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HEART RATE VARIABILITY AND ALLOSTASIS IN INDIVIDUALS WITH DEPRESSION AND ANXIETY SYMPTOMS

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By

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DEDICATION

This thesis is dedicated to my family for their undying love and support in everything that I do, and to Jess Margarito for all the wonderful opportunities you have given me over the last 7 years.
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ABSTRACT

HEART RATE VARIABILITY AND ALLOSTASIS IN INDIVIDUALS WITH DEPRESSION AND ANXIETY SYMPTOMS

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Objective: Allostasis suggests that variation in an organism’s physiological response to the environment optimizes functioning and balance, and when this variation is non-existent or impaired it can lead to negative health outcomes. Experiencing chronic negative emotion can impair this process, causing a “wear and tear” in the physical system. Anxiety and depression are both related to the experience of chronic negative emotion. It is believed that worry plays a role in anxiety by blunting emotional experience through hyper-arousal. This causes the system’s allostatic process to become impaired, something known as allostatic load. In depression, it is believed that a person will over-react to sad emotion due to its congruence to the disorder. Through heart rate variability as a measure of autonomic flexibility (how well the autonomic system responds to environmental changes), this study aims to investigate the effects of depressive and anxious symptoms on allostasis, through the use of emotion films.
Method: Participants included 64 college undergraduates, screened for subclinical depression, and anxiety. A control group was also included for comparison, resulting in 22 controls, 22 depressed, and 20 anxious. Participants underwent a series of tasks while being monitored by EKG, which included a rectangle counting tasks, one fear inducing film clip, and one sadness inducing film clip.

Results: Controls showed appropriate signs of autonomic flexibility in response to the sad and fear film clips. The anxious group was not physiologically responsive to any of the film clips, showing autonomic inflexibility, and the depressed group was equally inflexible except when reacting to the sad film, suggesting emotion specific reactivity to the sadness.

Conclusion: While the anxious group continues to exhibit autonomic inflexibility, the depressed group seems to physiologically respond to sad stimuli. This suggests that there is a physiological effect of sadness in individuals with depression that is not mirrored by anxiety and its reaction to fear.
CHAPTER I

Introduction

Emotions and Physiology

Emotions are a universal form of human experience that are described as ever-changing, affectively valenced (i.e. either negatively or positively), short-lived (lasting from minutes to seconds), and subjective states (Larsen et al., 2008). Humans are able to communicate and recognize emotions through facial expressions (Ekman, 1992), and they often motivate human behaviors that either change them or intensifies them. Moreover, emotions are crucial for mediating social relationships, because they are able to elicit physiological states that either inhibit these relationships or promote them (Porges, 2007).

Theories on physiology and emotion arose to explain what happens in the body when we experience an emotion (Canon, 1927). Taking their research on nervous system and its firing patterns Canon and Bard deduced that emotion occurs when we process stimuli first and the resulting evaluation of these stimuli is the emotion (Dalgleish, 2004). Moreover, areas in the brain having to do with stimuli processing fire first, and the areas related to emotion fire shortly after. This theory is a response to William James and Carl Lange’s theory on emotion which proposed that what we experience as emotion is actually the label we give our responses when an event occurs (Kalat, 2007). While these are differing theories, their premise is the same—that emotion affects physiology. Research now indicates that this relationship between the body, nervous system, and emotion is complex, but this complex relationship can affect our physical health.

Allostasis and stress

Allostasis refers to the body’s ability to “gain stability through change” (Sterling & Eyer, 1988). It references acquired stability through variable physiological processes that need to be ever-changing
in order allow appropriate behavioral responses to our environment (Mc Ewan & Wingfield, 2003; Porges, 2007). This process is often mentioned in a wide array of stress studies because stress can be sporadic and has a particularly unique effect on the body (Desok et al., 2008; Dantzer & Kelly, 1989). Under stress, for example, there is simultaneous activation of different systems within the body—heart rate in the circulatory system increases, which provides oxygen to the necessary muscles; the neuroendocrine system releases the stress hormones and glucocorticoids, which increases blood pressure and blood sugar in the body for quick use; and the immune system mobilizes a generalized immune response, whereby all-purpose immune cells are released to maximize protection against disease causing pathogens (Jezova, et al. 2002 ;Segerstrom & Miller, 2004; Glaser & Kiecolt-Glaser, 2005).

The autonomic nervous system (ANS) mediates the stress systems that are crucial in maintaining allostasis. It is subdivided into two systems, the sympathetic (SNS) and parasympathetic (PNS) nervous systems (Kalat, 2007). The ANS and these subsystems can be viewed as a brake system where the SNS requires quick activation, and PNS puts a slow and steady stop to normalize the body from SNS activation (Porges et al., 1994). More specifically, the ANS mediates the performance of different organs in the body such as the heart, lungs, stomach and digestive tract by activating, enhancing, or deactivating these organs depending on the environment (Porges, 1995). Hence, by assessing the performance of some of these organs, we can evaluate this “brake” system and how well the PNS is working to normalize the body from SNS influence (Porges, 2003). Research suggests that when this brake system is not working properly there can be detrimental consequences to one’s health by promoting physiological states that are conducive to illness (Lucini et al., 2006; Cuesta & Singer, 2012).

Allostasis is useful in stressful situations and is mobilized by the SNS, but constant activation of
allostatic cycles are detrimental to one’s health. For example, the positive effects of the cortisol released by the neuroendocrine system begin to have an adverse effect, lowering immune system functioning, and damaging cells in the hippocampus, the brain area known to influence memory (Sugawara, 1982; Mc Ewen & Sapolsky, 1995). This “wear and tear” caused by chronic stress exposure is known as allostatic load (Ogden, 2004). Increased allostatic load has been implicated in weakened immune response, accelerated disease, hypertension, heart disease, and other lieu of negative health outcomes (Mc Ewan, 2000; Taylor, 2006). Allostatic load provides a window to autonomic functioning during high chronic stress (Desok et al., 2008). Studies have observed that the physiological response in people with PTSD or similar stress disorders have become “blunted” or non-reactive (Cohen et al., 2000; Telles et al., 2010). These studies reason that the body is already in a generalized altered and aroused state where no further physiological activation can be observed and this is what led to this habituated response. In order to assess this physiological state, phasic experimental designs where challenge is introduced are often use in conjunction with physiological assessments. Such designs are important and telling of allostasis, because the responses through the different phases in the experiment can be observed, providing a window to one’s physiological functioning.

**Allostasis and Heart rate variability**

Allostasis is a theory of adaptation rooted in the organism’s physiological ability to predict and respond to future environmental demands (Logan & Barksdale, 2008). Allostasis assumes that the organism has a resting state, that this resting state changes to deal with environmental demands when challenge is introduced, and that this resting state is regained after the stressor has been removed (Mc Ewan & Wingfield, 2003; McEwan, 2000). There are observable experimental designs that test the efficiency of this mechanism. Essentially, these designs introduce conditions that activate allostatic cycles and physiological responses are observed (Taelman, et al., 2008). Under normal conditions,
individuals show appropriate physiological responses to stress, but certain individuals afflicted with medical illnesses, such as heart disease, and mental disorders, such as anxiety or depression, do not respond appropriately to stress (Wright et al., 2007; Gordon et al., 2012; Thornton & Hallas, 1999). For example, individuals with heart disease have been observed to over react to stress (Wright et al., 2007), while individuals with depression symptoms have an observed difficulty recovering to normal heart rate baseline after a stress task has lowered it (Gordon et al., 2012). These physiological responses can be measured using an array of indeces. One such index is known as heart rate variability (HRV), which can be used to assess allostasis in tandem with conditions designed to activate allostatic cycles.

HRV is a good measure of allostasis because it is a measure that is highly sensitive to environmental changes (Porges, 2007). HRV is the naturally occurring heart rhythm that reflects a variation in heart beat (R-R) intervals that readily change in response to environmental demands and stress (Task Force, 1996; Taelman, et al., 2008). It is a physiological assessment of the cardiovascular system that can be assessed by a wide set of physiological measures (Kawachi, 1997). Some range from generalized HRV that use time series methods, such as the standard deviation of normal (R-R) intervals (SDNN), to more specific assessments of neurological structures that influence the heart, such as respiratory sinus arrhythmia (RSA) as an assessment of the autonomic nervous system or frequency domain methods, such as power spectrum density (PSD), that look at the composition of plotted R-R waves to determine which subsystem in the autonomic nervous system is providing the signals that make up those plotted R-R waves (Bernston, Cacioppo, & Quigley, 1993). In frequency domain methods, parasympathetic nervous system signals make up the high frequency composition of the plotted R-R waves (around 0.18 to 0.4 Hz), known as high frequency heart rate variability (HF-HRV), and R-R waves plotted around low frequency (0.04 – 0.15) are thought to come from the sympathetic nervous system (Grossman, 1992). Evaluating the autonomic nervous system is important, because it
mediates the resting state. And using HRV, regardless of methodology, is a useful tool for that.

Despite the subtle differences in measuring HRV, these indices have been observed to assess allostatic load. For example, a meta-analysis of different time domain measures of HRV revealed that obtaining low numbers in these measures presented individuals with a high risk for developing cardiovascular disease (Thayer, Yamamoto, & Brosschot, 2010). Similarly, RSA and frequency domain measures indicate that low RSA and low HF-HRV are predictors of morbidity and mortality (Masi et al., 2007; Kristal-Boneh et al., 1995). These studies suggest that despite the measure used to assess cardiovascular functioning, the conclusion was the same. Chronic low HRV is related to negative health outcomes.

As mentioned, HRV is used to assess different parts of the ANS. HRV baseline is the phase of the experimental design where HRV is recorded during either a vanilla baseline task or no task at all (Jennings et al., 1992), and it is the phase mostly related to the physiological resting state, which is influenced by the PNS (Porges, 1994). HRV recordings during this phase are able to tell people with different characteristics apart. For example, medically sick groups with heart problems have been observed to consistently have low HRV compared to control groups (Tsuji et al., 1996; Greiser et al., 2005). This is consistent with studies previously mentioned (Thayer, Yamamoto, & Brosschot, 2010; Masi et al., 2007; Kristal-Boneh et al., 1995). Chronic low HRV is related to poor health outcomes, and using HRV baseline can help us assess which groups are at risk for such outcomes. Another important feature of HRV baseline is that it becomes and observable tool to assess allostasis by providing a point of comparison with other conditions in the same experiment. For example, due to allostasis, HRV will look different during the presentation of a challenging or stressful stimulus than when a minimally invasive stimulus or no stimulus is presented (HRV baseline).

HRV reactivity is an organism’s response to stressful events (Souzza et al., 2013). Reactivity is
often measured during a task that resembles a stressful situation. For example, HRV has been recorded when a person is presenting a speech in front of the experimenter (Beda et al., 2007). During these presentations, HRV decreases from the previous baseline task (Moses et al. 2007). These changes from baseline to task are thought to reflect adaptive changes to the environment where the organism, recognizing and detecting threat, undergoes physiological changes to help it deal with the threat (Bernston et al., 1997). These physiological changes allow the organism to handle stress by meeting metabolic demands in the body (Porges, 1994). HRV reactivity assesses allostasis because it is the phase of the experiment where challenge or stress is introduced and where physical changes occur relative to HRV baseline (Wagner et al., 2013). Allostatic load occurs when no HRV changes are observed in HRV reactivity when compared to baseline HRV. For example, RSA reactivity, a measure of HRV reactivity, has been observed to be blunted in depressed individuals who underwent tasks that elicited challenge (Rottenberg et al., 2007). Depression has been linked to illnesses related to the heart, such as cardiovascular disease (CVD) (Nemeroff et al., 1998). This observed blunting of HRV reactivity is suggestive of allostatic load or the “wear and tear” that may have a role in conditions such as CVD.

HRV recovery is the observed rise of HRV after a stressor has been removed (Hansen & Johnsen, 2013). This mechanism is often viewed as a return to baseline, and has been observed in the literature to be organism’s ability to return to variability after a stress inducing task (Movius & Allen, 2005; Rottenberg et al., 2007). Theoretically, HRV recovery mirrors the parasympathetic response whereby an organism regains their resting state after a stressor has been removed. Indeed HRV recovery has been observed in studies of individuals who underwent stress tasks (Hansen, Johnsen, & Thayer, 2003). Allostatic load in HRV recovery is detected when a blunting effect from HRV reactivity to HRV baseline is apparent. It can also be observed when there are leftover effects of stressful events
that carry over to non-stressful events. For example, Tanja and colleagues (2000) found that reduced HRV in the stressful workplace physiologically carried over into leisure time after work. Since HRV recovery is a sign of parasympathetic recovery and return to baseline, blunting of HRV recovery suggests impaired allostasis and an inability to regain parasympathetic influence to the body.

In summary, the combination of HRV baseline, reactivity, and recovery assess not only allostasis, but allostatic load. The three phases of HRV assess this load by pointing out impairments in any of the three phases. For example, if an individual has high HRV baseline but then has low HRV reactivity to a stimulus and continuously decreased HRV during recovery, then perhaps their parasympathetic nervous system is slow to activate to regain stability. This parasympathetic nervous system impairment is the “wear and tear” in reaction to prolonged levels of stress. On the other hand, perhaps other factors influence and mediate this allostatic response. Research has indicated that emotion has an effect on physiology that is similar to the stress response.

**Stress, allostasis, and negative emotion**

Studies of emotion and physiology indicate that negative emotion has a different physiological pattern than positive emotion (Ekman et al., 1983; 1992), negative emotions activate similar physiological systems of stress such as cortisol release and has similar heart marker patterns (Pietromonaco et al., 2013; Alvarez et al., 2013), and that prolonged experience of negative emotion is related to HRV patterns of dysregulation (Rottenberg, 2007). This dysregulation and this activation of various physiological systems might be indicative of the complex relationship between stress and emotion.

There is evidence that prolonged exposure to stressful situations can lead to mood and anxiety disorders, both of which are disorders of negative emotion. For example, people who are depressed tend to have greater number of stressful life events prior to onset of their disorder (Comer, 2008).
Similarly, a person who experiences a traumatic life event might then develop Post traumatic stress disorder (PTSD), a disorder marked by symptoms that are similar to a stress response (APA, 2013). Also, stressful events in life that would normally lead to depression such as bereavement (APA, 2013), seem to have a physiological impact on individuals who have experienced loss (O’Connor et al., 2000). This is suggestive in the increased mortality of an individual after spousal loss (Clayton, 1974; Krauss & Lilienfeld, 1959). Moreover, research suggests that both anxiety and mood disorders are thought to be involved in the development and exacerbation of heart disease, one of the leading causes of death (Watkins et al., 1999; Carney et al., 2001; Carney & Jaffe, 2002).

Experiencing negative emotion has an impact on the body that is similar to an allostatic stress response. Cardiovascular functioning markers, such as HRV, are low on average during the presentation of negatively charged emotional stimuli versus when no such stimulus is presented (Feldman et al., 1999). Acute experience of negative emotion, such as anger, increases blood sugar and adrenaline activity in the body in preparation to deal with such emotions (Cannon, 1914). These responses mirror allostatic cycles that are experienced during stress. If this is true, then allostatic dysregulation should also be evident on anxiety and mood disorders.

Like prolonged stress, experiencing long periods of negative emotion can lead to the body’s “wear and tear” process of allostatic load. This observed in autonomic dysregulation in anxiety and mood disorders (Nemeroff et al, 1998). Studies observed that mental disorders related to uncontrollable and dysregulated depression were related to low HRV when compared to control groups (Rechlin et al., 1994). Similarly, anxiety disorders, such as panic disorders or specific phobias, are notorious for eliciting and maintaining low HRV throughout the trajectory of the disorder (Monk et al., 2001; Kawachi et al., 1995). These disorders are not only related to dysregulated emotion, but they are also related to dysregulated physiological processes. For example, panic disorders and post-traumatic stress
disorders are related to HRV autonomic dysregulation during a stress task (Cohen et al., 2000). This suggests that people afflicted with such disorders might not respond physiologically appropriate to different environmental tasks and stimuli.

While physiological markers show that autonomic dysregulation occurs in anxiety and mood disorders, studies suggest that this dysregulation might be related to the nature of the disorder. For example, worry, which is related to anxiety, has been shown to blunt physiological responses to prevent one from experiencing further physiological reactivity to negative emotional stimuli (Bokorvec, 1990; Llera & Newman, 2010). Likewise, it has been observed that individuals who are depressed reacted much more emotionally to a sad film than a control group (Golin et al., 1977). This evidence points to potential differences in the way individuals with anxiety and mood disorders physiologically react to emotion.

**Negative potentiation hypothesis and physiological reactivity in depression**

Major Depressive Disorder (MDD) involves (1) depressed mood, (2) diminished interests, (3) significant diminishment or gain in weight or appetite, (4) insomnia or hypersonnia, (5) psychomotor agitation or retardation, (6) fatigue or loss of energy, (7) feeling worthless, (8) diminished ability to concentrate or think, (9) recurrent thoughts of death (DSM-5; APA, 2013). A noteworthy feature of MDD is that it is marked by extreme and uncontrollable sadness (APA, 2013). There is evidence that sadness has distinct behavioral and physiological presentations that differ from other emotions. For instance, the induction of sadness has been paradoxically characterized by high heart rate, and psychomotor retardation (Schartz et al., 1981). Other studies have also noted heart rate increases when sadness is elicited versus when happiness is elicited (Ekman et al., 1983; Bioten, 1996). If this effect is apparent with elicited sadness, then depressed individuals who experience chronic and extreme sadness should have a distinct physiological pattern of response to stimuli.
Allostasis in depression seems to be mediated by the emotional experience of depression. Emotional experience and its expression mimic the same physiological responses as stress. In depression, sadness is the predominant emotion experienced and it is often expressed through crying (Patel, 1993). Using RSA, Rottenberg and colleagues (2003) indicated that crying mediated allostasis in the control group by decreasing one’s physiological variability during crying spells and greatly increasing variability after such crying spell. The period after the crying spell was known as the crying resolution phase. The depressed group did not show RSA increases during the crying resolution task. This is a perfect example of allostatic impairment. Unlike the control group, the depressed group did not show RSA recovery after the crying spell, indicating impairment. Moreover, baseline was also assessed to compare it to the crying and crying resolution phases. The study found higher baseline in the control group relative to the depressed group. Studies mirror these results; highlighting depression’s impaired HRV baseline, reactivity, and recovery (Solomon in press; Solomon et al., 2009). While it seems depression might be related to allostatic impairment, other literature posits that mood congruence, cognition, and depression severity might affect the three phases of allostasis.

Aaron Beck (1979) proposes a cognitive component to depression whereby our thought processes reinforce negative mood and sadness. According to Beck, thoughts are intimately tied to emotion and depression occurs because people have negative thoughts about themselves, their world, and their future (Beck, 1979). These thoughts distort emotional processing in favor of sad or dejected mood. The more an individual thinks negative thoughts the more they become depressed. It is these thoughts that create physiological reactivity because they reinforce negative emotions that are accompanied by physiological changes (Rottenberg, Gross, & Gotlib, 2005).

Some evidence suggests that negative or emotionally congruent stimuli get an exaggerated physiological response from an individual afflicted with depression. The negative potentiation
hypothesis argues that depressed individuals show different reactions to distinct emotional stimuli. More specifically, the theory posits that people with MDD or similar mood disorders are particularly reactive to emotion stimuli that match the cognitive schema of the disorder (Rottenberg, Gross, & Gotlib 2005). In fact, depressed individuals tend to react above and beyond how a normal individual would react to negative stimuli (Sigmon & Nelson-Gray, 1992). If this is true then depressed individuals should be particularly reactive to sad stimuli as opposed to any other stimuli.

Physiological studies seem to support this hypothesis. A study shows that HRV predicted cognitive reactivity in individuals after a sad film even when controlling for the severity of depressive symptoms (Beevers et al., 2011). This indicates that even with or without depression, a person is able to react to negative stimuli when they are prompted. The study gives a glimpse at the mechanism in which MDD affects psychophysiology, namely through cognitive reactivity to sad stimuli and not the symptoms themselves. This study, however, did not look to see if this same cognitive reactivity holds with other emotions, rendering its implications limited.

Still, the literature remains inconclusive as to whether or not depressed individuals exhibit overall blunted cardiovascular responses to sad or stress inducing stimuli. A study by Hughes and colleagues (2000) shows that students with depressed mood tended to physiologically overreact to introduced stressors compared to a control group. Other studies indicate that depressed individuals have a blunted response to different stress tasks (Solomon et al., 2009). This pattern of responding is different from other observed patterns in depression. Other patterns show blunted responses to negative, sad stimuli (Rottenberg et al., 2003). This suggests that while there might be some supporting evidence to the negative potentiation hypothesis, it remains inconclusive. Some studies observe reactivity to stress tasks, and others do not.
Autonomic and emotion dysregulation in anxiety disorders

Anxiety disorders involve somatic symptom experiences that include apprehension, physical tension, experience of physical symptoms and dissociative anxiety (Healy, 2005). Most anxiety symptoms are related to autonomic hyperarousal. This is seen in the various disorders listed by the Diagnostic Statistical Manual for Mental Disorders, 5th edition (DSM-5; APA, 2013). For example, according to the DSM-5 (APA, 2013), Panic Disorder is characterized by panic attacks involving somatic symptoms such as chest pain, feelings of choking, heart palpitations, numbness, chills, or paraesthesia (APA, 2013). While anxiety is related to somatic symptoms, the emotions closely related to it are fear and worry. These emotions are motivational emotions that drive the behavior behind anxiety disorders.

The allostatic response in anxiety is similar to that in depression. For example, studies have observed decreased HRV baseline in groups with diverse anxiety disorders such as panic disorder, social anxiety disorder, obsessive compulsive disorder, and generalized anxiety disorder relative to controls (Pittig et al., 2012). The study attributes this low HRV baseline response to autonomic inflexibility, or the autonomic nervous system’s inability to alternate physiological states. Indeed, the theoretical features of anxiety have been related to physiological states that prevent individuals with anxiety disorders from alternating appropriate physiological states.

Pertinent to anxiety is the state of worry, and worry’s ability to affect our psychophysiological state. Worry is the cognitive state most related to anxiety disorders. For example, one symptom of generalized anxiety disorder is “excessive anxiety and worry” (APA, 2013). According to Friedman (2007) worry is a cognition that “alerts to immediate threat, keeps unresolved threat in consciousness, and anticipates future threat”. Unlike fear, which is a singular emotion in response to threat, worry is a collection of enduring thoughts and/or images that deal primarily with anticipation. For example,
people who suffer from anxiety often endorse worrisome thoughts like “a situation is not safe until proven safe” or “it is always better to assume the worst” (Comer, 2005). These thoughts not only promote worry, but also promote physical arousal that accompanies it. Worry has often been related to physiological changes, like increases autonomic arousal (Friedman, 2007). Hence, if a person is constantly worried, then their arousal will constantly be high, and this worry will affect the way they physiologically react to threatening and negative stimuli.

Worry plays a big factor in the way psychophysiological patterns may present themselves in people with anxiety disorders. It has been shown, for example, that worry blunts the cardiovascular response (physiological measure) to threatening emotional stimuli in people with a specific phobia (Borkovec, 1990). More specifically, when people with a specific phobia were told to worry prior to the presentation of the threatening stimuli, they showed no cardiovascular reactivity to the stimuli when compared to those individuals who were told to relax and had the similar phobia. This form of psychological “blunting” has been consistent across physiological studies and self reports, and is particular to the cognitive state of worry (Mennin et al., 2005; Bokorvec, 1990). The physiological blunting of worry in anxiety has major implications for allostasis.

First, worry affects allostasis because it is a form of preparedness, physiological or otherwise (emotional), which readies the individual to deal with how they will react to the stimuli. This preparedness may be responsible for the observed low HRV baseline, which signals an impaired resting state. Second, the autonomic arousal that comes from worry sets up general level of arousal in which no room is given for further arousal to other negative and/or threatening stimuli. This creates autonomic inflexibility and the observed reactivity blunting in anxiety disorders (Bernston et al., 1998). Finally, the constant state of worry of an individual who fails to physiologically react to a stimulus will carry over in HRV recovery, and no change will be apparent.
Affect, mood and anxiety disorders

Drawing clear distinctions between mental disorders can be challenging. There is high comorbidity, for example, between anxiety and depression and it is extremely difficult to separate one from the other (Clark, 1989). The tripartite model (Clark & Watson, 1991) attempts to make distinctions between these disorders through the symptoms associated with them. The theory states that depression and anxiety both have to do with negative affect, but depression has to do more with the lack of positive affect, whereas anxiety has to do more with physiological hyper arousal. These differences are in line with the symptoms for both disorders. For example, consistent with the tripartite model, the different anxiety disorders in the *Diagnostic Statistical Manual of Mental Disorders 4th edition* (APA, 2000) have high hyper arousal symptomatology.

Positive affect is particularly useful in assessing the difference between anxiety and depression, because those who are particularly anxious still experience it (Clark & Watson, 1991). Positive affect has been described by Watson as the level of pleasurable engagement to the environment (Watson, Clark, & Carey, 1988). This level of pleasurable engagement consists of different elements that show how much the individual is enjoying the engagement. For example, high levels of joy and interest, two of the elements of positive affect, might create motivational states that would allow a person to engage with his or her environment. Being that positive affect is a key feature in separating these two disorders, this study would use this theoretical feature of the tripartite model to separate anxiety and depression.

Hypotheses

Based on previous research, it is hypothesized that HRV will be affected by anxiety and depression in the following ways:

1. Baseline HRV in anxiety and depression will be lower than the control group, mirroring studies
that have observed the same phenomenon (Rottenberg, et al., 2007; Friedman, 2007)

2. Due to worry avoidance, no HRV reactivity and recovery will be observed in the anxious group (Mennin, 2000; Bokorvec, 1990).

3. Due to the negative potentiation hypothesis, HRV reactivity will be observed in the depressed group during the sad film only, but no HRV recovery will be observed (Rottenberg, Gross, & Gotlib, 2005).

4. HRV reactivity and recovery will be observed in the control group.
CHAPTER II

Methods

Participants

As part of the Department of Psychology’s pretesting, 64 students were administered a 25 item questionnaire consisting of items from the Beck Anxiety Inventory, the Beck Depression Inventory - II, and the Positive and Negative Affectivity Schedule-Positive Affect (BAI-/BDI-II/PANAS-PA) at the beginning of the three consecutive semesters. Individuals were preliminarily grouped using the BAI, BDI-II, PANAS-PA and their scores reflected the following: Persons considered in the high subclinical depression and low subclinical anxiety scored a 7-20 (moderate-severe depression) on the items of the BDI-II, a score of 0-7 (mild anxiety) on the items of the BAI, and a scores of 10-24 (low positive affect) on the PANAS-PA. Persons with high subclinical anxiety and low subclinical depression scored an 8-24 (moderate-severe anxiety) on the items of the BAI, a score of 0-7 (mild depression) on the items of the BDI-II, and a score of 25-50 (high positive affect) on the PANAS-PA. Persons considered in the control group scored from 0-7 (mild anxiety and depression) on the items of the BDI-II and the items of the BAI, and high scores on the PANAS-PA (25-50). Persons who meet these qualifications were invited to complete a 45-60 minute battery of tests. Participants that came into the study were not taking any medications, and did not have a history of stroke, heart problems or diabetes. The study involved adult students, who can read and understand English, and were given the right to withdraw from the study at any time, as specified in the study’s information and consent forms. If necessary, participants were reminded that they had the right to decline any further participation.
Procedure

Students who meet criteria in the pre-screener questionnaires, and were willing to participate were able to choose from a selection of session times during which they can come to a specified room of the CSUN Department of Psychology as managed by the Psychology Department subject pool software.

In the beginning of the study, the administrator introduced the study and provided participants with information forms. To insure anonymity and confidentiality, each participant received a subject code to track his or her forms throughout the study. Participants were asked to fill out the full versions of the BDI, BAI, and a demographic questionnaire. Their grouping depended on the full versions of the BAI and BDI-II, in which the individuals were grouped according to the highest score on either scale. If the individual, for example, scored higher on the BAI he/she was placed in the anxious group. Following this, EKG electrodes were placed on the participant’s wrists, and above the right side of the right ankle, and a respiratory band was placed around the participant’s chest. To maximize comfort, research assistants placed electrodes on same-gendered participants. Participant’s weight and height were also taken to calculate their BMI scores.

Participants were seated in a comfortable chair while recording devices were placed, and remained in that chair through the remainder of the study. Once placement concluded, instructions were being given for a “vanilla” baseline task (Jennings et al., 1992), which took approximately 4-5 minutes. This was immediately followed by either the fear film clip or the sad film clip, conditions which were counterbalanced across subjects. After the first emotion film clip, the participant was asked to fill out a sleepiness scale and another likert scale post film questionnaire that assessed the degree of self reported emotion experienced during film clip. A second “vanilla” baseline immediately preceded the second emotion clip. After both sadness and anxiety emotion inducing clips have been watched, an amusement
clip was shown in order to decrease the negative effects of the previous two films.

After completing the questionnaire, participants received a debriefing form and any questions they had were answered by the administrators. Participants were further instructed to address further questions to the co-principal investigator, who is well-versed with the details of the study. Participants were also be given the phone number for the university counseling office, as well as other similar resources.

**Measures**

1) *Items from the Beck Depression Inventory - II and the Beck Anxiety Inventory (BDI-II/BAI).* The prescreener questionnaire had a total of seven items from the Beck Depression Inventory - II (BDI-II: Beck, Steer, & Brown, 1996) and eight items from the Beck Anxiety Inventory (Beck, Epstein, Brown & Steer, 1988). These items have been validated by Stultz & Crits-Christoph (2010) to slightly increase the hit rate of correct diagnosis in people with anxiety and depression. Depressive factors looked at by this screener included: sadness, pessimism, past failure, self criticalness, loss of interest, worthlessness, and loss of interest in sex. Anxiety factors looked at by this screener included: numbness or tingling, heart pounding or racing, feeling of choking, shaky, difficulty breathing, faint, face flushed, and sweat (not due to heat). These measures together took approximately 6 minutes to complete.

2) *Positive and Negative Affectivity Scales (PANAS-PA).* The PANAS-PA is a selection of items from Clark and Watson’s (1991) overall PANAS-X (Clark, 1989; Clark & Watson, 1991, 1994). It is a 10-item self-report questionnaire that assessed the level of the participant’s positive affect. It is answered in Likert scale format rating from 1 (strongly disagree) to 5 (strongly agree). This measure took about 5 minutes to complete. Cutoff scores were determined by population comparison, and these include 10-32 low positive affect, and 33-50 high positive affect (Crawford & Henry, 2004). Different time frames are given in conjunction to the PANAS, but for our purposes the timeframe adopted was ‘for the
past two weeks’.

3) Beck Depression Inventory (BDI-II). Subjects enrolled in the full study received the full version of the Beck Depression Inventory – II (BDI-II: Beck, Steer, & Brown, 1996). The BDI-II is a 21-item self-report instrument used to assess the severity of depression in adults and adolescents age 13 years and older. The questionnaire was developed to indicate the presence and degree of cognitive, motivational, affective, and somatic symptoms of depression. The following recommended cut off scores for the BDI-II will be used: 0-9 normal; 10-15 dysphoric; 16-20 depressive states; 20-30 moderate depression; 30-63 severe depression (Kendall, Hollon, Beck, Hammen, & Ingram, 1987). This measure took about 5 minutes to complete.

4). Beck Anxiety Inventory (BAI). Subjects enrolled in the full study received the full version of the Beck Anxiety Inventory (BAI: Beck, Epstein, Brown & Steer, 1988): The BAI is a 21-item self-report instrument used to assess the severity of anxiety in adults and adolescents age 13 years and older. The questionnaire was developed to indicate the presence of somatic symptoms of anxiety. The following recommended cut off scores for the BAI will be used: 0-7 minimal level of anxiety; 8-15 mild anxiety; 16-25 moderate anxiety; 26-63 severe anxiety (Kendall, Hollon, Beck, Hammen, & Ingram, 1987). This measure took about 5 minutes to complete.

5) Physiological measures (HRV)

Heart rate and respiration were recorded through Aqknowledge (Version 4.2). The participants were asked to wear a respiration band around their chest right underneath their armpits. The band contains sensors that record breathing activity. Electrodes that record heart rate were placed on the participant’s wrists and one on the leg below the right ankle. Placement took approximately between 5 to 7 minutes.

Emotion clips from mainstream films

Participants were asked to view three video clips from popular movies (“When Harry Met
Sally”, “Silence of the Lambs”, and “The Champ”). These videos elicit the emotions of amusement, fear, and sadness (Gross & Levenson, 1995). The exposure to each film took approximately between 3 to 4 minutes on average.

Data collection and EKG analysis

Data was collected via EKG using BIOPAC software, Acqknowledge version 4.2 at a sample rate of 200 Hz, the minimum sample rate suggested by BIOPAC systems. Data collected was analyzed under Kubios software, a Matlab extension software developed by the University of Finland, Applied Physics Department. Kubios has been used in previous research to analyze EKG waveforms (Quintana et al., 2013), and it has been specifically used to analyze data having to do with depression and anxiety disorders (Kemp et al., 2012). Kubios is often used because it’s simple, has QRS detection algorithms to detect R-R waves, has trend removal analysis options, and calculates time and frequency domain features of HRV (Tarvainen et al., 2014).

Kubios processed the EKG waveform and calculated the standard deviation of normal intervals (SDNN), the distances between R waves, the wave with the biggest amplitude on the waveform. Data was filtered for outliers and an interpolation method under its correct artifact feature. The medium setting was selected, whereby Kubios identified which intervals were “abnormally” bigger or smaller than 0.25 second compared to the local average. This setting was chosen, because the results correlated highly to the raw data. This converted the RR series into an equidistantly sampled form, which allowed the data to be controlled for outliers, and for missing data. Although the time series SDNN method was the primary focus of the analysis, a smooth priors detrending method that can affect the power spectrum density, a frequency domain method, was implemented (Yoo et al., 2004). The smooth priors detrending method filtered out the very low frequency heart rate variability (LVF-HRV) signals from the analysis in the data, which amplified those signals sent out by the parasympathetic and sympathetic
nervous systems, HF-HRV and LF-HRV, respectively. The smooth priors approach has been shown to have an effect on time-series measures of HRV, such as SDNN (Tarvainen, et al., 2001). Since this method can affect time series SDNN, it was implemented to obtain HRV that consisted of strong autonomic nervous system signals.

Statistical analyses

To assess HRV level differences across groups, a multivariate analysis of variance (MANOVA) using groups (subclinical depression, sub-clinical anxiety, and control) as an independent between subjects variable and SDNN baseline as a dependent samples variable was conducted. Second, repeated measures ANOVAs for each of the three groups were used to detect potential differences in SDNN within each condition. In this case, the independent variable was condition and the dependent variable was SDNN. Finally, four follow up dependent samples t-tests were used to detect specific differences in conditions (baseline to fear, fear to baseline, baseline to sadness, sadness to baseline) and to assess reactivity and recovery if an effect of condition on SDNN was found in the repeated measures ANOVAs.
CHAPTER III

Results

Demographic and subclinical characteristics

64 participants were called in and asked to participate in the study. The overall average age of participants was 19.125 (SD = 2.00) years of age. In terms of overall ethnic categorization, the participants were 56.1% Hispanic/Latino, 22.7% Caucasian, 6.3% African American, 6.3% Asian, and 6.3% other, which included mixed and Middle Eastern participants. These overall percentages mirrored the percentages within each group (see Table 1). Participants were categorized into groups based on their scores on the full versions of the measures described above, and their average weights and BMI scores were also calculated (see Table 2).

Table. 1

Demographic characteristics divided into sex, ethnicity, and education per group

<table>
<thead>
<tr>
<th>Variable</th>
<th>Subclinical Depression (n = 22)</th>
<th>Subclinical Anxiety (n = 20)</th>
<th>Control (n = 22)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>%</td>
<td>n</td>
</tr>
<tr>
<td>Sex</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>5</td>
<td>22.7</td>
<td>6</td>
</tr>
<tr>
<td>Female</td>
<td>17</td>
<td>77.3</td>
<td>14</td>
</tr>
<tr>
<td>Ethnicity</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hispanic</td>
<td>14</td>
<td>63.6</td>
<td>11</td>
</tr>
<tr>
<td>Caucasian</td>
<td>5</td>
<td>22.7</td>
<td>6</td>
</tr>
<tr>
<td>Black</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Asian</td>
<td>3</td>
<td>13.6</td>
<td>1</td>
</tr>
<tr>
<td>Other</td>
<td>0</td>
<td>0</td>
<td>3</td>
</tr>
<tr>
<td>Education</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Freshman</td>
<td>11</td>
<td>50</td>
<td>11</td>
</tr>
<tr>
<td>Sophomore</td>
<td>6</td>
<td>27.3</td>
<td>6</td>
</tr>
<tr>
<td>Junior</td>
<td>3</td>
<td>13.6</td>
<td>1</td>
</tr>
<tr>
<td>Senior</td>
<td>2</td>
<td>9.1</td>
<td>2</td>
</tr>
</tbody>
</table>

Note: Other: Included participants that self identified as mixed race, and Middle Eastern.
Table. 2

Average Age, BMI, weight, and inventory scores per group

<table>
<thead>
<tr>
<th>Measures</th>
<th>Subclinical Depression n = 22</th>
<th>Subclinical Anxiety n = 20</th>
<th>Control n = 22</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>SD</td>
<td>M</td>
</tr>
<tr>
<td>Age</td>
<td>19.63</td>
<td>2.88</td>
<td>18.95</td>
</tr>
<tr>
<td>BMI</td>
<td>24.21</td>
<td>4.45</td>
<td>22.58</td>
</tr>
<tr>
<td>Weight</td>
<td>147.54</td>
<td>28.90</td>
<td>139.65</td>
</tr>
<tr>
<td>BDI-II-P</td>
<td>7.07</td>
<td>3.83</td>
<td>3.32</td>
</tr>
<tr>
<td>BAI-P</td>
<td>3.92</td>
<td>3.00</td>
<td>7.34</td>
</tr>
<tr>
<td>PANAS-PA</td>
<td>29.78</td>
<td>9.53</td>
<td>38.05</td>
</tr>
<tr>
<td>BDI-II</td>
<td>17.27</td>
<td>8.04</td>
<td>10.5</td>
</tr>
<tr>
<td>BAI</td>
<td>9.55</td>
<td>5.78</td>
<td>20.25</td>
</tr>
</tbody>
</table>


Data for analysis and Missing Data

The data collected had a total of 7 conditions, but for the purposes of our analyses we only ran 5, which were the intended conditions. The remaining two conditions were amusement film (not counterbalanced) and another vanilla baseline condition. These remaining conditions were used to the benefit the participant in an attempt to have them leave with no leftover effects of the sad or fear films. Data for 15 participants were unusable because of missing data due to placement issues, or individuals scored the same in the BDI-II and BAI (See Table 3). There were a higher number of unusable and missing data in the anxious group. This was due to training issues during a semester where more individuals with higher anxiety symptoms were called in to participate.
Table. 3

<table>
<thead>
<tr>
<th>Group</th>
<th>n</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>D</td>
<td>2</td>
<td>0.03</td>
</tr>
<tr>
<td>A</td>
<td>8</td>
<td>0.10</td>
</tr>
<tr>
<td>C</td>
<td>3</td>
<td>0.04</td>
</tr>
<tr>
<td>Com</td>
<td>2</td>
<td>0.03</td>
</tr>
<tr>
<td>Total</td>
<td>15</td>
<td>0.19</td>
</tr>
</tbody>
</table>

Note: C = Control, A= Anxious, D = Depressed, Com = Comorbid (participants that scored the same in BDI-II and BAI)

Grouping and Positive Affect

Regrouping was observed across the depressed and the anxious groups. Regrouping occurred as a result of those individuals who scored a certain way in their pre-screener measures and scored the opposite on the full scales. A summary of participant regrouping based on their scores may be found in Table 4. Six participants out of the twenty two placed in the depressed group had a PA score of 32 or higher. Five of those individuals in the depressed group who originally fulfilled the depression requirements for the subclinical depressed group, ended up being placed in the anxious group due to the higher scores in the BAI than BDI-II. These five individuals had PANAS scores below 32. One participant in the control group who also fulfilled the depression requirements was placed in the control group for scoring low in the overall BAI and BDI-II scores. These participants also had PANAS scores 32 or lower.

To assess the validity of the relationship between positive affect and depression, a correlation was performed using BDI-II scores and PANAS-PA scores as variables. There was a significant negative correlation between BDI-II scores and PANAS-PA scores, \( r (64) = -0.56, p < .001 \), indicating
that the lower the BDI-II scores, the higher PANAS-PA scores. This relationship has been validated in literature (Clark & Watson, 1991). Since there is a significant correlation between these two variables, it may be that positive affect also mediates some of the effect on SDNN, but this effect cannot be completely separated from the effects that depression might have on SDNN. Due to this, the scores in the PANAS-PA cannot be treated as a covariate.

Table. 4

<table>
<thead>
<tr>
<th>Regrouping between pre-screener and post screener</th>
<th>Group</th>
<th>Participants</th>
</tr>
</thead>
<tbody>
<tr>
<td>C to A</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>C to D</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>A to C</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>A to D</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>D to C</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>D to A</td>
<td>4</td>
<td></td>
</tr>
</tbody>
</table>

Note: C=Control, A=Anxious, D=Depressed.

Baseline Multivariate Analysis of Variance

To assess baseline HRV across groups, a multivariate analysis of variance (MANOVA) using the three baseline SDNNs as dependent variables and group as an independent variable was performed. There was a statistically significant difference in SDNN based on group, $F (6, 118) = 2.41, p < .05$, Wilks’ $\Lambda = 0.79$, partial $\eta^2 = 0.11$, indicating that baseline levels differed between the groups. Furthermore, univariate analysis of variance with group as the independent variable on each individual’s baseline also revealed a significance effect of group on all baseline conditions (See Table 5). Tukey HSD post hoc analysis indicated that across baseline 1 and baseline 3, the control group’s baseline SDNN was significantly higher than those of the depressed group ($p < .05$) and the anxious groups ($p < .05$). The analysis, however, failed to find a significant difference in baseline 2, where baseline SDNN in the control group was not significantly different from those of either the depressed or
anxious groups ($p > .05$). Furthermore, the analysis failed to find significant differences in baseline SDNN between the anxious and depressed groups across all three baseline conditions ($p > .05$), indicating that baseline for the anxious group was similar to that of the depressed group. The Tukey HSD analysis revealed that across groups, the baseline condition was higher in the control group than for the depressed and anxious groups (See Table 6 and Figure 1).

**Table. 5**

Univariate analysis of variance

<table>
<thead>
<tr>
<th>Source</th>
<th>$df$</th>
<th>$F$</th>
<th>$p$</th>
<th>Partial $\eta^2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Groups</td>
<td>Baseline 1</td>
<td>2</td>
<td>5.90$^{**}$</td>
<td>0.00</td>
</tr>
<tr>
<td></td>
<td>Baseline 2</td>
<td>2</td>
<td>3.24$^*$</td>
<td>0.05</td>
</tr>
<tr>
<td></td>
<td>Baseline 3</td>
<td>2</td>
<td>5.87$^{**}$</td>
<td>0.00</td>
</tr>
</tbody>
</table>

Note: $^{**}p < .001$; $^*p < .05$

**Table. 6**

Multiple group comparisons using Tukey HSD

<table>
<thead>
<tr>
<th>Dependent Variable</th>
<th>Mean Difference</th>
<th>SE</th>
<th>$p$</th>
<th>95% Confidence Interval</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Lower Bound</td>
<td>Upper Bound</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline 1</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>D and A</td>
<td>±2.90</td>
<td>5.92</td>
<td>0.88</td>
<td>±17.11</td>
</tr>
<tr>
<td>C and D</td>
<td>±18.51$^*$</td>
<td>5.77</td>
<td>0.01</td>
<td>±4.64</td>
</tr>
<tr>
<td>C and A</td>
<td>±15.61$^*$</td>
<td>5.92</td>
<td>0.03</td>
<td>±1.39</td>
</tr>
<tr>
<td>Baseline 2</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>D and A</td>
<td>±1.07</td>
<td>5.97</td>
<td>0.98</td>
<td>±15.42</td>
</tr>
<tr>
<td>C and D</td>
<td>±13.43</td>
<td>5.83</td>
<td>0.06</td>
<td>±0.56</td>
</tr>
<tr>
<td>C and A</td>
<td>±12.36</td>
<td>5.97</td>
<td>0.10</td>
<td>±1.98</td>
</tr>
<tr>
<td>Baseline 3</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>D and A</td>
<td>±2.53</td>
<td>5.67</td>
<td>0.90</td>
<td>±16.14</td>
</tr>
<tr>
<td>C and D</td>
<td>±17.60$^*$</td>
<td>5.53</td>
<td>0.01</td>
<td>±4.32</td>
</tr>
<tr>
<td>C and A</td>
<td>±15.07$^*$</td>
<td>5.67</td>
<td>0.03</td>
<td>±1.45</td>
</tr>
</tbody>
</table>

Note: C = Control, A = Anxious, D = Depressed, $^p < .05$
Assessing change in SDNN within tasks and assessing covariates

To assess whether or not there was potential for reactivity or recovery within each group, a repeated measures ANOVA was conducted on each of the groups to measure the change in SDNN within each of the conditions. Each of the groups met assumptions of sphericity, indicating that variances in SDNN within each task were not significantly different from one another. Second, a slew of potentially confounding variables such as weight, BMI, sex, order of how the task was given, education, and ethnicity were treated as covariates. The effects of each of these on the task and SDNN were non-significant.

The analysis found a statistically significant effect of task on SDNN for both the depressed group, $F(4, 80) = 2.85, p < .05$, partial $\eta^2 = 0.12$, and the control group, $F(4, 84) = 5.45, p < .05$, partial $\eta^2 = 0.21$. But no statistical significance was observed in the anxious group, $F(4, 76) = 1.86, p > .05$, partial $\eta^2 = 0.09$, indicating no SDNN change in whatever task was presented. Since there was an effect of task on SDNN for the control and depressed groups, this warrants more specific tests to see where this difference lies.
**Reactivity and Recovery analysis of SDNN within each group**

To assess specifically for HRV reactivity (SDNN decrease from baseline to condition), two paired-samples *t*-tests were used. These compared the baseline SDNN with the condition SDNN in the depressed and control groups, where a difference had been detected due to the repeated-measures ANOVA. There was a significant decrease in the control group’s SDNN between the baseline (*M* = 60.42, *SD* = 22.49) and fear (*M* = 54.57, *SD* = 19.86) conditions, *t* (22) = 2.35, *p* > .0125. There was also a significant decrease between the baseline (*M* = 61.22, *SD* = 20.30) and sadness (*M* = 54.91, *SD* = 19.29) conditions, *t* (22) = 2.53, *p* > .0125. For the depressed group there was a non-significant drop from the baseline SDNN (*M* = 41.90, *SD* = 16.07) to the fear SDNN (*M* = 44.58, *SD* = 17.48), *t* (21) = -1.12, *p* > .05. Meanwhile, there was a significant difference between the baseline SDNN (*M* = 47.78, *SD* = 19.32) and the sadness SDNN (*M* = 42.91, *SD* = 17.72), *t* (21) = 2.40, *p* < .05 in the depressed group.

To assess for HRV recovery (SDNN increase from condition to baseline), two paired-samples *t*-tests were used to compare condition SDNN with the baseline SDNN in the depressed and control groups. The test found that a rise in SDNN was evident in the control group when comparing both the fear condition (*M* = 54.57, *SD* = 19.86) to baseline (*M* = 61.22, *SD* = 20.30), *t* (22) = -2.88, *p* < .0125, and the sadness condition (*M* = 54.91, *SD* = 19.29) to baseline (*M* = 64.12, *SD* = 21.60), *t* (22) = -3.88, *p* < .0125. These effects were not observed in the depression group. That is, there was a non-significant effect when comparing the fear condition (*M* = 44.58, *SD* = 17.48) to baseline (*M* = 47.78, *SD* = 19.32), *t* (21) = -1.64, *p* > .0125, and the sadness condition (*M* = 42.91, *SD* = 17.72) to baseline (*M* = 46.52, *SD* = 14.28), *t* (21) = -1.62, *p* > .0125 in the depressed group.
**Table 7.**

*SDNN Reactivity and recovery in the control group*

<table>
<thead>
<tr>
<th>Condition</th>
<th>Baseline</th>
<th>Condition</th>
<th>t</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>SD</td>
<td>M</td>
</tr>
<tr>
<td>Fear reactivity</td>
<td>60.42</td>
<td>22.49</td>
<td>54.57</td>
</tr>
<tr>
<td>Sadness reactivity</td>
<td>61.22</td>
<td>20.3</td>
<td>54.91</td>
</tr>
</tbody>
</table>

*p < .05. **p < .01 Note: M = Mean. SD = Standard Deviation. Condition for fear reactivity is fear film clip. Condition for Sadness reactivity is Sad film clip.

<table>
<thead>
<tr>
<th>Condition</th>
<th>Baseline</th>
<th>Condition</th>
<th>t</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>SD</td>
<td>M</td>
</tr>
<tr>
<td>Fear recovery</td>
<td>54.57</td>
<td>19.86</td>
<td>61.22</td>
</tr>
<tr>
<td>Sadness recovery</td>
<td>54.91</td>
<td>19.29</td>
<td>64.12</td>
</tr>
</tbody>
</table>

*p < .05. **p < .01 Note: M = Mean. SD = Standard Deviation. Condition for Fear recovery is fear film clip. Condition for Sadness recovery is sad film clip.

**Table 8.**

*SDNN Reactivity and recovery in the depressed group*

<table>
<thead>
<tr>
<th>Condition</th>
<th>Baseline</th>
<th>Condition</th>
<th>t</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>SD</td>
<td>M</td>
</tr>
<tr>
<td>Fear reactivity</td>
<td>41.90</td>
<td>16.07</td>
<td>44.58</td>
</tr>
<tr>
<td>Sadness reactivity</td>
<td>47.78</td>
<td>19.32</td>
<td>42.91</td>
</tr>
</tbody>
</table>

*p < .05. **p < .01 Note: M = Mean. SD = Standard Deviation. Condition for Fear reactivity is fear film clip. Condition for Sadness reactivity is sad film clip.

<table>
<thead>
<tr>
<th>Condition</th>
<th>Baseline</th>
<th>Condition</th>
<th>t</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>M</td>
<td>SD</td>
<td>M</td>
</tr>
<tr>
<td>Fear recovery</td>
<td>44.58</td>
<td>17.48</td>
<td>47.78</td>
</tr>
<tr>
<td>Sadness recovery</td>
<td>42.91</td>
<td>17.72</td>
<td>46.52</td>
</tr>
</tbody>
</table>

*p < .05. **p < .01 Note: M = Mean. SD = Standard Deviation. Condition for Fear recovery is fear film clip. Condition for Sadness recovery is sad film clip.
CHAPTER IV

Discussion

The purpose of this study was to assess the features of allostasis and allostatic load in anxiety and depression and how these compare with a control group using HRV baseline, reactivity, and recovery. Previous studies suggest that individuals with depression and anxiety disorders have decreased HRV baseline relative to controls (Friedman & Thayer, 1998(a)(b); Pittig et al., 2013; Rottenberg et al., 2007). This same effect was predicted and observed in this study. HRV baseline in the depressed and anxious groups was decreased relative to the control group. More specifically, the anxious and depressed groups presented continuously decreased HRV that was significantly different from the control group during all baseline conditions. No significant change was observed between any of the baseline HRV conditions between the depressed and anxious groups, indicating that the MANOVA effect is driven by the difference between the control group and the two mentioned groups. This suggests that anxious and depressed individuals may have chronic hyperarousal patterns and autonomic inflexibility due to allostatic load.

It was further predicted that there would be an effect of condition on SDNN in the control and depressed groups, but not the anxious group. The repeated measures ANOVA revealed an effect of condition in the depressed and control groups, but not the anxious groups. This indicated that there was SDNN change in one or more conditions and indicated reactivity and recovery in the control and depressed groups. Specific differences in the control group lie before and after both film conditions. When the control group was presented with a sad or fear film their HRV decreased compared to their previous baseline. Their HRV also increased immediately after the film clips when presented with the subsequent baseline condition. This is indicative of healthy autonomic flexibility and an appropriate allostatic response (Mc Ewan & Stellar, 1993; Souza et al., 2013).
The depressed group seemed to physiologically react specifically to the sad film. As predicted, depressed individuals showed reactivity during the sad film, but did not show reactivity during the fear film nor did they show recovery after both the sad and fear film clips. This reactivity to the sad film occurred when HRV decrease after baseline during the sad film. This same effect has been observed in previous research, and has been shown to predict future recovery from depression compared to depressed individuals who do not have reactivity during the sad film (Rottenber et al., 2006). Unlike the anxious group, the depressed group in this study physiologically responded based on the emotional nature of the stimulus. While there seems to be autonomic inflexibility and an apparent allostatic load in depression, there seems to be an effect of emotion in this instance.

The anxious group did not show this reactivity or recovery. Indeed meta-analyses, theoretical papers, and studies on diverse anxiety disorders continue to support the notion of poor autonomic inflexibility and high allostatic load in anxiety disorders that are apparent in baseline, reactivity and recovery periods (Gorman & Sloan, 2000; Licht et al. 2009; Mc Ewan, 2003). As further predicted, the anxious group showed unchanging HRV during the baseline, film clip, and subsequent baseline conditions. This supports the notion of allostatic load in the anxiety group. Their constant hyper-arousal creates a dysfunctional physiological state that does not allow the individual to physiologically react to differing emotional stimuli.

**Potential Factors affecting the study**

It is important to mention that although order did not have a strong influence in the study, changing the order of the dependent samples t-test did seem to influence reactivity and recovery and changes the implications of the study. We combined the variance for both fear-sadness and sadness-fear conditions by running the four pairs dependent samples t-test as if all participants underwent the fear-sadness condition. The reason the analysis was done like this was because there was no statistically
significant effect of order, and because the baseline conditions presented were exactly the same for both fear-sadness and sadness-fear conditions. Since this was true, we could assume that the SDNN changes would remain the same regardless if the conditions were flipped. This did not happen.

There was a slight difference in interpretation when the data was analyzed under the assumption that all participants underwent a sadness-fear condition. When this happened, there appeared to be no reactivity when comparing the first baseline to sad film in the control group. However, there continued to be reactivity for the fear film and the SDNN remained changed after each film (recovery) and its significance remained robust and in-tact in the control group. This suggests that while order might have had a slight effect in HRV reactivity in the control group, it did not affect the robust effect of HRV recovery.

The anxious group continued to show blunted HRV reactivity and recovery regardless of the analysis. And in the depressed group, the effect of this change in analysis was curious. When the analysis was inverted and sadness-fear condition was given first, there appeared to be no reactivity, or decline from baseline SDNN to sad film clip SDNN, but there appeared to be recovery, or SDNN increase during the subsequent baseline task after the sad film clip. Given this effect of order in the analysis, the implications for reactivity in the control and depressed groups must be interpreted with caution.

Potential reasons for this discrepancy in analyses when the conditions are switched for the depressed group could have been due to depression severity and whether this factor was distributed normally in both groups. An independent samples t-test that was used to assess BDI-II score differences in the depressed group found slight, albeit non significant, differences in severity scores between depressed individuals who were placed in the fear-sadness than those placed in the sadness-fear conditions. In this analysis the mean scores for the fear-sadness depressed group were lower the mean.
score for the sadness-fear depressed group. Studies indicate that depression severity can have a substantial effect on HRV (Stein et al., 2000) and this seemed to be observed in this study.

Although changes were observed when the condition analysis was flipped, there was still a significant difference in some of the baseline-condition, or condition to baseline comparisons. In the control group reactivity in the fear film and recovery after both films was still maintained, even if reactivity on the sad condition was not evident anymore. In the depressed group there was recovery after the sad film indicating that the depressed group still reacted particularly to the sad condition and not fear. While this may limit the reactivity and recovery implication of this study, there is still validity in assuming that individuals in the depressed group had a particular reaction to the sad film, and that the control group maintains autonomic flexibility due to its reactivity to the fear film and its subsequent recovery to both films.

The grouping of the variables could be another factor affecting the study. The use of the two short pre-screeners and PA scales were not entirely successful at separating individuals with anxiety and depression. This sometimes resulted in individuals having scores in the anxiety and depression inventories that were close to each other. This closeness in anxiety and depression can reflect a comorbidity of these clinical constructs in some of the participants. Studies have observed a greater HRV decrease in comorbid anxiety and depression when compared to depression alone (Kemp et al., 2012). Therefore, perhaps the effect of the depressed group was driven by the comorbidity. Alternatively, the same was true for some individuals in the anxious group. Their scores in the anxiety inventory were also near the scores in the depression inventory. This suggests that, while there might be a problem of comorbidity, the issue was spread out in the groups, cancelling each effect out.

Another potential factor that could have affected the study was positive affect (PA). In an attempt to separate depression and anxiety, PA was used in conjunction with other established
measures of depression and anxiety. It has been theorized that PA is low in depression but not anxiety, allowing the researcher to separate these disorders. This study failed to successfully separate those individuals with depression and anxiety using this method, with some individuals belonging to the opposite group from what was originally thought (as assessed by the full anxiety and depression inventories). Furthermore, positive affect was found to be negatively correlated with BDI-II scores, attesting to their robust relationship.

**Future Directions**

Future studies would do well to duplicate this study addressing the limitations found, which were counterbalancing and the pre-screening participants. Our study attempted to screen our participants with a quick screener used in previous research (Stultz & Crits-Christoph, 2010). The screener divided questions from the full scales BAI and BDI-II, theorizing that separating these items will increase their diagnosing hit rate among clinical populations. Their hit rate increased 83%, which was only slightly behind the full scales. We had a different experience. Some of the participants screened with this measure did not reliably remain in the group they were intended. For example, some individuals might have scored high in depression in the shorter measure, but ended up scoring low in the full measure, making them fit into one of the opposing groups. Perhaps studies can use a greater number of measures and clinical interviews to appropriately screen for those individuals with depression. Also, when counterbalancing, future studies should also randomize their participants to make sure that depression severity or any other variable is evenly distributed in groups. This randomization would prevent an effect of order that was observed in this study.

Given that persons with anxiety disorders often show blunted reactivity to any stimuli, it would be interesting to see if this finding generalizes to other disorders. This is of particular interest when it comes to the effect of worry, and its blunted effects on physiology (Borkovec, 1990). Since worry is
present in most anxiety disorders, perhaps it has a mediating effect on the way these anxiety disorders influence HRV. Other research, however, suggest that it is the nature of the disorder that has a great impact on HRV. Friedman (2007) indicated that physiological impact of anxiety disorders can be seen as hierarchical, with worsening results if the symptoms of the anxiety disorder are that of hyper arousal (heart palpitations, hyperventilation, sweating). This is evident in that HRV has been observed to be lower in panic disorder than social phobia and generalized anxiety disorder. The current study did not assess the effect of worry on HRV, but it did ask the participants about their symptoms. Perhaps teasing apart these anxiety disorders and assessing their physiological reactivity to different emotion stimuli can evaluate the degree into which anxiety physiologically affects emotional experience.

Studies can also concentrate on the phenomenon of reactivity in depression. Since depressed individuals reacted more to the sad film, it would be interesting to see whether severity of depression has an effect on this phenomenon. Other studies with clinical populations suggest the depressed individuals have an attenuated response to sad stimuli much like the anxious group in this study with the two film conditions. Another study suggests that physiologically reacting to sad stimuli in depression is a marker and predictor for future recovery (Rottenberg et al., 2006). Perhaps the more severe one’s symptoms, the more attenuated one’s response to emotionally congruent stimuli becomes. A meta-analysis (Bylsma, Morris, and Rottenberg, 2008) observed that MDD had an overall blunting effect on reactivity as opposed to hyperactivity (unusual increase in HRV to negative stimuli) or negative reactivity (normal decrease in HRV to negative stimuli). This study used a normal college population that experiences depression symptoms, but is still functional given their academic obligations. By using groups taken from clinical populations and comparing them with groups from academic populations such as this one, we can have a better understanding of the way depression affects physiological experience of emotion.
Also, studies should concentrate on the mediating effects of positive affectivity on physiology. Positive affect was found to be greatly correlated with the BDI-II, regardless of what group the individual ended up being placed. Given this relationship, we were unable to use positive affect as a covariate. The variables were just too closely related. Initially, it was used to separate anxiety from depression, but there was a grouping issue where most people who scored low on PA and high on the BDI-II-P ended up scoring higher on the full versions of the anxiety and depression scales. Perhaps future studies can look at the relationship between PA and how that affects depression and anxiety in physiological measures. More specifically, they can separate depression and anxiety disorders into two distinct groups using PA as a mediator. For example, comparing HRV measures across those depressed individuals with high positive affect and vice versa. A comparison between these two groups can give researchers a hint on the effects of PA on depression. In my study, there was no differentiation of whether the depressed group or lack of positive affect had the effect on SDNN.

Finally, future studies would do well in using other physiological markers to assess allostasis. Cortisol, for example, is a marker that has similar phasic properties like HRV. Cortisol has been assessed during cognitive performance task, with increase release when task is presented versus when it was not (Bohnen et al., 1990). Moreover, the phasic problems in HRV are also found in cortisol. For example, blunted cortisol reactivity has been observed with populations that undergo prolonged stress due to stress causing medical conditions (Hebert & Lupien, 2007). Considering cortisol has more direct effects on the body, suppresses immune response and impairs memory (Sugawara, 1982; Mc Ewen & Sapolsky, 1995), it would be beneficial to assess how cortisol reactivity profiles differ in anxiety and depression, providing a bigger scope of health functioning in these groups.

In conclusion, the present study attempted to assess allostasis in people who experience anxiety and depression symptoms using HRV. Results indicated that allostasis in anxiety and depression is
impaired compared to controls. The results of this study have implications for emotion and the physiological experience of emotion. It indicates that the presentation of emotional stimuli creates specific physiological changes in the heart, that psychological disturbances can affect these changes and disrupt these processes, and that these psychological disturbances may disrupt the physiological experience of emotion.
References


APPENDIX A

Demographic Questionnaire

Name: ___________________

Participant Code Number: ______________________

Date: ______________________

Sex: [ ] Male [ ] Female

Age: __________

DOB: __________

Education Level (Freshman, Sophomore, Junior, Senior): ____________

Ethnicity: __________________
APPENDIX B

Becks Depressive Inventory – II Purified (BDI-II-P)

Instructions: This questionnaire consists of 7 groups of statements. Please read each group of statements carefully, and then pick out the one statement in each group that best describes the way you have been feeling during the past two weeks including today. Circle the number beside the statement you have picked. If several statements in the group seem to apply equally well, circle the highest number for that group. Be sure that you do not choose more than one statement for any group.

1. Sadness

0  I do not feel sad
1  I feel sad much of the time
2  I am sad all the time
3  I am so sad or unhappy that I can’t stand it.

6. Loss of Interest in Sex

0  I have not noticed any recent change in my interest in sex
1  I am less interested in sex than I used to be.
2  I am much less interested

2. Pessimism

0  I am not discouraged about my future.
1  I feel more discouraged about my future than I used to be.
2  I do not expect things to work out for me.
3  I feel my future is hopeless and will only get worse.

7. Worthlessness

0  I do not feel I am worthless
1  I do not consider myself as worthwhile and useful as I used to
2  I feel more worthless

3. Past Failure

0  I do not feel like a failure.
1  I have failed more than I should have.
2  As I look back, I see a lot of failures.
3  I feel I am a total failure as a person.

4. Self Criticalness
as compared to other people

0 I don’t criticize or blame myself more than usual.
1 I am more critical of myself than I used to be.
2 I criticize myself for all of my faults.
3 I blame myself for everything bad that happens.

TOTAL:_______

5. Loss of Interest

0 I am no more irritable than usual
1 I am more irritable than usual
2 I am much more irritable than usual.
3 I am irritable all the time.
APPENDIX C

Beck’s Anxiety Inventory Purified (BAI-P)

Below is a list of common symptoms of anxiety. Please carefully read each item in the list. Indicate how much you have been bothered by that symptom during the past week, including today, by circling the number in the corresponding space in the column next to each symptom.

<table>
<thead>
<tr>
<th>Not At All</th>
<th>Mildly, but didn’t bother me much</th>
<th>Moderately-It wasn’t pleasant at times</th>
<th>Severely-It bothered</th>
</tr>
</thead>
<tbody>
<tr>
<td>Numbness and Tingling</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Heart pounding/racing</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Feeling of Chocking</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Shaky/Unsteady</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Difficulty in Breathing</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Faint/Lightheaded</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Face Flushed</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Hot/Cold Sweats</td>
<td>0</td>
<td>1</td>
<td>2</td>
</tr>
</tbody>
</table>

TOTAL:  

Grand Total: 

53
APPENDIX D

Positive and Negative Affective Schedule – Positive Affect (PANAS-PA)

This scale consists of a number of words and phrases that describe different feelings and emotions. Read each item and then mark the appropriate answer in the space next to that word. Indicate to what extent you have felt this way during the past two weeks. Use the following scale to record your answers:

1  2  3  4  5
Very slightly, a little moderate quite a bit extremely
Not at All

_____active
_____alert
_____attentive
_____determined
_____enthusiastic
_____excited
_____inspired
_____interested
_____proud
_____strong

TOTAL: _______
APPENDIX E

Beck’s Depression Inventory - II

Instructions: This questionnaire consists of 21 groups of statements. Please read each group of statements carefully, and then pick out the one statement in each group that best describes the way you have been feeling during the past two weeks, including today. Circle the number beside the statement you have picked. If several statements in the group seem to apply equally well, circle the highest number for that group. Be sure that you do not choose more than one statement for any group, including Item 16 (Changes in Sleeping Pattern) or Item 18 (Changes in Appetite).

1. Sadness
   0 I do not feel sad.
   1 I feel sad much of the time.
   2 I am sad all the time.
   3 I am so sad or unhappy that I can’t stand it.

2. Pessimism
   0 I am not discouraged about my future.
   1 I feel more discouraged about my future than I used to be.
   2 I do not expect things to work out for me.
   3 I feel my future is hopeless and will only get worse.

3. Past Failure
   0 I do not feel like a failure.
   1 I have failed more than I should have.
   2 As I look back, I see a lot of failures.
   3 I feel I am a total failure as a person.

4. Loss of Pleasure
   0 I get as much pleasure as I ever did from the things I enjoy.
   1 I don’t enjoy things as much as I used to.
   2 I get very little pleasure from the things I used to enjoy.
   3 I can’t get any pleasure from the things I used to enjoy.

5. Guilty Feelings
   0 I don’t feel particularly guilty.
   1 I feel guilty over many things I have done or should have done.
   2 I feel quite guilty most of the time.
   3 I feel guilty all of the time.

6. Punishment Feelings
   0 I don’t feel I am being punished.
   1 I feel I may be punished.
   2 I expect to be punished.
   3 I feel I am being punished.

7. Self-Dislike
   0 I feel the same about myself as ever.
   1 I have lost confidence in myself.
   2 I am disappointed in myself.
   3 I dislike myself.

8. Self-Criticalness
   0 I don’t criticize or blame myself more than usual.
   1 I am more critical of myself than I used to be.
   2 I criticize myself for all my faults.
   3 I blame myself for everything bad that happens.

9. Suicidal Thoughts or Wishes
   0 I don’t have any thoughts of killing myself.
   1 I have thoughts of killing myself, but I would not carry them out.
   2 I would like to kill myself.
   3 I would kill myself if I had the chance.

10. Crying
    0 I don’t cry anymore than I used to.
    1 I cry more than I used to.
    2 I cry over every little thing.
    3 I feel like crying, but I can’t.
Beck Depression Inventory

11. Agitation
0  I am no more restless or wound up than usual.
1  I feel more restless or wound up than usual.
2  I am so restless or agitated that it's hard to stay still.
3  I am so restless or agitated that I have to keep moving or doing something.

12. Loss of Interest
0  I have not lost interest in other people or activities.
1  I am less interested in other people or things than before.
2  I have lost most of my interest in other people or things.
3  It's hard to get interested in anything.

13. Indecisiveness
0  I make decisions about as well as ever.
1  I find it more difficult to make decisions than usual.
2  I have much greater difficulty in making decisions than I used to.
3  I have trouble making any decisions.

14. Worthlessness
0  I do not feel I am worthless.
1  I don't consider myself as worthwhile and useful as I used to.
2  I feel more worthless as compared to other people.
3  I feel utterly worthless.

15. Loss of Energy
0  I have as much energy as ever.
1  I have less energy than I used to have.
2  I don't have enough energy to do very much.
3  I don't have enough energy to do anything.

16. Changes in Sleeping Pattern
0  I have not experienced any change in my sleeping pattern.
  1a I sleep somewhat more than usual.
  1b I sleep somewhat less than usual.
  2a I sleep a lot more than usual.
  2b I sleep a lot less than usual.
  3a I sleep most of the day.
  3b I wake up 1–2 hours early and can't get back to sleep.

17. Irritability
0  I am no more irritable than usual.
1  I am more irritable than usual.
2  I am much more irritable than usual.
3  I am irritable all the time.

18. Changes in Appetite
0  I have not experienced any change in my appetite.
  1a My appetite is somewhat less than usual.
  1b My appetite is somewhat greater than usual.
  2a My appetite is much less than before.
  2b My appetite is much greater than usual.
  3a I have no appetite at all.
  3b I crave food all the time.

19. Concentration Difficulty
0  I can concentrate as well as ever.
1  I can't concentrate as well as usual.
2  It's hard to keep my mind on anything for very long.
3  I find I can't concentrate on anything.

20. Tiredness or Fatigue
0  I am no more tired or fatigued than usual.
1  I get more tired or fatigued more easily than usual.
2  I am too tired or fatigued to do a lot of the things I used to do.
3  I am too tired or fatigued to do most of the things I used to do.

21. Loss of Interest in Sex
0  I have not noticed any recent change in my interest in sex.
1  I am less interested in sex than I used to be.
2  I am much less interested in sex now.
3  I have lost interest in sex completely.
APPENDIX F

Beck’s Anxiety Inventory

Instructions: Below is a list of common symptoms of anxiety. Please carefully read each item in the list. Indicate how much you have been bothered by each symptom during the Past week, including today, by placing an X in the corresponding space in the column next to each symptom.

(I) THE BECK ANXIETY INVENTORY (BAI)

<table>
<thead>
<tr>
<th>Name of the symptom</th>
<th>Not At All</th>
<th>Mildly, but it didn’t bother much</th>
<th>Moderately – it was unpleasant at times</th>
<th>Severely – it bothered me a lot</th>
</tr>
</thead>
<tbody>
<tr>
<td>Numbness or tingling</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Feeling hot</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Wobbliness in legs</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Unable to relax</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Fear of worst happening</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Dizzy or lightheaded</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Heart pounding/racing</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Unsteady</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Terrified or afraid</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Nervous</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Feeling of choking</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Hands trembling</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Shaky/Unsteady</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Fear of losing control</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Difficulty in breathing</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Fear of dying</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Scared</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Indigestion</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Paint/lightheaded</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Face flushed</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>Hot/cold sweats</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>COLUMN SUM</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>GRAND TOTAL</td>
<td></td>
<td></td>
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</table>