCID; Spitzer, Williams, Gibbons, & First; 1989) for Axis I disorders. Combat status and diagnosis were used to determine eligibility for study participation. Veterans who met the criteria for current alcohol or substance dependence, psychotic disorders, or lifetime PTSD in the control group, were excluded (Beckham et al., 2000).

Eighty-three consecutive outpatient males, diagnosed with PTSD, were invited to participate in the parent study. Five declined to participate, six were excluded due to high doses of medication with known cardiovascular effects ($\geq$ 100 mg Amitriptyline and/or other anticholinergic medication), one PTSD veteran did not complete the study, and three were ineligible due to positive drug screens (Beckham et al, 2000). Of the remaining 68 PTSD veterans, 48 had complete data available (both anger speech and anger imagery data) and were included in this study.

Veterans without PTSD were recruited through mailings to age appropriate patients who had sought services at the Veterans Affairs Medical Center within the last year. Local area Vietnam Veterans groups were also included in recruitment efforts. Sixty-seven responders met the study criteria for the parent study (i.e., Vietnam veteran, theater service, CES >0 and Mississippi PTSD Scale <90). Two participants were ineligible due to lifetime PTSD, one for alcohol abuse, and seven due to current PTSD symptoms (Beckham et al., 2000). Of the remaining 57 non-PTSD veterans, 51 had complete data available (both anger speech and anger imagery data) and were included in this study.

Analysis using independent samples t-tests (two participants were excluded from this analysis due to missing data) showed beta and calcium channel blockers were impacting participants' cardiovascular responding during speech conditions. For example, when comparing veterans taking beta blockers (n=12) to veterans not taking beta blockers (n=87), medicated veterans exhibited significantly lower cardiovascular responding than non-medicated participants during anger speech [SBP $t(97)=1.91$, $p=.05$; $M=151$ mm/Hg for non-medicated and $M=137$ mm/Hg for medicated participants; DBP $t(97)=2.32$, $p=.02$; $M=88$ mm/Hg for non-medicated and $M=79$ mm/Hg for medicated participants; HR $t(97)=2.19$, $p=.03$; $M=76$ for non-medicated and $M=69$ for medicated participants]. Thus, participants were excluded from further analyses if they were taking beta or calcium channel blockers. This resulted in the exclusion of ten participants from the PTSD group (nine taking beta-blockers and one taking calcium channel blockers) and nine participants from the non-PTSD group (three taking beta-blockers and six taking

calcium channel blockers). The remaining sample used in the analyses consisted of 38 participants in the PTSD group and 42 participants in the non-PTSD group.

Among the veterans being treated with hypertensive medication, PTSD veterans were more likely to be taking beta blockers and non-PTSD veterans were more likely to be taking calcium channel blockers. However, a chi-square test for independence using the Yates' Correction for Continuity showed the proportion of PTSD and non-PTSD veterans who were taking any medications known to reduce heart rate and blood pressure were similar [15.8% of the PTSD group and 14.3% in the non-PTSD group, χ^2(1, n=80)=0.00, p=1.0].

Materials

Participants completed the following measures of hostility: the Cook-Medley Hostility Scale (Cook & Medley, 1954), the Buss-Durkee Inventory (Buss & Durkee, 1957), the Spielberger Anger Expression Scale (Spielberger et al., 1985) and the Rotter Interpersonal Trust Scale (Rotter, 1967). Prior factor analysis of these four scales using this veteran sample yielded three factors: Covert Hostility, Overt Hostility, and Hostile Beliefs. In the prior study only Covert Hostility was significantly related to cardiovascular responding (Beckham et al., 2002).

Finger arterial pressure and heart rate were collected using an Ohmeda Finapres Blood Pressure Monitor (Model 2300). The finger cuff was affixed to the middle phalange of the middle finger of the non-dominant hand and the subject's arm was immobilized at heart level by a sling. Prior research has demonstrated the accuracy of the Finapres (Imholtz, Wieling, van-Montfrans, & Wesseling, 1998). Measures of heart rate,

systolic blood pressure, and diastolic blood pressure were recorded beat-by-beat on a microcomputer through a serial interface using custom software. The data were later converted to a real-time metric (Graham, 1978) for analysis.

Design and Procedure

Data used in this study were collected at the Durham Veterans Affairs Medical Center for the purpose of studying the cardiovascular responses to relived anger when that anger is thought about or imagined. This current study seeks to further explore the cardiovascular responses to anger using data collected during the recall (verbalized) session, during which information regarding the subject's autobiographical anger event/episode was collected. While physiological data was collected during this anger verbalization task, it has not been examined prior to this current study.

Data were collected across three sessions: an interview session, anger verbalization session, and anger reliving session.

Interview Session I: In Session one the SCID and aforementioned questionnaires (e.g. hostility measures) were administered as well as additional measures of health and affect that are not included in these results.

Anger Speech Session II: Participants sat in a reclining arm chair. Once the Finapres was in place, ten minutes of baseline readings were collected. At the conclusion of baseline readings the participants read aloud a neutral passage for ninety seconds followed by two minutes of reading recovery readings. The purpose of the neutral read aloud passage was to provide a baseline speech condition upon which to compare the

anger verbalization data. Toward the end of the two minutes of post reading recovery, instructions for anger memory retrieval/verbalization were given to the participants.

Participants were instructed to recall a personal anger memory concerning an event during which they felt angry, frustrated, or upset with someone and were unhappy with the conclusion (see Appendix A for detailed instructions). Once the subject retrieved a particular memory, the experimenter used prompts such as "What happened? Tell me everything about that day- time, place, weather, and so on. How were you feeling then? and What did you say?" The participants discussed the anger memory for a minimum of five minutes.

Upon completion of the anger retrieval periods, participants rated their anger, at the time of the event, on a scale of zero to ten, as well as their current level of anger using the same scale. Following ratings, five minutes of post-anger physiological data was collected and the anger speech protocol concluded.

Anger Imagery Session III: Participants were again seated in the reclining arm chair. Participants were able to view a video tape on a television screen through which the anger reliving instructions were presented. The experimenter explained that a "big X" would appear on the screen and they would hear the following instructions: "Now we want you to think about that time when you felt so angry you wanted to explode (see Appendix B for detailed instructions)." The anger memory that participants were asked to imagine/relive was the same anger memory discussed during anger verbalization in Session II.

After these instructions were reviewed, the Finapres was put in place and ten minutes of baseline physiological readings were collected. At the end of the ten minute baseline participants were given instructions to relax and … "Clear your mind. Don't think about anything in particular". During this time an "X" was displayed on the TV. After two minutes of relaxation participants were given ten seconds of instructions and told to begin reliving the anger. During this time a picture of fireworks was displayed on the TV. Also at that time the experimenter paused the video until the participant gave the pre-arranged signal that he was reliving the anger at which point the experimenter restarted the video. After receiving the anger signal from the participant, the experimenter played a recording of 90 seconds of ocean wave scenery, which indicated to the participant to stop reliving their anger. After the ocean wave scenery, five minutes of post-anger physiological data was collected and the anger reliving protocol was concluded.

Data Reduction & Statistical Data Analysis for Research Hypotheses

Dependent variables used in the analyses consisted of heart rate, systolic blood pressure, diastolic blood pressure, and self-reported anger ratings at the conclusion of the anger speech recall on a scale of zero to ten (zero= least angry, ten=most angry).

Data Reduction:

Physiological data were collected and reduced for the following time periods:

Anger Speech Session II: This session consisted of a ten minute baseline, 90 second read aloud of neutral passage, two minute reading recovery, anger verbalization for six minutes, and five minute anger recovery period. Once the physiological data (HR,

SBP, & DBP) were converted from beat-by-beat to a real-time metric (Graham, 1978) they were condensed in the following manner: the ten-minute baseline period was reduced to ten readings representing the mean for each minute; the 90-second neutral read aloud period was reduced to nine ten-second means; the two minute reading recovery was reduced to twelve ten-second means; the six minute anger verbalization period was reduced to thirty-six ten-second means; and the five minute anger recovery period was reduced to thirty ten-second means.

Not all participants spoke for the full six minutes of the anger verbalization condition (e.g. six PTSD and six non-PTSD subjects stopped recalling their anger between the five and six minute marks). Given that the length of time verbalizing anger varied among participants, but that all participants engaged in at least five minutes of anger speech, data were used from the first five minutes of the anger verbalization period in the analyses. Due to experimenter variation, the anger recovery period was shortened for thirteen participants. Each participant completed at least four minutes and twenty seconds of the anger recovery period; the first four minutes of recovery data were used in the analyses.

Anger Imagery Session III: This session consisted of a ten minute resting baseline, two minutes of pre-anger relaxation, the variable length anger imagery period, 90 seconds of post-anger ocean wave scenery video, and a five minute anger recovery period. Once data were converted from beat-by-beat to a real-time metric (Graham, 1978) they were condensed in the following manner: the ten minute resting baseline period was reduced to ten readings representing the mean (HR, SBP, & DBP) for each

minute of data; the two minute relaxation/pre-anger period was reduced to twelve, ten second means; the imagery period was reduced to nine, ten second means from the first 90 seconds of imagery data; the 90 seconds of post-anger ocean wave scenery was reduced to nine, ten second means; and the five minute anger recovery period was reduced to five, one minute means.

Because the length of the imagery period varied for each individual, for this study the mean DBP, SBP, and HR for each person's last sixty seconds prior to raising their finger (to indicate they were feeling anger) was calculated and used in the analyses. It is this last sixty seconds of each participant's data that constitutes the anger imagery period referred to in the remainder of the study.

Data Analysis:

Manipulation Check: The purpose of this analysis was to verify the physiological effect of the anger verbalization task. Mean heart rate and blood pressures for each condition (baseline, neutral read aloud, reading recovery, anger verbalization, and anger recovery) were calculated. For each dependent variable (HR and BPs) a repeated measures ANOVA, using all veterans, was conducted. Greenhouse-Geisser corrected p-values and ε are reported to correct for violations of the sphericity assumption. It was expected that cardiovascular activity would increase from initial baseline during reading of the neutral passage and decrease during the post-reading recovery. After reading recovery, it was predicted that cardiovascular responding would rise during anger verbalization, to a level greater than that present during the neutral reading passage, and again decrease during the recovery period.

I. Comparing the Cardiovascular Response to Anger During Imagery and Speech

This step of analysis focused on comparing the last minute of anger verbalization and last minute of anger imagery conditions in the entire sample of veterans. First, change scores for each subject for each dependent variable (HR and BPs) in the anger verbalization condition were created by calculating the difference in means between the last minute of anger verbalization data and the neutral reading passage . The purpose of creating this change score was to control for the metabolic activity associated with speech.

Second, change scores for each subject for each dependent variable (HR and BPs) in the anger imagery condition were created by calculating the difference between the means of the last minute of anger imagery and the last minute of resting baseline data. The purpose of creating this change score was to control for baseline metabolic activity. For each dependent variable (systolic and diastolic blood pressure and heart rate) a paired samples t-test was used to compare cardiovascular responding during anger verbalization and anger imagery in the full sample of veterans.

In addition to analyzing differences between anger verbalization and anger imagery, cardiovascular responding (SBP, DBP, and HR) during the pre-anger imagery resting baseline was compared to cardiovascular responding during anger imagery to confirm that heart rate and blood pressure had increased significantly from baseline while reliving anger. For each dependent variable (systolic and diastolic blood pressure and heart rate) a paired samples t-test was used to compare cardiovascular mean responding

during the last minute of the pre anger imagery baseline to cardiovascular mean responding during the last sixty seconds of anger imagery in the full sample of veterans.

II. Cardiovascular Response to Spoken Anger in PTSD and non-PTSD Veterans

The purpose of this analysis was to compare the differences in cardiovascular response during neutral speech and anger speech and to compare the cardiovascular responding between the two groups during anger speech. Mean heart rate and blood pressures were analyzed in separate 2 Group (PTSD vs. non-PTSD) X 2 Speech (neutral read aloud vs. anger speech) ANCOVAs using the last minute of resting baseline data for SBP, DBP, and HR as covariates in the respective analyses.

A significant main effect for speech was predicted, with greater cardiovascular responding occurring during the anger verbalization condition than the neutral read aloud condition. In addition, it was predicted that the two groups would differ in the magnitude of their cardiovascular response during anger verbalization. Specifically, it was predicted that if the bio-information approach to spoken anger was accurate then veterans with PTSD would show greater CVR during anger speech than veterans without PTSD; on the other hand, if the emotion dysregulation approach to spoken anger was accurate, then veterans with PTSD would show attenuated CVR during anger speech compared to veterans without PTSD.

Veterans self-reported anger ratings were also expected to differ. On a scale of zero to ten, with zero equaling least angry and ten equaling most angry, it was predicted that veterans with PTSD would report greater anger during spoken anger than veterans without PTSD. Anger ratings were analyzed using an independent samples t-test.

III. Cardiovascular Recovery from Spoken Anger in PTSD and non-PTSD Veterans

The final step of the analysis focused on comparing cardiovascular responding (SBP, DBP, and HR) between PTSD and non-PTSD veterans during the post anger verbalization recovery period. First, change scores were created using the mean from each minute during the anger recovery period and subtracting the mean from the last minute of resting baseline to show the magnitude of the remaining discrepancy, over time, between resting and recovery cardiovascular responding.

Secondly, heart rate and blood pressures were analyzed in separate 2 Group (PTSD vs. non-PTSD) X 4 Time (minute one change score, minute two change score, minute three change score, and minute four change score) ANOVAs. Greenhouse-Geisser correct p-values and ε are reported to correct for violations of the sphericity assumption. A significant main effect for time was predicted, with the magnitude of cardiovascular responding decreasing across the recovery period. In addition, it was predicted that the two groups would differ in the magnitude of their cardiovascular responding during the recovery period.

Results

Demographics

The demographic characteristics and statistical comparisons for the eighty participants (n=38 PTSD and 42 non-PTSD) are summarized in Table 1 on page 34. In the PTSD group, 44.7% were African-American (52.6% Caucasian) and in the non-PTSD group 23.8% were African-American (76.2% Caucasian). Veterans with PTSD had significantly less education and lower SES, a higher unemployment rate, and were more likely to have current major depression (as well as higher total scores in the Beck Depression Inventory though this analysis included 37 participants with PTSD and 19 without PTSD due to missing data).

Participants diagnosed with PTSD also had significantly greater combat exposure, lower scores on the Rotter Interpersonal Trust Scale, higher scores on the three hostility factors derived from factor analysis of the hostility inventories (see Beckham, Feldman, et al., 2000 for details of covert hostility, covert hostility, and hostile beliefs factors), and higher scores on the Buss-Durkee Inventory, the Anger In and Anger Out scores of the Spielberger Anger Expression Scale, and the Cook-Medley Hostility Scale. These differences in demographic characteristics and hostility/anger measures are consistent with the literature on disparities between Vietnam veterans with and without PTSD.

Preliminary Analysis: Paired samples t-tests were performed to investigate group differences in the baseline (including the neutral reading passage) measures of SBP, DBP, and HR. There were no significant differences on any cardiovascular measures

Table 1

Participant Characteristics

	PTSD Group (n-=38)			non-PTSD Group (n=42)				
	%	M	SD	%	M	SD	Test Statistic (df)	p
Age (in years)		49.50	3.68		49.71	4.56	t(78)=-.230	.81
Education (in years)		14.23	2.60		15.38	2.42	t(78)=-2.03	.04*
SES (Hollingshead)		48.13	15.89		34.02	13.57	t(78)=4.28	.00*
% African-American	44.7%			23.8%			$\chi^2(1)$=3.90	.04*
% Caucasian	52.6%			76.2%			$\chi^2(1)$=4.86	.02*
% married	73.7%			85.7%			$\chi^2(1)$=1.80	.17
% employed	57.9%			85.7%			$\chi^2(1)$=7.74	.00*
% Current Major Depression	15.8%			00.0%			$\chi^2(1)$=5.07	.02*
Combat Exposure		29.15	8.57		20.76	11.22	t(78)=3.73	.00*
Body Mass Index		28.07	3.86		28.77	4.41	t(78)=-.75	.45
Beck Depression Inventory-II Total		27.92	9.94		6.97	5.40	t(54)=8.53	.00*
Beck Depression Inventory-II Cognitive		18.57	7.38		2.90	3.39	t(54)=8.75	.00*
Beck Depression Inventory-II Somatic		9.36	3.46		4.03	2.90	t(54)=5.73	.00*
Covert Hostility Factor		0.52	0.58		-0.63	0.77	t(78)=7.44	.00*
Overt Hostility Factor		0.56	1.01		-0.31	1.01	t(78)=3.84	.00*
Hostile Beliefs Factor		0.82	0.81		-0.05	0.88	t(78)=4.60	.00*
Buss-Durkee Hostility Inventory		44.00	12.68		20.32	7.77	t(78)=10.17	.00*
Rotter Interpersonal Trust Scale		3.37	2.44		6.14	3.31	t(78)=-4.21	.00*
Spielberger Anger In		20.52	3.46		12.71	3.71	t(78)=9.70	.00*
Spielberger Anger Out		18.78	4.88		13.23	3.23	t(78)=6.04	.00*
Cook-Medley Hostility Scale		32.30	9.24		15.13	8.16	t(78)=8.82	.00*

An asterisk (*) indicates a significant difference between groups.

between the PTSD and non-PTSD veterans during the two separate ten minute baseline

conditions (both pre anger verbalization and pre anger imagery). Nor were there any

significant differences between the PTSD and non-PTSD veterans on any cardiovascular

measures during reading of the neutral passage (during the anger verbalization

experiment); see Table 2 on pg. 40 for baseline and neutral reading means.

Veterans showed consistency in cardiovascular responding across the two resting

baseline measures (both pre anger verbalization and pre anger imagery). Scatterplots

were first examined to check for violations of linearity and homoscedasticity assumptions

and for the presence of outliers. Correlations were large and positive for SBP [r=.78,

n=80, p=.01], DBP [r=.84, n=80, p=.01], and HR [r=.93, n=80, p=.01].

For all subsequent analyses, relevant preliminary assumption testing was

conducted as needed to check for normality, linearity, univariate outliers, homogeneity of

variance-covariance matrices, and multicollinearity, with no serious violations noted.

Manipulation Check: The purpose of this analysis was to verify the physiological

effect of the anger verbalization manipulation. This involved exploring changes in

systolic and diastolic blood pressure, and heart rate across the five conditions (baseline,

neutral read aloud, reading recovery, anger verbalization, and anger recovery; see Figures
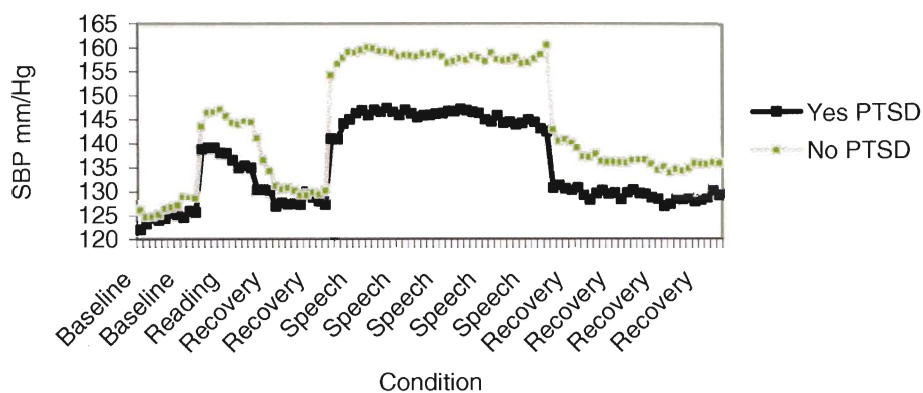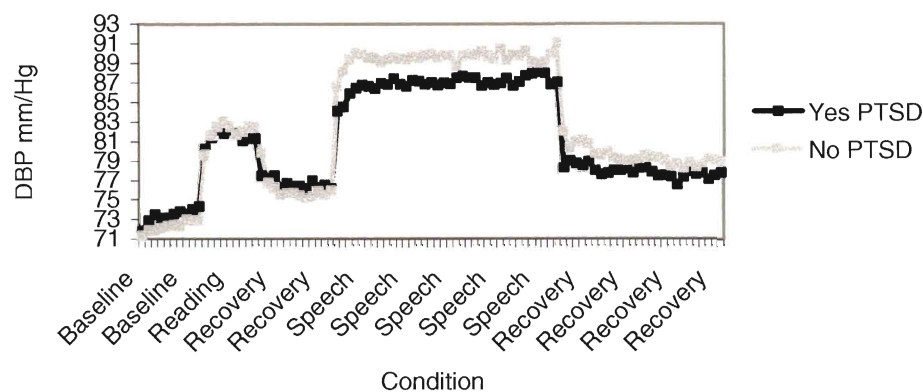
Figure 1: Systolic BP/ Anger Verbalization Task

Figure 2: Diastolic BP/ Anger Verbalizaton Task

Figures 1-3: Display SBP , DBP, and HR means for PTSD and non-PTSD veterans across the five conditions. Baseline reflects 10, one min. means; means for all other time periods were calculated for every ten seconds of data. As described under heading Session I of the Data Reduction section, to handle missing data the anger speech period was shortened by 30 sec, thus data are not continuous between the anger speech and anger recovery conditions. Although baseline means were comparable, participants without PTSD achieved greater increases in SBP, DBP, and HR during read aloud and anger verbalization conditions than did participants with PTSD.

Figure 3: Heart Rate/ Anger Verbalization Task
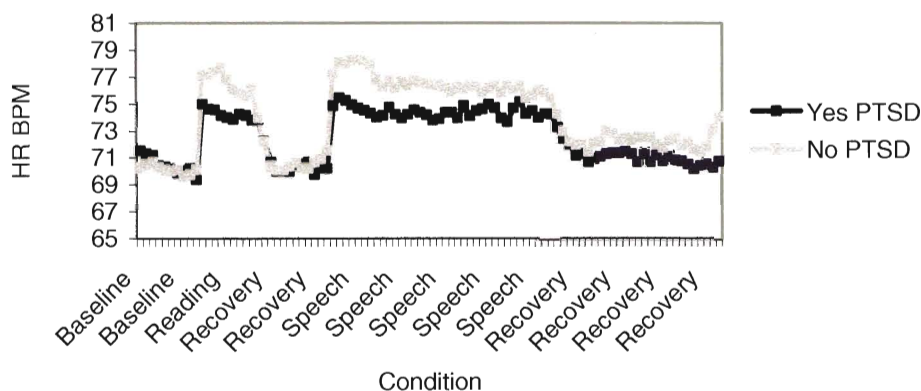
1-3, pg. 38). The first column in Table 2 (pg. 40) contains means and standard deviations for SBP, DBP, and HR for each period for the entire sample of veterans.

The results support the effectiveness of the verbalization paradigm in eliciting significant changes in cardiovascular responding when participants recall and voice an autobiographical anger memory. Separate repeated measures one-way ANOVAS, across each of the five conditions, indicated there was a significant change in the level of cardiovascular responding across conditions for all three cardiovascular variables [SBP, $F(4,76)=57.28$, $p=.00$, partial eta squared=.75, $\varepsilon=0.741$; DBP, $F(4,76)=99.70$, $p=.00$, $\varepsilon=0.758$, partial eta squared= .72; and HR, $F(4,76)=40.73$, $p=.00$, $\varepsilon=0.743$, partial eta squared=.68].

Post hoc analysis using the Simes variation on Bonferroni (Simes, 1986) revealed, as predicted, that heart rate and blood pressure increased significantly from baseline to reading the neutral passage and then decreased significantly during the rest period after reading. After post-reading rest, heart rate and blood pressure again increased

Table 2

Means and Standard Deviations of Cardiovascular Variables During Anger Verbalization Experiment

	Full Sample (n=80)		PTSD Veterans (n=38)		non-PTSD Veterans (n=42)	
	Mean	Std. Dev.	Mean	Std. Dev.	Mean	Std. Dev.
Systolic Blood Pressure:						
Baseline	127.26 $_A$	21.26	124.44	22.54	126.76	18.06
Neutral Read Aloud	141.39 $_C$	24.97	137.18	26.76	145.19	22.86
Reading Recovery	130.30 $_{AB}$	19.86	128.39	19.68	132.04	20.11
Anger Verbalization	152.16 $_D$	24.63	145.40	21.78	158.28	25.69
Anger Recovery	133.24 $_B$	18.20	129.17	15.55	136.92	19.77
Diastolic Blood Pressure:						
Baseline	73.54 $_B$	11.73	73.34	12.53	72.26	10.46
Neutral Read Aloud	81.71 $_A$	12.92	81.42	14.38	81.97	11.62
Reading Recovery	76.32 $_C$	10.84	76.63	11.15	76.04	10.67
Anger Verbalization	88.16 $_D$	12.20	86.68	12.39	89.55	12.02
Anger Recovery	78.72 $_A$	10.52	77.87	10.77	79.50	10.37
Heart Rate:						
Baseline	69.78 $_C$	10.26	70.45	9.94	70.17	10.01
Neutral Read Aloud	75.55 $_A$	10.11	74.27	9.73	76.71	10.43
Reading Recovery	70.86 $_B$	9.42	70.74	9.38	70.97	9.57
Anger Verbalization	75.62 $_A$	9.97	74.43	9.44	76.70	10.43
Anger Recovery	71.79 $_B$	9.74	71.16	9.73	72.36	9.83

Note: Blood pressure (BP) units are mm/Hg, and heart rate units are beats per minute. Within each cardiovascular variable, if conditions share a common subscript they are not significantly different. All significance levels were $p \leq .03$.

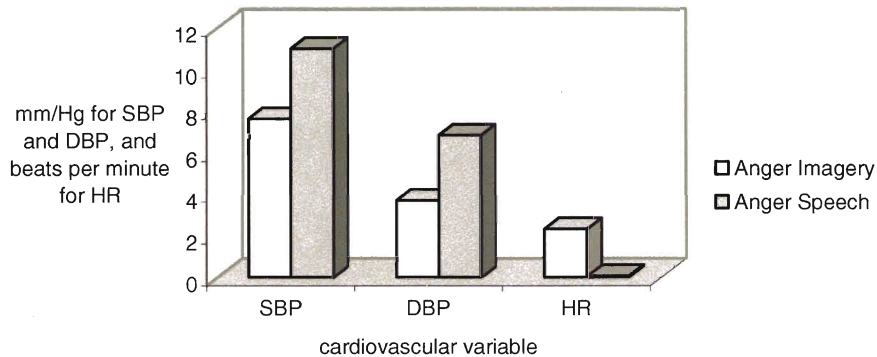Figure 4: Change Scores for Anger Imagery & Anger Speech

Figure 4: Displays SBP, DBP, and HR change scores for anger imagery and anger speech. DBP differences are significant (p=.004) and SBP differences approach significance (p=.071) in the expected direction; blood pressure is greater during anger speech than anger imagery even after controlling for the metabolic activity associated with speech. HR differences are also significant (p=.001), though not in the expected direction; this is explained in the text as a consequence of similar levels of heart rate responding during neutral and anger speech.

significantly during verbalization and then decreased significantly during the post-anger recovery period.

   I. Comparing the Cardiovascular Response to Anger During Imagery and Speech: In this hypothesis it was predicted that the anger verbalization condition would evoke greater cardiovascular responding than the anger imagery condition, across the entire sample of veterans, even after controlling for baseline metabolic activity. Separate paired samples t-tests using change scores (as outlined in the Data Reduction Section: mean anger verbalization minus mean neutral reading passage and mean anger imagery minus mean resting baseline) were conducted to evaluate whether cardiovascular responding in the imagery and verbalization conditions differed.

   As predicted, the results showed that verbalizing an anger memory results in greater increases in blood pressure than anger imagery, even after controlling for the

Figure 5: SBP Resting Baseline and Anger Imagery
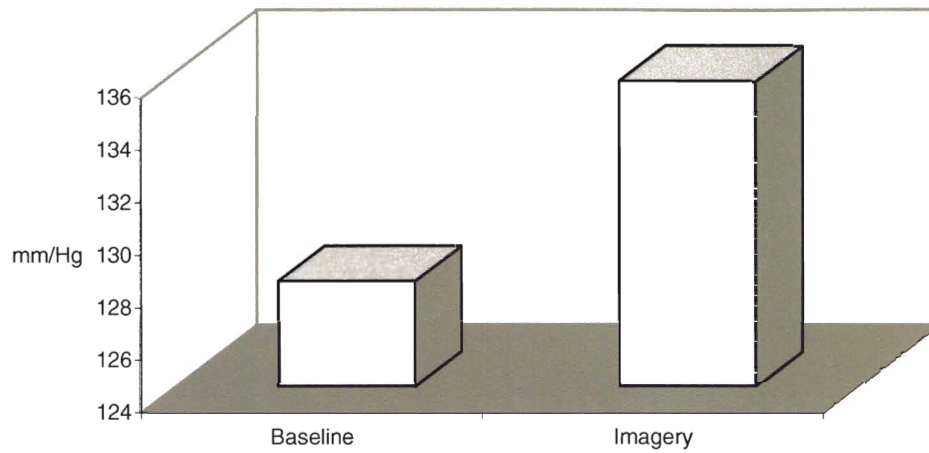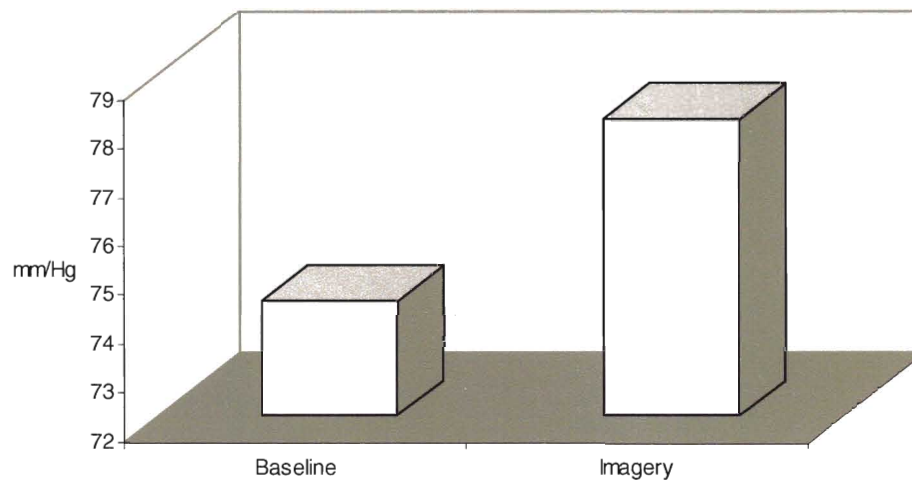

Figure 6: DBP Resting Baseline and Anger Imagery

Figures 5-7: Display the means for the full sample of veterans for SBP, DBP, and HR during the last minute of the resting baseline and the last minute of anger imagery. Differences between resting baseline and anger imagery for each cardiovascular variable are significant at $p < .01$.

Figure 7: HR Resting Baseline and Anger Imagery
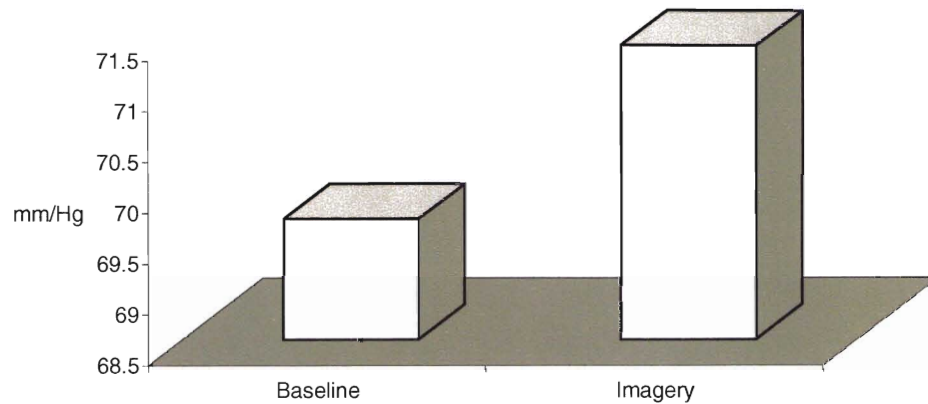
metabolic activity associated with speech (see Figure 4 pg. 41). This finding was significant for diastolic blood pressure [$t(79)=-2.94$, $p=.004$, eta squared=.09] and approached significance for systolic blood pressure [$t(79)=-1.82$, $p=.071$, eta squared=.04].

Contrary to the findings involving blood pressure, heart rate was greater during anger imagery than it was during anger verbalization (imagery change score M=2.33 beats/min, SD=3.97, verbalization change score M=.073 beats/min, SD=3.77) and this effect was significant [$t(79)=3.54$, $p=.001$, eta squared=.14]. The finding that heart rate was greater in imagery than in verbalization was primarily due to comparable levels of reactivity during both speech conditions. As can be seen in Figure 3 (pg. 39), in both the neutral and anger speech conditions heart rate reactivity was similar (M=75.52 beats/min, during neutral speech; M=75.62 beats/min, during anger speech). Conversely, heart rate during the last minute of pre-imagery resting baseline (M=69.06 beats/min) and the last

minute of anger imagery (M=71.40 beats/min) differed significantly and resulted in a greater residual change score, $\underline{t}$(79)=-5.26, $\underline{p}$<.001.

Although anger speech elicits greater CVR than anger imagery, it was predicted that anger imagery would still elicit significantly greater CVR than resting baseline. To evaluate this hypothesis, separate paired samples t-tests were conducted comparing mean responding during the last minute of pre-anger imagery resting baseline (for the entire sample of veterans) to mean responding during the last minute of anger imagery. As expected, cardiovascular responding showed a significant increase from resting baseline to anger imagery (see Figures 5-7, pg. 42). This finding was significant for systolic blood pressure [$\underline{t}$(79)=-6.77, $\underline{p}$<.01, eta squared =.36], diastolic blood pressure [$\underline{t}$(79)=-5.64, $\underline{p}$<.01, eta squared=.29], and heart rate [$\underline{t}$(79)=-4.23, $\underline{p}$<.01, eta squared=.18].

II. Cardiovascular Response to Spoken Anger in PTSD and non-PTSD Veterans: In the second hypothesis it was predicted that cardiovascular responding would be greater during anger speech than neutral speech. It was also predicted that if the bio-information approach to spoken anger was accurate then veterans with PTSD would show greater CVR during anger speech than veterans without PTSD; on the other hand, if the emotion dysregulation approach to spoken anger was accurate, then veterans with PTSD would show attenuated CVR during anger speech compared to veterans without PTSD. To evaluate whether cardiovascular responding differs between the two groups and two speech conditions separate 2 (PTSD vs. non-PTSD) X 2 (neutral read aloud vs. anger verbalization) ANCOVAs were conducted on systolic blood pressure, diastolic blood

pressure, and heart rate. The last minute of resting baseline data for SBP, DBP, and HR was entered as a covariate in the respective analyses.

As expected, the results demonstrate that talking about an anger memory produces greater increases in blood pressure than neutral speech (see Table 2, pg. 38, for means). While the speech (neutral read aloud vs. anger verbalization) main effect was significant for SBP [$F(1,77)=14.29$, $p=.00$; partial eta squared= .16] and DBP [$F(1,77)=11.67$, $p=.001$; partial eta squared=.13] there was not a significant main effect for HR [$F(1,77)=0.45$, $p=.50$].

This analysis also showed that combat veterans *without* PTSD had significantly higher blood pressure during the speech conditions than did combat veterans with PTSD. Again, this main effect (PTSD vs. non-PTSD) was significant for SBP [$F(1,77)=9.00$, $p=.004$, partial eta squared= .11] and DBP [$F(1,77)=5.25$, $p=.025$, partial eta squared= .06] but not for HR [$F(1,77)=1.20$, $p=.28$].

It was predicted that the PTSD and non-PTSD veterans would manifest differing levels of cardiovascular responding specifically during the anger verbalization condition (see Table 2, pg. 38, for means). There was a trend toward significance in the Group x Condition interaction for systolic and diastolic blood pressures [for SBP: $F(1,77)=2.63$, $p=.10$, partial eta squared=.03; for DBP: $F(1,77)=2.18$, $p=.14$, partial eta squared=.02], however these analyses were underpowered (power equaled .36 for SBP and .30 for DBP).

Given the lack of power to detect an interaction effect, follow-up analyses were done for exploratory purposes and should be interpreted with caution. Separate PTSD v.

Table 3

Mean Levels and Standard Deviations of PTSD, non-PTSD, and Combined Groups for Cardiovascular Variable Change Scores During Recovery from Anger Verbalization

	PTSD Group (n=38)		non-PTSD Group (n=42)		Full Sample (n=80)	
	M	SD	M	SD	M	SD
Systolic Blood Pressure:						
Minute 1 Recovery	4.82	16.65	11.53	16.06	8.35 $_A$	16.58
Minute 2 Recovery	3.61	17.32	7.92	14.89	5.87 $_B$	16.13
Minute 3 Recovery	3.72	16.27	7.31	13.73	5.60 $_B$	15.00
Minute 4 Recovery	2.26	15.14	6.14	13.77	4.30 $_B$	14.48
Diastolic Blood Pressure:						
Minute 1 Recovery	4.25	7.25	8.01	7.57	6.22 $_A$	7.61
Minute 2 Recovery	3.57	7.46	6.55	7.10	5.13 $_B$	7.38
Minute 3 Recovery	3.47	8.17	6.36	6.92	4.99 $_B$	7.63
Minute 4 Recovery	3.06	7.11	5.86	6.98	4.53 $_B$	7.14
Heart Rate:						
Minute 1 Recovery	2.42	3.78	2.37	5.41	2.39 $_A$	4.68
Minute 2 Recovery	1.94	3.39	2.42	4.46	2.19 $_{AB}$	3.97
Minute 3 Recovery	1.67	3.47	2.19	4.28	1.94 $_{AC}$	3.90
Minute 4 Recovery	1.15	3.52	1.95	4.07	1.57 $_C$	3.82

Note: Blood pressure (BP) units are mm/Hg, and heart rate units are beats per min. Within each cardiovascular variable, if minutes of recovery share a common subscript they are not significantly different. All significance levels were $p \leq .01$ with the exception of heart rate where the significance level was $p = .03$.

non-PTSD (for both neutral and anger speech conditions) ANCOVAs using the last minute of baseline systolic and diastolic blood pressures as covariates were undertaken. There was no significant group difference in blood pressure during neutral speech for SBP [$F(1,77)=2.37$, $p=.13$, partial eta squared= .03] or DBP [$F(1,77)=1.92$, $p=.17$, partial eta squared= .02]. Veterans without PTSD (for SBP M=158 mm/Hg, SD=25 mm/Hg; for DBP M=89 mm/Hg, SD=12 mm/Hg) had greater systolic and diastolic blood pressure during anger verbalization than veterans with PTSD (for SBP M=145 mm/Hg, SD= 21 mm/Hg; for DBP M=86 mm/Hg, SD=12 mm/Hg). This effect was significant for both SBP [$F(1,77)=7.33$, $p=.008$, partial eta squared= .09] and DBP [$F(1,77)=6.50$, $p=.013$, partial eta squared= .08].

If it is accurate that veterans without PTSD had significantly greater blood pressure during anger verbalization, this finding stands in contrast to differences in the experience of anger. It was predicted that veterans with PTSD would report greater anger during spoken anger than veterans without PTSD. On a scale from zero to ten (zero=least anger, ten=most anger), veterans *with* PTSD (M=5.05, SD=2.27) reported more anger during the anger verbalization task than did veterans without PTSD, [M=3.74, SD=1.9; $t(78)=2.81$, $p=.006$, eta squared=.09].

III. Cardiovascular Recovery from Spoken Anger in PTSD and non-PTSD Veterans: In the final hypothesis it was predicted that heart rate and blood pressure would decrease throughout the four minute post anger verbalization recovery period and that there would be differences between the PTSD and non-PTSD veterans during this recovery. Change scores were created subtracting the mean from the last minute of

resting baseline from the mean of each minute of post anger recovery (for a total of four change scores per physiological variable). These change scores were then entered into separate 2 Group (PTSD vs. non-PTSD) X 4 Time (change scores across recovery) repeated measures ANOVAs for each cardiovascular variable.

Consistent with the prediction, cardiovascular responding decreased considerably during the recovery period. This effect was significant for all of the cardiovascular measures: for SBP [F(3,76)=7.74, p=.00; partial eta squared=.23, ε=.667], for DBP [F(3,76)=7.72, p=.00; partial eta squared=.23, ε=.728], and for HR [F(3,76)=2.88, p=.05; partial eta squared=.10, ε=.663]. Table 3 on pg. 46 lists means and standard deviations for the change scores for each cardiovascular variable during each minute of the anger recovery period. As shown in Table 3, post hoc comparisons using the Simes variation on the Bonferroni correction (Simes, 1986) revealed a similar pattern of cardiovascular recovery for both systolic and diastolic blood pressure. The largest reduction in blood pressure occurred between minute 1 and 2, after which blood pressure gradually decreases throughout the remaining recovery time. These changes are represented graphically in Figures 8-10 (pg. 49).

This analysis also showed that veterans *without* PTSD had higher blood pressure change scores during recovery than veterans *with* PTSD (see Table 3 for means). While this effect was significant for DBP [F(1,78)=3.82, p=.05; partial eta squared =.05] it did not reach significance for SBP [F(1,78)=1.9, p=.17; partial eta squared =.02]. Both of these tests, in particular the analysis of SBP, were underpowered (DBP=.48, SBP=.27) and this may account for the non-significant result for systolic blood pressure. Consistent
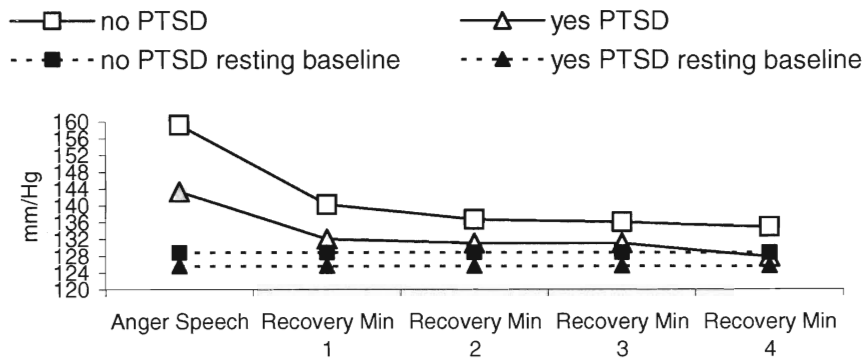
Figure 8: Systolic BP: Anger Speech Recovery

—□— no PTSD   —△— yes PTSD
- - ■ - - no PTSD resting baseline   - - ▲ - - yes PTSD resting baseline

Figure 9: Diastolic BP: Anger Speech Recovery

—□— no PTSD   —△— yes PTSD
- - ■ - - no PTSD resting baseline   - - ▲ - - yes PTSD resting baseline
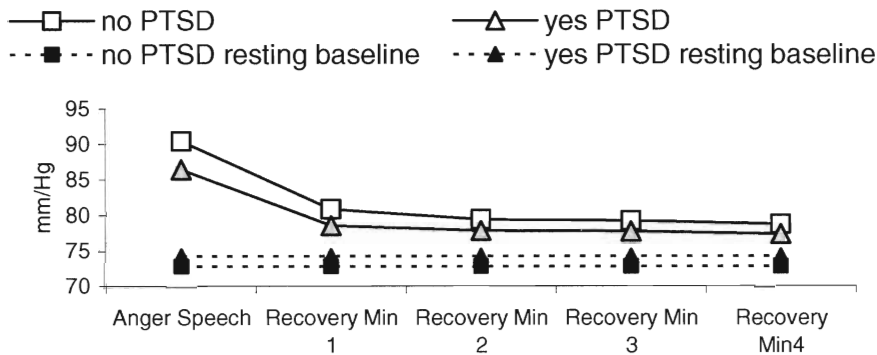
Figure 10: Heart Rate: Anger Speech Recovery

—□— no PTSD   —△— yes PTSD
- - ■ - - no PTSD resting baseline   - - ▲ - - yes PTSD resting baseline
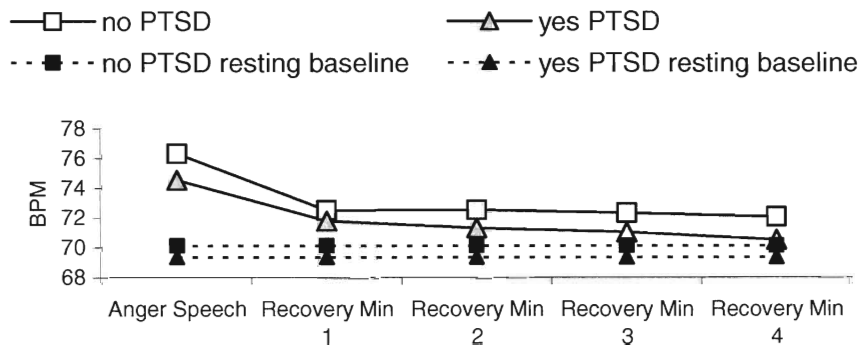
Figures 8-10: Display SBP, DBP, and HR means for PTSD and non-PTSD veterans during the last minute of anger speech and the four minutes during the post-anger verbalization recovery period. Although cardiovascular measures decreased during recovery from anger, there is still a significant difference between veterans blood pressures and heart rate at minute four during anger recovery compared with the last minute of resting baseline.

with findings in the imagery study (Beckham et al., 2002), there was no difference in HR between PTSD groups during recovery from anger.

Though it was clear that blood pressure and heart rate decreased during anger recovery for both PTSD and non-PTSD veterans, it was unclear whether these measures had returned to pre-anger levels. To determine whether mean cardiovascular responding at minute four of recovery differed from mean resting baseline levels separate 2 (PTSD vs. non-PTSD) by 2 (last minute of resting baseline vs. last minute of anger recovery) ANOVAs were conducted. Results indicated that even after four minutes of rest the veterans' blood pressure and heart rate were higher than resting baseline levels. These results were significant for systolic blood pressure [$F(1,78)=6.76$, $p=.01$; partial eta squared=.08], diastolic blood pressure [$F(1,78)=32.08$, $p=.00$; partial eta squared=.29], and heart rate [$F(1,78)=13.18$, $p=.001$; partial eta squared=.14]. There were no significant differences between PTSD and non-PTSD veterans.

Discussion

The first goal in undertaking this study was to directly compare the cardiovascular effects of thinking about an anger memory to the cardiovascular effects of voicing an anger memory. The second goal was to compare neutral to anger speech and explore differences that may exist between veterans with and without PTSD in the cardiovascular response to spoken anger. Few studies have directly compared anger imagery to anger speech (Siegman & Snow, 1997), and the cardiovascular effects of spoken anger have not been explored in a trauma population with elevated levels of hostility.

I. Comparing the Cardiovascular Response to Anger During Imagery and Speech: Even after controlling for the metabolic activity associated with speech, the results indicate that cardiovascular responding is greater during spoken anger than it is during anger that is recalled but not verbalized. This finding, that anger speech is accompanied by greater elevations in blood pressure than anger imagery, is consistent with prior work that directly compared these conditions (Siegman & Snow, 1997). However, in this prior work, the metabolic activity of speech was not controlled for, and this called into question whether the differences between thinking about and talking about anger could be entirely attributed to cardiovascular output associated with talking (rather than anger). The current findings support the prior study, and advance the conclusion that spoken anger has a greater impact on CVR than anger that is not verbalized. If interpreted from a bio-informational perspective (Lang, 1993), the results suggest that verbalizing an anger memory may provide additional cues, or increased cue intensity, that more fully activate the anger memory structure than does thinking about the event.

Although anger speech elicits greater cardiovascular reactivity than anger imagery, this study also shows that anger imagery is sufficient to raise heart rate and blood pressure significantly from a resting baseline. Although this is contrary to one study that directly compared anger during speech and imagery (Siegman & Snow, 1997), it is consistent with findings from numerous emotion imagery studies (e.g., Prkachin, 1999; Sinha et al., 1992). One reason that Siegman & Snow (1997) may have failed to find elevated CVR during anger imagery is that their study did not rely on continuous measurement of blood pressure and heart rate as the emotion imagery studies have done. Intermittent monitoring of cardiovascular responding is disruptive and may have distracted participants from the anger imagery task; intermittent monitoring could also fail to detect brief but significant increases in blood pressure or heart rate.

Importantly, participants in the current study were not screened for emotional intensity or imagery ability or given emotion scripts embedded with response details, as prior imagery studies have done, yet they still manifest significant increases in CVR when thinking about their past anger event. If cardiovascular reactivity (Finney et al., 2002) or chronic low level cardiac activation (Pieper & Brosschot, 2005) to anger are pathways to cardiovascular disease, the current findings suggest merely recalling the event, which elevates CVR, could have (cumulative) negative health consequences. This effect would likely be enhanced in populations with greater levels of anger and hostility such as people with PTSD (Beckham et al., 2002), in particular if they are prone to ruminating about these past anger-inducing incidents, as this would increase the frequency of their exposure to heightened blood pressure and heart rate.

II. Cardiovascular Response to Neutral Speech and Spoken Anger in PTSD and non-PTSD Veterans: Results from these analyses show that a spoken anger memory elicits significantly greater blood pressure than talking about a neutral topic, and that veterans *without* PTSD experience greater CVR during spoken anger than veterans *with* PTSD.

Similar to the results from the current investigation, some studies have found that vocalized anger evokes greater CVR than neutral speech (Siegman et al., 1990, 1992; Siegman & Boyle, 1993), while others have only partially supported this conclusion (Drummond & Quah, 2001). Drummond and Quah (2001) found that when speaking slowly and softly, anger speech elicited greater blood pressure than neutral speech, however, there was no difference in blood pressure between neutral and anger speech when speaking loudly and quickly. The researchers suggest the cardiovascular effect of loud and fast talking during anger speech is due to the metabolic activity of speech rather than anger, and that this accounts for the lack of significant difference in CVR between (loud and fast) neutral and anger speech. It is important to note that the participant's report of anger increased only about 20% from neutral to anger material in Drummond and Quah's study, which corresponds to "slightly angry" on their scale. Thus the similar levels of CVR during (loud and fast) neutral and anger speech could also be explained by lack of significant anger on the part of participants.

Unlike these past anger speech studies, the current study did not manipulate speech style; subjects were simply asked to talk about a time when they felt angry, frustrated, or upset with someone and were unhappy with the conclusion. Leaving

speech style up to the participants, regardless of whether they were speaking loudly and quickly or softly and slowly, captures a more natural account of spoken anger. Overall the results indicate that increases in blood pressure while talking during anger cannot be solely attributed to the metabolic activity associated with speech.

Compared with non-PTSD veterans, veterans with PTSD displayed lower blood pressure during anger speech. While it might be argued that attenuated CVR in PTSD veterans could suggest they were not fully engaging in the anger speech task, but rather were avoiding or experiencing emotional numbing associated with PTSD, this account seems unlikely given that veterans with PTSD reported greater levels of anger during the anger speech task than veterans without PTSD.

The finding of attenuated CVR in veterans with PTSD, who are known to be higher in hostility, is consistent with results from two investigations of hostility and laboratory induced stress (Anderson et al., 2005; Finney et al., 2002), and would appear to support a model of emotional dysregulation similar to that proposed in anger studies with alexithymics (Linden et al., 1996; Neumann et al., 2004; Newton & Contrada, 1994; Wehmer et al., 1995). Emotion dysregulation and concomitant disruption of sympathetic and parasympathetic control (for a review see Thayer & Lane, 2000) is associated with several types of psychopathology such as depression, alexithymia, generalized anxiety disorder, panic disorder, and hostile personality. This disruption in sympathetic activation is thought to be a consequence of disinhibition of the parasympathetic nervous system, which is indexed by vagal tone (expressed as heart rate variability). So, for example, when participants with and without Generalized Anxiety Disorder (GAD) are

asked to worry, those with GAD show significantly less heart rate variability (i.e. lower vagal tone) than participants without GAD (Thayer, Friedman, & Borkovec, 1996). Even during baseline, GAD participants exhibit reduced levels of heart rate variability compared to controls. Thus evidence of physiological dysregulation is present both during and outside of presentation of the provoking stimuli (i.e. worry), suggesting both trait and state effects of emotion dysregulation.

Prior studies have proposed different reasons for why, or how, greater hostility would be associated with lower reactivity during anger. For example, in attempting to explain *why,* Finney and colleagues (2002) suggest that higher levels of hostility are protecting African-American men from the effects of oppression-related sources of stress. The researchers also propose that methodological differences, in the form of African-American experimenters, contributed to attenuated CVR in higher hostility African-American participants who may have felt less stressed by the procedure due to "solidarity" effects.

In attempting to explain *how* higher hostility would be associated with lower reactivity, Anderson and colleagues (2005) suggest that attenuated reactivity *and* prolonged recovery in high hostile participants may be attributed to changes in blood pressure regulation. Their conclusion is that blood pressure in high hostility individuals has come to be controlled more by changes in peripheral resistance and less by cardiac output (Brosschot & Thayer, 1998), however, the authors emphasize this conclusion is speculative since their study did not measure blood pressure regulation.

Brosschot and Thayer (1998) argue that the level of reactivity to anger is less important than recovery from anger, and that recovery from anger is delayed because most anger must be inhibited since anger expression is rarely socially sanctioned. The authors point out that individuals high in hostility experience more anger producing situations, which, over time, requires more inhibition of anger that results in more time spent recovering from anger. In other words, high hostility individuals would be spending a good deal of time suppressing their anger (even if they would prefer to express it) and recovering from it.

The mechanism by which habitual anger would lead to physiological dysregulation that results in suppressed cardiovascular reactivity during anger speech is unknown. A measure of vagal tone (i.e. heart rate variability) would likely shed light on this process by revealing differential autonomic influences, however, heart rate variability was not analyzed in the current study. As Brosschot and Thayer (1998) summarize, the vagus nerve is responsible for parasympathetic control and prolonged recovery from anger signals lack of vagal tone/control. Over time this brings about sympathetic dominance of cardiac control and the entire cardiac system is eventually characterized by reduced flexibility. It is speculative, but it may be that blunted sympathetic activation (Neuman et al., 2004) observed in the PTSD veterans in the current study, is a sign of sympathetic and parasympathetic dysregulation. This would be a result of frequent anger and attempted inhibition and delayed recovery from that anger. Blunted sympathetic activation may reflect an attempt by the nervous system to compensate for frequent exposure to anger outburst by suppressing CVR during anger.

The finding that PTSD veterans were *less* reactive during anger speech than non-PTSD veterans may seem inconsistent with the prior work with this sample showing that PTSD veterans were *more* reactive during anger imagery. This pattern of reactivity can still be viewed through a lens of emotion dysregulation, in particular from a blunted sympathetic activation viewpoint. Anger imagery provokes a significant increase in CVR in both groups of veterans, however, the PTSD veterans respond with even greater blood pressure under this condition. PTSD veterans are more hostile and this contributes to greater exposure to anger producing situations. This learning history, rich in hostility and anger, lowers the threshold for activation of anger memories. Thus, as was found in the prior study (Beckham et al., 2002), veterans with PTSD are more reactive during anger imagery.

Both groups of veterans are more reactive during anger speech than anger imagery, however, in the anger speech condition non-PTSD veterans now evidence greater mean blood pressure than veterans with PTSD. It may be that over repeated exposure to excessive levels or excessive frequency of anger, a physiological attempt to maintain homeostasis results in blunted sympathetic activation or reduced autonomic flexibility during anger. This would explain why PTSD veterans reported being angrier during anger speech even though their physiological responding was attenuated compared to the "less angry" non-PTSD veterans.

A dynamic view of emotion regulation (Hoeksma, Oosterlaan, & Schipper, 2004) suggests that the physiological effects concomitant to feeling anger may be mitigated by behavior (e.g. modifying the situation, distracting oneself, or cognitive re-appraisal) that

returns the person to a non-anger state, reducing CVR and facilitating a quick recovery from anger. Thus cardiovascular reactivity to anger could be seen as less harmful over time if it is infrequent, brief, (Davidson, Stuhr, Dixon, MacGregor, & MacLean, 2000; Rozanksi & Kubansky, 2005) and followed by a speedy recovery. This could partially explain why the dysregulatory process (i.e. blunted affect or reduced autonomic flexibility) would not be present in the non-PTSD veterans; in other words, the non-PTSD group may be more likely to "do" something that facilitates recovery. In addition, the non-PTSD group would not experience anger episodes with the same frequency as the veterans with PTSD. Given the chronicity of anger in the PTSD population, it is unlikely that their reactivity to anger is infrequent, brief, or quickly resolved. It is possible then that neurovisceral changes occur as a way of adapting to the cardiovascular demands of chronic anger regardless of whether it is voiced or experienced internally.

III. Cardiovascular Recovery from Spoken Anger in PTSD and non-PTSD Veterans: These results show that the greatest reduction in CVR occurs in the first two minutes of recovery from anger. Despite these reductions, all veterans were still significantly higher at minute four of recovery than they were at resting baseline. It is also notable that non-PTSD veterans continued to have greater DBP during recovery (as they did during spoken anger) compared to PTSD veterans.

Prolonged recovery from a negative emotion, such as anger, is a way in which cardiovascular health is thought to be impacted by psychosocial stressors (for a review see Brosschot & Thayer, 1998; Pieper & Brosschot, 2005). The logic behind this approach is that the duration and not the magnitude of the stressor has cumulative

negative effects on health. One point that has been made in the hostility and anger

speech literature is that while lower levels of hostility may be associated with attenuated

CVR *during* spoken anger (Anderson et al., 2005; Brosschot & Thayer, 1998; Finney et

al., 2002), participants higher in hostility maintain elevated levels of CVR for a longer

time during recovery (Anderson et al., 2005; Neuman et al., 2004b) than participants

lower in hostility.

Such was not the case in the present study where the non-PTSD group (which is

lower in hostility) either displayed approximately the same level of recovery as the PTSD

group, or in the case of DBP, was actually higher during recovery. Unfortunately the

recovery period was too brief to determine whether veterans without PTSD would have

eventually returned to baseline sooner that veterans with PTSD.

The difference between the recovery results of the current study and prior studies

could be due to a number of variables. Rumination has been proposed as a mechanism

by which CVR recovery is delayed (Glynn, Christenfeld, & Gerin, 2002), however, it

would be speculation to suggest that rumination delayed the recovery of veterans in this

study since rumination was not measured.

The difference in the present results and past studies could possibly be associated

with age-related changes in cardiovascular recovery, since increased age is associated

with less autonomic flexibility and this would likely delay recovery from anger for both

groups of veterans. Participants in the prior studies (Anderson et al., 2005; Finney et al.,

2002) were undergraduates in college and were significantly younger than the mean age

of fifty in the current study. Thus a brief recovery period in younger subjects may reveal

group differences whereas the same time period with older participants would not be sufficient to reveal group differences.

Another tenable possibility is that delayed recovery in the current study could be associated with the participants' weight. A recent study (Steptoe & Wardle, 2005) showed that greater body mass index (BMI) predicts delayed blood pressure recovery from a laboratory stressor. The mean BMI for both PTSD and non-PTSD subjects in the current study is 28, which is considered the borderline between being overweight and obese. Steptoe and Wardle (2005) found that participants with greater levels of abdominal fat and higher BMI were still above baseline levels of blood pressure 45 minutes after completion of a stress inducing task.

Collectively these explanations for extended recovery in both groups suggest that age-related changes and/or health problems, such as obesity, may have delayed recovery from the anger speech task. It may be that delayed recovery from stressors has multiple causes (i.e. rumination after anger, sympathetic nervous system dysregulation due to frequent anger episodes, obesity) that are working independently or in concert to inhibit a speedy return to baseline cardiovascular functioning for the entire group of veterans.

Strengths and Limitations

The naturalistic manner in which verbalized anger was measured is a strength in this study. While some studies have been concerned with isolating speech style from anger, this seems an arbitrary distinction. Whether anger speech is naturally loud and fast or slow and quick, the essential information is that when anger is allowed to unfold naturally it elicits greater CVR than just talking in general.

Saliency of anger and hostility in this *clinical* sample is also posited as a considerable strength in the present work. Other work that has looked at the link between hostility and CVR during spoken anger (Anderson et al., 2005; Finney et al., 2002; Neuman et al., 2004b) has used non-clinical samples (i.e. undergraduates). The current study extends the CVR and spoken anger findings into a population with greater than normal levels of anger, hostility, and violence (Beckham et al., 1996; Chemtob et al., 1994; Kulka et al., 1990; Lasko et al., 1994; Novaco & Chemtob, 2002). The findings from this study suggest that excessive reactivity alone does not characterize the response to anger in a high hostility trauma population. Lower level but chronic cardiovascular and sympathetic nervous system activation, as well as parasympathetic disinhibition, may better describe what occurs physiologically in high hostility individuals when they are angry.

Although use of a clinical sample for this investigation is considered a strength, there are restrictions to the generalizability of this study. This sample was restricted to men; specifically to men who served a combat role during the Vietnam conflict. It is unclear whether a sample of women with PTSD would respond similarly, or whether a younger sample of male or female PTSD participants would reveal similar results. In addition, due to the effect of beta and calcium-channel blockers on CVR, participants taking these medications were excluded from the analyses. It is unknown how this might affect the applicability of the results to the population of Vietnam veterans with or without PTSD.

A limitation also exists in the anger recovery analysis. The time period used to assess recovery, four minutes, was insufficient to determine whether non-PTSD or PTSD veterans recover more quickly from spoken anger. Future work should include a lengthier time period in order to capture these differences, if they exist. In addition, rumination may delay recovery from negative emotional states, whereas cognitive coping may facilitate recovery (Glynn et al., 2002; Hoeksma et al., 2004). It would be helpful in interpreting group differences in recovery if it were known whether participants engaged in rumination or cognitive coping.

Summary and *Conclusions*

Spoken anger produces greater elevations in blood pressure than anger that is recalled but not voiced. Some researchers suggest that spoken anger creates a feedback mechanism that increases anger and CVR. From a bio-information perspective, the results suggest that greater activation of anger memory networks is achieved by talking about the anger episode rather than by thinking about the anger episode.

While talking about anger produces greater increases in blood pressure it is not necessary to voice anger to experience increases in blood pressure; merely thinking about anger, in particular for PTSD veterans, produces a significant cardiovascular response. It could be that *infrequent* reactivity followed by immediate coping that attenuates CVR is not pathological, whereas chronic high *or* lower-level anger-related increases in CVR, which are maintained by an absence of coping, are pathological by virtue of delaying recovery from anger. This in turn results in increased exposure to elevated CVR and subsequent dysregulation of the central nervous system (i.e. low vagal tone, blunted

sympathetic activation, and/or reduced autonomic flexibility). Thus people with high levels of hostility, such as those diagnosed with PTSD, who think about or re-experience anger more frequently than others, could be exposing themselves to persistent elevated levels of CVR, which may have adverse consequences for their cardiac health.

It is important to gain a better understand of how different methods of re-experiencing anger affects the cardiovascular system of people with PTSD. Learning more about this phenomenon has implications for better treatment of PTSD-associated anger, and better understanding of the pathway(s) from psychosocial stressors, such as anger, to poor health.

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Appendix A
Subject Instructions for Session II Anger Speech/Verbalization

I want you to try to think of a time when you got really angry, frustrated, or upset with another person; a situation in which you were not happy with the outcome. Something that still frustrates or angers you when you think about it. Can you think of anything like that that has happened?

Just give me a word or two about what that was about. Can you think of another time? How long ago was that? How long ago was the second incident? Tell me about the (first or second) incident. (When patient begins speaking, interviewer begins timing for at least six minutes).

Examples of Prompts:

What happened?

Tell me everything about that day, time, place, weather, etc.

How were you feeling then?

What did you say? What did the other person say?

What led up to this?

How did the other person respond to the incident?

Have you had any contact with that person since?

Why do you feel that the situation remains unresolved?

At the end of the six minutes the subject rates:

1) how angry he was then on a scale of 1 to 10

2) how angry he is now on a scale of 1 to 10

Appendix B

Subject Instructions for Session III Anger Imagery

For this part of the study, which will last about 20 minutes, you will be guided by what you see on the TV. Written instructions on the TV set will tell you when the session begins and what you should do when.

At one point during the video tape, you will receive instructions to relive that angry situation we talked about earlier today. The time when you were really angry with _____ (fill in name of person). At other times during the tape, you'll be asked to just relax and think about nothing in particular. These periods only last for two or three minutes, so please be patient with them. The purpose of these rest periods are to get some baseline measures of your blood pressure; thus, we are collecting some very important data even though it seems you are not doing anything. When the session is over, the TV will say so.

When the time comes when you are to relive the angry situation that we talked about, the written instructions on the TV screen will say, "Now we want yo to think about that time when you felt so angry you wanted to explode." Relive this situation in your mind by thinking about it and reliving it in your mind. When you are able to feel your anger, please raise your left hand like this (experimenter demonstrates the hand signal).

Do you have any questions? Please do not speak during the video. O.K. Let's begin.

Appendix C

Additional Statistical Analysis: Hostility and PTSD During Anger Verbalization

In a prior publication exploring the response to anger with this sample of veterans (Beckham et al., 2002) a relationship was found between hostility, PTSD status, and cardiovascular responding during anger imagery. Those results indicated a significant interaction between group status and hostility such that only at higher levels of hostility did veterans with PTSD have greater diastolic blood pressure during anger imagery than veterans without PTSD. This relationship was similar, though not significant, for systolic blood pressure.

As with the prior findings, multiple regression was used to assess whether this relationship would apply to relived anger that is verbalized rather than relived through imagery. The main effects of hostility (the total from the Cook-Medley Hostility Inventory) and PTSD status were entered into step 1 of the model and the Group X Hostility interaction was entered into step two of the model. The dependent variables were systolic and diastolic blood pressures.

Unlike the results from the imagery data, higher levels of hostility did not interact with PTSD status to produce greater responding during anger retrieval (verbalization). The full model with PTSD status, hostility, and their interaction approached significance for systolic blood pressure [$F(3,76) = 2.40$, p=.074, R^2=.09] and was not significant for diastolic blood pressure [$F(3,76) = .642$, p=.591, R^2=.02].

Vita

Jennifer Runnals was born in Brunswick, Maine on April 25th, 1971. She completed high school in Ayer, Massachusetts and obtained her baccalaureate degree in Industrial and Organizational Psychology at Christopher Newport University in 1993. In 1999 Ms. Runnals obtained a masters degree in Clinical Psychology from Augusta State University. She is currently pursuing a doctoral degree in Clinical Psychology with a specialization in Behavioral Medicine at Virginia Commonwealth University.