Running head: AUTONOMIC EFFECTS OF ANXIETY AND DEPRESSION

Cognitive Biases and Autonomic Responding in Anxiety and Depression

Aimee K. Santucci

Thesis Submitted to the Faculty of the Virginia Polytechnic Institute and State University in partial fulfillment of the requirements for the degree of

Master of Science

in

Psychology

Bruce H. Friedman, Ph. D., Chair
Helen J. Crawford, Ph. D.
Martha Ann Bell, Ph.D.

April 29, 2001
Blacksburg, Virginia

Keywords: Anxiety, Depression, Heart Rate Variability

Cognitive Biases and Autonomic Responding in Anxiety and Depression

Aimee K. Santucci

ABSTRACT

The present study addressed cognitive biases in anxiety and depression using the emotional Stroop task, and explored both the affective space and autonomic underpinnings of these disorders. In previous studies, anxiety has been associated with both an attentional bias toward threat information and low cardiac vagal control, as reflected in heart rate variability (HRV) indices. Depression has been linked to a memory bias for negative information; however, findings of low HRV for depression are mixed. The high comorbidity of these disorders renders such findings as difficult to interpret. In the present study, it was hypothesized that the negative affect groups (anxious, depressed, comorbid anxious/depressed) would have lower vagally mediated HRV across tasks compared to the control group and that the anxiety and depression groups would show biases for group specific words on the Stroop task. Results for the Stroop tasks generally support previous findings of an attention bias in anxiety. The comorbid anxiety/depression group generally showed lower vagal control across tasks compared to the other groups, although comparisons between the “pure” anxiety and depression groups and the controls were not significant. It is suggested that this is because the comorbid group had higher depression and anxiety than either of the “pure” groups.

ACKNOWLEDGEMENTS

I extend sincere appreciation to my mentor, Dr. Bruce Friedman, for his continued support and invaluable guidance throughout all the phases of this project. I also thank the members of my committee, Dr. Martha Ann Bell and Dr. Helen Crawford, for their advice and input on this project. I would also like to thank all the members of my lab, especially Elena Kyrgos and Israel Christie, for their assistance with data input and analyses. Last, but not least, I would like to thank all of my friends and family for their support, advice, and encouragement throughout this project.

TABLE OF CONTENTS

ABSTRACT II

ACKNOWLEDGEMENTS III

TABLE OF CONTENTS IV

INTRODUCTION 1

Information Processing Models of Anxiety and Depression 2
Models of Affective Space 8
Physiological Correlates of Anxiety and Depression 12
Design and Hypotheses 22

METHOD 25

Participants 25
Materials 26
Procedure 30

ANALYSES 32

Physiological measures 32
Stroop Task Word Groups 33

DISCUSSION 35

Physiological Measures 35
Stroop Task Words 39

REFERENCES 43

TABLE 1 52

TABLE 2 53

TABLE 3 54

TABLE 4 55

FIGURE CAPTIONS 56

FIGURE 1 57

FIGURE 2 58

FIGURE 3 59

APPENDIX A 60

BAI 60

APPENDIX B 61
BDI 61
APPENDIX C 64
Handedness Questionnaire 64
APPENDIX D 65
Marlowe-Crowne SDS 65
APPENDIX E 67
SAI: Form Y-1 67
Form Y-2 68
APPENDIX F 70
Emotion Report Form 70
APPENDIX G 71
AIM 71
APPENDIX H 73
Stroop task words 73
APPENDIX I 74
Electrode Placement Diagram 74
APPENDIX J 75
Informed Consent Forms 75
APPENDIX K 79
Demographic Form 79
CURRICULUM VITAE 80

Cognitive Biases and Autonomic Responding in Anxiety and Depression

INTRODUCTION

Emotion and cognition are intimately linked. Emotions may direct attention, guide decision-making, stimulate learning, and trigger behavior (Cacioppo & Berntson, 1999). For example, negative affect can act as an important signal by directing the organism to attend to information so that current state or activity can be changed or adjusted (Pratto & John, 1991). While this may be considered an adaptive response for an organism, continued emotional dysregulation (e.g., anxiety and depression) impairs normal functioning and may lead to a preoccupation with upsetting experiences. Specifically, biases in the cognitive system may play an important role in the etiology and/or maintenance of both anxiety and depression (Mogg & Bradley, 1999). Anxiety has been linked with an attention bias toward threat or danger, and depression is associated with feelings of failures or worthlessness as well as a memory bias for negative information (Williams et al., 1997).

In addition to these information processing biases, the physiological patterns associated with anxiety and depression were the focus of the present study. Autonomic nervous system functioning has been found to be compromised in these disorders, with well-documented effects of anxiety (e.g., see Friedman & Thayer, 1998b, for a review), but with less clear effects from depression. While there may be a specific autonomic mediator of depression (Hugdahl, 1998), most of the studies on depression and heart rate variability (HRV) have been conducted on cardiovascular disease patients and its relationship to depression or have been on the cardiovascular effects of pharmacological treatment.

Past research has attempted to link information processing with physiological responding. Specifically, it has been proposed that autonomic measures may reflect relevant aspects of

information processing, such as attention (Porges, 1992). For example, infants with poor sustained attention (i.e., who are easily distractible) have been shown to have low tonic HRV (Richards & Casey, 1992). This is similar to the pattern found in adult generalized anxiety, where the process of worry is associated with decreased HRV (Thayer, Friedman, & Borkovec, 1996). This finding is congruent with previous studies where low HRV as an index of cardiac vagal tone has been found to be associated with poor attention regulation. However, the link between information processing and the physiological effects of depression remains unclear. One goal of the present study is to further explore the relationship between physiological responding and information processing in both anxiety and depression.

Information Processing Models of Anxiety and Depression

A potential means for understanding the unique characteristics of depression and anxiety is through information processing models. Cognitive approaches to anxiety and depression have focused not only on the content of cognitions, but also on the cognitive structure of information processing. This view is exemplified by Beck's (1976) cognitive theories of emotional disorders, which suggests that individuals with anxiety and depression have cognitive contents specific to their respective disorder and that these thoughts are automatic (i.e., nonvolitional). Depression is associated with transient automatic thoughts, interpretation, and imagery that center around the theme of self-deprecation and negative attitudes about the past; however, in anxiety, cognitions are characterized by the theme of possible danger (i.e., experiences are misread as threatening and the probability of future danger is overestimated). Central to this model is the prediction that for both anxiety and depression there will be similar mood-congruent biases in all aspects of information processing, including selective attention, memory, and reasoning (Mogg & Bradley, 1999).

Williams et al. (1997) have suggested that the cognitive content-specificity hypothesis has received inconsistent support, specifically questioning the role of automatic thoughts in depression. Several studies have suggested that anxiety is primarily associated with a bias in early aspects of processing such as attention, and depression is associated with a bias in later stages of processing, such as memory (e.g., retrieval) (see Williams et al., 1997, for a review).

Anxiety. Williams et al. (1997) have proposed a model in which the bias in anxiety operates at an automatic, preattentive stage. Processing resources are drawn toward threatening material before that information has reached conscious awareness (Mathews & MacLeod, 1994). Various tasks have been used to assess pre-attentive biases in anxiety, such as the visual dot probe (Mogg, Bradley, & Williams, 1995) and dichotic listening (Mathews & MacLeod, 1986). These studies have shown anxiety to be consistently marked by a preconscious bias towards perceiving negative information, which leads to hypervigilance towards threat in the environment. According to the Williams et al. (1997) model, since priority to threat material occurs at a preconscious stage of processing, anxious individuals should not be aware of this effect, resulting in an implicit memory bias for threat-relevant information. Implicit memory refers to the nonconscious effects of previous experiences on current performance (Schacter, 1995). This implicit memory bias in anxiety has been demonstrated in stem-completion tasks (e.g., Mathews, Mogg, May, & Eysenck, 1989).

Adaptations of the Stroop task (Stroop, 1935) have also generally supported the hypothesis that threatening words command more processing resources in anxious subjects (e.g., MacLeod, Mathews, & Tata, 1986; Mogg, Bradley, Williams, & Mathews, 1993). In the modified Stroop task used in these studies, participants are presented with neutral and emotionally-relevant

words and asked to indicate the color or the word. Words that command more processing resources will result in longer response latencies for the color-naming of that word.

In the Stroop paradigms, it is difficult to separate perceptual from response bias explanations of the data because both early and late stages of information processing are involved (Dalgleish & Watts, 1990). That is, if the cognitive systems of emotionally disturbed people are programmed to *respond* to stimuli related to their concern, this may not reflect an *attentional* bias but a *response* bias. It has been suggested that this response bias can be the result of response inhibition or cognitive avoidance (de Ruiter & Brosschot, 1994).

Cognitive avoidance has also been implicated in the longer response latencies found for emotionally valenced information in the Stroop task by repressors (de Ruiter & Brosschot, 1994). Repressors score high on the Marlowe-Crowne Social Desirability Scale (MC-SDS; Crowne & Marlowe, 1964) and low on the State-Trait Anxiety Inventory (STAI; Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983). This coping disposition is marked by a low capacity to tolerate emotional arousal from threatening information, so the repressor withdraws attention from threatening aspects of situations (Hock, Krohne, & Kaiser, 1996). This results in denial of anxiety to avoid the threatening information, even though repressors often respond nonverbally as if they are highly anxious (i.e., both physiologically and behaviorally) (Weinberger, Schwartz, & Davidson, 1979). Repressors were shown to have greater interference for anger, anxiety, and grief words on an emotional Stroop task than high trait anxious subjects and have memory deficiencies for personal, affect-laden memories (Davis, 1987). These studies support the idea that repressors engage in cognitive avoidance; that is, they avoid processing emotionally negative information. This response bias can be seen in the Stroop interference effect: the effort or time needed to cognitively process threatening information increases response latencies.

The role of worry in anxiety may function as a cognitive avoidance response to threatening stimuli (Borkovec, Ray, & Stober, 1998). Worry can be used as a cognitive avoidance response to the occurrence of aversive images. For GAD individuals, worry about superficial things may also serve to distract these individuals from the real problem. Although worrying is used to avoid threatening material, worrying can also contribute to maintenance in or an increase in the emotional disturbance generated by those events. It has also been suggested that anxiety may impair the functioning of the central attention or executive system of working memory. Impairment of the efficiency of this system may be attributable to the increased worry and other forms of task-irrelevant processing that characterize anxious individuals (Eyesenck, MacLeod, & Mathews, 1987).

The attentional bias and cognitive avoidance that have been demonstrated in anxiety may have a reciprocal relationship. The strong motivation that anxious individuals have to cognitively avoid threatening information at a preattentive level of awareness may lead to an attention bias for stimuli to be avoided. This attentional bias may be adaptive in that it enables a subject to come up with an avoidance response (Lavy & van den Hout, 1994).

This process of avoiding threatening information in anxiety can be conceptualized in terms of Freud's description of repression. (Freud, 1915, cited in Postman, 1962). According to Freud, at the level of perception, repression requires the paradoxical process of perceiving information so that this information will not be perceived. This can be adaptive means of protecting oneself against stimuli that are threatening, distractive, or disruptive. This idea was expanded by McGinnies (1949, cited in Postman, 1962), who suggested that perceptual defense is a mechanism whereby unpleasant or dangerous stimuli are avoided. This idea is based on McGinnies' findings of increased emotionality before recognition of subliminally presented taboo words (measured by

increased galvanic skin response). However, perceptual defense has been criticized because this concept suggests that the processes of "knowing" and "not knowing" occur simultaneously. That is, on some level, the individual must be aware of the information in order to avoid it. This is an important issue to consider in light of the fact that the bias in anxiety is often presented as "preattentive" or "automatic". The mechanism of perceptual defense may assist in supporting the view that highly anxious individuals do not have a preattentive bias but a response bias (de Ruiter and Brosschot, 1994), where the cognitive systems of individuals with trait emotional dysregulation are programmed to respond to stimuli related to their concern.

Depression. Depressed individuals, according to the Williams et al. (1997) model, have a bias for negative information that occurs at later, controlled stages of processing (i.e., information bias has entered conscious awareness). There is considerable evidence of recall bias in depression (Mathews & MacLeod, 1994). This is an explicit memory bias, a process whereby material is consciously recollected (Schacter, 1995). This biasing effect of mood on recall has been found in clinical depression as well as in depressed mood, showing lower recall for neutral words and/or higher recall for negative words (Dalgleish & Watts, 1990). Similar studies using anxious subjects have not consistently found evidence of memory bias for threatening words (e.g., Mogg, Mathews, & Weinman, 1987). However, a recent study (Friedman, Thayer, & Borkovec, 2000) found an explicit memory bias toward recall of threat words by a GAD group. Additionally, studies with word lists have shown less powerful results for memory bias in depression than studies that use person memories that have particular emotional significance (Dalgleish & Watts, 1990) (e.g., memories of failure or rejection).

Attention may play a role in the memory bias seen in depression. Depressed individuals have been shown to have both an explicit memory bias for depression-relevant information

(Williams et al., 1997) and deficiencies in episodic memory (Ellis, Thomas, & Rodriguez, 1984). This may be due to the role of worry in depression: depressive rumination can result in a decrease in available attentional capacity, which may result in failure to engage in effortful strategies that support memory of information that is not depression-relevant (Borkovec et al., 1998). The explicit memory bias shown for depression-relevant information may be the result of attention deployment to mainly depression-relevant information.

Evidence for an attention bias in depression that is similar to the one in anxiety has yielded mixed results, though there have been relatively few studies of this phenomenon. However, these studies have often been confounded by group selection (e.g., anxious subjects as depressed as the depression group) and use of stimulus materials that were not depression-relevant (e.g., MacLeod et al., 1986). In general, a bias for depression-relevant or negative information has not been found with the Stroop paradigm (e.g., Mogg et al., 1993), although one study did demonstrate longer response latencies for depressed-content words by non-clincially depressed individuals (Gotlib & McCann, 1984). Most of the theories suggesting reduced attentional resources in depression would predict this outcome based on general behavioral slowing, motivational deficits, or narrowing of attention to focus mainly on task irrelevant information (see Thomas, Goudemand, & Rousseaux, 1999, for a review). Studies of reduced attentional resources in depression have revealed inconsistent and contradictory findings, such as improved performance on double tasks compared to poor performance on simple tasks (Krames & McDonald, 1985). It remains unclear whether depression interferes with decision-making processes (e.g., as is generally suggested by Stroop task performance) because these tasks require more demand from automatic or effortful processes or because they account for a specific deficit in depressive disorders (Thomas et al., 1999).

Models of Affective Space

Information processing models provide means to differentiate anxiety and depression by demonstrating how individuals with anxiety or depression differentially attend to and reflect on their environments. Models of affective space can be used to further understand the affective disturbance associated with these disorders. Research on the structure of affective space of both anxiety and depression has been a source of controversy. This research has been characterized by two models, the tripartite model, and the circumplex model of emotion, which differ in terms of the proposed underlying dimensions used to represent the organization of emotions.

Tripartite Model of Anxiety and Depression

Research on pathological anxiety and depression has shown the two disorders to have overlapping and distinguishing features. For example, self-report measures of depressive and anxious symptoms correlate .62 to .70, on average (Clark, Steer, & Beck, 1994). Clinical rating scales (e.g., Hamilton Rating Scale for Anxiety, Hamilton Rating Scale for Depression) have better discrimination between constructs with correlations that range from .40 to .45 (Clark et al., 1994).

Clark and Watson (1991) have proposed a tripartite model of depression and anxiety to account for the high correlation between anxiety and depression measures. This model recognizes a common feature of both depression and anxiety to be negative affect: a nonspecific distress factor characterized by tendencies to be distressed, worried, anxious, and have a negative view of oneself. The distinguishing feature of depression is a loss of positive affect (i.e., anhedonia – the loss of pleasure in activities that were once reinforcing). In contrast, anxiety is associated with "hyperarousal" (i.e., global autonomic arousal), and does not necessary lead to a loss of positive affect. The validity and applicability of the tripartite model was investigated using both outpatient and student samples. Analyses indicated that depression and anxiety share 40% common variance.

Low positive affect was found to be only associated with the depression factor whereas physiological symptoms were found to be specific only to anxiety.

Circumplex Model of Emotion. Although the tripartite model is supported by the distinction between anxiety and depression, other models question the validity of representing positive and negative affect as separate dimensions (i.e., orthogonal). Furthermore, although research suggests a strong autonomic component of anxiety (Clark et al., 1994), the tripartite model does not include the dimension of arousal of the affective space. This depiction of global autonomic arousal in the tripartite model is also misleading, for it is difficult to draw a straightforward relationship between arousal and ANS functioning. Arousal is a vague term to apply to ANS functioning because the branches of the ANS do not each fire as a whole but in patterns of discrete localized activity (Wolf, 1995). Furthermore, there is a lack of a strong relationship between different measures of arousal (i.e., behavioral, autonomic, and cortical; Hugdahl, 1998), suggesting that arousal in one system does not always imply arousal in other systems.

Additionally, Russell (1980) proposed that the dimensions of affect are not orthogonal, but rather are highly systematically related. In the circumplex model of emotion, the structure of affect is best represented as a two-dimensional affective space: arousal (low to high) and pleasure-displeasure (emotionality). Affect is not described as clustering around the axes of the dimensions, but as being some combination of the emotionality and arousal components (Russell, 1980). The organization of affect is depicted as emotion terms arranged on the circumference of the circle. The boundaries between each emotion are not discrete, but rather are vague or “fuzzy” (Russell, 1997). The circumplex model is supported by studies of how the layperson conceptualizes affective states as well as multivariate analyses of self-reported affective states (see Russell, 1980,

1997, Larsen & Diener, 1992, for a review). A confirmatory factor analysis also strongly supported the independence of hedonic level and emotional intensity (Thayer & Miller, 1988).

The manner in which Russell has conceptualized arousal is not the same as the global physiological arousal associated with anxiety in the tripartite model. The arousal dimension in the circumplex model refers to activation, the energy level of an emotional experience (Russell, 1980). High activation is associated with high affect intensity and high activity level, whereas high emotional control is related to low activation (Larsen & Diener, 1992). This activation aspect of emotional experience will be addressed in the current study by administering the Affect Intensity Measure (AIM; Larsen & Diener, 1987)

The other component of this model is the positive-negative dimension. Although adherents of the tripartite model propose that positive and negative affect are orthogonal, proponents of the circumplex model suggest that positive and negative affect are strongly negatively correlated, and that results from factor analyses demonstrate that measurement error masks bipolarity in affect ratings (Green & Salovey, 1999). This measurement error appears to be reflected in the use of the terms positive and negative affect (PA and NA) in the Clark and Watson model. The PA dimension at one end, contains adjectives such as *peppy* and *elated* and, at the other end, adjectives such as *drowsy* and *dull*. The high-end anchors of this dimension do appear to be positive or pleasant; however, pleasant mood adjectives such as *happy* and *contented* are not included as part of this dimension, even though these are important positive emotions (Larsen & Diener, 1992). Furthermore, the low end of PA contains unpleasant adjectives that are low activation. Similar problems exist with the NA dimension, where the lower end contains pleasant items, such as *pleasant* and *relaxed* (Larsen & Diener, 1992).

The terms PA and NA make conceptual claims about the nature of the construct; however, the content shows different information (Larsen & Diener, 1992). Although the terms NA and PA in the Clark and Watson model refer to valenced states, the adjective sets used to represent PA and NA reflect more than hedonic valence: they also reflect activation. This dimension of activation is reflected in the circumplex model. However, although the circumplex model does capture important aspects of emotional experience (emotionality and arousal), it should not be regarded as a total theory of the way emotions differ because other dimensions, such as action readiness, may be as important as the arousal and emotionality dimensions (Larsen & Diener, 1992).

Both models appear to provide important information about the dimensions of affective space, and the PA/NA model and the circumplex model may be useful in different experimental contexts (Larsen & Diener, 1992). For example, research on event-related potentials of depressed individuals has shown that depression may be the result of neglect of positive information (Miller, 1996). However, in ANS studies of musically-induced emotions (happiness, sadness, serenity, and agitation), and affective reactions to laboratory tasks, the emotions appeared to be better defined by the arousal-valence model (Friedman, Takayama, Long, & Thayer, 1997; Nyklicek, Thayer, & Van Doornen, 1997).

One goal of the present study is to explore differences in autonomic responding in anxiety and depression. Since autonomic responses appear to be more sensitive to the arousal dimension, it is consistent to use the circumplex model to represent affective space in these disorders. In this model, anxiety is depicted as high arousal negative affect and depression as low arousal negative affect. Thus, there is still a shared component of NA between anxiety and depression but arousal differentiates the disorders (as opposed to PA).

Physiological Correlates of Anxiety and Depression

Physiological correlates of anxiety and depression can be used to substantiate the use of the circumplex model to describe anxiety and depression. As discussed previously, although Clark and Watson (1991) suggested that anxiety is characterized by "hyperarousal" (i.e., a global autonomic arousal), this perspective on anxiety overlooks the complex patterns of autonomic nervous system (ANS) activity that occur in anxiety. Physiological correlates may also be used to clarify the term "low arousal" used in conjunction with depression in the circumplex model. Although the terms high and low arousal used in the circumplex model can serve to better differentiate anxiety and depression than the Clark and Watson model, these terms still reflect generalized ANS activity. ANS measures change as situationally-adaptive response patterns, but not necessarily in unison (Wolf, 1995). Therefore, the term high arousal that is associated with anxiety should not be interpreted as a general increase in all measures of ANS activity, nor should low arousal, associated with depression, be viewed as a general decrease in ANS activity. One goal of the present study is to further explore differences in ANS activity in anxiety and depression.

Physiological differentiation of anxiety and depression reflects the question of ANS specificity of emotions. If anxiety and depression are different emotions, as depicted by their placement on the circumplex, do they have different physiological patterns? The ANS changes in emotion that have been found to assist in dealing with situations where lengthy appraisal would threaten survival, influence the way emotions subjectively "feel", motivate behavior aimed at modulating their levels (e.g., thrill-seeking), serve as contextual cues that bind networks of association and memory, and play an important role in the processes of mental and physical health and disease (Levenson, 1992). As such, establishing specificity does not mean that every emotion

has a unique pattern, since closely related emotions may have similar patterns. For example, anxiety and depression appear to have a shared NA component; however, differences in the arousal dimension may distinguish the emotions physiologically, and specifically may increase with increasing intensity of emotions (Stemmler, 1989). Therefore, the circumplex model provides a useful framework for investigating anxiety and depression in terms of arousal and affect, both of which appear to be important areas for investigating the physiological correlates of anxiety and depression.

Anxiety. Anxiety has been linked with global sympathetic nervous system activity (see Friedman & Thayer, 1998b, for a review of these models and alternatives). In these models, the ANS is viewed as reacting from a steady state (i.e., global sympathetic arousal in response to anxiety disrupts homeostasis). Although autonomic dysregulation does occur in anxiety, these perspectives on anxiety overlook the complex patterns of ANS activity that occur in anxiety as well as the widely different patterns found in the various anxiety disorders. For example, panic anxiety can be described as a pattern of elevated sympathetic activity and vagal withdrawl (Friedman & Thayer, 1998a). In contrast, blood phobia, a specific phobia, has very different physiological effects than panic, and is associated with a strong vasovagal response (Friedman & Thayer, 1998a).

A physiological measure that is useful in depicting sympathetic and vagal cardiac regulation is heart rate variability (HRV). Changes in heart rate (HR) are due to feedback from internal mechanisms and rhythms are mediated by an interplay of cardiac sympathetic and vagal activity (Saul, 1990). The parasympathetic nervous system is primarily responsible for the beat-to-beat variability of HR, and the heart responds more gradually to sympathetic stimulation. Since ANS functioning is an index of the integrity of ANS/CNS integration, absence of or decrease in

this beat-to-beat variability is indicative of abnormal central nervous system modulation of HR (Saul, 1990). Furthermore, greater HRV indicates high levels of cardiac vagal control, which is important for self-regulation and flexibility in meeting environmental demands (Porges, 1992). Conversely, decreased heart rate variability has predictive value for mortality in healthy adults, there has been found to be a consistent association of decreased vagally mediated HRV with both sudden death and coronary heart disease mortality in middle-aged and elderly men (Stein & Kleiger, 1999).

Alterations in HRV can be seen in a variety of psychological conditions. For example, in comparing the autonomic characteristics of panickers, blood phobics, and nonanxious controls on a variety of laboratory stressors, panickers were found to have the highest HR and lowest HRV compared to blood phobics and controls, although controls had significantly higher HRV than blood phobics (Friedman & Thayer, 1998). Other studies have supported this finding of decreased HRV in panic disorder as well as in post-traumatic stress disorder (PTSD) (Klein, Cnaani, Harel, Braun, & Ben Haim, 1995; Cohen et al., 2000). In Generalized Anxiety Disorder (GAD), which is characterized by persistent and uncontrollable worry (DSM-IV, American Psychiatric Association, 1994) and is maintained by hypervigilance for threat information in the environment (Thayer, Friedman, Borkovec, Johnson, & Molina, 2000), worry has been linked to reduced vagal control (Thayer, Friedman, & Borkovec, 1996). Taken together, these studies demonstrate that reduced HRV is a broadly patholitic marker for a variety of psychological conditions.

Depression. The effect of depression on autonomic functioning, especially HRV is less clear. Previous studies have found that depression has been associated with reduced HRV in patients with coronary heart disease (CHD) (Watkins, Grossman, Krishnan, & Blumenthal, 1999; Krittayaphong et al, 1997). Decreased heart rate variability, which is associated reduced vagal

control, reflects dysregulation of the parasympathetic nervous system and has been linked to increased risk for cardiac morbidity or mortality. This has important implications for coronary artery disease (CAD) patients who are depressed; these individuals have been found to have significantly lower HRV than CAD patients who are not depressed (Carney et al., 1995).

It is possible, then, that depression is a risk factor for CVD, through dysregulated cardiac ANS control. One proposal is that if disturbances in autonomic cardiac regulation occur, they are more likely to be found in patients with major depression than in patients with brief reactive depression (Rechlin, Weis, Spitzer, & Kaschka, 1994). This hypothesis was supported in that patients with reactive depression did not differ significantly from the normal subjects on any of the physiological measures (e.g., spectral analysis of HRV, root mean squared successive differences). Although patients with amitriptyline-treated melancholic depression showed the lowest HRV, which may be due in part to the drug's anticholinergic effects, the patients with major depression (non-medicated), melancholic type, were found to differ significantly from the normal subjects and the other diagnostic groups. However, Yeragani et al. (1992) failed to find a difference in HRV between untreated patients with major depression and matched control subjects.

Failure to find an effect of major depression on HRV may be due, in part, to the age of the participants. In a study of comparing the HRV of patients with major depression and control subjects, in which no differences in HRV were found, the patients' mean age was considerably higher than that of the controls (Rechlin, Claus, & Weis, 1994). Similarly, in a study which failed to find differences in HRV between depressed individuals and panickers (Yergani, Balon, Pohl, & Ramesh, 1995), the patients with depressed were older compared to the panic group (Yeragani et al., 1992). Since age reduces HRV parameters (Kleiger, Stein, Bosner, & Rottman, 1995), minor alterations caused by affective disorders may not be visible in older patients (Rechlin et al., 1994).

Furthermore, gender differences may moderate the relationship between depression and heart rate variability. While depression has been linked to decreased heart period variability (HPV) in men, depressed female subjects have been shown to have increased HPV compared to their nondepressed counterparts (Thayer, Smith, Rossy, Sollers, & Friedman, 1988).

Although some authors argue that the association of depression with low HRV is related to reduced physical activity in these patients (Watkins et al., 1999), one study restricted activity level and still found that HRV differed significantly between depressed and age and sex-matched nondepressed CHD patients (Carney et al., 1995). Similarly, in a sample of healthy young women, the presence of a subclinical depressive state (measured via Beck Depression Inventory) was associated with both increased cardiac (i.e., shorter pre-ejection period) and peripheral sympathetic activity (i.e., increase in systolic and diastolic blood pressure) as well as reduced vagally mediated HRV across rest and stressor tasks (Light, Kothandapani, & Allen, 1998).

Cardiovascular Functioning and Information Processing

As was discussed previously, anxiety has been shown to be associated with both attentional deployment to threatening information in the environment and decreased vagally mediated HRV (i.e., reduced autonomic flexibility) (Friedman & Thayer, 1998b). This suggests that both psychological and physiological functioning are compromised in anxiety. Several models have been proposed that address a possible link between physiology and affect. For example, Eppinger and Hess (1910, cited in Porges, 1988) proposed that the regulation of autonomic functioning may be linked to affect regulation, specifically suggesting that the parasympathetic branch may be important mediator of physiological and psychological responses.

Models of Emotion, Attention, and HRV

More recently, Richards and Casey (1991) and Porges (1992, 1995) have proposed how autonomic regulation, attention, and emotion are linked. During attention, limbic structures for CV control (i.e., hypothalamus and amygdala), which have descending projections to the vagus and also project to the solitary nucleus controlling respiration, as well as cardioinhibitory centers in the frontal cortex, inhibit the medulla and pons, which control respiratory activity (Richards & Casey, 1991, 1992). This results in decreased respiratory sinus arrhythmia (RSA). RSA reflects the cardiac-respiratory relationship because, during inspiration, vagal influence on the sinoatrial node is diminished, but during expiration, the intervals between heart beats become longer and heart rate decreases, the fluctuations in heart rate that coincide with respiration. Therefore, RSA is a measure of the variability in HR between inspiration and expiration and reflects vagal influences on the heart (Hugdahl, 1998).

The vagus conveys efferent information from brain stem structure to the sinoatrial (SA) node of the heart, and changing vagal influences on the SA node control most of the rapid changes in heart rate (Saul, 1990). Therefore, it is expected that there is a strong relationship between the functional status of the vagal system (reflecting status of the brain-stem) and both autonomic and behavioral reactivity elicited by environmental and cognitive stimuli. As a function of this relationship, attentional processing has been proposed to disrupt normal functioning (i.e., rapid changes in heart rate from vagal influences) and to result in reduced parasympathetic control; therefore, attending to demands from the environment would be enhanced by vagal withdrawl (Porges, 1992).

Early studies with adults supported the idea that attentional processing in adults can be indexed via heart rate variability (e.g., Porges & Raskin, 1969). More recently, a study with school-age children (Suess, Porges, & Plude, 1994) supported the hypothesis that high vagal

control is associated with good attentional capacity. Children with higher resting HRV (indexed by Porges' V) and slower resting heart rates performed better on a continuous performance task (CPT) and, across all subjects, HRV decreased during the CPT, suggesting that change in HRV is a means to determine mental effort and attention.

These studies suggest that high vagal control is associated with good attentional capacity whereas, in infants, low vagal tone has been shown to associated with poor attention and affect regulation (Porges, 1992a,b). The limbic structures that are responsible for CV control (i.e., amygdala and hypothalamus), and which have links to the attentional system (i.e., frontal areas, parietal attentional system, and mesencephalic reticular formation; Richards & Casey, 1991) are also important in the regulation of emotion and emotional behavior (Hugdahl, 1998).

In his polyvagal theory, Porges (1995) describes why emotion and heart rate variability are associated. The parasympathetic nervous system regulates homeostasis by giving negative feedback to the peripheral ANS in response to sympathetic activity, which occurs in many emotional states. Therefore, vagal control may be important in regulation of affective states; this proposal is supported by the proximity of the origin of the facial nerves (i.e., trigeminal), which are important for outward expression of emotion, and vagus nerve. Therefore, individual differences in vagal control may serve as an indicator of ability to appropriately regulate emotion.

Thayer and Friedman (in press) have recently proposed a model that focuses on the roles of both central [γ-aminobutyric acid (GABA) –ergic] and peripheral (HRV) inhibitory processes and suggests how these are important for adaptive behavior and emotion regulation. A focus of this model is the central autonomic network (CAN), a functional unit within the central nervous system that appears to support goal-directed behavior and adaptive functioning, through control of visceromotor, neuroendocrine, and behavioral responses. The structures of the CAN include

the anterior cingulated, insular, and ventromedial prefrontal cortices, the central nucleus of the amygdala, the paraventricular and related nuclei of the hypothalamus, the periaquaductal gray matter, the parabrachial nucleus, the nucleus of the solitary tract, the nucleus ambiguus, the ventrolateral medulla, and the medullary tegmental field. The output of the CAN is mediated through the preganglionic sympathetic and parasympathetic neurons that innervate the heart through the stellate ganglia and the vagus nerve. The variability in heart rate is the results of these inputs to the SA node of the heart. Therefore, the output of the CAN is linked to HRV.

Other CAN structures, the orbital and medial prefrontal cortices, are associated with affective and attentional regulation. There are direct and indirect pathways linking these cortical areas with parasympathetic motor output regions. An intact frontal cortex may tonically inhibit subcortical (amygdala) activity, and decreased medial prefrontal cortex activity may be a means of linking perseverative thinking to anxiety and health. Thus, the CAN model provides a framework for ANS-CNS integration and suggests how inhibitory processes that are an integral aspect of ANS and CNS functioning may subserve adaptive emotion states.

Anxiety, Depression, and HRV

The relationship between emotion, attention and decreased HRV may be applied to the study of worry and attention in anxiety. Worry, in anxious individuals, is a persistent awareness of possible future danger, which is repeatedly rehearsed without being resolved (Mathews, 1990). This may lead to a tendency to select the more emotionally threatening interpretation of cues relating to possible aversive events (i.e., hypervigilance for threat information in the environment).

Hypervigilance and worry result in maladaptive attentional deployment, which is related to low vagal tone (Porges, 1992).

Although decreased HRV in anxiety has been widely supported, the relationship between HRV and depression is less clear. The effects of depression on HRV may be mediated by the role of worry. Individuals with depression tend to engage in a type of worry that can be described as "depressive rumination", a factor that can maintain the depressive state (Borkovec et al., 1998). Depressive rumination involves thoughts of past negative events, which reflects sustained attention to this information. HRV was significantly reduced during sustained attention (Porges & Raskin, 1969). Although sustained attention is necessary to increase information processing, as the duration and intensity of sustained attention increase, so do costs to the organism (Porges, 1992). However, the relationship between depression, HRV, and sustained attention is a speculative one, and HRV may not be the appropriate measure to differentiating anxiety and depression. Discerning differential physiological effects of anxiety and depression will be addressed by using a variety of physiological measures in the current study.

To determine more clearly how autonomic functioning may be compromised in anxiety or depression, both electrocardiogram (ECG) and impedance cardiography will be used in the current study. From ECG, both HR and root mean square difference of successive interbeat intervals (MSSD) were calculated. For MSSD, slow sources of HRV are filtered leaving only faster (i.e., vagal trends). Impedance cardiography is a noninvasive technique for measuring thoracic blood volume changes from which cardiac output, systolic time intervals (pre-ejection period, PEP, and left ventricular ejection time, LVET), and myocardial contractility (Heather index) can be derived (Sherwood et al., 1990). RSA, a measure of vagal influences on the heart, was also derived from impedance cardiography; this is a time-domain peak-to-trough procedure

using inspiration and expiration periods as windows for determining the cardiac-respiratory relationship (Hugdahl, 1998). Of particular interest for the current study, in addition to HR, MSSD, and RSA, is the PEP measure, which is interpreted as a sensitive measure of beta-adrenergic (i.e., cardiac sympathetic) influences on the heart. In previous studies, Light et al. (1998) found shorter PEP (i.e., increased sympathetic activity) in a sample of sub-clinically depressed young women; therefore, PEP may prove to be a useful measure for distinguishing anxiety and depression

Comorbid anxiety/depression

In addition to both "pure" anxiety and depression groups, a comorbid anxiety/depression group was included in the current study. Comorbidity can be broadly defined as the presence of more than one disorder in a person in a defined period of time (Wittchen, 1996a). In a recent study, 59% of the sample who had depression also had anxiety disorder, and anxiety disorders are also the most common primary disorders associated with major depressive disorder (Wittchen, 1996b). Similarly, in a World Health Organization collaborative study, half of the cases of clinically diagnosable anxiety and depression appeared in the same patients at the same time and depressive disorders were more likely to co-occur with anxiety disorders than any other disorder (Sartorius, Ustun, Lecrubier, & Wittchen, 1996).

These studies support the inclusion of a comorbid anxiety/depression group in studies of anxiety and depression. Yeragani et al. (1995), in a study of depression and heart rate variability, did suggest that future studies should include a group with symptoms of both depression and panic disorder. However, this group has not often been previously included in physiological research on anxiety and depression, which is surprising since previous studies have found that these conditions are comorbid 50-60%. One study did find that depressed/high trait anxiety

patients had significantly lower HRV than non-anxious/depressed controls, suggesting that further studies are needed to determine the link between comorbid anxiety/depression and HRV more clearly (Tulen et al., 1996).

However, there are currently are no studies on the relationship between comorbid anxiety/depression and HRV in a young, healthy population, so one focus of the current study was to attempt to find the relationship between these variables in this particular group. Since anxiety and depression are so highly comorbid (e.g., Wittchen, 1996b), it was anticipated that it would be more difficult to find "pure" depressed or anxious participants for the current study. This was not considered a limitation to the current study since the sample sizes found appear to reflect true population differences (e.g., Angst & Dobler-Mikola, 1985; Lydiard & Brawman-Mintzer, 1998).

Design and Hypotheses

The purpose of the present study was to further explore cognitive biases in both anxiety and depression and to assess how these disorders affect cardiovascular functioning in non-clinical populations. This was explored by administering an emotional Stroop task and by assessing physiological responses measured via ECG and impedance cardiography. Response latency to the word groups in the emotional Stroop task will be determined. The design of this study is a 4 (group) X 3 (condition) mixed design, with group as the between subjects factor. The variable word-type (for the Stroop task) is nested within condition.

Physiological measures

The Stroop task has often been used as a laboratory stressor in studies of cardiovascular reactivity (e.g., Manuck, Kasprowicz, & Muldoon, 1990). In the present study, participants experienced tasks of rest, Stroop task stressor, and recovery. It was hypothesized that both

anxious and depressed individuals will show decreased heart rate variability (measured by MSSD and RSA) across all three tasks, which would serve as a marker for low vagal control. A co-mordid anxiety-depression group, which has not been previously included in Stroop research, will also be included in this study. One study compared co-morbid anxious/depressed subjects with depressed-only subjects and found that HRV was significantly lower in the co-morbid group (Tulen et al, 1996). Therefore, it is hypothesized that this group will also have decreased vagally mediated HRV across tasks compared to controls. The control group, who is defined as having low levels of anxiety and depression, should show the highest HRV across tasks. HR and impedance cardiography measures will also be taken during each of these tasks.

Since panickers have been shown to have elevated sympathetic activity and higher HR than depressed individuals (see Friedman & Thayer, 1998a for a review), impedance measures, which provide an index of cardiac sympathetic influences, may be useful in distinguishing between the anxiety and depression groups across tasks. It is hypothesized that the anxiety group and the comorbid anxiety/depression group will have significantly higher HR and shorter PEP across conditions compared to controls.

Change scores (rest-stressor difference) will also be compared between the groups for MSSD, RSA, HR, and PEP. Since the Stroop task is a behavioral stressor (Manuck et al., 1990), it is expected that, generally, exposure to this stressor would result in increased HR, shorter PEP, and decreased MSSD and RSA. Therefore, since it was hypothesized that the anxious, depressed, and comorbid anxious/depressed groups would have decreased MSSD and RSA across conditions, it is predicted that these groups would have significantly less change between rest and stressor than the control group. This was predicted because it was expected that the negative affect groups would have lower resting vagal control than the control group and, as a function of

this, should not show as much change between rest and stressor. Similarly, it is expected that the anxiety and comorbid anxiety/depression groups will have significantly less change between rest and stressor than the depression and control groups for HR and PEP.

Categories of Stroop Words

Past researchers using the emotional Stroop task have used anxiety and depression words generated by the researchers. In the current study, word groups will be generated using the circumplex model of emotion (Russell, 1980). In this model, anxiety is seen as high arousal-negative affect and depression as low-arousal negative affect. Since the emphasis in the circumplex model is on the ecological validity of representations of emotions, college students generated the word groups in the current task from ratings of groups of words. This is in contrast to previous studies of the emotional Stroop, in which word lists were generated by the experimenters (e.g., Bradley, Mogg, & Williams, 1993).

It was hypothesized that the anxious individuals should show greater response inhibition (i.e., longer response latency) to the high arousal-negative affect words. Depressed individuals may have greater response inhibition to low arousal-negative affect words (based on the circumplex model) or to the positive words, since depression may result in the neglect of positive information (Dalgleish & Watts, 1990). The effect of word group has not been widely substantiated in depression. However, in several emotional Stroop studies, anxious individuals tended to show a bias (i.e., longer response latency) for negative words (Mogg et al., 1993). This suggests that depression and anxiety-relevant words chosen for these studies may be more closely related to the common experience of negative affect shared by these disorders. Since the word groups in the present study were carefully chosen to represent the specific characteristics of anxiety and depression (high vs. low arousal), these word groups may show better discrimination

between these two groups. It was further hypothesized that the co-morbid anxiety-depression group should show longer response latencies to all negative words and that the control group should not show response bias for any of the word groups.

METHOD

Participants

Right-handed females (age range: 18 to 23 years) who were not currently taking antidepressants (N = 302) were recruited from undergraduate students for the first stage of this study; extra credit was offered for participation. Data from 27 subjects were not used because the questionnaire responses were incomplete. Females were chosen for this study based on the finding that they are far more likely than men to report both anxiety and depression (Culbertson, 1997). Subjects were initially assessed for their level of anxiety and depression using the Beck Anxiety Inventory (BAI; Beck, Epstein, Brown, & Steer, 1988; Appendix A) and Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961; Appendix B). These self-report measures and a lateral-preference inventory (Coren, Porac, & Duncan, 1979; used to confirm that all were right-handed) (Appendix C) were group-administered by the experimenter. Based on the BAI and BDI scores (>9 is mild anxiety or depression, >16 moderate, >20 severe; see description of inventories in the materials section), four groups were chosen for the second stage of the study: "pure" anxiety group (BAI>9; BDI<9), "pure" depression group (BAI<9; BDI>9), co-morbid anxiety depression group (BAI & BDI >9), and low anxious/depressed (BAI & BDI <9) (See Table 1 for questionnaire means by group). All participants abstained from caffeine and alcohol for 12-hr before the study, and received compensation in the form of extra credit points for participation in both parts of the study.

Average questionnaire scores, age, weight, and alcohol and caffeine intake by group are reported in Table 1. Several of the initial low anxiety/depression subjects (n=5) were identified as repressors based on reports of low trait anxiety and high social desirability (Hock, Krohne, & Kaiser, 1996). For this sample, repressor status was defined as a trait anxiety score less than or equal to 30 (possible low=20, possible high=80) and a social desirability score [Marlowe-Crowne Social Desirability Scale (SDS), reference] (Appendix D) greater than or equal two 22 (out of a possible 33). The average social desirability score in this sample was 23.6. The BAI, BDI, state and trait anxiety (STAI) (Appendix E) means for this group were as follows: 2.8, 1.0, 23.6, and 26.4. Thus the repressor group had the highest rating for social desirability but the lowest for anxiety (state, trait, and BAI) as well as depression compared to the other four groups. The data for these subjects was removed from the low anxiety/depression group. This decision was based on previous research that suggests that repressors often respond both physiologically and behaviorally as if they are highly anxious (Weinberger et al., 1979).

Data from 8 subjects who participated in the second stage of the study were not included. This was due to equipment problems, physiological measures out of normal range (e.g., one subject whose data was not included had an average MSSD of 280 ms), or subject error (e.g., participating in the study directly after exercising).

Materials

Screening Stage Questionnaires

The BDI is a 21-item self-report measure that assesses cognitive, motivational, and physiological symptoms of depression that was used to identify levels of depression in this sample (Beck et al., 1961) (Appendix B). Participants are asked to indicate which of four self-evaluative statements they agree with. Items assess mood, pessimism, guilt, irritability, crying spells, sleep

and appetite disturbance and loss of libido. Each item is scored from 0 to 3; 0 indicates an absence of depression (e.g., “I don’t feel I am worse than anybody else”) and 3 indicates severe depression (e.g., “I blame myself for everything bad that happens”). Possible scores range between 0-63. Adults scoring 0-9 show no indication of a depressed state, those scoring 10-15 only a mild level, 16-23 reflects a moderate state, and scores ranging from 24-63 indicate a severe level of depression.

The BAI (Beck et al., 1988) (Appendix A) is a 21-item self-report measure that assesses the presence and severity of anxious symptoms. The overlap between symptoms of anxiety and depression was recognized during the development of the BAI, and attempts were made to avoid such overlap in the generation of items. Fourteen somatically related symptoms and seven cognitive and subjective features related to anxiety and panic were developed. The scoring procedure for the BAI is the same as for the BDI described in the previous section.

Lateral Preference Inventory (Appendix C). The lateral preference inventory is an 11 item self-report battery for the assessment of hand, eye, foot, and ear preference (Coren, Porac, & Duncan, 1979). Left preference is scored with –1, both right and left preference as zero, and right preference as +1. Total positive scores indicate right laterized preference and negative scores indicate left lateralized preference.

Experimental Stage Questionnaires

The BAI and BAI were also used in the experimental stage of the study.

Emotion Adjective Questionnaire. The emotion adjective questionnaire is a list of twelve emotion adjectives with a 5-point likert scale for each item (1=experiencing none of the emotion, 5=experiencing a lot of the emotion) (Nyklicek et al., 1997) (Appendix F). The questionnaire was

designed to broadly sample the domain of affective space. This questionnaire was administered after the Stroop stressor task.

State-Trait Anxiety Inventory (STAI), Form Y. The STAI is a 40-item two-section questionnaire that assesses both trait anxiety, relatively stable individual differences in anxiety proneness, and state anxiety, current experiences of anxiety (Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983) (Appendix E). The state anxiety scale consists of 20 statements that evaluate how the respondent feels right now; responses range from 1 ("not at all") to 4 ("very much so"). The trait scale consists of 20 statements that assess how the person generally feels, with responses also from 1 through 4. Scores range from 20-80 on each section, with higher scores representing a greater amount of anxiety. Speilberger et al. (1983) reported that mean state anxiety for college students was 36.47 for males and 38.76 for females, and mean trait anxiety was 38.3 for males and 40.4 for females. The test-retest reliability (104 days) for college students was .75 for trait anxiety and .32 for state anxiety, with alpha coefficients of .92 for state anxiety and .91 for trait anxiety.

Marlowe-Crowne Social Desirability Scale (MC-SDS). The MCSDS is a 33 item true-false questionnaire designed to measure both social desirability and avoidance of disapproval (Crowne & Marlowe, 1960) (Appendix D). Scores range from 0 to 33 with higher scores representing higher need for approval (Crowne & Marlowe, 1964). Means of 14.0 and 12.3 have been found in normal and depressed respondents (N=503). Alpha coefficients have ranged between .73 to .88 in various samples, with test-retest reliability of .88 across 1 month.

Affect Intensity Measure (AIM) (Appendix G). Affect intensity relates to stable individual differences in the intensity or magnitude of experienced emotion (Larsen & Diener, 1987). Those who score high on affect intensity experience both positive and negative emotions quite strongly

compared to low affect intensity individuals who tend to experience emotions mildly with few fluctuations. The AIM was included in the current study to address the arousal dimension in the circumplex model, in which high arousal/activation is associated with anxiety. Since high activation is also correlated with high affect intensity (Larsen & Diener, 1992), the AIM was included in the current study to determine if high affect intensity is related to anxiety. The AIM questionnaire is a 40-item self-report measure that assesses the degree of affect intensity, with higher scores representing high affect intensity. In previous studies, test-retest reliabilities for the AIM at 1, 2, and 3 month intervals were .80, .81, and .81, respectively, and α coefficients ranged between .90 and .94 across four separate samples (Larsen & Diener, 1987).

Word Groups on Stroop Task

A list of 58 words was generated by one graduate student and one upper-level undergraduate student on the basis of these words matching the following categories: high arousal-negative emotionality (e.g., distressed, nervous), low arousal-negative emotionality (e.g., alone, despondent), high arousal-positive emotionality (e.g., joyful, euphoric), low arousal-positive emotionality (e.g., pleasant, tranquil), and neutral/low arousal-low emotionality (e.g., vague, impartial). These words were rated by 37 undergraduates. The 58 words were randomly listed on a page. Each word was rated on a five-point Likert-type scale (1=low, 5=high) on the dimensions of arousal, emotionality, negative affect, and positive affect. Word groups were chosen by calculating the means for each word on each of the four dimensions. Low and high criteria for each of the four dimensions was calculated by using the median for each of the four dimensions across words. The range of distances from the median was similar across all groups. Ten words were generated for each of the five groups (Appendix H). Word groups did not significantly differ in length, $F(4, 45) = 1.275$, $p = .294$. However, the mean word length for the

low arousal/negative word group was slightly higher (M = 8.5 letters) than the other groups and slightly lower (M = 6.6) in the low arousal/positive group (range of means for other groups, M = 7.2-7.8), so several words were substituted with shorter or longer words of similar frequency in the English language. After the word substitution occurred, there continued to be no significant difference in word length between the groups, $F(4, 45) = .055$, $p = .994$, or frequency in the language, $F(4, 45) = .316$, $p = .866$

Ambulatory Monitoring System (AMS). ECG and thoracic impedance variables were recorded using an AMS monitor (Vrije Universiteit, Department of Psychophysiology, Amsterdam, the Netherlands). The validity of this device is similar to that of a non-ambulatory device for measuring these variables (de Geus, Willemsen, Klaver, & van Doornen, 1995).

Procedure

Electrode attachment. Upon arrival, all subjects first completed informed consent and a screening form, which included questions regarding handedness, caffeine and alcohol consumption, exercise habits, and medication intake. Handedness was used to assess hemispheric dominance, and all subjects who participated in the initial screening and the second session were required to be right-handed. Since right-handed individuals tend to have more lateralized functioning (Kolb & Whishaw, 1996), and the left and right cerebral cortex contribute differently to the regulation of emotion (Davidson, 1992), it is important to control hemispheric dominance in studies involving emotion. It has also been proposed that hemispheric laterality appears to be an important variable for understanding individual differences in processing negative affective stimuli, including threat stimuli in anxious patients (Otto, McNally, Pollack, Chen, & Rosenbaum, 1994).

Electrodes were placed on the subject's torso and connected to the AMS monitor, in accordance with the guidelines described in the User Manual v.1.2 for the Ambulatory Monitoring

System (Appendix I). All subjects and experimenters who assisted in the process of electrode placement were gender-matched. The Suretrace Ag/AgCl electrodes (ConMed Corporation) are single-use and pre-gelled with adhesive on one side. Each area where an electrode is placed was lightly abraded with Omniprep and then swabbed with alcohol. The subject was asked to sit quietly in a chair until the AMS monitor found a steady signal (M = 3 min).

Conditions. During the first condition, the subject was asked to sit quietly in a chair with eyes closed for 5 min. while physiological variables were recorded. There was an average of a 1.5 min delay between the time the subject moved to the new seating location and the beginning of the next task. The Stroop stressor task (software written for the purposes of this study; copyright B. Pumphrey, 1999) was then administered; the response format was explained to the subject at this time, who was asked not to begin until prompted by the experimenter. All stimulus words were written in uppercase Arial 12 point font and were displayed on a 23.5 cm x 30 cm computer monitor with a black screen. Each word was presented on the screen until the subject responds by pressing one of the color-coded keys. In the practice task, 20 color-name words were presented; color of the word was indicated by pressing 1 of 4 buttons on a computer keyboard that correspond to the colors red, yellow, green, and blue. In the emotional Stroop task, the 50 stimuli words were presented in a new random order for each participant; color of each word was indicated by pressing a button on the keyboard. A free recall task was administered after the emotional Stroop task to determine any explicit memory bias that may be occurring for specific word groups (e.g., depression-relevant words for individuals with depression). The subject returned to the seating location from the first task. There was an average of 1.5 minute delay before the next task began. For the final condition, subjects were once again asked to sit quietly in a chair (recovery) for 5 min. Each subject completed several questionnaires after the recovery

task: the BDI, BAI, AIM, MC-SDS, and the STAI. Informed consent forms and screening form are located in the appendices (Appendix J and K).

ANALYSES

Physiological measures

Because the cell sizes were unequal, all the following univariate analysis of variance tests were done utilizing Type III sum of squares using estimated marginal means with a Sidak post-hoc test, where applicable (SPSS, v. 9.0, 2000). The Type III sum of squares was used because this approach is recommended for an unbalanced model with no missing cells; this method calculates the sum of squares of an effect in the design as the sum of squares adjusted for any other effects that do not contain it (SPSS, v. 9.0, 2000). Although the Sidak is a less powerful than more commonly used post-hoc tests (e.g., Tukey), it adjusts the observed significance level for the fact that multiple comparisons are made and provides tighter bounds than the Bonferroni test.

To analyze the physiological measures between groups and across tasks, data were standardized within subject and aggregated over tasks (rest, Stroop, and recovery. The standardization procedure is used when averaging across conditions to avoid confounding between and within subjects' variability (Friedman & Thayer, 1998b). Although differences were in the predicted direction of effect, there was not a significant difference between the groups for average MSSD, $F(3, 57) = 1.16$, $p = .34$, or average RSA, $F(3, 56) = 1.62$, $p = .20$ (see Table 2 and Figure 1 for group means). However, contrast analyses comparing each of the three NA groups with the control group showed significantly lower average MSSD, $t(42) = -2.031$, $p = .048$, and average RSA, $t(42) = -1.075$, $p = .01$ in the comorbid anxious/depressed group than the control group. Contrast analyses between the other NA groups and the control group were not

significant. There were also no group differences for RSA during rest, $F(3, 57) = 2.33$, $p = .08$; however, during recovery, co-morbid anxious-depressed had significantly lower RSA ($M = 49.48$) than the low anxiety-depression group ($M = 78.9$), $F(3, 57) = 3.48$, $p = .02$ ($p = .01$ for post-hoc analysis). No statistically significant differences were found for HR for either rest, $F(3, 58) = 1.561$, $p = .21$, recovery, $F(3, 58) = 1.49$, $p = .23$, or average HR across tasks, $F(3, 62) = 1.14$, $p = .24$. Similar outcomes were found for PEP in rest, $F(3, 63) = .625$, $p = .601$, Stroop, $F (3, 58) = 1.46$, $p = .24$, recovery, $F(3, 58) = .45$, $p = .72$, or average PEP across tasks, $F (3, 58) = .35$, $p = .79$.

Change scores were computed by subtracting the rest from the stressor conditions; this was done to determine the changes in ANS functioning that occurred as result of exposure to the stressor. These were not significant for changes in HR, $F(3, 58) = .79$, $p = .50$, or PEP, $F(3, 58) = 1.31$, $p = .28$. Results did approach significance for MSSD change, $F (3, 58) = 2.307$, $p = .09$, and were significant for RSA difference, $F (3, 56) = 3.826$, $p = .015$, with the comorbid anxious-depressed group significantly smaller change in RSA ($M = 16.09$) than the control group ($M = 67.50$), $p = .009$. See Figures 2 and 3 for change scores by group.

Since low HRV in depression has often been presupposed to be a function of reduced physical activity, it is important to control for activity level by determining if physical activity is mainly associated with any one group. There was not a significant relationship between group and regular or non-regular exercise, $\chi^2(N=67, 3) = 2.264$, $p = .519$.

Stroop Task Word Groups

For the negative low arousal words, the effect of group was significant, $F(3, 58) = 4.10$, $p = .01$, with the anxiety group having significantly longer response times than the controls, $p = .03$ (see Table 2 for means). The effect of group was also significant for the positive, high arousal

words, F(3, 58) = 5.69, p = .002, with the anxiety group showing significantly longer response latencies than the depressed (p = .01) and control groups (p = .01). There were no significant group differences for positive low arousal words, F(3, 57) = 2.14, p = .11, or neutral words, F(3, 57) = 2.06, p = .12. However, the effect of group did approach significance for negative high arousal words, F (3, 58) = 2.342, p = .08. Averaged across all word types, the effect of group was significant, F(3, 58) = 5.515, p =.002, with the anxiety group (M = 1033.17) showing significantly longer response latencies than either the depressed (M = 807.42) or control groups (M = 872.99). Although not significantly different from the control group, the depressed group overall had the shortest response latencies. See Table 3 for word-type means by group.

Questionnaires

The BAI and BDI scores across the four groups were significantly correlated with the initial scores from the screening session (BAI, r = .505, p = .0001; BDI, r = .915, p = .0001). However, the screening stage BAI and second stage BDI scores were also highly correlated (r = .637, p = .0001) as well as the second stage BDI and BAI scores (r = .699, p = .0001), attesting to the high comorbidity of the disorders. These correlations between anxiety and depression are similar to those found in previous studies (Clark et al., 1994). Affect intensity was not significantly correlated with any of the other measures but social desirability (MC-SDS) was significantly negatively correlated with State anxiety (r = -.298, p =.014), Trait anxiety (r = -375, p=.002), anxiety measured by the BAI (r = -.449, p =.0001) and depression (r = -.325, p = .007). On the Emotion Adjective Questionnaire (Nyklicek et al., 1997), the co-morbid anxiety-depression group reported feeling significantly less peaceful, F(3, 63) = 7.438, p = .0001, happy, F(3, 63) = 9.468, p = .0001, and more sad, F(3, 63) = 13.603, p = .0001, than the other three groups. The co-morbid was also significantly more tired, F(3, 63) = 3.969, p = .01, and more

uneasy, $F(3, 63) = 5.729$, $p = .002$, than the control group. See Table 4 for mean word emotion adjective responses by group.

Multivariate Analyses

A discriminant analysis was done using the questionnaire totals (AIM, MC-SDS, SAI, TAI), physiological measures within and across conditions (HR, MSSD, PEP, HI, LVET, RSA), and reaction times on Stroop task by word group (positive/high arousal, negative/high arousal, positive/low arousal, negative/low arousal, and neutral). Using these variables, 94.6% of the original group cases were correctly classified. The greatest misclassification problems were in the depression group: three of the four subjects in this group were classified in the control group. The anxious group ($n = 9$) and the comorbid group ($n = 23$) each had 100% classification in their original groups. For the control group, 94.7% were classified in the original group, with only one subject classified in the depression group.

DISCUSSION

Physiological Measures

The main hypothesis was that the negative affect (anxious, depressed, and comorbid) groups would have lower vagally mediated HRV across tasks than the low anxious/depressed group. The most robust findings were for the comorbid anxiety/depression group. This group not only had significantly lower average RSA and MSSA than the control group, but also had significantly lower RSA during recovery and smaller RSA change scores than the control group. Significant differences were not found between either of the "pure" groups (anxious or depressed) and the control group, although the means were in the expected direction of effect (See Figures 1 and 2 and Table 2). RSA and MSSD were both consistently lower in the NA groups than in the nonanxious/nondepressed control group.

Disparate group sizes and large standard deviations (see Table 1) may have contributed to the failure to reach significance for either of the "pure" NA groups. The comorbid group also tended to have lower MSSD and RSA both within and across conditions compared to the other NA groups. This finding is consistent with clinical research: subjects with diagnoses of both anxiety and depression are more severely affected in general (i.e., often have more severe presentation of symptoms, longer duration of therapy, and poorer treatment outcome) (Angst & Dobler-Mikola, 1985). Based on these findings, it is not surprising that the comorbid group would have the lowest HRV; this is consistent with this group having the most impaired psychological functioning.

In the current study, the self report of emotion on the Emotion Adjective Questionnaire substantiated that co-morbid anxiety-depression appears to be a more severe presentation of both disorders: this group not only reported less positive emotion than the other three groups but also more uneasiness, which is an arousal component. This pattern of results (comorbid group with lowest vagally mediated HRV, control with highest, and "pure" NA groups between these two groups) are also consistent with findings by Tulen et al. (1996), who found in a clinical sample that the HRV of depressed/low anxious patients was between that of the controls and the depressed/high anxiety patients. These results taken together demonstrate that HRV may be an important measure in serving as a broadly patholitic marker in a variety of psychological conditions, including anxiety, depression, comorbid anxiety/depression, panic disorder, and PTSD (Friedman & Thayer, 1998b).

Previous studies have suggested that panickers have elevated sympathetic activity and higher HR than depressed individuals (Rechlin et al., 1994), and that subclinical depression may also be associated with increased cardiac sympathetic activity (Light et al, 1998). This hypothesis

was not supported in that none of the group differences for HR or PEP were significant. However, the small change scores (rest-stressor) for HR, PEP, and MSSD suggest that the Stroop may not have been a potent stressor. This may have been partially a function of the short duration of the stressor task (participants averaged 42-s to complete the Stroop task). The impedance monitor collected the ICG complexes at 30-s intervals; therefore, often only one or two ICG waveforms were available from which to derive the cardiac sympathetic measures. This situation may be ameliorated by either using a potent stressor than the Stroop, or by increasing the length (i.e., adding additional emotional words) and difficulty of the Stroop task to make the task a more powerful stressor.

Additionally, it was noted anecdotally that some subjects had difficulty distinguishing the yellow and green words on the screen, even though these colors were selected by hue and intensity to represent a pure bright yellow or green. A means to potentiate the stressor effect may be to increase the ambiguity of the colors as the Stroop task continues, forcing the participant to select a response for a color that is a blend of two stimulus colors. An increased stressor may also be more efficacious for producing different recovery outcomes in the final resting task (i.e., recovery). In this framework, reactivity to a stressor can be viewed as a means of determining an organism's responsiveness (Friedman & Thayer, 1998b). Organisms who are unable to respond to change with flexibility and resiliency may continue to show decreased vagally mediated HRV even after the stressor is no longer being presented (i.e., during recovery). Greater vagal control seen in the recovery task would suggest a more adaptive CV system that can respond flexibly to changing environmental demands.

Examination of the change scores also reveals that change in the variable of interest did not always occur in the expected direction. Although a stressor often results in an increase in

sympathetic activity or vagal withdrawl, this was not always the case for every participant: several subjects showed parasympathetic activation during the stressor task. This response appeared the most often in the co-morbid anxious/depressed group (8 participants), and in four additional participants in the other NA groups. This response may not fit the expected reaction to a stressor if the goal is to uncover nomothetic norms in physiological responding. However, when couched in terms of individual response stereotopy (IRS), individuals are expected to show idiosyncratic responding to laboratory tasks and demonstrate these characteristic individual responses across conditions (Malmo & Shagass, 1949, cited in Engel, 1972). In this model, subjects can show opposite responses to the same stimulus, as was demonstrated in the response to the Stroop task stressor in the current study.

The relationship between exercise, affect, and low HRV did not appear to be an issue in the current study: exercise or lack thereof was not significantly related to any one particular group. This was important to determine since a criticism of past studies on depression and HRV is that this relationship is mainly a function of reduced physical activity (Carney et al., 2000). However, participants were classified into the categories of "regular" vs. "not regular" exercisers on the basis of their responses on the screening questionnaire. Thus, regular physical activity included a wide range of the type, duration, and intensity of the activity. Future studies will include a much more precise rendering of the activity level of the groups through the use of fitness questionnaires.

Future research on autonomic effects of anxiety should include a more thorough screening for different types of anxiety, which is important because subtypes of anxiety, such as panic and blood phobia, have very different autonomic characteristics (Friedman & Thayer, 1998a). While two measures of anxiety were used in the current study, the BAI items focus on very diverse

physiological effects that may characterize any one of the subtypes of anxiety, and the STAI items have many common characteristics with the BDI (i.e., does not appear to focus on the unique characteristics of anxiety).

Stroop Task Words

It was hypothesized that the anxious individuals should show greater response inhibition (i.e., longer response latency) to the high arousal-negative affect words and the depressed group for low arousal-negative affect words. For the negative low arousal words, the effect of group was significant, with the anxiety group having significantly longer response times than the controls. The effect of group was also significant for the positive, high arousal words, with the anxiety group showing significantly longer response latencies than the depressed and control groups. Therefore, the anxiety group also had the longest response latencies for both negative low arousal and positive high arousal words. While past studies have generally supported the idea that negative words command more processing resources in anxious subjects (MacLeod et al., 1986; Mogg et al., 1993, this effect has not been found with positive words.

While the current findings for the anxiety group seem inconsistent with past research, the present results support the use of the circumplex model of emotion. Since the arousal dimension differentiates anxiety and depression, the Stroop findings suggest that the anxious group may be threatened by high intensity emotions, whether negative or positive. This is consistent with the proposal that emotion intensity is a stable characteristic of a person and generalizes across stimulus conditions and emotional domains (Larsen & Diener, 1987).

The concept of cognitive avoidance that has been demonstrated in anxiety may also be why the effect was found for positive words in the anxiety group. Anxious individuals have a strong motivation to cognitively avoid threatening information, which may lead to an attention

bias for stimuli to be avoided. This attentional bias may be adaptive in that it enables a subject to come up with an avoidance response (Lavy & van den Hout, 1994). Dalgleish and Watts (1990) have proposed that presentations of emotion words may have a more powerful effect, such as in the Stroop paradigm, when they are relevant to the person's emotional situation. The positive, high arousal words may have appeared threatening because they were the exact opposite of what the person was feeling (i.e., negative). However, the PA/NA model proposes that while depression is marked by a lack of PA, anxiety is not. Future research is needed to explore the relationship between PA and NA in anxiety, as well as to substantiate the finding of a bias for positive words in the Stroop task.

Interpretation of performance on tasks such as the emotional Stroop can be difficult because there may be different types of processing involved at different levels that contribute to the final response. Adaptations of the Stroop with sub-threshold presentation of stimuli have generally supported the idea that the attentional bias in anxiety is automatic in nature. However, delayed response on Stroop-like tasks may reflect both the strength of involuntary response tendencies competing with the correct response, as well as the amount of voluntary effort needed to suppress the competing response (Matthews & Wells, 1999). However, others disagree and suggest that this demonstrates not an attentional bias but a response bias. This response bias can be the result of response inhibition or cognitive avoidance (de Ruiter & Brosschot, 1994).

The difficulty of interpretation of Stroop task performance can be seen in the current study with the performance of the depression group. This group consistently had the shortest response latencies across all word groups, which may suggest that this group did not have either an attention or response bias for any of the word types. However, these findings are consistent with the idea that depression is characterized as a state where external information makes little impact

on conscious processing, because the individual is occupied by internal thoughts (Rothbart, Posner, & Rosicky, 1994). This would suggest that a depressed individual may not have been paying attention to the words during the Stroop task, resulting in the short response latencies. For the comorbid anxious/depressed group, the findings were interesting because this group did not have any significant effects for the categories of Stroop words. One possible explanation is that this may have been due to an averaging effect from both conditions (i.e., the anxiety group had the longest latencies and the depression group had the shortest). However, a comorbid anxious/depressed group has not been previously used in emotional Stroop research, so this interpretation is only speculative.

Although there were not many significant group differences for responses to individual Stroop words, the investigation of group (e.g., anxious, depressed) response biases may become a more suitable research topic when the list of Stroop words used in this study is validated. Determining the individual response latencies for each of the words in the Stroop task may also help to determine words that are disorder-relevant for future studies. Past studies tended to examine the effects of word groups, but individual words may have differentially contributed to the overall response. By determining response latencies to individual words, ineffective words (e.g., a high arousal-negative word that had a similar response latency to neutral word for anxious individuals) can be discarded and effective words retained for future studies.

Conclusions

One focus of this study was to explore the use of circumplex model (Russell, 1980) to represent affective and autonomic space for anxiety and depression. The comorbid anxiety/depression group was found to have significantly lower HRV than the nonanxious/nondepressed control group both within and across conditions. This suggests that the

arousal/activation dimension may be an important aspect of a model that depicts the autonomic and affective space of both anxiety and depression and that low HRV may be common to NA states. Although results were not significant for the pure anxiety and depression groups, means for each group were in the predicted direction of effect (i.e., mean MSSD and RSA within and across conditions was lower in both these groups than the controls). These findings suggest that reduced cardiac vagal control can be considered a broadly patholitic marker for a wide range of disorders.

Thayer and Lane (in press) propose that HRV is an index of neurovisceral integration and reflects self-regulatory ability, which is responsiveness to changes in the environment, both internal and external, which includes the capacity to attend to salient events and disregard irrelevant information. The affective styles of anxious and depressed individuals are detrimental to functioning because the ability to appropriately respond to environmental demands is compromised. This can be seen in the Stroop results: the anxiety group appeared to be threatened by high intensity stimuli of both valences and the depression group may have been preoccupied by negative internal thoughts. Future research should also investigate comorbid anxiety/depression as a separate group to gain a better understanding of the unique characteristics of this group. In sum, a comprehensive model of anxiety, depression, and comorbid anxiety/depression should include a thorough understanding of compromised functioning at many different levels, including affective, cognitive, and physiological. This proposal is supported in the results from the discriminant analysis, which showed that differences between the groups in this study may be better represented by a range of variables.

REFERENCES

Angst, J., & Dobler-Mikola, A. (1985). The Zurich study:VI. A continuum from depression to anxiety disorders? European Archives of Psychiatry and Neurological Sciences, 235, 179-186.

Beck, A.T. (1976). Cognitive therapy and emotional disorders. New York: International Universities Press.

Beck, A.T., Epstein, N., Brown, G., & Steer, R.A. (1988). An inventory for measuring clinical anxiety: Psychometic properties. Journal of Consulting and Clincial Psychology, 56, 893-897.

Beck, A.T., Ward, C.H., Mendelson, M., Mock, J., & Erlbaugh, J. (1961). An inventory for measuring depression. Archives of General Psychiatry, 4, 53-61.

Borkovec, T. D., Ray, W.J., & Stober, J. (1998). Worry: A cognitive phenomenon intimately linked to affective, physiological, and interpersonal behavioral processes. Cognitive Therapy and Research, 22, 561-576.

Bradley, B.P., Mogg, K., & Williams, R. (1993). Implicit and explicit memory for emotional information in non-clinical subjects. Behaviour Research and Therapy, 32, 65-78.

Cacioppo, J.T., & Bernston, G.G. (1999).The affect system: Architecture and operating characteristics. Current Directions in Psychological Science, 8, 133-137.

Clark, D.A., Steer, R.A., & Beck, A.T. (1994). Common and specific dimensions of self-reported anxiety and depression: Implications for the cognitive and tripartite models. Journal of Abnormal Psychology, 103, 645-654.

Clark, L.A., & Watson, D. (1991). Tripartite model of anxiety and depression: Psychometric evidence and taxonomic implications. Journal of Abnormal Psychology, 100, 316-336.

Cohen, H. Benjamin, J., Geva, A.B., Matar, M.A., Kaplan, Z., & Kotler, M. (2000). Autonomic dysregulation in panic disorder and in post-traumatic stress disorder: Application of power spectrum analysis of heart rate variability at rest and in response to recollection of trauma or panic attacks. Psychiatry Research, 96, 1-13.

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

Crowne, D.P., & Marlowe, D. (1964). Studies in evaluative dependence. New York: Wiley.

Culbertson, F.M. (1997). Depression and gender. American Psychologist, 52, 25-31.

Dalgleish, T., & Watts, F.N. (1990). Biases of attention and memory in disorders of anxiety and depression. Clinical Psychology Review, 10, 589-604.

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

Davis, P.J. (1987). Repression and inaccessibility of affective memories. Journal of Personality and Social Psychology, 53, 585-593.

de Geus, E.J.C., Willemsen, G.H.M., Klaver, H.A.M., & van Doornen, L.J.P. (1995). Ambulatory measurement of respiratory sinus arrhythmia and respiration rate. Biological Psychology, 41, 205-227.

de Ruiter, C., & Brosschot, J.F. (1994). The emotional Stroop interference effect in anxiety: Attentional bias or cognitive avoidance? Behaviour Research and Therapy, 3, 315-319.

Ellis, H.C., Thomas, R.L., & Rodriguez, I.A. (1994). Emotional mood states and memory: Elaborative encoding, semantic processing, and cognitive effort. Journal of Experimental Psychology: Learning, Memory, and Cognition, 10, 470-482.

Eysenck, M.W., MacLeod, C., & Mathews, A. (1987). Cognitive functioning and anxiety. Psychological Research, 49, 189-195.

Freud, S. (1915). Repression. Translated in Collected Papers Vol. IV. London: Hogarth.

Friedman, B.H., Takayama, E., Long, D.L., & Thayer, J.F. (1997, May). Affective and cardiac reactions to quiet rest, shock avoidance, facial cooling, and combined shock avoidance/facial cooling. Poster presented at the Biological Basis of Behavior preconference, annual meeting of the American Psychological Society, Washington DC.

Friedman, B.H., & Thayer, J.F. (1998a). Autonomic balance revisited: Panic anxiety and heart rate variability. Journal of Psychosomatic Research, 44, 133-151.

Friedman, B.H., & Thayer, J.F. (1998b). Anxiety and autonomic flexibility: A cardiovascular approach. Biological Psychology, 49, 303-323.

Friedman, B.H., Thayer, J.F., & Borkovec, T.D. (2000). Explicit memory bias for threat words in generalized anxiety disorder. Behavior Therapy, 31, 745-756.

Gotlib, I.H. & McCann, C.D. (1984). Construct accessibility and depression: An examination of cognitive and affective factors. Journal of Personality and Social Psychology, 47, 427-439.

Green, D.P., & Salovey, P. (1999). In what sense are positive and negative affect independent?: A reply to Tellegen, Watson, and Clark. Psychological Science, 10, 304-306.

Hugdahl, K. (1998). Psychophysiology: The mind-body perspective. Cambridge, MA: Harvard University Press.

Katon, W., & Roy-Byrne, P.P. (1991). Mixed anxiety and depression. Journal of Abnormal Psychology, 100, 337-345.

Kleiger, R.E., Stein, P.K., Bosner, M.S., & Rottman, J.N. (1995). Time-domain measurements of heart rate variability. In M. Malik & A.J. Camm (Eds.), Heart rate variability (pp. 33-45). Armonk, NY: Futura Publishing Company.

Klein, E., Cnaani, E., Harel, T., Braun, S., & Ben-Haim, S.A. (1995). Altered heart rate variability in panic disorder patients. Biological Psychiatry, 37, 18-24.

Kolb, B. & Whishaw, I.Q. (1996). Fundamentals of human neuropsychology. New York: W.H. Freeman and Company.

Larsen, R.J., & Diener, E. (1987). Affect intensity as an individual difference characteristic: A review. Journal of Research in Personality, 21, 1-39.

Larsen, R.J., & Diener, E. (1992). Promises and problems with the circumplex model of emotion. In M.S. Clark (Ed.), Review of personality and social psychology: Emotion (Vol. 13) (pp. 25-59). New York: SAGE Publications.

Lavy, E.H., & van den Hout, M.A. (1994). Cognitive avoidance and attentional bias: Causal relationships. Cognitive Therapy and Research, 2, 179-191.

Levenson, R.W. (1992). Autonomic nervous system differences among emotions. Psychological Science, 3, 23-27.

Light, K.C., Kothapandi, R.V., & Allen, M.T. (1998). Enhanced cardiovascular and catecholamine responses in women with depressive symptoms. International Journal of Psychophysiology, 28, 157-166.

Lydiard, R.B., & Brawman-Mintzer, O. (1998). Anxious depression. Journal of Clinical Psychiatry, 59, 10-17.

MacLeod, C., Mathews, A., & Tata, P. (1986). Attentional bias in emotional disorders. Journal of Abnormal Psychology, 95, 15-20.

Manuck, S.B., Kasprowicz, A.L., & Muldoon, M.F. (1990). Behaviorally-evoked cardiovascular reactivity and hypertension: Conceptual issues and potential associations. Annals of Behavioral Medicine, 12, 17-29.

Mathews, A. (1990). Why worry? The cognitive function of anxiety. Behavior Research and Therapy, 28, 455-468.

Mathews, A., & MacLeod, C. (1986). Discrimination of threat cures without awareness in anxiety states. Journal of Abnormal Psychology, 95, 131-138.

Mathews, A., & MacLeod, C. (1994). Cognitive approaches to emotion and emotional disorders. Annual Review of Psychology, 45, 25-50.

McGinnies, E. (1949). Emotionality and perceptual defense. Psychological Review, 56, 244-251.

Miller, G.A. (1996). How we think about cognition, emotion, and biology in psychopathology. Psychophysiology, 33, 615-628.

Mogg, K., Bradley, B.P., & Williams, R. (1995). Attentional bias in anxiety and depression: The role of awareness. British Journal of Clinical Psychology, 34, 17-36.

Mogg, K., Bradley, B.P., Williams, R., & Mathews, A. (1993). Subliminal processing of emotional information in anxiety and depression. Journal of Abnormal Psychology, 102, 304-311.

Mogg, K., Mathews, A., & Weinman, J. (1987). Memory bias in clinical anxiety. Journal of Abnormal Psychology, 96, 94-98.

Nyklicek, I., Thayer, J.F., & Van Doornen, L.J.P. (1997). Cardiorespiratory differentiation of musically-induced emotions. Journal of Psychophysiology, 11, 304-321.

Otto, M.W., McNally, R.J., Pollack, M.H., Chen, E., & Rosenbaum, J.F. (1994). Hemispheric laterality and memory bias for threat in anxiety disorders. Journal of Abnormal Psychology, 103, 828-831.

Porges, S.W. (1992). Autonomic regulation and attention. In B.A. Campbell & H. Hayne (Eds.), Attention and information processing in infants and adults: Perspectives from human and animal research (pp. 201-223). Hillsdale, NJ: Lawrence Earlbaum Associates.

Porges, S.W., & Raskin, D.C. (1969). Respiratory and heart rate components of attention. Journal of Experimental Psychology, 81, 497-503.

Postman, L. (Ed.) (1962). Psychology in the making. New York: Alfred A. Knopf.

Pratto, F. & John, O.P. (1991). Automatic vigilance: The attention-grabbing power of negative social information. Journal of Personality and Social Psychology, 61, 380-391.

Rechlin, T., Claus, D., 7 Weis, M. (1994). Heart rate analysis in 24 patients treated with 150 mg amitriptyline per day. Psychopharmacology, 110, 110-114.

Rechlin, T., Weis, M., Spitzer, A., & Kaschka, W.P. (1994). Are affective disorders associated with alterations of heart rate variability? Journal of Affective Disorders, 32, 271-275.

Richards, J.E., & Casey, B.J. (1991). Heart rate variability during attention phases in young infants. Psychophysiology, 28, 43-53.

Richards, J.E., & Casey, B.J. (1992). Development of sustained visual attention in the human infant. In B.A. Campbell, H. Hayne, & R. Richardson (Eds.), Attention and information processing in infants and adults (pp. 30-60). Hillsdale, NJ: Lawrence Earlbaum Associates.

Russell, J.A. (1980). A circumplex model of affect. Journal of Personality and Social Psychology, 39, 1161-1178.

Russell, J.A., (1997). How shall an emotion be called? In R. Plutchik & H.R. Conte (Eds.), Circumplex models of personality and emotion (pp. 205-220). Washington, DC: American Psychological Association.

Sartorius, N., Ustun, T.B., Lecrubier, Y., & Wittchen, H. (1996). Depression comorbid with anxiety: Results from the WHO study on psychological disorders in primary health care. British Journal of Psychiatry, 168 (suppl. 30), 38-43.

Schacter, D.L. (1995). Implicit memory: A new frontier for cognitive neuroscience. In M.S. Gazzaniga (Ed.), The cognitive neurosciences (pp. 815-824). Cambridge, MA: MIT Press.

Sherwood, A., Allen, M.T., Fahrenberg, J., Kelsey, R.M., Lovallo, W.R., & van Doornen, L.J.P. (1990). Methodological guidelines for impedance cardiography. Psychophysiology, 27,1-23.

Spielberger, C.D., Gorsuch, R.L., Lushene, R., Vagg, P.R., & Jacobs, G.A. (1983). Manual for the State-Trait Anxiety Inventory. Palo Alto, CA: Consulting Psychologists Press.

SPSS [computer software]. (2000). Chicago, IL: SPSS Inc.

Stemmler, G. (1989). The autonomic differentiation of emotions revisited: Convergent and discriminant validation. Psychophysiology, 26, 617-632.

Stroop, J.R. (1935). Studies in interference in serial verbal reactions. Psychological Monographs, 50, 38-48.

Stein, P.K. & Kleiger, R.E. (1999). Insights from the study of heart rate variability. Annual Review of Medicine, 50, 249-261.

Tanaka-Matsumi, J., & Kemeoka, V.A. (1986). Reliabilities and concurrent validities of popular self-report measures of depression, anxiety, and social desirability. Journal of Consulting and Clinical Psychology, 54, 328-333.

Tellegen, A., Watson, D., & Clark, L.A. (1999). On the dimensional and hierarchical structure of affect. Psychological Science, 10, 297-303.

Thayer, J.F., Friedman, B.H., & Borkovec, T.D. (1996). Autonomic characteristics of generalized anxiety disorder and worry. Biological Psychiatry, 39, 255-266.

Thayer, J.F., Friedman, B.H., Borkovec, T.D., Johnsen, B.H., & Molina, S. (2000). Phasic heart period reactions to cued threat and non-threat stimuli in generalized anxiety disorder. Psychophysiology, 37, 361-368.

Thayer, J.F., & Lane, R.D. (in press). A model of neurovisceral integration in emotion regulation and dysregulation. Journal of Affective Disorders.

Thayer, J.F., & Miller, M.L. (1988). Further evidence for the independence of hedonic level and emotional intensity. Personality and Individual Differences, 9, 425-426.

Thayer, J.F., Smith, M., Rossy, L.A., Sollers, J.J., & Friedman, B.H. (1998). Heart period variability and depressive symptoms: Gender differences. Biological Psychiatry, 44, 304-306.

Tulen, J.H.M., Bruihn, J.A., de Man, K.J., van der Velden, E., Pepplinkhuizen, L., Man in't Veld, A.J. (1996). Anxiety and autonomic regulation in major depressive disorder: An exploratory study. Journal of Affective Disorders, 40, 61-71.

Watson, D., & Tellegen, A. (1985). Toward a consensual structure of mood. Psychological Bulletin, 98, 219-235.

Williams, J.M.G., Watts, F.N., MacLeod, C., & Mathews, A. (1997). Cognitive psychology and emotional disorders (2nd ed.). New York: John Wiley and Sons.

Wittchen, H. (1996a). What is comorbidity – Fact or artefact? British Journal of Psychiatry, 168 (suppl. 30), 7-8.

Wittchen, H. (1996b). Critical issues in the evaluation of comorbidity of psychiatric disorders. British Journal of Psychiatry, 168 (suppl. 30), 9-16.

Wolf, S. (1995). Dogmas that have hindered understanding. Integrative Physiological and Behavioral Science, 30, 3-4.

Yeragani, V.K., Balon, R., Pohl, R., & Ramesh, C. (1995). Depression and heart rate variability. Biological Psychiatry, 38, 768-770.

Yeragani, V.K., Pohl, R., Balon, R., Ramesh, C., Glitz, D., Jung, I., & Sherwood, P. (1992). Heart rate variability in patients with major depression. Psychiatry Research, 46, 35-46.

TABLE 1

Means for Experimental Stage Questionnaires by Group

	Anxious		Depressed		Anxious/ Depressed		Low Anxious/ Depressed	
n	11		7		23		21	
	M	SD	M	SD	M	SD	M	SD
BAI	13.55	3.8	4.85	3.53	18.13	6.19	4.62	2.22
BDI	5.82	2.99	12.86	2.61	18.79	7.07	3.26	2.62
AIM	159.91	29.69	148.86	15.61	155.87	29.13	153.02	20.9
STAI (State)	33.91	5.92	33.43	9.11	47.61	11.68	28.19	4.95
STAI (Trait)	38.54	6.92	42.86	8.03	58.09	9.52	30.74	5.71
MC-SDS	15.36	5.45	16.86	4.18	13.43	4.03	16.57	4.48

TABLE 2

Means and Standard Deviations for Physiological Measures Within and Across Conditions by Group

	Anxious		Depressed		Anxious/ Depressed		Low Anxious/Depressed	
	M	SD	M	SD	M	SD	M	SD
average MSSD	49.82	18.78	50.74	22.62	42.74	24.73	59.97	39.01
MSSD rest	61.47	38.17	55.61	20.82	44.04	24.31	65.01	44.48
MSSD Stroop	50.91	37.36	44.64	26.77	40.02	26.74	45.83	29.13
MSSD recovery	56.79	24.47	51.96	24.12	44.17	27.52	69.05	46.72
MSSD difference*	10.55	19.19	10.97	14.38	4.02	18.2	19.18	21.17
average RSA	94.71	47.57	86.98	49.39	76.12	48.59	115.22	64.21
RSA rest	104.23	55.28	107.38	58.11	83.19	51.12	136.44	86.49
RSA Stroop	68.61	36.27	54.86	35.08	67.11	52.47	68.94	43.88
RSA recovery	111.31	59.82	98.69	65.13	78.06	49.48	140.28	78.9
RSA difference*	38.48	39.34	52.52	40.26	16.09	34.25	67.5	70.14
average HR	69.41	12.21	75.07	6.35	78.78	15.25	75.2	9.64
HR rest	68.03	11.74	72.44	4.96	77.77	16.15	74.12	9.25
HR Stroop	72.44	12.89	79.14	10.72	81.43	14.26	77.99	11.59
HR recovery	67.77	12.39	73.63	5.63	77.19	15.64	73.49	8.66
HR difference*	4.41	3.71	6.71	8.58	3.71	4.01	3.87	4.1
average PEP	120.06	14.75	124.13	8.74	118.44	14.36	120.89	12.79
PEP rest	123.78	16.31	124.05	9.41	118.76	15.16	121.32	12.78
PEP Stroop	116.73	22.06	121.15	7.11	119.57	14.26	122.06	12.78
PEP recovery	119.66	16.91	123.21	10.59	117.01	14.31	119.28	14.17
PEP difference*	7.05	27.31	-1.09	5.05	-0.81	3.89	-0.75	4.04

*all change scores were calculated by rest-stressor

TABLE 3

Table 3
Means and Standard Deviatons in ms for Groups Responses on Emotional Stroop Task

	Anxious		Depressed		Anxious/ Depressed		Low Anxious/Depressed	
	M	SD	M	SD	M	SD	M	SD
negative/low arousal	977.82	133.05	782.29	110.99	937.48	149.67	851.19	143.87
negative/high arousal	1020.18	181.05	815.14	141.35	1001.56	279.58	875.19	195.47
positive/low arousal	978.86	161.29	840.63	127.9	962.34	159.26	879.82	146.4
positive/high arousal	1029.18	99.37	815.43	81.53	967.39	160.29	864.24	139.7
neutral	1025.27	195.29	850.29	122.61	1069.23	397.78	894.53	162.46
average response for all words	1006.26	154.01	807.43	108.97	994.67	157.99	872.99	140.79
number of words recalled	4	2.86	4.57	1.39	5.04	2.79	3.14	1.74

TABLE 4

Means for Individual Items on Emotion Adjective Questionnaire by Group*

	Group							
	Anxious		Depressed		Anxious/ Depressed		Low Anxious/ Depressed	
	M	SD	M	SD	M	SD	M	SD
Peaceful	3.75	0.75	3.86	0.38	2.74	0.86	3.57	0.81
Interest	4	0.97	3.86	0.69	4.04	0.76	4.09	0.96
Relaxation	3.69	0.79	3.57	0.53	3.04	0.88	3.43	1.12
Excitement	3.38	0.86	2.29	0.95	2.3	1.11	2.67	0.8
Happiness	3.56	0.96	3.71	0.76	2.56	1.04	3.95	0.74
Uneasiness	2.31	1.14	1.71	0.76	2.87	1.18	1.62	0.92
Anger	1.06	0.25	1	0	1.09	0.42	1	0
Sadness	1.06	0.25	1.29	0.49	2.13	1.06	1	0
Tiredness	2.75	1.29	2.86	0.9	3.56	0.84	2.64	0.82
Enjoyment	3	0.63	3	1	2.31	1.06	3.14	0.73
Pleasantness	3.44	0.73	3.14	1.07	2.91	1.16	3.67	0.91
Arousal	2.5	1.41	1.71	0.95	2.17	1.07	2.05	1.07

* measured on a Likert-type scale from 1 through 5

(1= experiencing very little of the emotion, 5=experiencing a lot of the emotion)

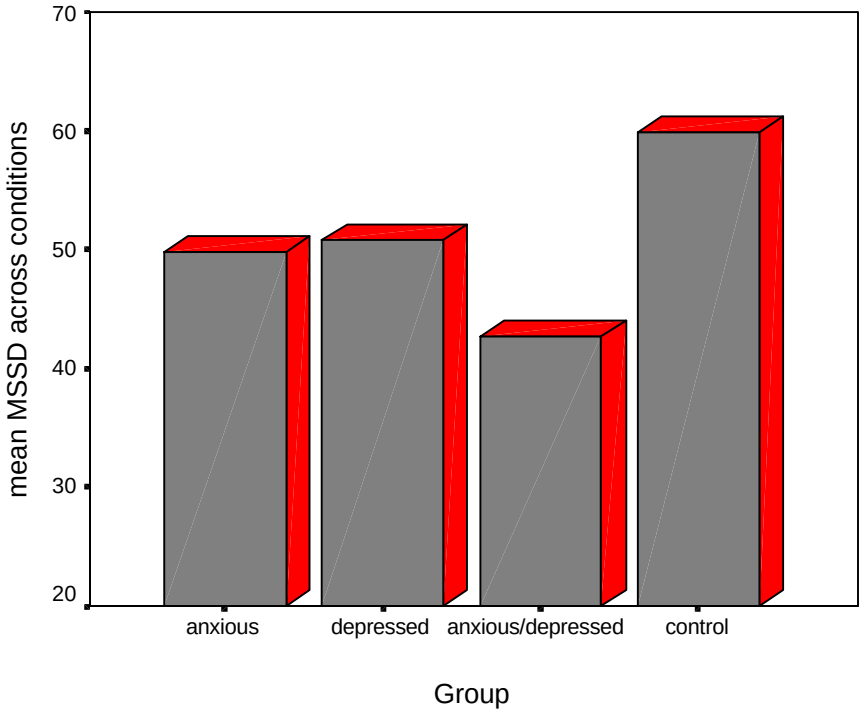
FIGURE CAPTIONS

Figure 1. Mean MSSD and mean RSA averaged across conditions (rest, stressor, and recovery) by group (anxious, depressed, comorbid anxious/depressed, and nonanxious/nondepressed controls).

Figure 2. Mean MSSD and RSA change scores, calculated by subtracting rest from stressor, by group (anxious, depressed, comorbid anxious/depressed, and nonanxious/nondepressed controls).

Figure 3. Mean HR and PEP change scores, calculated by subtracting rest from stressor, by group (anxious, depressed, comorbid anxious/depressed, and nonanxious/nondepressed controls).

FIGURE 1


70
60
50
40
30
20
mean MSSD across conditions
anxious
depressed
anxious/depressed
control
Group

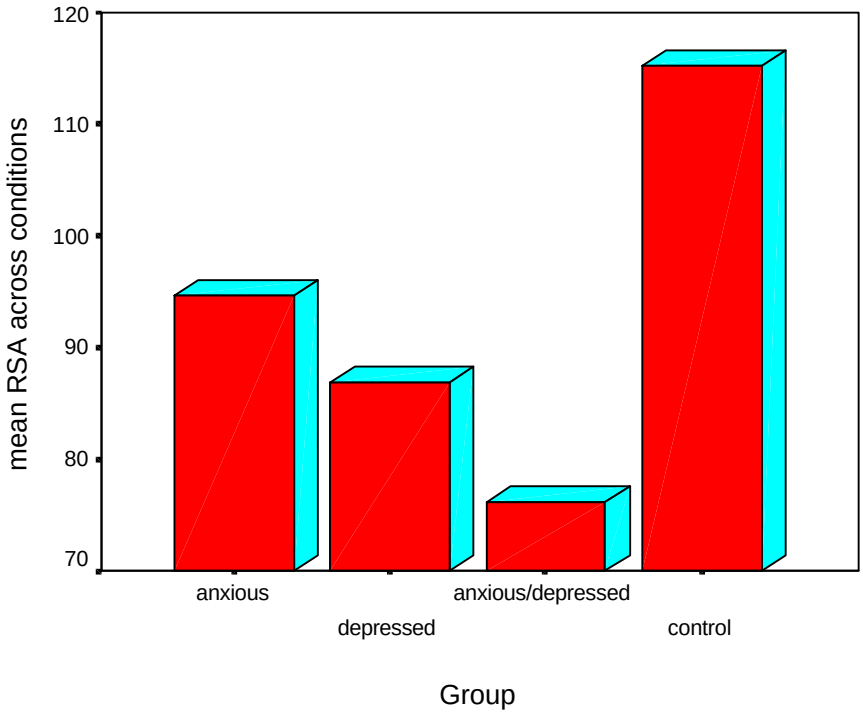
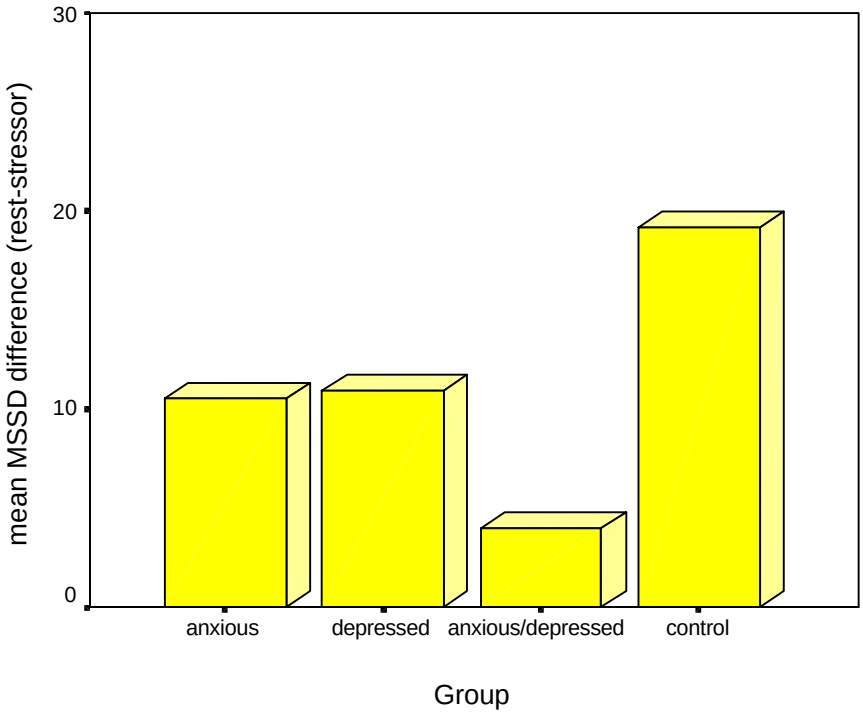

120
110
100
90
80
70
mean RSA across conditions
anxious
depressed
anxious/depressed
control
Group

FIGURE 2


30
20
10
0
mean MSSD difference (rest-stressor)
anxious
depressed
anxious/depressed
control
Group

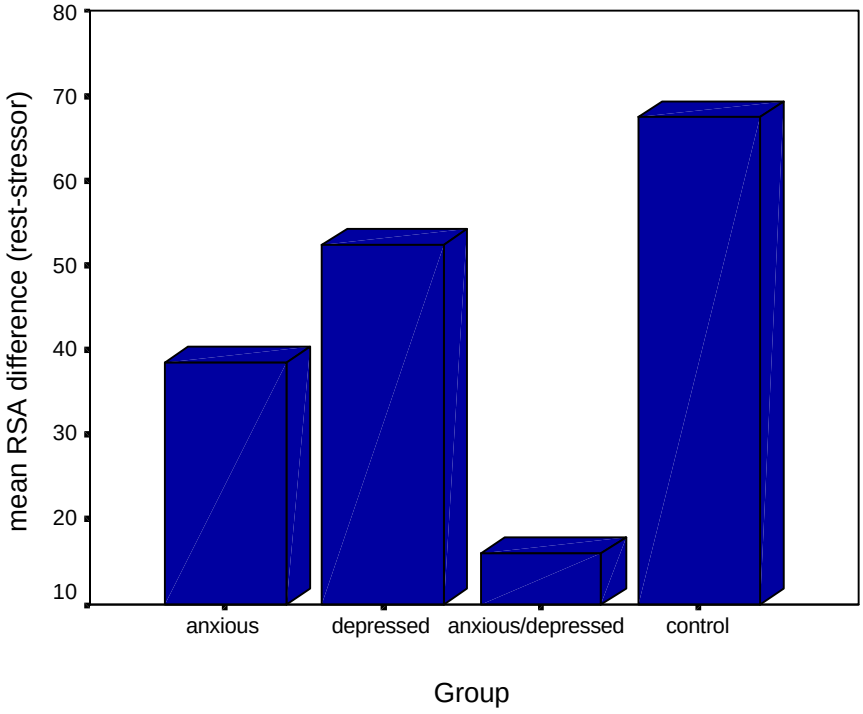
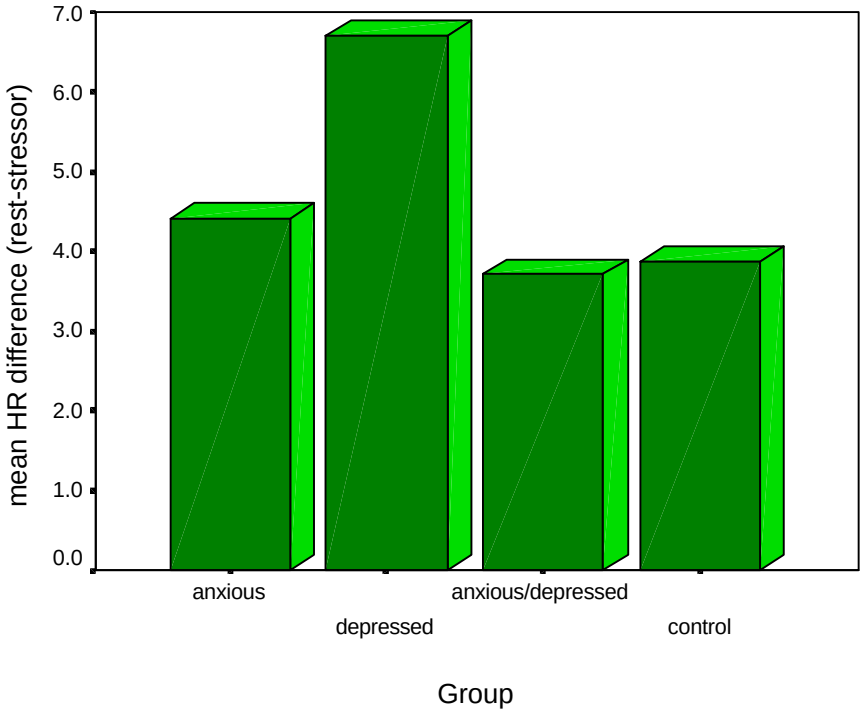
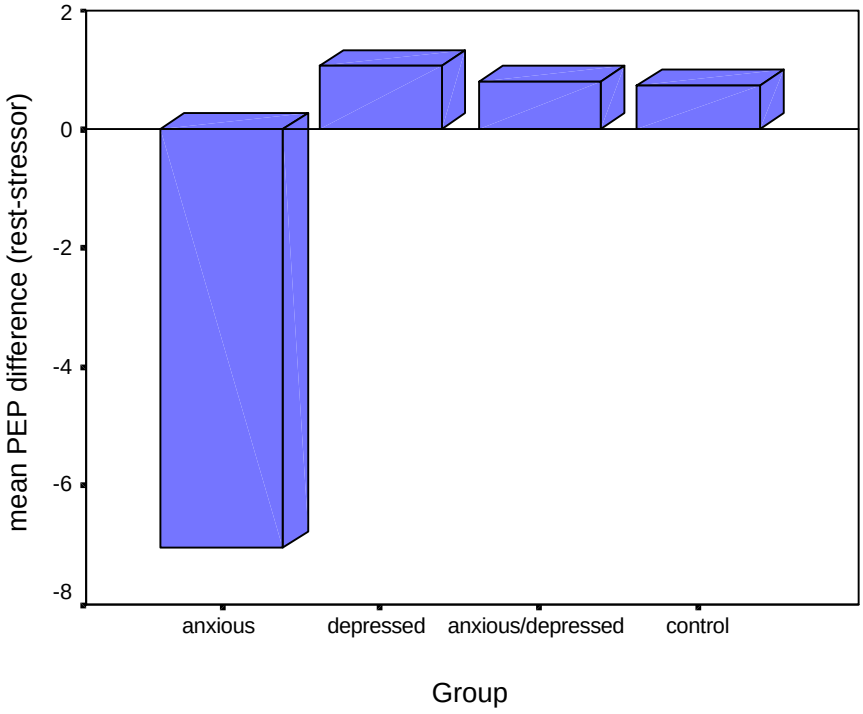

80
70
60
50
40
30
20
10
mean RSA difference (rest-stressor)
anxious
depressed
anxious/depressed
control
Group

FIGURE 3


7.0
6.0
5.0
4.0
3.0
2.0
1.0
0.0
mean HR difference (rest-stressor)
anxious
depressed
anxious/depressed
control
Group


2
0
-2
-4
-6
-8
mean PEP difference (rest-stressor)
anxious
depressed
anxious/depressed
control
Group

APPENDIX A

BAI

Below is a list of common symptoms of anxiety. Please read each item in the list carefully. Indicate how much you have been bothered by each symptom during the PAST WEEK, INCLUDING TODAY by indicating A (not at all), B (mildly), C (moderately), or D (severely) on your ***purple scantron sheet*** for each of the 21 items.

	Not at All	**Mildly It did not bother me much**	**Moderately It was very unpleasant but I could stand it**	**Severely I could barely stand it**
Symptom	A	B	C	D
1. numbness or tingling				
2. feeling hot				
3. wobbliness in legs				
4. unable to relax				
5. fear of the worst happening				
6. dizzy or lightheaded				
7. heart pounding or racing				
8. unsteady				
9. terrified				
10. nervous				
11. feelings of choking				
12. hands trembling				
13. shaky				
14. fear of losing control				
15. difficulty breathing				
16. fear of dying				
17. scared				
18. indigestion or discomfort in abdomen				
19. faint				
20. face flushed				
21. sweating (not due to heat)				

APPENDIX B

BDI

On this questionnaire are groups of statements. Please read each group of statements carefully. Then pick out the one statement in each group which best describes the way you have been feeling the PAST WEEK, INCLUDING TODAY! Circle the number beside the statement you picked. If several statements in the group seem to apply equally well, circle each one. Be sure to read all the statements in each group before making your choice.

1. A. 0 I do not feel sad.
   B. 1 I feel sad.
   C. 2 I am sad all the time and I can't snap out of it.
   D. 3 I am so sad or unhappy that I can't stand it

2. A. 0 I am not particularly discouraged about the future
   B. 1 I feel discouraged about the future.
   C. 2 I feel I have nothing to look forward to.
   D. 3 I feel that the future is hopeless and that things cannot improve

3. A. 0 I do not feel like a failure.
   B. 1 I feel I have failed more than the average person.
   C. 2 As I look back on my life, all I can see is a lot of failures.
   D. 3 I feel I am a complete failure as a person.

4. A. 0 I don't get as much satisfaction out of things as I used to.
   B. 1 I don't enjoy things the way I used to.
   C. 2 I don't get real satisfactions out of anything anymore.
   D. 3 I am dissatisfied or bored with everything.

5. A. 0 I don't feel particularly guilty.
   B. 1 I feel guilty a good part of the time.
   C. 2 I feel quite guilty most of the time.
   D. 3 I feel guilty all of the time.

6. A. 0 I don't feel I am being punished.
   B. 1 I feel I may be punished.
   C. 2. I expect to be punished.
   D. 3. I feel I am being punished.

7. A. 0 I don't feel disappointed in myself.
   B. 1 I am disappointed in myself.
   C. 2 I am disgusted with myself.
   D. 3 I hate myself

8. A. 0 I don't feel I am any worse off than anyone else
   B. 1 I am critical of myself for my weakness or mistakes.
   C. 2 I blame myself all the time for my faults.
   D. 3 I blame myself for everything bad that happens.

9. A. 0 I don't have any thoughts of killing myself.
   B. 1 I have thoughts of killing myself, but I would not carry them out.
   C. 2 I would like to kill myself.
   D. 3 I would kill myself if I had the chance.

10. A. 0 I don't cry any more than usual.
    B. 1 I cry more now than I used to.
    C. 2 I cry all the time now.
    D. 3 I used to be able to cry, but now I can't cry even though I want to.

11. A. 0 I am no more irritated now than I ever am.
    B. 1 I get annoyed or irritated more easily than I used to.
    C. 2 I feel irritated all the time now.
    D. 3 I don't get irritated at all by the things that used to irritate me.

12. A. 0 I have not lost interest in other people.
    B. 1 I am less interested in other people than I used to be.
    C. 2 I have lost most of my interest in other people.
    D. 3 I have lost all of my interest in other people.

13. A. 0 I make decisions about as well as I ever could.
    B. 1 I put off making decisions more than I used to.
    C. 2 I have greater difficulty in making decisions than before.
    D. 3 I can't make decisions at all anymore.

14. A. 0 I don't feel I look any worse than I used to.
    B. 1 I am worried that I am looking old or unattractive.
    C. 2 I feel that there are permanent changes in my appearance that make me look unattractive.
    D. 3 I believe that I look ugly.

15. A. 0 I can work about as well as before.
    B. 1 It takes an extra effort to get started at doing something.
    C. 2 I have to push myself very hard to do anything.
    D. 3 I can't do any work at all.

16. A. 0 I can sleep as well as usual.
    B. 1 I don't sleep as well as I used to.
    C. 2 I wake up 1-2 hours earlier than usual and find it hard to get back to sleep
    D. 3 I wake up several hours earlier than I used to and cannot get back to sleep

17. A. 0 I don't get more tired than usual.
    B. 1 I get tired more easily than I used to.
    C. 2 I get tired from doing almost anything.
    D. 3 I am too tired to do anything.

18. A. 0 My appetite is no worse than usual.
    B. 1 My appetite is not as good as it used to be.
    C. 2 My appetite is much worse no
    D. 3 I have no appetite at all anymore

19. A. 0 I haven't lost much weight, if any, lately.
    B. 1 I have lost more than 5 pounds.

C. 2 I have lost more than 10 pounds
D. 3 I have lost more than 15 pounds.

20. A. 0 I am no more worried about my health than usual.
B. 1 I am worried about physical problems such as aches and pains: or upset stomach; or constipation.
C. 2 I am very worried about physical problems and it's hard to think of much else.
D. 3 I am so worried about my physical problems that I cannot think about anything else.

21. A. 0 I have not noticed any recent change in my interest in sex.
B. 1 I am less interested in sex than I used to be.
C. 2 I am much less interested in sex now.
D. 3 I have lost interest in sex completely.

APPENDIX C

Handedness Questionnaire

Fill in the appropriate letter for each item on the purple scan form.

	Right	Both	Left
	A	**B**	**C**
1. With which hand would you throw a ball to hit a target?.....	1	0	-1
2. With which hand do you draw?.....	1	0	-1
3. With which hand do you use an erase on paper?....	1	0	-1
4. With which hand do you remove the top card when dealing?.....	1	0	-1
5. With which foot do you kick a ball?.....	1	0	-1
6. If you wanted to pick up a pebble with your toes, which foot would you use?.....	1	0	-1
7. If you had to step up onto a chair which foot would you place on the chair first?.....	1	0	-1
8. Which eye would you use to sight down a rifle?...	1	0	-1
9. If you wanted to listen to a conversation going on behind a closed door which ear would you place against the door?.....	1	0	-1
10. If you wanted to listen to someone’s heartbeat, which ear would you place against their chest?.....	1	0	-1
11. Into which ear would you place the earphone of a transistor radio?.....	1	0	-1

APPENDIX D

Marlowe-Crowne SDS

Listed below are a number of statements concerning personal attitudes and traits. Read each item and decide whether the statement is TRUE or FALSE about you personally. Circle the "T" if an item is TRUE for you. Circle the "F" if the item is FALSE or not true for you. Please answer all the questions.

		TRUE	FALSE
1.	Before voting I thoroughly investigate the qualifications of all the candidates.	T	F
2.	I never hesitate to go out of my way to help someone in trouble.	T	F
3.	It is sometimes hard for me to go on with my work if I am not encouraged.	T	F
4.	I have never intensely dislike anyone.	T	F
5.	On occasion I have had doubts about my ability to succeed in life.	T	F
6.	I sometimes feel resentful when I don't get my way.	T	F
7.	I am always careful about my manner of dress.	T	F
8.	My table manners at home are as good when I eat out in a restaurant.	T	F
9.	If I could get into a movie without paying and be sure I was not seen I would probably do it.	T	F
10.	On a few occasions, I have given up doing something because I thought too little of my ability.	T	F
11.	I like to gossip at times.	T	F
12.	There have been times when I felt like rebelling against people in authority even though I knew they were probably right.	T	F
13.	No matter whom I'm talking to, I'm always a good listener.	T	F
14.	I can remember "playing sick" to get out of something.	T	F
15.	There have been occasions when I took advantage of someone.	T	F

16.	I'm always willing to admit it when I make a mistake.	T	F
17.	I always try to practice what I preach.	T	F
18.	I don't find it particularly difficult to get along with loud mouthed, obnoxious people.	T	F
19.	I sometimes try to get even rather than to forgive and forget.	T	F
20.	When I don't know something I don't at all mind admitting it.	T	F
21.	I am always courteous, even to people who are disagreeable.	T	F
22.	At times I have really insisted on having things my own way.	T	F
23.	There have been occasions when I felt like smashing things.	T	F
24.	I would never think of letting someone else be punished for my wrongdoing.	T	F
25.	I never resent being asked to return a favor.	T	F
26.	I have never been irked when people expressed ideas very different from my own.	T	F
27.	I never make a long trip without checking the safety of my car.	T	F
28.	There have been times when I was quite jealous of the good fortune of others.	T	F
29.	I have almost never felt the urge to tell someone off.	T	F
30.	I am sometimes irritated by people who ask favors of me.	T	F
31.	I have never felt that I was punished without cause.	T	F
32.	I sometimes think when people have a misfortune they only got what they deserved.	T	F
33.	I have never deliberately said something that hurt someone's feelings.	T	F

APPENDIX E

SAI: Form Y-1

Directions: A number of statements which people have used to describe themselves are given below. Read each statement and then blacken in the appropriate circle to the right of the statement to indicate how you feel right now, that is, at this moment. There are no right or wrong answers. Do not spend too much time on any one statement but give the answer which seems to describe your present feelings best.

		NOT AT ALL	SOMEWHAT	MODERATELY SO	VERY MUCH SO
1.	I feel calm	①	②	③	④
2.	I feel secure	①	②	③	④
3.	I am tense	①	②	③	④
4.	I feel strained	①	②	③	④
5.	I feel at ease	①	②	③	④
6.	I feel upset	①	②	③	④
7.	I am presently worrying over possible misfortunes	①	②	③	④
8.	I feel satisfied	①	②	③	④
9.	I feel frightened	①	②	③	④
10.	I feel comfortable	①	②	③	④
11.	I feel self-confident	①	②	③	④
12.	I feel nervous	①	②	③	④
13.	I am jittery	①	②	③	④
14.	I feel indecisive	①	②	③	④
15.	I am relaxed	①	②	③	④
16.	I feel content	①	②	③	④
17.	I am worried	①	②	③	④
18.	I feel confused	①	②	③	④
19.	I feel steady	①	②	③	④
20.	I feel pleasant	①	②	③	④

Form Y-2

Directions: A number of statements which people have used to describe themselves are given below. Read each statement and then blacken in the appropriate circle to the right of the statement indicate how you generally feel. There are no right or wrong answers. Do not spend too much time on any one-statement buy give the answer which seems to describe how you generally feel.

		NOT AT ALL	SOMEWHAT	MODERATELY SO	VERY MUCH SO
21.	I feel pleasant	①	②	③	④
22.	I feel nervous and restless	①	②	③	④
23.	I feel satisfied with myself	①	②	③	④
24.	I wish I could be as happy as others seem to be	①	②	③	④
25.	I feel like a failure	①	②	③	④
26.	I feel rested	①	②	③	④
27.	I feel “calm, cool, and collected”	①	②	③	④
28.	I feel that difficulties are piling up so that I can not overcome them…….. .	①	②	③	④
29.	I worry too much over something that really doesn’t matter	①	②	③	④
30.	I am happy	①	②	③	④
31.	I have disturbing thoughts	①	②	③	④
32.	I lack self-confidence	①	②	③	④
33.	I feel secure	①	②	③	④
34.	I feel make decisions easily	①	②	③	④
35.	I feel inadequate	①	②	③	④
36.	I am content	①	②	③	④
37.	Some unimportant thought runs through my mind and bothers me……. .	①	②	③	④
38.	I take disappointments so keenly that I can’t put them out of my mind…….. .	①	②	③	④
39.	I am a steady person	①	②	③	④

40. I get in a state of tension or turmoil as I think over my resent concerns and interests ……. ① ② ③ ④

APPENDIX F

Emotion Report Form

Indicate, by circling on the answer sheet, how much of each emotion you feel right now If you do not feel any of a particular emotion, circle 1. If you feel a lot, circle 5, or an intermediate amount, circle 3, etc.

1. Peacefulness	1	2	3	4	5
2. Interest	1	2	3	4	5
3. Relaxation	1	2	3	4	5
4. Excitement	1	2	3	4	5
5. Happiness	1	2	3	4	5
6. Uneasiness	1	2	3	4	5
7. Anger	1	2	3	4	5
8. Sadness	1	2	3	4	5
9. Tiredness	1	2	3	4	5
10. Enjoyment	1	2	3	4	5
11. Pleasantness	1	2	3	4	5
12. Arousal	1	2	3	4	5

APPENDIX G

AIM

Directions: The following questions refer to the emotional reactions to typical life events. Please indicate how YOU react to these events by placing a number from the following scale in the blank space preceding each item. Please base your answers on how YOU react, *not* on how you think others react or how you think a person should react.

NEVER	ALMOST NEVER	OCCASIONALLY	USUALLY	ALMOST ALWAYS	ALWAYS
1	2	3	4	5	6

1. _____ When I accomplish something difficult I feel delighted or elated.
2. _____ When I feel happy it is a strong sense of exuberance.
3. _____ I enjoy being with other people very much.
4. _____ I feel pretty bad when I tell a lie.
5. _____ When I solve a small personal problem, I feel euphoric.
6. _____ My emotions tend to be more intense than those of most people.
7. _____ My happy moods are so strong that I feel like I'm "in heaven".
8. _____ I get overly enthusiastic.
9. _____ If I complete a task I thought was impossible, I get ecstatic.
10. _____ My heart races at the anticipation of some exciting event.
11. _____ Sad movies deeply touch me.
12. _____ When I'm being happy it's a feeling of being untroubled and content rather than being zestful and aroused.
13. _____ When I talk in front of a group for the first time my voice gets shaky and my heart races.
14. _____ When something good happens, I am usually much more jubilant than others.
15. _____ My friends say I'm emotional.
16. _____ The memories I like the most are those of times when I felt content and peaceful rather than zestful and enthusiastic.
17. _____ The sight of someone who is hurt badly affects me strongly.
18. _____ When I'm feeling well it's easy for me to go from being in a good mood to being really joyful.
19. _____ "Calm and cool" could easily describe me.
20. _____ When I'm happy I feel like I'm bursting with joy.
21. _____ Seeing a picture of some violent care accident in a newspaper makes me feel sick to my stomach.
22. _____ When I'm happy I feel very energetic.
23. _____ When I receive an award I become overjoyed.
24. _____ When I succeed at something, my reaction is calm contentment.

25. _____ When I do something wrong I have strong feelings of shame and guilt.
26. _____ I can remain calm even on the most trying days.
27. _____ When things are going good I feel “on top of the world.”
28. _____ When I get angry it’s easy for me to still be rational and not overreact.
29. _____ When I know I have done something very well, I feel relaxed and content rather than excited and elated.
30. _____ When I do feel anxiety it is normally very strong.
31. _____ My negative moods are mild in intensity.
32. _____ When I am excited over something I want to share my feelings with everyone.
33. _____ When I feel happiness, it is a quiet type of contentment.
34. _____ My friends would probably say I’m a tense or “high-strung” person.
35. _____ When I’m happy I bubble over with energy.
36. _____ When I feel guilty, this emotion is quite strong.
37. _____ I would characterize my happy moods as closer to contentment than to joy.
38. _____ When someone compliments me, I get so happy I could “burst.”
39. _____ When I am nervous I get shaky all over.
40. _____ When I am happy the feeling is more like contentment and inner calm than one of exhilaration and excitement.

APPENDIX H

Stroop task words

Negative/ Low Arousal	Negative/ High Arousal	Positive/ Low Arousal	Positive/ High Arousal	Neutral
lonely	crying	serene	joyful	vague
unhappy	scared	calm	rapturous	colorless
despondent	worried	nonpartisan	blissful	indeterminate
low	alarmed	satisfied	euphoric	impartial
depressed	alone	peaceful	happy	equitable
melancholy	frightened	tranquil	delighted	chair
desolate	distressed	pleasant	ecstatic	doorknob
undecided	miserable	placid	elated	furniture
downcast	disturbed	quiet	giddy	table
sadness	fearful	mild	exhilarated	carpet

APPENDIX I

Electrode Placement Diagram

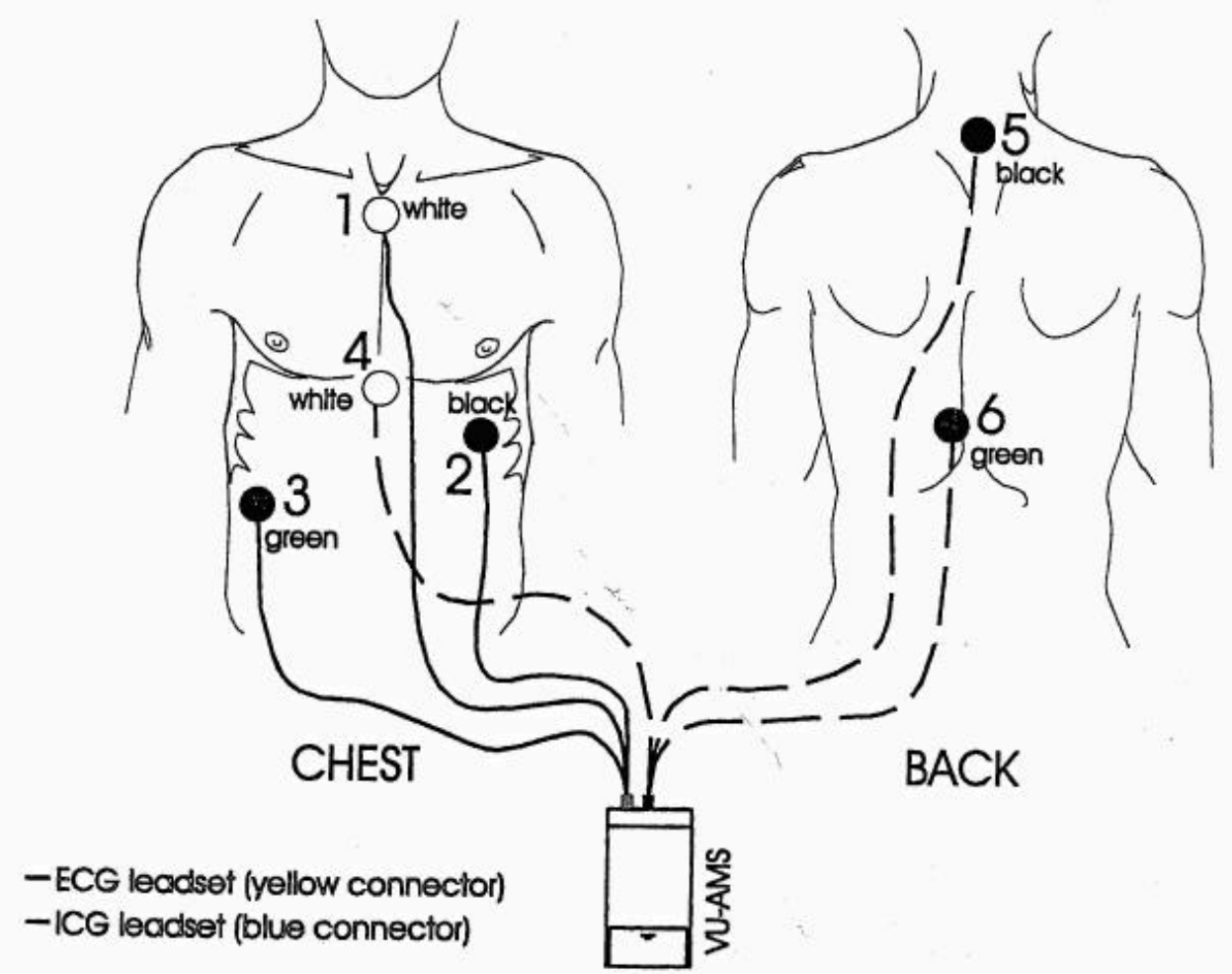


Figure 2. Electrode placement and leadwire attachment

(Vrije Universiteit, Department of Psychophysiology, Amsterdam, the Netherlands)

APPENDIX J

Informed Consent Forms

Virginia Polytechnic Institute and State University

Informed Consent for Participants
Of Investigative Projects

TITLE OF PROJECT: **COGNITIVE BIASES AND AUTONOMIC RESPONDING IN ANXIETY AND DEPRESSION**

Principle Investigators: Aimee K. Santucci, Ben G. Pumphrey, Bruce Friedman, Ph.D.

1. The purpose of this research:

The purpose of this study is to investigate: 1) information processing in anxiety and depression; and 2) autonomic nervous system responding in anxiety and depression via heart rate variability and impedance cardiography.

The first stage, which you are about to complete, is the screening stage. This stage involves approximately 500 subjects, including yourself. The purpose of this stage is to measure levels of anxious and depressed symptomotology. Based on your scores you may be asked to complete further measures that will be administered at a later time. If you are selected for the second stage, you will be asked to complete a computerized task, fill out some questionnaires, and allow for measures to be taken of heart rate, respiration, and thoracic impedance.

2. Procedures

I understand that in this first stage I will be asked to complete the following self-report questionnaires: Beck Depression Inventory, Beck Anxiety Inventory, the Marlowe-Crowne SDS, the State-Trait Anxiety Inventory, and a Handedness Questionnaire. I understand that it will take approximately 45 to 60 minutes to complete these questionnaires in this phase of the study. If selected for the second half of the study I also understand that it will take approximately one hour and 30 minutes to complete.

3. Risks:

The risks of this study are minimal. However, I understand that these questionnaires and measures may evoke uncomfortable feelings and images. If this occurs, services are available on campus through the Psychological Services Center and the on-campus health facility. Safeguards that will be used to minimize my risk or discomfort are that I will be able to contact one of these services, if I so desire.

Should you report that you may harm yourself or others (on the Beck Depression Inventory), the researcher has the obligation to break confidentiality and report this information to the appropriate agency.

4. Benefits of this Project:

I understand that my participation in this study will help evaluate the information processing aspects and autonomic underpinnings of anxiety and depression.

I understand that I am able to receive a synopsis of the results when completed. If I choose to receive a summary of the findings, I understand that I must provide a self-addressed stamped envelope to the investigators.

5. Extent of Anonymity and Confidentiality:

I understand that the results of this study will be kept strictly confidential. The information I provide will have my name removed and only a subject number will identify me during the analyses and any written reports of the research.

6. Compensation:

I understand that I will receive one extra credit point for the first stage and two extra credit points if I am selected and complete the second stage. Extra credit will be assigned to the psychology class in which I am enrolled and will accept extra credit.

7. Freedom to Withdraw:

I understand that I am free to withdraw from this study at any time. If I choose to withdraw, I will not be penalized by reduction in points or grade from any psychology course.

8. Approval of Research:

This research has been approved, as required, by the Institutional Review Board for projects involving human subjects at Virginia Polytechnic Institute and State University, and by the Human Subjects Committee in the Department of Psychology.

9. Participant's Permission:

I have read and understood the Informed Consent and conditions of this project. I have had all my questions answered. I hereby acknowledge the above and give my voluntary consent for participation in this project. I agree to abide by the rules for this project.

Aimee K. Santucci,231-4428, asantucci@vt.edu
Ben G. Pumphrey
Bruce H. Friedman, Ph.D., 231-9611
David W. Harrison, Chair, Human Subjects Committee, 231-4422
David Moore, Chair, Institutional Review Board, Research Division, 231-4991

Participant Signature: _____ Date: _____

Witness: _____ Date: _____

Virginia Polytechnic Institute and State University

Informed Consent for Participants
Of Investigative Projects

TITLE OF PROJECT: **COGNITIVE BIASES AND AUTONOMIC RESPONDING IN ANXIETY AND DEPRESSION**

Principle Investigators: Aimee K. Santucci, Ben G. Pumphrey, Bruce Friedman, Ph.D.

1. The purpose of this research:

The purpose of this study is to investigate: 1) information processing in anxiety and depression; and 2) autonomic nervous system responding in anxiety and depression via heart rate variability and impedance cardiography.

In the second stage of this study, there are approximately 60 subjects, including yourself.

2. Procedures

This study will take approximately one hour to complete. I understand that I will have six electrodes placed on my chest and back via adhesive and then be hooked up to an ambulatory monitoring system. For this part of the study, all participants and experimenters will be gender-matched. I will be asked to complete a computerized task designed to measure my emotional state, and will allow continuous measures of my heart rate, respiration, and thoracic impedance to be taken. I will then be asked to complete the Beck Depression Inventory, the Beck Anxiety Inventory, the Marlowe-Crowne SDS, the Affect Intensity Measure, and the State-Trait Anxiety Inventory.

3. Risks:

The risks of this study are minimal. However, I understand that these questionnaires and measures may evoke uncomfortable feelings and images. If this occurs, services are available on campus through the Psychological Services Center and the on-campus health facility. Safeguards that will be used to minimize my risk or discomfort are that I will be able to contact one of these services, if I so desire.

Should you report that you may harm yourself or others (on the Beck Depression Inventory), the researcher has the obligation to break confidentiality and report this information to the appropriate agency.

4. Benefits of this Project:

I understand that my participation in this study will help evaluate the information processing aspects and autonomic underpinnings of anxiety and depression.

I understand that I am able to receive a synopsis of the results when completed. If I choose to receive a summary of the findings, I understand that I must provide a self-addressed stamped envelope to the investigators.

5. Extent of Anonymity and Confidentiality:

I understand that the results of this study will be kept strictly confidential. The information I provide will have my name removed and only a subject number will identify me during the analyses and any written reports of the research.

6. Compensation:

I understand that I will receive two extra credit points if I complete this second stage. Extra credit will be assigned to the psychology class in which I am enrolled.

7. Freedom to Withdraw:

I understand that I am free to withdraw from this study at any time. If I choose to withdraw, I will not be penalized by reduction in points or grade from any psychology course.

8. Approval of Research:

This research has been approved, as required, by the Institutional Review Board for projects involving human subjects at Virginia Polytechnic Institute and State University, and by the Human Subjects Committee in the Department of Psychology.

9. Participant's Permission:

I have read and understood the Informed Consent and conditions of this project. I have had all my questions answered. I hereby acknowledge the above and give my voluntary consent for participation in this project. I agree to abide by the rules for this project.

Aimee K. Santucci, 961-3659
Ben G. Pumphrey
Bruce H. Friedman, Ph.D., 231-9611
David W. Harrison, Chair, Human Subjects Committee, 231-4422
David Moore, Chair, Institutional Review Board, Research Division, 231-4991

Participant Signature: _____ Date: _____

Witness: _____ Date: _____

APPENDIX K

Demographic Form

Participant # _____

Information: PLEASE DO NOT MARK ANY OF THIS INFORMATION ON THE SCAN FORMS

Age: _____

Weight: _____

Height: _____

Year in School: _____

Major Area of Study: _____

Are you currently taking any medication? If so, please describe.
_____

_____

How much caffeine do you consume in an "average" day? When was the last time you had caffeine?

_____

_____

Do you smoke? Yes _____ No _____
If yes, how many cigarettes do you smoke daily? _____
When was the last time you smoked? _____

Average weekly alcohol consumption (approximate number of alcoholic beverages consumed weekly):
_____

Are you actively trying to lose weight? Yes _____ No _____
If you answered "yes", please describe how (Examples: diet, exercise, prescribed or over the counter medications):
_____

_____

Do you have any know medical problems? (Examples: heart conditions, high or low blood pressure, bouts of dizziness or fainting, diabetes, asthma, neurological disorders):
_____

CURRICULUM VITAE

Aimee K. Santucci

Addresses

Office:
5091 Derring Hall
Department of Psychology
Virginia Tech
Blacksburg, VA 24060-1436

Home:
500 Houston St. #16
Blacksburg, VA 24060

Phone
Office: (540) 231-4428
Home: (540) 961-3659
Fax: (540) 231-4428

E-mail:
asantucci@vt.edu

Education

Virginia Polytechnic Institute and State University, M.S. in Psychology, 2001
University of West Florida, M.A. in Psychology, 1999
Bloomsburg University of Pennsylvania, B.A. in Psychology,
B.A. in French, minor in Anthropology, 1995

Academic Employment

8/98 - Present, Graduate Teaching Assistant, Department of Psychology, Virginia Tech, Blacksburg, VA
Lab in Developmental Psychology, Introductory Psychology, Developmental Psychology, Advanced Developmental Psychology
5/99 - 8/99, Research Assistant, Department of Psychology, Mind-Body Lab
8/96-12/97, Graduate Teaching Assistant, Department of Psychology
University of West Florida, Pensacola, FL

Research Interests

Psychophysiology of emotion and mood states
Psychophysiological measures as a means to study conscious and nonconscious emotional processes
Attentional mechanisms and the role of attention in consciousness and awareness
Cognitive and physiological aspects of meditative states
Teaching of attention and awareness skills and stress reduction techniques

Publications

Published Abstracts:

Santucci, A.K. (2001). Autonomic characteristics of anxiety, depression, and co-morbid anxiety-depression. Graduate Student Assembly: 17th Annual Research Symposium of Virginia Tech, 63.

Santucci, A.K. (2001). Autonomic characteristics of anxiety, depression, and co-morbid anxiety-depression. Psychosomatic Medicine, 63, 157.

Christie, I.C., Friedman, B.H., & Santucci, A.K. (2000). Comparative assessment of two heart rate variability measures. Psychophysiology, 37 (Suppl. 1), S32.

Santucci, A.K., Friedman, B.H., & Pumphrey, B.G. (2000). Heart rate variability in anxiety and depression during rest and stroop. Psychophysiology, 37 (Suppl. 1), S86.

Friedman, B., Santucci, A. & Christie, I. (2000). Chain P-technique Factor Analysis of Cardiovascular Activity. Annals of Behavioral Medicine, 22, (Suppl. 1), S136.

Santucci, A., Friedman, B., Curtis, E., & Pumphrey, B. (2000). Cardiovascular and Affective Responses to Relaxing and Arousing Tasks. Annals of Behavioral Medicine, 22, (Suppl. 1), S144.

Friedman, B., Santucci, A., Curtis, E., & Pumphrey, B. (1999). Idiodynamic Profiles of Cardiovascular Activity. Psychophysiology, 36, (Suppl. 1), S52.

Other Publications

Santucci, A.K. (1999). "Precis1, Section 6: Cognition". Exploring Psychology: Reader Workbook. New York: McGraw-Hill Companies, Inc.

Santucci, A.K. (1999). "Precis 2, Section 5: Memory". Exploring Psychology: Reader Workbook. New York: McGraw-Hill Companies, Inc.

Conference Presentations

Santucci, A.K., Friedman, B.H., & Kyrgos, E.C. (2001). Autonomic and cognitive characteristics of anxiety, depression, and co-morbid anxiety-depression. To be presented at the 13th annual meeting of the American Psychological Society, Toronto, Ontario, Canada.

Santucci, A.K., Friedman, B.H., & Kyrgos, E.C. (2001). Autonomic and cognitive characteristics of anxiety, depression, and co-morbid anxiety-depression. Presented at the 2001 Virginia Psychological Association Annual Meeting, Roanoke, VA

Santucci, A. K., & Mikulas, W. L. (2000). Cultivating mindfulness through body awareness. Presented at the 71st annual meeting of the Eastern Psychological Society, Baltimore, MD.

Santucci, A.K., & Mikulas, W.L. (1998). Cultivating mindfulness in a workshop setting. Presented at the 39th annual meeting of the Southeastern Psychological Association, Mobile, AL.

Tloczynski, J., Santucci, A., & Astor-Stetson, E. (1995). The perception of visual illusions by novice and long-term meditators. Presented at the 66th annual meeting of the Eastern Psychological Association, Boston, MA.

Astor-Stetson, E., Waggoner, J., Santucci, A., & Wueschinski, S. (1994). Perception of holusions in set and no-set conditions. Presented at the 1994 University of Scranton Conference.

Grants and Funding

Galper Fund Award, Virginia Tech, April 2001: for professional development activities: $250
Graduate Student Assembly Travel Fund Award, October 2000, $300
Galper Fund Award, Virginia Tech, April 2000: for professional development activities: $200
Thesis Grant, University of West Florida, January 1997-December 1997: "Cultivating Mindfulness Through Body Awareness": $500

Academic Awards

Virginia Tech Graduate School 17th Annual Research Symposium: 2nd place winner for poster presentation in the Arts and Sciences: $200