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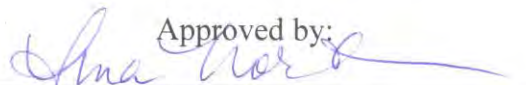
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Stress in Young Adults: Implications of Mandala-Coloring on Anxiety

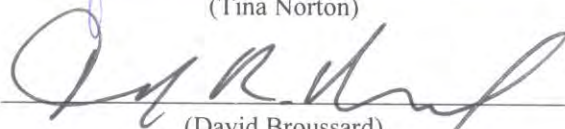
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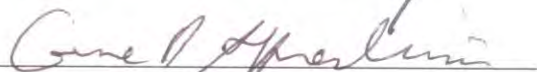
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Stress in Young Adults: Implications of Mandala Coloring on Anxiety

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Abstract

The 20th century has been deemed the “age of anxiety” as research indicates that there has been a substantial rise in anxiety since the 1950’s (Twenge, 2000). The purpose of this experiment is to assess the effectiveness of mandala-coloring paired with a validated mindfulness technique such as focused breathing as a possible therapeutic tool to be used especially with people experiencing stress or anxiety. It is primarily predicted that coloring mandalas paired with focused breathing will reduce anxiety greater than controls. It is further predicted that self reports of anxiety along with physiological responses will be different between experimental and control groups. The procedure in this study consists of four phases which include baseline (sitting and standing), speech 1 and speech 2 which represent psychosocial stressors validated through the Trier Social Stress Test, and post-stress manipulation which consists of either 7 minutes of mandala-coloring or no-task depending on group. Self-reports of state anxiety, positive and negative affect, and blood pressure were assessed once after each phase, whereas pulse, skin conductance levels (SCL), and heart rate (HR) were measured throughout the duration of the experiment. Results indicated that state anxiety and negative affect were lower in the mandala-coloring (experimental) group as compared to the no-task (control group) following the psychosocial stressor. This suggests modest support for the effectiveness of mandala-coloring paired with already validated focused breathing as an effective technique for reducing self-reported anxiety. Further research will be needed to assess the combination of these techniques in clinical settings.

Keywords: Stress, state anxiety, physiological reactivity, coloring therapy, mandala, mindfulness

Stress in Young Adults: Implications of Manadala-Coloring on Anxiety

Approximately 18.1% of adults suffer from anxiety disorders (Sandmire, Gorham, Rankin, & Grimm, 2012). This makes them the most prevalent lifetime psychological disorders in the United States (Kessler et al., 2005). The specific disorders to fall under this broad category include panic disorder, generalized anxiety disorder, specific phobias, agoraphobia, social phobia, and posttraumatic stress disorder. Furthermore, other psychological disorders such as brief reactive psychosis, adjustment disorders, mood disorders, and some forms of schizophrenia all possess stress-related components (Everly & Lating, 2013). Due to the prevalence of stress and anxiety, the 20th century has been deemed the “age of anxiety” as research indicates that there has been a substantial rise in anxiety since the 1950’s (Twenge, 2000). Ultimately, since stress and anxiety are so prevalent in today’s society, it is important for clinicians to have many validated therapeutic tools in order to help people reduce levels of stress and anxiety.

Historical Context

Stress is a historical term that was originally defined by Hans Selye as, “the state manifested by a specific syndrome which consists of all the nonspecifically induced changes within a biologic system,” (Selye, 1956). Selye also emphasized the importance of defining what stress is not. He explains that stress is not tension, a result of damage, and is not avoidable (Selye, 1973). The most important word in this definition is “non-specific.” Selye believed that many things could act as stressors and bring about the stress response.

General adaptation syndrome. Selye went on to develop the General Adaptation Syndrome (GAS), which he described as a biological syndrome as a result of some stressor (Selye, 1975). This system consists of three phases which include the alarm reaction, stage of resistance, and stage of exhaustion. The alarm reaction is the body’s initial response to a stressor.

Selye described the alarm reaction as a somatic response of the body's defensive forces, or "call to arms," (Selye, 1975). Next, the body enters a stage of resistance which is wholly opposite to the alarm reaction. In the stage of resistance, the body is preparing for continuous exposure to the stressor and bodily functions move above normal resistance levels (Selye, 1975). Eventually, the stage of exhaustion occurs. In this stage, the organism has been exposed to the stressor for a considerable amount of time. It happens that the adaptation energy which increases in the stage of resistance is now depleted and the organism becomes worn out (Selye, 1975).

"Fight or flight." Selye's development of the alarm reaction was greatly influenced by Walter Cannon's description of the "fight or flight" reaction of the sympathetic nervous system. Cannon sought to explain the origin of intense emotions such as surprise, terror, and anger. Cannon first points out that primitive vertebrate animals react to their surroundings by uncontrollable reflexes (Cannon, 1949). This uncontrollable reflex compares to the human sympathetic nervous system which is a widely distributed and involuntary system that connects things like organs, muscles, and sweat glands. (Cannon, 1949). The sympathetic nervous system becomes active at times of intense emotional responses. Some examples of sympathetic activation include constricted blood vessels, inhibition of digestion, increased muscular activity, and other salivary and fluid excretions (Cannon, 1949). Cannon was able to conclude that the presence of danger evokes a strong emotional response such as fear. In a time of fear, the person may feel an impulse to run away or to fight an enemy if necessary (Cannon, 1949). Activation of the sympathetic nervous system is what prepares the body for this fight or flight response. The fight or flight response is what Selye describes in his alarm reaction phase of the GAS.

Biological Stress Response

Sympathetic Nervous System. One of the main biological systems that responds to stress is the sympathetic nervous system which is also known as the “fight or flight” nervous system. The sympathetic nervous system is part of the autonomic nervous system. This is a fast and immediate response that allows the body to prepare to react to a threatening situation. Activation of this system, which is part of the autonomic nervous system in the brainstem, results in the discharge of norepinephrine throughout the brain which results in enhanced arousal, vigilance, and anxiety (Chrousos & Gold, 1992). Furthermore, the hippocampus and amygdala are the major brain regions involved in this system along with their relationship between the mesocortical and mesolimbic dopamine pathways that are activated during the stress response (Chrousos & Gold, 1992). During the stress response, 35% of epinephrine and norepinephrine are secreted from the adrenal medulla on the kidneys with the remaining being directly released in the blood stream by sympathetic nerve endings (al’Absi, 2007). Furthermore, catecholamines such as epinephrine impact organ systems by being transported throughout the body (al’Absi, 2007). Overall, the sympathetic nervous system is activated by a stressor and catecholamines impact the brain and organ system in order to generate the fight or flight response.

Parasympathetic Nervous System. A branch of the autonomic nervous system that works contrary to the sympathetic nervous system (SNS) is the parasympathetic nervous system (PNS). The PNS innervates the same organs and parts of the body as the SNS, but in most cases works to diminish the “fight or flight” response (Stern et al., 2001). For example, if the sympathetic nervous system is activated during a time of threat then heart rate increases. Once the threat is gone, the PNS will be activated in order to reduce heart rate. Although, it is important to note that the SNS and PNS do not only have to work in a reciprocal manner, they

can also work in a coactivational manner meaning they are both active at the same time (Stern, et al., 2001). The relationship between the PNS and SNS is highly complex, and not as straightforward as once thought.

Hypothalamus-pituitary-adrenal (HPA) axis. Another important regulator system involved in the stress response is the HPA axis. Compared to the sympathetic response, the HPA axis works more slowly in responding to stress. Upon stimulation by a stressful stimulus, the paraventricular nucleus of the hypothalamus (PVN) commences secretion of corticotrophin-releasing hormone (CRH) (al'Absi, 2007). CRH then triggers adrenocorticotropin (ACTH) from the pituitary gland (al'Absi, 2007). ACTH travels through the bloodstream to the adrenal cortex where it triggers the secretion of cortisol, a glucocorticoid which is a steroid that helps regulate glucose (al'Absi, 2007). The functioning of the HPA axis is regulated by a negative feedback loop in which cortisol loops back to the hypothalamus and anterior pituitary in order to inhibit CRH from being released and thus stopping the process. The main effects of cortisol include the redirection of energy consumption among organs, increase of cardiovascular functioning, affecting the immune system as well as mood and cognitive process, along with aiding in metabolic demand while the organism is under stress (al'Absi, 2007). Overall, the HPA axis is an important system in the allostatic state of supporting homeostasis especially when the organism is under stress.

Homeostatis & Allostasis

In order to understand the unstable biological response of the body as a result of a stressor, it is first important to understand the body's normal stability. Homeostasis can be defined as the stability of physiological systems such as body temperature, pH, glucose and oxygen levels that are absolutely necessary to maintain life (McEwen & Wingfield, 2003).

Allostasis is a process that supports homeostasis by achieving stability in response to environmental or life changes (McEwen & Wingfield, 2003). Examples of the effects of allostasis include hormones of the hypothalamus-pituitary-adrenal (HPA) axis, and catecholamines such as epinephrine and norepinephrine that are released as a result of sympathetic nervous system activation (McEwen & Wingfield, 2003). Furthermore, an allostatic state refers to the altered activity levels that need to be maintained in order to respond to challenges or a changing environment (McEwen & Wingfield, 2003). An allostatic state can be loosely associated with Seyle's stage of resistance. Moreover, an allostatic state is when the body increases its biological resources in order to survive an environmental change (McEwen & Wingfield, 2003). A collective result of an allostatic state can be described as an allostatic load in which either energy demands exceed energy income, or energy income exceeds demands (McEwen & Wingfield, 2003). The body's biological response to stress represents an allostatic state in which the body is attempting to return to homeostasis.

Cannon-Bard Theory of Emotion

Before defining anxiety, it is essential to note the connection between stress and anxiety. In his studies, Walter Cannon defined emotion as "feelings" and other affective experiences (Cannon, 1949). Therefore, anxiety can be considered an emotion. In Cannon's experiments with cats, he observed that removal of their SNS resulted in the termination of their physiological reactions. However, Cannon observed that that cat's emotional responses and temperament were unchanged (Cannon, 1949). From these observations, Cannon theorized that an eliciting stimulus would cause subcortical activity in the thalamus (current research emphasizes the cortex) which would then cause autonomic arousal and the presence of emotion (Cannon, 1949). The most

important aspect of this theory is that physiological arousal and emotion are separate systems, and not dependent on each other.

Anxiety

Anxiety is a specific concept that can result from stress. As the concept developed, it was broken into two spheres. Trait anxiety represents stable individual differences in anxiety-proneness, and the tendency for people to perceive stressful situations as threatening (Spielberger, 1985). State anxiety represents more of a reaction than a basic tendency. State anxiety consists of an emotional reaction and a stream of subjective feelings that a person might experience during a situation in which they perceive as threatening. State anxiety can also be defined as a level of intensity corresponding to the activation of the automatic nervous system (Spielberger, 1985).

Physiological Stress Response

In a threatening situation, the sympathetic system is responsible for inducing the stress response. The stress response can be defined as a range of psychophysiological responses such as increased heart rate, blood pressure, respiration, and electrodermal activity as a result of frightening stimuli (Kirschbaum, 1993). Stress response dysfunction is apparent in many clinical populations, but also occurs in normal controls. For example, studies have reported that panic disorder patients indicate increased heart rate and blood pressure levels, and thus overactive sympathetic systems resulting in chronic anxiety (Martinez, Garakani, Kaufmann, Aaronson, & Gorman, 2010). It has also been shown that individuals with intellectual disability (ID) show higher levels of stress as a result of social and psychological experiences (Schrade et al., 2011). This increase in stress is probably a result of prolonged poor social conditions, and ridicule or stigma towards their disability (Schrade et al., 2011). Furthermore, patients with fibromyalgia

syndrome (FMS) report disruption in psychosocial functions with symptoms of anxiety, depression, and poor quality of life (Schmidt et al., 2013). These symptoms could be a result of stress brought on by their chronic pain. Therefore, stress and anxiety are reported in many populations with the underlying result of the physiological stress response.

Cardiovascular system. This system revolves around how the heart moves blood to various organs around the body. The cardiovascular system can be described in terms of heart rate, cardiac output, as well as blood pressure, volume, and flow (Stern, Ray, & Quigley, 2001). Changes in cardiovascular activity are linked to stressors along with motor activity and body movement. More specifically, activation of the sympathetic nervous system produces arousal responses within the cardiovascular system (Stern et al., 2001). Some of these responses include increase in heart rate and blood pressure.

Heart rate. Heart rate can be defined as the number of heart beats that occur per minute (Stern et al., 2001). Thus, as a result of a stressor, heart rate increases in beats per minute (bpm).

Blood pressure. Another response that increases as a result of sympathetic activation is blood pressure (BP). BP can be described as the necessary pressure that the heart must produce in order to move blood through arteries, capillaries, and veins (Stern et al., 2001). Furthermore, the maximum amount of blood pressure occurs when the heart contracts (Stern et al., 2001). This is referred to as systolic BP. Conversely, the minimum amount of blood pressure occurs after contraction when the heart is in relaxation (Stern et al., 2001). This is referred to as diastolic BP. It is important to note that diastolic BP is most sensitive to cardiovascular assessment. A normal blood pressure of a healthy college student is 120/80 mmHg (Stern et al., 2001). However, factors such as diet, age, and weight impact an individual's BP. Further, mean arterial pressure

can be calculated by adding 1/3 systolic BP to 2/3 diastolic BP. Mean arterial pressure represents that average arterial pressure of a single cardiac cycle which includes contraction and relaxation.

Pulse volume. Pulse is another cardiovascular response that is affected by sympathetic activation. Pulse volume is the change in blood flow as it relates to the pumping of the heart (Stern et al., 2001). Specifically, pulse volume is a measurement of the amplification of single pulse (Stern et al., 2001).

Electrodermal activity. Electrodermal activity refers to the electrical activity occurring in a person's skin. More specifically, skin conductance level (SCL) refers to the tonic level of electrical conductivity of the skin, and typical values range anywhere from 2-20 microsiemens (μS) (Stern et al., 2001). As a result of sympathetic activation, sweat rises toward the skin's surface in varying amounts from varying glands (Stern et al., 2001). The hydration of the skin with sweat increases SCL (Stern et al., 2001). Therefore, SCL should increase as a result of sympathetic activation resulting from a stressor.

Biopsychosocial Paradigm

The biopsychosocial perspective has been taken on by many professional fields in order to most thoroughly explain health and illness. This paradigm implies that biological, psychological, and social forces work together in order to determine a person's health or vulnerability to disease (Straub, 2012). In other words, this perspective upholds a systems model which posits that many different aspects of a system need to work together in order to create an outcome. The belief in the biopsychosocial paradigm classifies the mind and body as entities that influence each other. The relationships between the theoretical perspectives that fall under the biopsychosocial paradigm and that are relevant to this research are explained in Figure 1.

Mind-Body Therapy (MBT)

MBT can be defined as healing practices with the aim to use the mind's ability in order to affect biological functioning (Bertisch, Wee, Phillips, & McCarthy, 2009). Thus, MBT uphold the biopsychosocial model. MBT is typically an alternative therapy meaning that it is used in conjunction with another clinically supported therapy. MBT includes practices such as meditation, mindfulness, deep breathing, muscle relaxation, guided imagery, biofeedback, and yoga, etc., and is reported to be used by about 16.6% of Americans to treat medical or psychological conditions (Bertisch et al., 2009).

Mindfulness

One mechanism of MBT is mindfulness which plays a role in treatment of psychological disorders such as borderline personality disorder, anxiety, and depression, along with use in non-clinical populations (Arch & Craske, 2006). Mindfulness can be described as fostering concentration, attention, and acceptance toward what a person is experiencing at the present moment (Craft & Craske, 2006).

Focused Breathing

Further, focused breathing is a type of mindfulness technique. The premise of focused breathing is to have people become aware of the sensations of breathing while paying attention to experiences in the present moment (Craft & Craske, 2006). Thus, focused breathing has been used to aid the parasympathetic nervous system in order to return the body to homeostasis after a stress response (Linehan, 1993). Focused breathing is commonly used as a stress reduction technique especially in with people with Borderline Personality Disorder (Linehan, 1993).

Meditation

For clarification, meditation also falls under the category of mind-body therapies. Meditation can be described as a relaxing method which limits stimulus input and centers attention on a constant object of focus (Carrington et al., 1980). Therefore, the basic principles of meditation are similar to that of mindfulness, but mindfulness is more easily achieved with less practice. There is some research that states that meditation can be used as a relaxation or cognitive technique that could be of therapeutic benefit to anxiety sufferers (Bogart, 1991).

Mandalas

A mandala is a circular art form that resembles geometric stained glass. Mandalas were used in Eastern cultures as a form of meditation. In Sanskrit, the word mandala means “healing circle” (Schrade, Tronsky, & Kaiser, 2011). Carl Jung was the first to use the mandala as a therapeutic tool. He proposed that the drawing of a mandala had a calming and healing effect on its creator (Henderson, Rosen & Mascaro, 2007). Consequently, it is suggested that the drawing or coloring of mandalas creates a trance-like state, similar to a meditative state that is effective in reducing stress. The mandala is a tool that is sought to organize a person’s inner-chaos (Curry & Kassar, 2005). In other words, drawing, coloring, or tracing the mandala’s structured pattern helps to elicit structure within the person’s thoughts ultimately creating a meditative state.

Art Therapy

Art therapy is a treatment option that is highly unstudied, but could be extremely useful in the treatment of anxiety disorders. Creativity is a function exclusive to humans (Pinker, 2012). Art therapy channels the use of creativity and of art making to help elicit self-expression. This form of expression helps people create a visual representation of their mental state (Curry & Kassar, 2005). By physically expressing anxiety through art making processes, people can

organize, make sense of, and even correct their feelings. Coloring therapy, in particular, is the combination of art therapy with meditation (Curry & Kasser, 2005). In other words, it is argued that the act of coloring not only encourages self-expression, but also produces a meditative state that could alleviate sentiments of anxiety. One specific means to create this meditative state is through the use of a mandala.

The purpose of this experiment is to test the effectiveness of mandala coloring enhanced by focused breathing techniques as a possible therapeutic technique especially with people experiencing anxiety. It will specifically examine how the use of mandalas influences anxiety with an emphasis on the effectiveness of art therapy. It is broadly hypothesized that self-reported and physiological anxiety will change over time as a result of experimental manipulation. The primary hypothesis predicts that coloring mandalas paired with focused breathing will reduce anxiety greater than controls. It is further predicted that self reports of anxiety along with physiological responses will be demonstrate differences between experimental and control groups. Demographic information such as gender and declared major and minor will also be considered. In general, this study seeks to address the question whether people could consider coloring or other art making processes an effective art therapy technique. Art therapy along with mindfulness techniques like focused breathing are still growing and developing within the field of psychology.

Methods

Participants

Of the 37 participants in the current sample, 81% were female and 19% were male. Participants were primarily Caucasian (81%). Other races that were represented were Asian (8%), African American (5%), and Hispanic (5%). The average age of participants was 19 years

(SD=1.22, range = 18-21). The sample was comprised of 54% freshmen, 11% sophomores, 16% juniors, and 19% seniors. The participants were undergraduate students attending a small liberal arts college in North Central Pennsylvania. The participants were notified of the experiment via posters hung across campus advertising the study. All participants were compensated \$10 cash for participation in the study funded by the Joanne and Arthur Haberberger Research Fellowship. All procedures were approved by Lycoming College Institutional Review Board.

Materials

Self-reported affect.

State Anxiety. The State Trait Anxiety Inventory (STAI) Form Y-1 was used to assess state anxiety (Spielberger, 1985). This instrument is a survey consisting of 20 questions answered on a 4-point Likert scale which ranges from 1 being “not at all” to 4 being “very much so”. Some of the questions asked were “I feel tense” or “I feel calm.” A high score indicates a high level of state anxiety, and a low score represents a low level of anxiety. The STAI is a validated measure that has been widely used across a variety of studies throughout the field of psychology (Spielberger, 1985; Curry & Kasser, 2005). STAI scores will be one dependent variable of self-reported affect in this study.

Positive and Negative Affect. The Positive and Negative Affect Schedule Short Form (PANAS-SF) was used to assess positive and negative mood (Watson, Clark, & Tellegan, 1988). Both positive and negative affect scores will be dependent variables of the research. This instrument is a self-report survey consisting of 20 descriptor words in no particular order. Participants were instructed to rate the words dependent on their feelings in that present moment. Words were rated on a 5-point Likert scale ranging from 1 being “very slightly or not at all” to 5 being “extremely.” A high positive score indicates that participants are in an energetic and

pleasurable mood where as a high negative score would indicate a nervous or aversive mood (Watson, et al., 1988). It is important to note that these two mood factors are negatively correlated (Watson, et al., 1988). This means that when negative scores are high, then positive scores should be low and vice versa. The PANAS-SF is a validated measure widely used in the field of psychology (Watson, et al., 1988). Both PANAS positive and negative scores will be used as a dependent variable to measure self-reported affect.

Physiological Measures. iWorx psychological physiology equipment was used to measure all physiological data. This data was recorded into Labscribe which is software used to analyze data.

Blood Pressure. A ReliOn® blood pressure monitor was used to measure participant's blood pressure. This cuff was secured above the elbow of the participant's dominant arm, the opposite arm that the electrodes were attached. BP measurements will be another dependent variable of the study. Blood pressure measurements were taken at four points throughout the experiment. These include once during baseline, after speech #1, after speech #2, and immediately following the seven minute post-stress manipulation. Blood pressure will be assessed in terms of systolic BP, diastolic BP, and mean arterial pressure. Systolic indicates the time in which the heart is pumping where as diastolic refers to the time when the heart is relaxed or not pumping. Mean arterial pressure is calculated by combining one-third systolic BP and two-thirds diastolic BP.

Heart rate & pulse. The plethysmograph electrode was used to measure pulse and heart rate. This electrode was attached to the volar surface of the middle finger of the participant's non-dominant hand. Heart rate was recorded in beats per minute (bpm), and pulse was recorded in milivolts (iWorx, 2013). Dependent variables will include mean measurements of HR and

pulse. This data will be generated at 30 second intervals for the duration of the three minute sitting baseline, three minute standing baseline, both three minute speeches, and the seven minute post-stress manipulation. Physiological recording will be analyzed at 30 second intervals. However, in order to eliminate data that has been severely flawed by movement artifact, only the largest and most accurate piece of recording during that specific 30 seconds will be used in data analysis.

Electrodermal activity. The skin conductance level (SCL) equipment consists of two connected electrodes used to measure skin conductance. These electrodes were attached to the volar surface (where the fingerprints are located) on two non-adjacent fingers. In this study, the electrodes were attached to the participant's index and ring fingers of non-dominant hand (iWorx, 2013). Electrodermal measurements will include mean SCL. This data will be generated at 30 second intervals for the duration of the three minute sitting baseline, three minute standing baseline, both three minute speeches, and the seven minute post-stress manipulation. SCL measurement intervals will directly correlate with pulse and HR measurements so that movement artifact can be eliminated and intervals can stay consistent across variables.

Procedure

The procedure is further explained in Figure 2. Prior to the beginning of the study, participants were randomly assigned into one of two groups. Upon arrival to the experiment, participants were explained the consent form. Once informed consent was obtained, the baseline phase began.

Baseline. Participants were asked to fill out the demographic survey, State Trait Anxiety Inventory (STAI; Spielberger, 1985) for state anxiety, and Positive and Negative Affect Schedule (PANAS-SF; Watson, Clark, & Tellegan, 1988). Participants were instructed to fill out

all surveys as they felt in the present moment. Following this, the participant's blood pressure was taken. Next, the participants were asked to wash their hands with soap and water in order to ensure effective physiological recording. Next, the electrodes were hooked up to the participant's non-writing hand. Pedicure foam was placed between the participant's fingers in order to ensure the electrodes did not touch. GSR electrodes connected to the index and ring fingers, while the pletysmograph electrode connected to the middle finger. The participants were asked to not move their hand while resting it on a table. At this point the participants were sitting, and 3 minute sitting baseline was recorded. Next, the participants were asked to stand on the opposite side of the table. They were told to rest their hand on the desk or against their thigh in order to ensure most comfort and least amount of movement. A 3 minute standing baseline was recorded. The standing baseline was taken as participants will be standing for the Trier Social Stress Test.

Trier Social Stress Test. All participants underwent the Trier Social Stress Test (TSST). This is a validated measure which increases anxiety level (Kirshbaum, Pirke, & Hellhammer, 1993). This test consisted of two public speaking tasks. Participants were told they had two minutes to prepare for their speech without writing anything down, and that they had three minutes to deliver the speech. Participants were also told that their speech and other nonverbal behaviors were going to be rated by lab assistants wearing white lab coats, and that they were going to be video recorded.

Speech #1. The participants were then given their first prompt, "You must take on the role of a job applicant who was invited to a personal interview with the company's hiring manager. Introduce yourself and convince the manager why you are the perfect applicant for the vacant position." Following the two minute preparation period, a lab assistant wearing a white lab coat and holding a clipboard entered the room. At this time the camera light was turned on,

but no video was actually recorded. An egg timer was also set for three minutes. If participants stopped talking before the end of their three minutes, the lab assistant instructed them to continue. Following the first speech presentation, blood pressure was measured, and the STAI and PANAS were filled out.

Speech #2. Next, the second prompt was delivered with the same directions. The prompt stated, “You have just been caught and accused of stealing. Defend yourself and convince the police why you are innocent and should not be arrested.” All protocol used during the first speech was also implemented during the second. Following the second speech, blood pressure measure was recorded, and participants filled out STAI and PANAS.

Post-stress manipulation. The participants underwent activities dependent upon group assignment. Participants in the experimental group received a pre-drawn mandala and were instructed on a focused breathing technique (Linehan, 1993). In this focused breathing technique, participants were asked to breathe as evenly and gently as possible. They were asked to pay attention to their breath and the way their stomach and lungs were moving while still remaining aware of their coloring task. Participants had seven minutes to complete the activity, and were presented with crayons, markers, and colored pencils. Participants in the control group were asked to sit quietly for seven minutes, and not use their cell phone. The control group did not participate in coloring or meditation activities. A no-task control group was chosen in order to maximize differences between groups as a result of no differences found in a previous study which used an unstructured coloring group (Muthard & Williams, 2012). After the final phase ended, all participants again completed the STAI and PANAS and blood pressure was measured. Following the final measures, the experiment was complete and participants were debriefed and compensated.

Data Analysis

All data were analyzed according to procedural phase, and transition between phases. For procedural phase analysis, repeated measures ANOVAs and independent samples t-tests were used. Transitions between phases were analyzed using a paired samples t-test. Specifically, t-tests were used on self-report measurements and BP because these variables were only measured once following each phase.

Baseline

An independent t-test was used separately on STAI scores, PANAS positive scores, PANAS negative scores, systolic BP, diastolic BP, and mean arterial pressure in order to ensure there were no differences between groups. Furthermore, a repeated measures ANOVA was used separately on pulse, SCL, and HR means in order to assess change over time, time by group interaction, and differences between groups in each variable during baseline. It is important to note that only the measurements taken from the sitting baseline are used in all further analyses. The sitting baseline was chosen as opposed to the standing baseline because these data were most stable.

Baseline to Speeches

In order to assess transitions from baseline to speeches, paired samples t-tests were used to validate that the psychosocial stressor was effective in increasing self-report and physiological measures. Further, grand means of pulse, SCL, and HR were generated for the sitting baseline, speech 1, and speech 2 phases so paired samples t-tests could be conducted. Grand means were created by averaging the six time points of pulse, SCL, and HR measurements separately to achieve one grand mean measurement of these variables during baseline, speech 1, and speech 2. Furthermore, repeated measures ANOVAs were used on the six time points (thirty second

intervals) of pulse, SCL, and HR measurements taken during both speeches in order to assess change over time, time by group interactions, and differences between groups.

Post-Stress Manipulation

An independent t-test was used on STAI scores, PANAS positive scores, PANAS negative scores, systolic BP, diastolic BP, and mean arterial pressure along with grand means of pulse, SCL, and HR in order to assess differences between groups during the post-stress manipulation phase. Furthermore, repeated measures ANOVAs were used on the seven time points (one minute intervals) of pulse, SCL, and HR measurements taken during the post-stress manipulation in order to assess change over time, time by group interactions, and differences between groups. Paired-samples t-tests were used to assess differences between sitting baseline and post-stress manipulation phase. Paired samples t-tests were used on all dependent variables. For the pulse, SCL, and HR measurements, grand means were generated by averaging the seven time points of each variable to have one grand mean of pulse, SCL, and HR during the post-stress phase. Finally, repeated measures ANOVA were used on all variables to assess changes over, time by group interaction, and differences between groups from speech 1 to post-stress manipulation, and speech 2 to post-stress manipulation.

Results

Demographics

Chi Square analyses were completed on demographic variables as a randomization check to confirm no significant differences in composition of control and experimental group. Analyses confirmed no differences in frequency of gender, race, and graduation year, $\chi^2(1, N=37)=.116$, $p=.734$; $\chi^2(3, N=37)=2.441$, $p=.486$; $\chi^2(3, N=37)=.983$, $p=.805$. Furthermore, an independent

samples t-test revealed that there were no differences in mean age between groups, $t(35)=.532$, $p=.598$ (Demographic variables are listed in Table 1).

Baseline

Self-reported affect. Independent samples t-tests were conducted on self-report measures to ensure that there were no group differences at baseline. There are no significant differences in mean state anxiety, positive affect, or negative affect between groups, $t(35)=-.271$, $p=.788$; $t(35)=.469$, $p=.642$; $t(35)=.860$, $p=.396$.

Blood Pressure. Independent t-tests were used to confirm that there were no significant group differences in blood pressure measures at the baseline phase. There were no difference in mean systolic blood pressure, mean diastolic blood pressure, or mean arterial pressure between groups, $t(34)=-.410$, $p=.684$; $t(34)=-.544$, $p=.590$; $t(35)=-1.125$, $p=.268$.

Physiological measures. A repeated measures ANOVA was used to assess the six time points (30 second intervals) of pulse, SCL and HR during the sitting baseline phase to analyze change over time and differences in change across time by group. Between subjects analyses revealed no significant differences in mean pulse between groups during baseline, $F(1,35)=.323$, $p=.573$. Within-subjects analyses revealed that there was a decrease in mean pulse measurements (millivolts) across time, $F(5,175)=4.404$, $p=.001$. However, the time by group interaction was not significant suggesting that both groups changed similarly across time, $F(5,175)=.523$, $p=.759$ (see Figure 3). Between subjects effects indicated no significant difference between groups of mean SCL during baseline, $F(1,35)=1.071$, $p=.308$. Within-subjects effects revealed that there were no significant changes across time of baseline, and no group by time interactions in mean SCL measurements (microsiemens), $F(5,175)=.633$, $p=.675$; $F(5,175)=.578$, $p=.717$ (see Figure 4). Between subjects effects indicated no differences between groups in mean HR during

baseline, $F(1,35)=1.894$, $p=.178$. Within-subjects effects revealed a significant increase in mean HR measurements (beats per minutes) across time, $F(5,175)=2.775$, $p=.019$. However, there were no time by group interactions in mean HR indicating that the groups changed similarly across time, $F(5, 175)=1.393$, $p=.229$ (see Figure 5). In summary, analyses indicated a general decrease in pulse and increase in HR during baseline, but the groups changed similarly across time. Further, SCL did not change much over baseline, with no groups also moving similarly.

Transition to Speech 1

Self-reported affect. A paired samples t-test was used to confirm that participant's anxiety level significantly increased in transition from baseline to speech 1 as a result of the psychosocial stressor. Overall, there was a significant increase in mean state anxiety for all participants from baseline to after the first speech 1, $t(34)=-6.121$, $p=.001$. Mean state anxiety scores increased from 32.17 (SD=7.55) during baseline to 41.11 (SD=11.43) following the first speech. There was a significant decrease in mean positive affect scores from baseline to the first speech, $t(34)=4.4586$, $p=.001$. Mean positive affect scores decreased from 30.94 (SD=6.76) during baseline to 27.57 (SD=6.80) following the first speech. There was a significant increase in mean negative affect scores from baseline to the first speech, $t(34)=-2.639$, $p=.012$. Mean negative affect scores increased from 12.26 (SD=2.69) during baseline to 15.17 (SD=2.63) following the first speech.

Blood pressure. Paired samples t-tests were used to analyze blood pressure changes in transition from baseline to after the first speech as a result of the psychosocial stressor. There was a significant increase in diastolic BP of all participants from baseline to after the first speech, $t(33)=-10.015$, $p=.001$. Mean diastolic BP increased from 67.76 mmHg (SD=6.75) during baseline to 77.44 mmHg (SD=6.75) following the first speech. However, there were no

differences in systolic BP or mean arterial pressure from baseline to after the first speech, $t(33)=-1.023, p=3.14$; $t(36)=-.467, p=.643$.

Physiological measures. A grand mean was calculated for pulse, SCL and HR variables for sitting baseline and speech 1 measurements. These grand means were analyzed using paired samples t-tests to indicated changes in pulse, SCL, and HR in all participants from baseline through speech 1. There was a significant decrease in pulse measurements from sitting baseline to after the first speech, $t(36)=3.269, p=.002$. Mean pulse decreased from .01 millivolts ($SD=.005$) during baseline to .003 millivolts ($SD=.01$) following the first speech. There was a significant increase in SCL from sitting baseline to after the first speech, $t(36)=-6.531, p=.001$. Mean SCL increased from 2.73 microsiemens ($SD=1.81$) to 4.66 microsiemens ($SD=3.00$) following the first speech. There was a significant increase in HR from sitting baseline to after the first speech, $t(36)=-9.558, p=.001$. Mean HR increased from 80.5 bpm ($SD=14.19$) during baseline to 105.35 bpm ($SD=14.95$) following the first speech. In summary, this demonstrates that pulse counter intuitively decreased from baseline to speech 1, but SCL and HR increased from baseline to speech 1 as anticipated.

Procedural Phase of Speech 1

A repeated measures ANOVA was used on the six time points (thirty second intervals) of pulse, SCL, and HR to verify that there were no differences across time or any time by group interactions during speech 1. Between-subjects effects revealed that the experimental group had a significantly higher pulse than controls during speech 1, $F(1,35)=6.366, p=.016$. Within-subjects effects revealed no significant differences in mean pulse measurements across time along with no significant time by group interactions suggesting the groups moved similarly, $F(5,175)=.504, p=.773$; $F(5,175)=.835, p=.527$ (see Figure 6). Between-subjects analyses

confirmed that there were no group differences in SCL during speech 1, $F(1,35)=.098, p=.759$. Within-subjects effects confirmed that there was a significant decrease in mean SCL across time, but there was no significant group by time interaction, $F(5,175)=14.638, p=.001; F(5,175)=.837, p=.525$ (see Figure 7). Between-subjects effects confirmed no group differences in HR during speech 1, $F(1,35)=.995, p=.325$. Within-subjects effects also confirmed no significant differences across time or any time by group interactions in HR measurements during speech 1, $F(5,75)=1.633, p=.153; F(5,175)=1.114, p=.355$ (see Figure 8). In summary, other than pulse, SCL and HR measurements held fairly constant, and groups moved similarly and were not different during speech 1.

Transition to Speech 2

Self-reported affect. Paired samples t-tests were used to confirm that all subject's stress levels increased in transition from baseline to following the second speech as a result of the TSST. There was a significant increase in mean state anxiety from baseline until after the second speech, $t(36)=-5.062, p=.001$. Mean state anxiety scores increased from 32.54 (SD=7.5) during baseline to 40.43 (SD=11.37) following the second speech. There was a significant decrease in positive affect from baseline through the second speech, $t(36)=5.058, p=.001$. Mean positive affect scores decreased from 31.03 (SD=6.58) during baseline to 26.59 (SD=6.98) following the second speech. There was a significant increase in negative affect from baseline through the second speech among all participants, $t(36)=-2.812, p=.008$. Mean negative affect scores increased from 12.21 (SD=2.63) during baseline to 14.62 (SD=5.60) following the second speech. In summary, results indicate that participants increased in state anxiety and negative affect, and decreased in positive affect from baseline to speech 2.

Blood pressure. Paired samples t-tests were used on blood pressure measures to analyze changes from baseline to speech 2 in all participants. There were no differences in systolic blood pressure between baselines and speech 2 measures, $t(35)=-.836, p=.409$. There was a significant increase from baseline to speech 2 in diastolic BP measures, $t(35)=-9.664, p=.001$. Mean diastolic BP increased from 68.03 mmHg (SD=8.8) during baseline to 77.75 mmHg (SD=7.73) following the second speech. There was a significant increase in average mean arterial pressure from baseline to after the second speech, $t(36)=-8.347, p=.001$. Average mean arterial pressure increased from 82.67 mmHg (SD=16.72) during baseline to 89.49 mmHg (SD=17.12) following the second speech.

Physiological measures. Grand means were calculated for pulse, SCL, and HR variables of measurements from the sitting baseline and from speech 2. These grand means were used in paired samples t-tests to analyze the transition from sitting baseline through the second speech. There was a significant decrease in grand mean pulse measurements from the sitting baseline to the end of the second speech which may have been the result of an outlier, $t(36)=2.435, p=.020$. There was a significant increase in grand mean SCL measurements from sitting baseline through the second speech, $t(36)=-7.875, p=.001$. There was a significant increase in grand mean HR measurements from the sitting baseline through the second speech, $t(36)=-10.224, p=.001$.

Procedural Phase of Speech 2

Furthermore, repeated measures ANOVA were used on the six pulse, SCL, and HR time points (thirty second intervals) to confirm that groups changed similarly across time during speech 2. Between subjects analysis confirmed no groups differences in pulse during speech 2, $F(1,35)=1.6, p=.214$. Within-subjects effects revealed no significant differences in pulse measurements across time and no significant time by group interactions, $F(5,175)=1.360$,

$p=.242$; $F(5,175)=1.409$, $p=.202$ (see Figure 9). Between-subjects analyses confirmed no group differences in SCL during speech 2, $F(1,35)=.571$, $p=.455$. Within-subjects effects revealed a significant decrease across time in SCL during speech 2, $F(5,175)=14.137$, $p=.001$. However, there was no group by time interaction in mean SCL during speech 2 suggesting that groups changed similarly across time, $F(5,175)=.725$, $p=.605$ (see Figure 10). Between-subjects confirmed no significant differences between groups in HR during speech 2, $F(1,35)=.814$, $p=.373$. Within-subjects effects revealed that there were no significant differences across time or any time by group interactions in mean HR measurements during speech 2, $F(5,175)=1.730$, $p=.130$; $F(5,175)=.197$, $p=.963$ (see Figure 11). In summary, SCL decreased during speech 2, whereas pulse and HR held fairly stable. Most importantly, in pulse, SCL, and HR groups moved similarly across time.

Post Stress

Self-reported affect. It is important to note that self-reported measures were only administered once during the post-stress phase which occurred at the end of the seven minute post-stress manipulation. Independent samples t-tests were used to assess differences between experimental and controls groups during the seven-minute post-stress manipulation phase. There was a trend toward significance in mean STAI score between groups, $t(35)=-1.762$, $p=.087$ (see Figure 12). Mean state anxiety of the experimental group was 30.37 (SD=7.31), and where as mean state anxiety of the control group was 35.17 (SD=9.19). There were no significant differences in mean positive affect between groups, $t(35)=1.441$, $p=.159$ (see Figure 13). These independent samples t-tests also revealed a significant difference in mean negative affect between groups, $t(35)=-2.027$, $p=.050$ (see Figure 14). Mean negative affect scores for the

experimental group were 11.05 (SD=1.87) where as mean negative affect scores for the control group were 12.94 (SD=3.59).

Blood pressure. Independent t-tests were used to analyze differences in blood pressure measurements between experiment and control groups during the post-stress manipulation. There were no significant differences in mean systolic BP, diastolic BP, or mean arterial pressure between groups, $t(34)=-1.472, p=.150$; $t(34)=-.772, p=.445$; $t(35)=-1.473, p=.150$ (see Figures 15, 16, and 17).

Physiological measures. Furthermore, an independent t-test was used on grand means created for pulse, SCL, and HR measures throughout the post-stress manipulation phase. Analyses indicate no significant differences in pulse, SCL, and HR between groups, $t(35)=-.855, p=.398$; $t(35)=1.320, p=.195$; $t(35)=.127, p=.900$ (see Figures 18, 19, 20).

Repeated measures ANOVA were used on seven time points (one minute intervals) of pulse, SCL, and HR measures taken during the post-stress manipulation phase to reveal differences between experimental and control group across time. It is important to note that post stress data were collapsed into one minute intervals (as opposed to thirty seconds) to make analyses more manageable. Between-subjects analyses revealed no group differences in pulse measurements during post-stress manipulation, $F(1,35)=.731, p=.398$. Within-subjects effects reveal no significant differences in mean pulse across time, and no significant group by time interactions suggesting that groups moved similarly across time, $F(6,210)=.979, p=.440$; $F(6,210)=1.606, p=.147$ (see Figure 21). Between-subjects effects revealed no group differences in SCL during post-stress manipulation, $F(1,35)=1.743, p=.195$. Within-subjects analyses on mean SCL indicated a decrease across time during the post-stress manipulation, $F(6,210)=2.466, p=.025$. However, there was no group by time interaction indicating that the groups moved in the

same direction, $F(6,210)=.991, p=.432$ (see Figure 22). Between-subjects analyses indicated there were no significant differences between groups of mean HR during post-stress manipulation, $F(1,35)=.016, p=.900$. Within-subjects effects of the repeated measures revealed no significant difference across time, but there was a group by time interaction in mean HR between groups during the post-stress manipulation, $F(6,210)=.617, p=.717$; $F(6,210)=3.797, p=.001$ (see Figure 23). Post hoc independent t-tests reveal that both the control and experimental group have a marginally significant change from the 0 second to 1 minute time point to the 2 minute to 3 minute time point, $t(17)=1.899, p=.075$; $t(18)=-1.908, p=.072$ (see Figure 23). The means indicate that the control group's HR decreased from 85.02 (SD=13.47) to 82.83 (SD=13.39) while the experimental group's mean HR increased from 79.97 (SD=9.99) to 83.47 (SD=11.83) suggesting that these changes can account for most of the time by group interaction (see Figure 23). Yet it is important to mention that the mean of the experimental group was higher than the control group during the last five minutes of the post-stress manipulation, $M=83.76, SD=10.86$; $M=81.74, SD=14.32$.

Change Over Baseline, Speech 1, Speech 2, and Post-Stress Manipulation

A repeated measures ANOVA was conducted on all dependent variables to assess change over time throughout all four phases (baseline, speech 1, speech 2, post-stress manipulation) of the experiment. This also acts as a manipulation check in order to verify that stress and anxiety increased as a result of the psychosocial stressor.

Self-Reported Affect

Between-subjects analyses on state anxiety, positive affect, and negative affect indicated no differences between group, $F(1,33)=.261, p=.613$; $F(1,33)=.312, p=.580$; $F(1,33)=.014, p=.907$. Within-subjects analyses on self-reported affect indicate a significant change across the

entire experiment for state anxiety, positive affect, and negative affect, $F(3,99)=23.702, p=.001$; $F(3,99)=10.326, p=.001$; $F(3,99)=6.819, p=.001$ (See Figures 24, 25, and 26). There was a time by group interaction only for positive affect, $F(3,99)=2.857, p=.041$. There were no time by group interactions for state anxiety or negative affect, $F(3,99)=1.408, p=.245$; $F(3,99)=1.452, p=.232$. Post hoc analyses indicated that for state anxiety and negative affect measurements increased from baseline to speech 1, did not change between speeches, and decreased from speech 2 to post-stress, $t(34)=-6.121, p<.05$; $t(34)=.500, p>.05$; $t(36)=5.450, p<.05$; $t(34)=-2.639, p<.05$; $t(34)=.849, p>.05$; $t(36)=3.334, p<.05$. Post hoc analyses also indicated that positive affect decreased from baseline to speech 1, did not change between speeches, and increased from speech 2 to post-stress, $t(34)=4.9, p<.05$; $t(34)=1.708, p>.05$, $t(36)=-1.957, p<.05$.

Blood Pressure

Between-subjects analyses reveal that there were no significant differences between groups for systolic BP, diastolic BP, and mean arterial pressure, $F(1,32)=1.266, p=.269$; $F(1,32)=.069, p=.794$; $F(1,35)=1.625, p=.211$. Within-subjects analyses indicated a significant change over time for systolic BP, diastolic BP, and mean arterial pressure, $F(3,96)=6.435, p=.001$; $F(3,96)=76.013, p=.001$; $F(3,105)=3.393, p=.021$ (See Figures 27, 28, and 29). Furthermore, there was no time by group interaction for any of the blood pressure variables $F(3,96)=.836, p=.477$; $F(3,96)=.546, p=.652$; $F(3,105)=.028, p=.994$. Post hoc analysis on diastolic BP indicated that diastolic BP significantly increased from baseline to speech 1, did not change between the speeches, and significantly decreased from speech 2 to post-stress, $t(33)=-10.015, p<.05$; $t(33)=-.035, p>.05$; $t(35)=11.925, p<.05$. However, post hoc analyses indicated that systolic BP and mean arterial pressure did not change from baseline to speech 1, or speech 1 to speech 2, but did decrease from speech 2 to post-stress, $t(33)=-1.023, p>.05$; $t(33)=-.312,$

$p > .05$; $t(35)=4.113$, $p > .05$; $t(36)=-.467$, $p > .05$; $t(36)=-1.362$, $p > .05$; $t(36)=11.282$, $p < .05$. This suggests that the psychosocial stressor may have not been effective at increasing systolic BP or mean arterial pressure.

Physiological Measures

Between subjects analyses reveal a trend towards difference between groups in mean pulse, but there were no group differences in SCL or HR, $F(1,35)=3.799$, $p=.059$; $F(1,35)=.720$, $p=.402$; $F(1,35)=.039$, $p=.844$. Within-subjects analyses also indicated significant change across time for pulse, SCL, and HR variables, $F(3,99)=3.725$, $p=.014$; $F(3,105)=42.985$, $p=.001$; $F(3,105)=89.770$, $p=.001$ (See Figures 30, 31, and 32). There was a time by group interaction only for HR measurements, $F(3,105)=2.863$, $p=.040$. There were no time by group interactions for pulse or SCL, $F(3,105)=1.765$, $p=.158$; $F(3,105)=1.366$, $p=.257$. Furthermore, post hoc analyses indicated that pulse significantly decreased from baseline to speech 1 and between speeches, but did not change from speech 2 to post-stress, $t(36)=-9.455$, $p < .05$; $t(36)=9.480$, $p < .05$; $t(36)=-1.354$, $p > .05$. These results may have been affected by an outlier. Further, post hoc analyses revealed that SCL significantly increased from baseline to speech 1, did not change between speeches and from speech 2 to post-stress, $t(36)=-6.531$, $p < .05$; $t(36)=-1.926$, $p > .05$; $t(36)=1.246$, $p > .05$. Lastly, post-hoc analyses indicated that HR significantly increased from baseline to speech 1, did not change between speeches, and significantly decreased from speech 2 to baseline, $t(36)=-9.558$, $p < .05$; $t(36)=-1.139$, $p > .05$; $t(36)=10.420$, $p < .05$. In summary, these analyses indicate that all dependent variables changed over time as a result of experimental manipulation with the exception of systolic BP, mean arterial pressure, pulse not increasing due to the psychosocial stressor.

Discussion

The purpose of this experiment was to assess the effectiveness of mandala-coloring as a potential art therapy technique in conjunction with the already validated mindfulness technique of focused breathing. By linking art therapy and mindfulness techniques, this study seeks to bring some validation to the art making process, especially coloring, as an effective tool to help reduce stress and anxiety. It was hypothesized that self-reported affect and physiological stress would change over time as a result of experimental manipulation which included the psychosocial stressor. The primary hypothesis of this research was that mandala-coloring paired with focused breathing techniques in the experimental group would reduce stress greater than the control group.

It is first important to conclude that the TSST effectively worked as a psychosocial stressor in this experiment, which supports the hypothesis that self-reported affect and physiological stress would change over time. Results indicated that from baseline to both speech 1 and speech 2 independently, all self-reports changed in the proper direction to indicate that participants experienced psychosocial stress. Specifically, state anxiety and negative affect scores increased during as a result of the TSST, while positive affect scores decreased. In analyzing the effect of the TSST on blood pressure, results indicated that diastolic BP increased from baseline to both speeches, and mean arterial pressure increased from baseline to speech 2. However, there were no changes in systolic BP, which is consistent with the literature as diastolic BP is more susceptible to stress (Stern et al, 2001). Furthermore, HR and SCL both significantly increased from baseline to both speeches demonstrating that the psychosocial stressors worked effectively. However, pulse decreased from baseline to both speeches which may have been a result of overall variability in pulse measurements during the psychosocial

stressor. In this case, it is important to note that there is much heterogeneity of the stress response, and that there are individual differences in responses (Kudielka, Hellhammer, & Wust, 2009; Stern et al, 2001). It is also important to note that the variability may have been a result of the integrity of equipment functioning. The findings that the TSST significantly increased stress are consistent with other research that demonstrated the same trend in both self-reported and physiological measures of stress (Kirschbaum et al., 1993; Kelly et al., 2008; Campisi, Bravo, Cole, & Gobeil, 2012). Overall, these conclusions support the hypothesis that stress would change over time due to the psychosocial stressor.

From the results, it is possible to conclude the findings regarding mandala-coloring and focused breathing are mixed, which lends partial support to the hypothesis that mandala-coloring would reduce stress greater than a control. However, it is important to note that it cannot be determined whether mandala-coloring or focused breathing individually contributed to the reductions since they were used together. Self-reports, especially negative affect and state anxiety demonstrate a trend toward stress reduction in the experimental group in the predicted direction. These results are consistent with research that has also demonstrated a decrease in state anxiety after mandala-coloring (Curry & Kasser, 2005; Bell & Robbins, 2007; van der Vennet, & Serice, 2012). At this point, there is not enough consistent literature to explain the underlying mechanisms as why mandala-coloring seems to be most effective when measured by self-reports.

However, physiological measures did not demonstrate any differences between groups, thus not supporting the primary hypothesis. This suggests that there was a disconnect between self-reported affect and physiological measures. A very common model of anxiety proposes that anxiety functions on a three-way systems model which include physiological, behavioral, and cognitive anxiety (Lang, 1978). Therefore, each aspect has its own individual response system

that can help to register different aspects of anxiety as a whole (Wilhelm & Roth, 2001). For example, the behavioral system might register avoidance whereas the physiological system may physically increase heart rate. Viewing anxiety as a systems model justifies why this study found effects in self-reported affect, but not in physiological measures. The Cannon-Bard Theory of Emotion (1927) further explains these results by indicating that autonomic arousal and conscious emotions are separate systems which do not result from one another. Therefore, the finding that differences were found only in self-reported affect can be explained by the literature.

Furthermore, there was a slight trend that moved in the opposite direction than predicted. SCL levels during the entire post-stress manipulation, and HR measurements during the latter portion of the post-stress manipulation were actually higher in the experimental group than the control group which would suggest that the control group was more physiologically aroused. There are two reasons that might explain this trend of HR and SCL moving in the opposite direction than anticipated in the experimental group. One possibility is that the experimental group had a task to complete compared to the control group which had no task. The presence of the task itself may be more arousing than sitting with no task. Initially, a no-task control group was incorporated into the study design as it was thought to maximize differences between control and experimental groups. Secondly, there is some research that suggests that mindfulness techniques actually increase arousal as opposed to decreasing arousal. One of the fundamental components of mindfulness is that it requires an attention that is characterized by observing one's moment to moment experiences (Carmody, Baer, Lykins, & Olendzki, 2009). Therefore, if mindfulness requires attentional processes this may explain why the experimental group's physiological arousal was higher. Other studies conclude that heart rate during meditational activities also causes higher arousal (Peng et al., 1999; Jevning, Wallace, & Beidbach, 1992).

Overall, even though these findings are not consistent with the hypothesis of this study, there is literature that supports that mediation creates an attentive state which may cause increases in physiological measures.

One limitation of this study was that the sample was predominately female. This could have contributed to the lack of differences found in physiological measures between groups as expected. Research supports that females may be more likely to respond to the tend and befriend model rather than the fight or flight model (Taylor, 2000). The tend and befriend model states that some stressors may cause a release of oxytocin which might prompt females to respond with affiliative behaviors like protecting offspring or seeking other's social support for protection (Taylor, 2000). It is important to note that females still experience SNS arousal, yet the male fight or flight response may be heightened by the presence of androgens (Taylor, 2000). Since affiliative responses were not measured in this study, it is impossible to determine whether or not these responses were present. Yet, if the predominantly female sample did respond to the stressor in a more social way, then mandala-coloring and focused breathing would probably not have an impact on these social responses which may explain why no differences were detected. There is also a body of research that suggests that females tend to be more expressive in their response to stress. One research study concluded that women were more expressive about both positive and negative emotions than men (Kring & Gordon, 1998). Due to the fact that the current study was predominately women, it helps explain why results were only significant in self-reported affect.

Another limitation regarding demographics was that the sample consisted of a very narrow age range. Thus, the results from this study are constrained to young adults, and may not apply to wider audiences of varied ages such as very young or very old populations. Thirdly, this study was limited because factors that could affect stress response such as sleep deprivation,

caffeine use, and exercise were not measured. However, these factors were controlled for as a result of random assignment to groups. Lastly, this research was limited because the participants were not experienced in focused breathing or mindfulness techniques. Therefore, it cannot be guaranteed that participants were practicing the focused breathing exercises as instructed.

In conclusion, this study found limited support for the effectiveness of the combination of mindfulness techniques with art therapy in reducing anxiety as compared to controls. Yet it is important to note, that because focused breathing and mandala-coloring were completed at the same time it is not possible to conclude whether one technique led to the self-reported stress reduction more than the other. Combining lesser validated art therapy color techniques with relatively highly studied mindfulness techniques could potentially help improve the validation of art therapy techniques, giving practitioners additional therapeutic techniques to use with clients. Further research will need to be completed in order to completely understand whether mandala-coloring can effectively reduce stress and anxiety.

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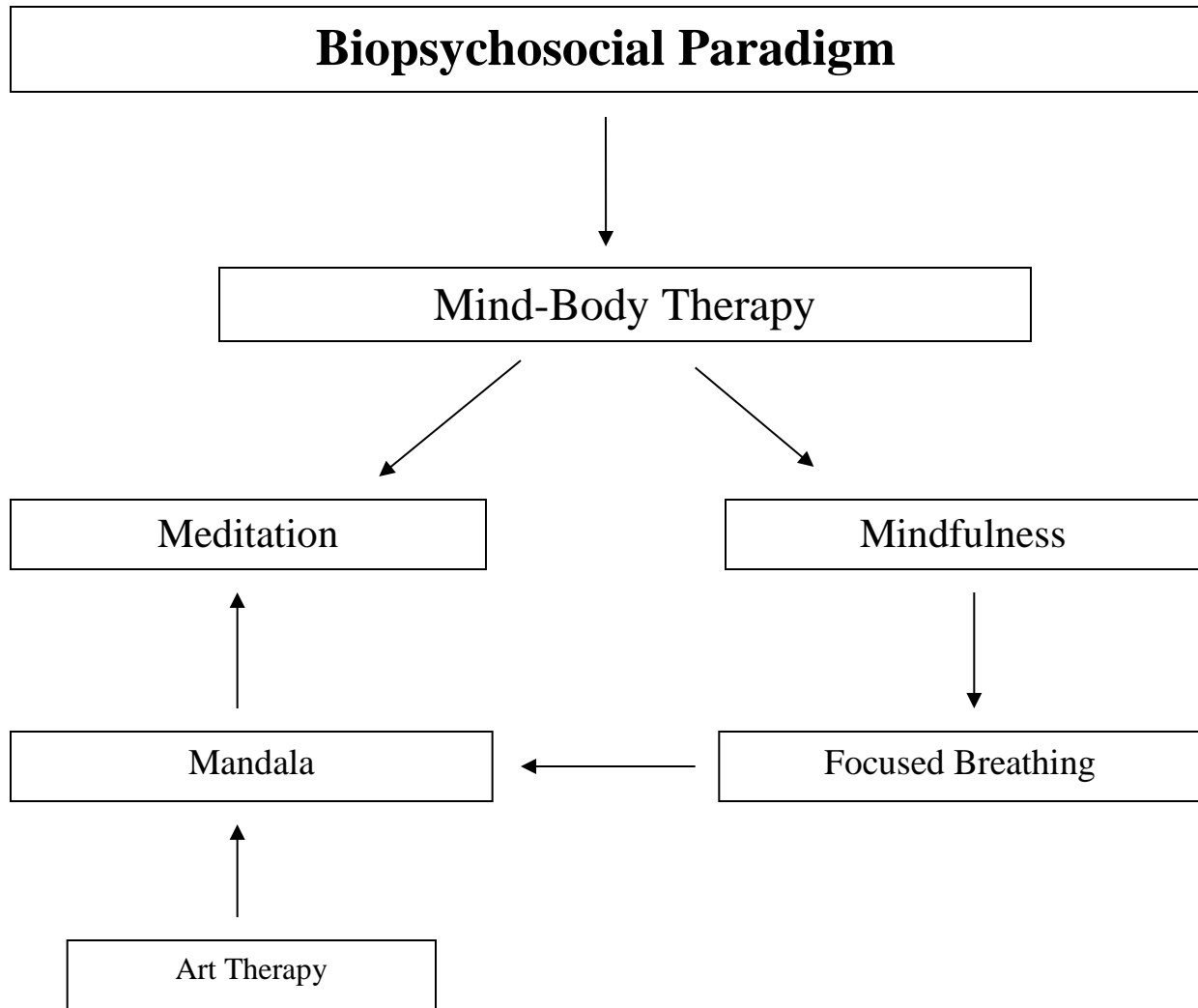


Figure 1: Relationship between theoretical perspectives addressed in this research.

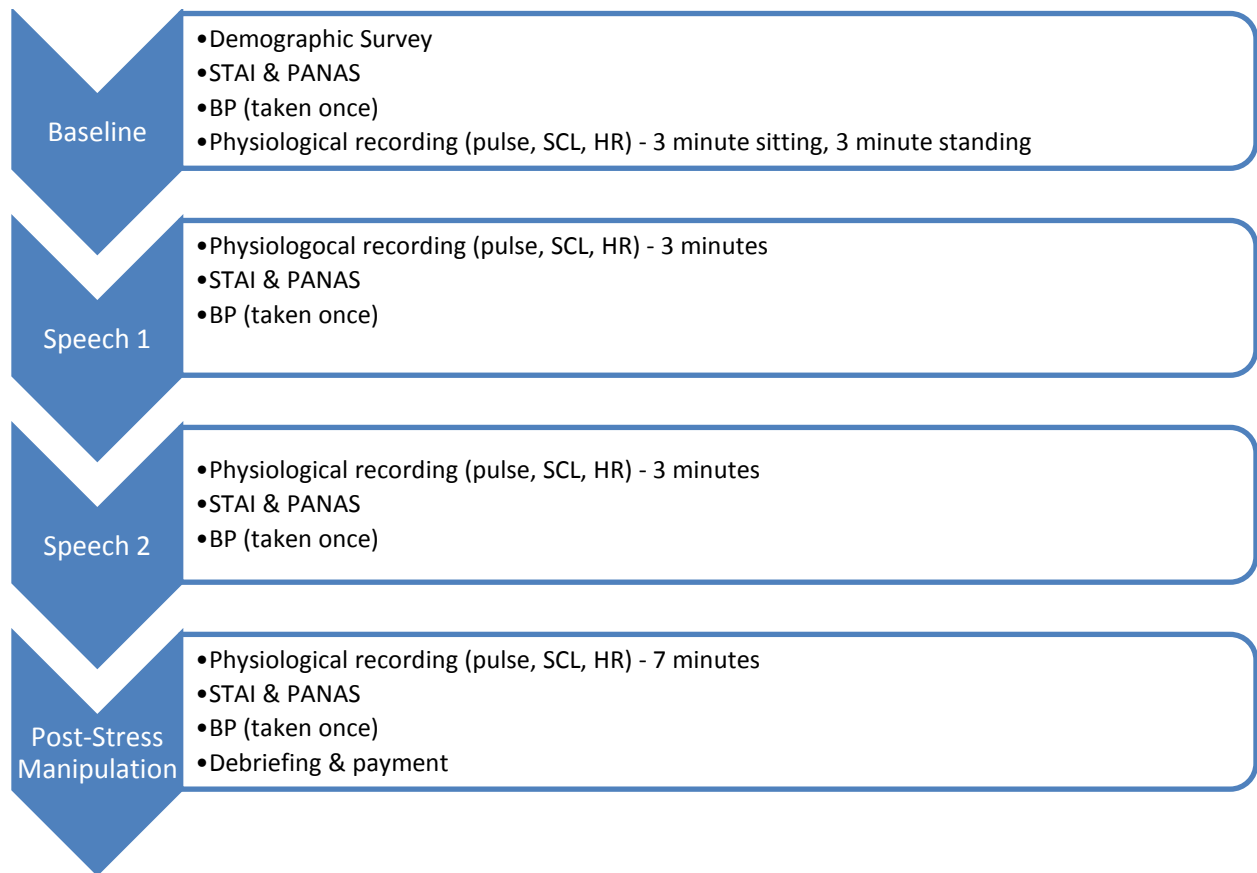


Figure 2: Sequential progression of procedure.

(N=37)	Overall	Experimental	Control
Age in years M (SD)	19 (1.22)	19(1.24)	18(1.23)
Gender N (%)			
Female	30 (81%)	15 (79%)	15 (83%)
Male	7 (19%)	4 (21%)	3 (17%)
Race N (%)			
Caucasian	30 (81%)	14 (74%)	16 (89%)
Asian	3 (8%)	2 (11%)	1 (6%)
African American	2 (5%)	2 (11%)	0 (0%)
Hispanic	2 (5%)	1 (5%)	1 (6%)
Graduation Year N(%)			
2014	7 (19%)	4 (21%)	3 (17%)
2015	6 (16%)	2 (11%)	4 (22%)
2016	4 (11%)	2 (11%)	2 (11%)
2017	20 (54%)	11 (58%)	9 (50%)

Table 1. Demographics table.

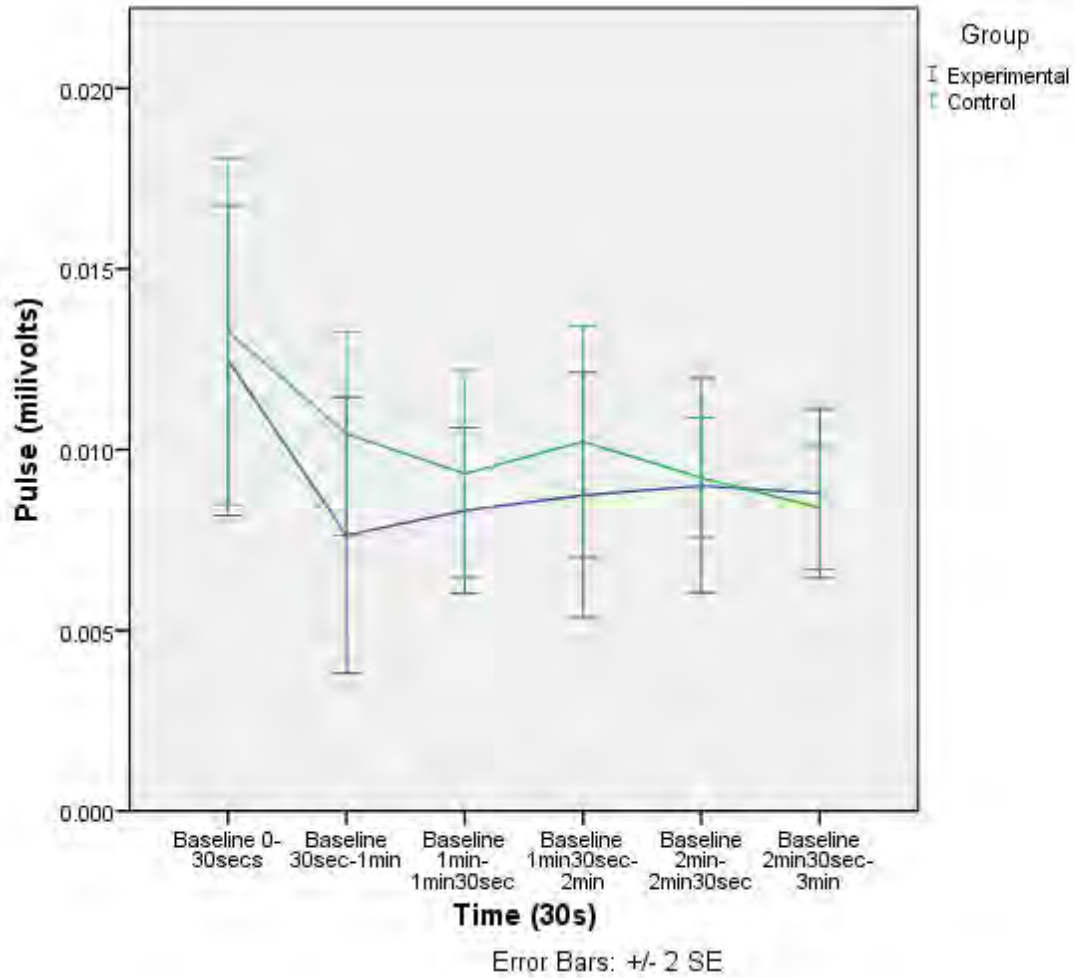


Figure 3. Pulse during baseline. Pulse measurements decreased over time during the sitting baseline, but there were no time by group interactions, $F(5,175)=4.404, p=.001$; $F(5,175)=.523, p=.759$.

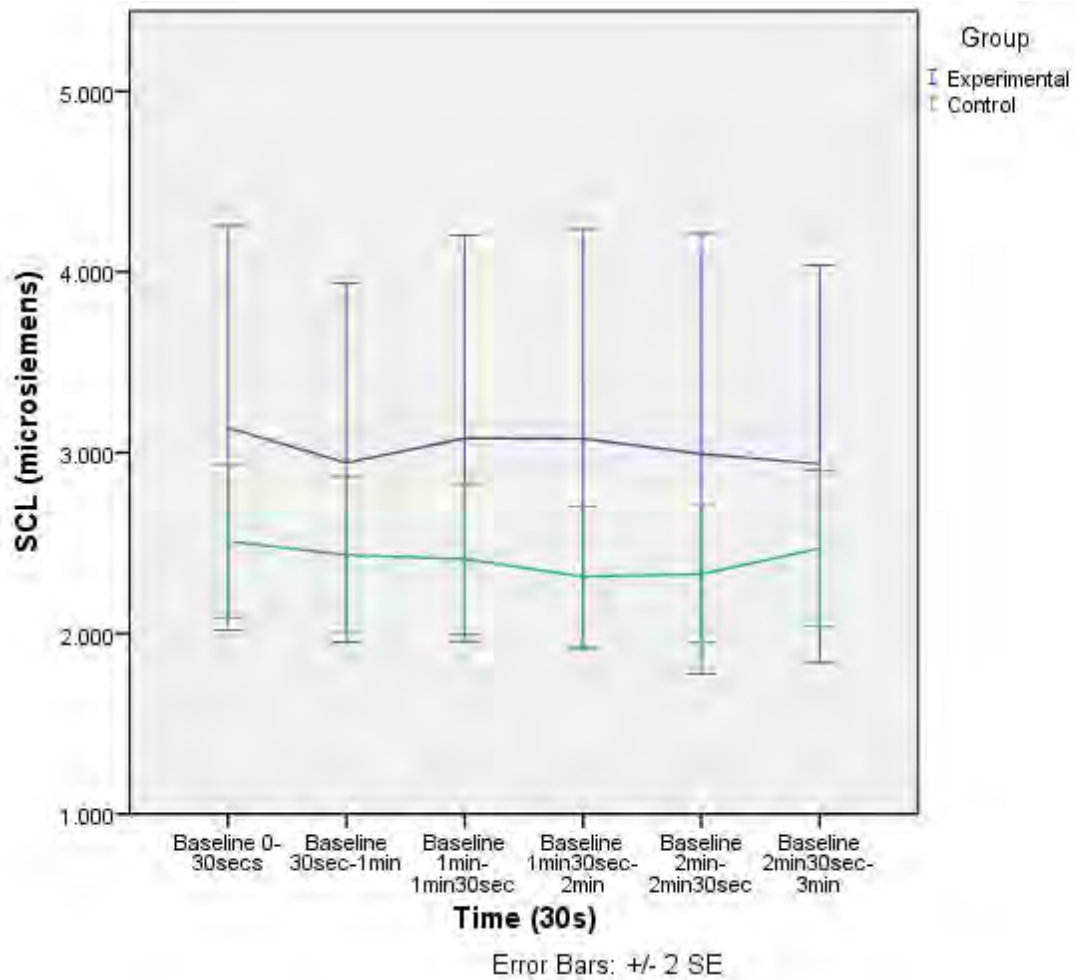


Figure 4. SCL during baseline. There were no significant changes in SCL across time, and there were no time by group interactions, $F(5,175)=.633, p=.675$; $F(5,175)=.578, p=.717$.

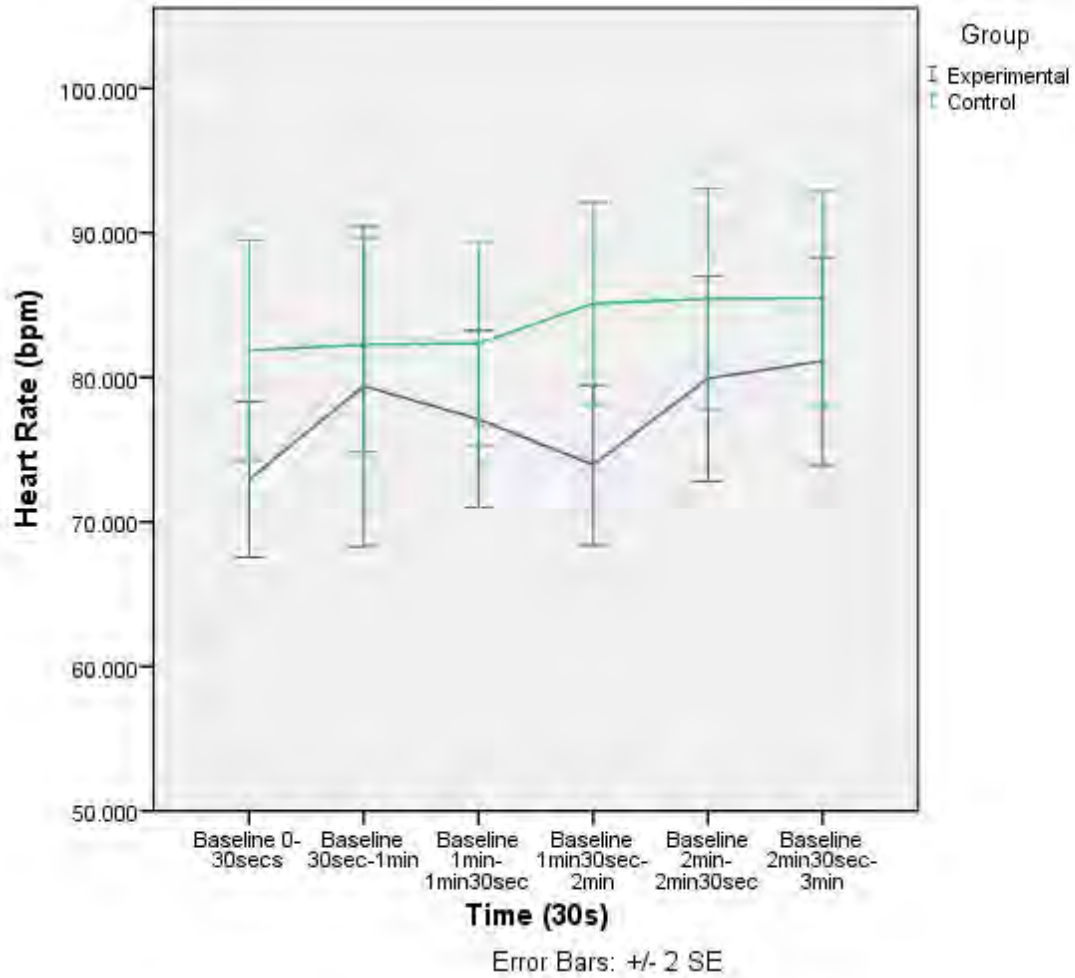


Figure 5. HR during baseline. HR increased across time, but there were no time by group interactions, $F(5,175)=2.775, p=.019$; $F(5, 175)=1.393, p=.229$.

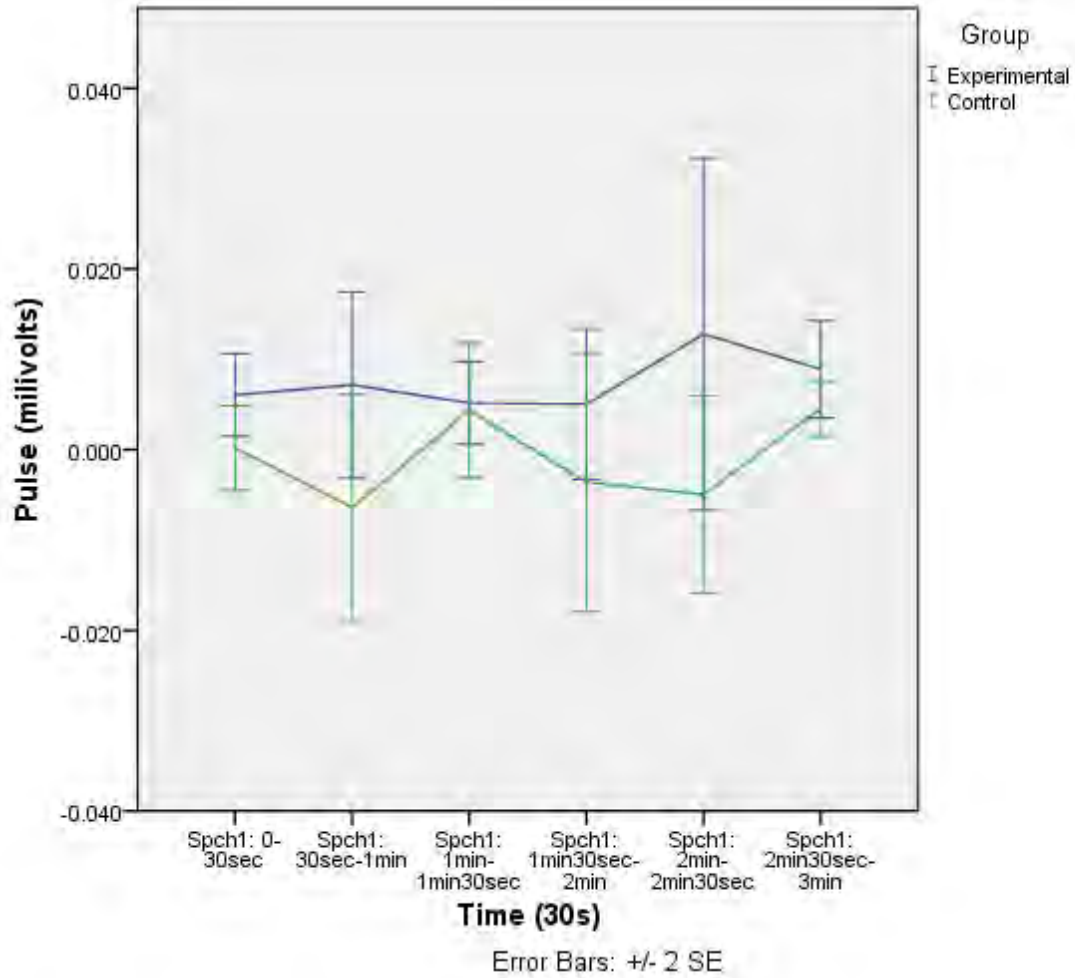


Figure 6. Pulse during speech 1. There were no significant differences across time, and no time by group interactions during the first speech of the TSST, $F(5,175)=.504, p=.773$; $F(5,175)=.835, p=.527$.

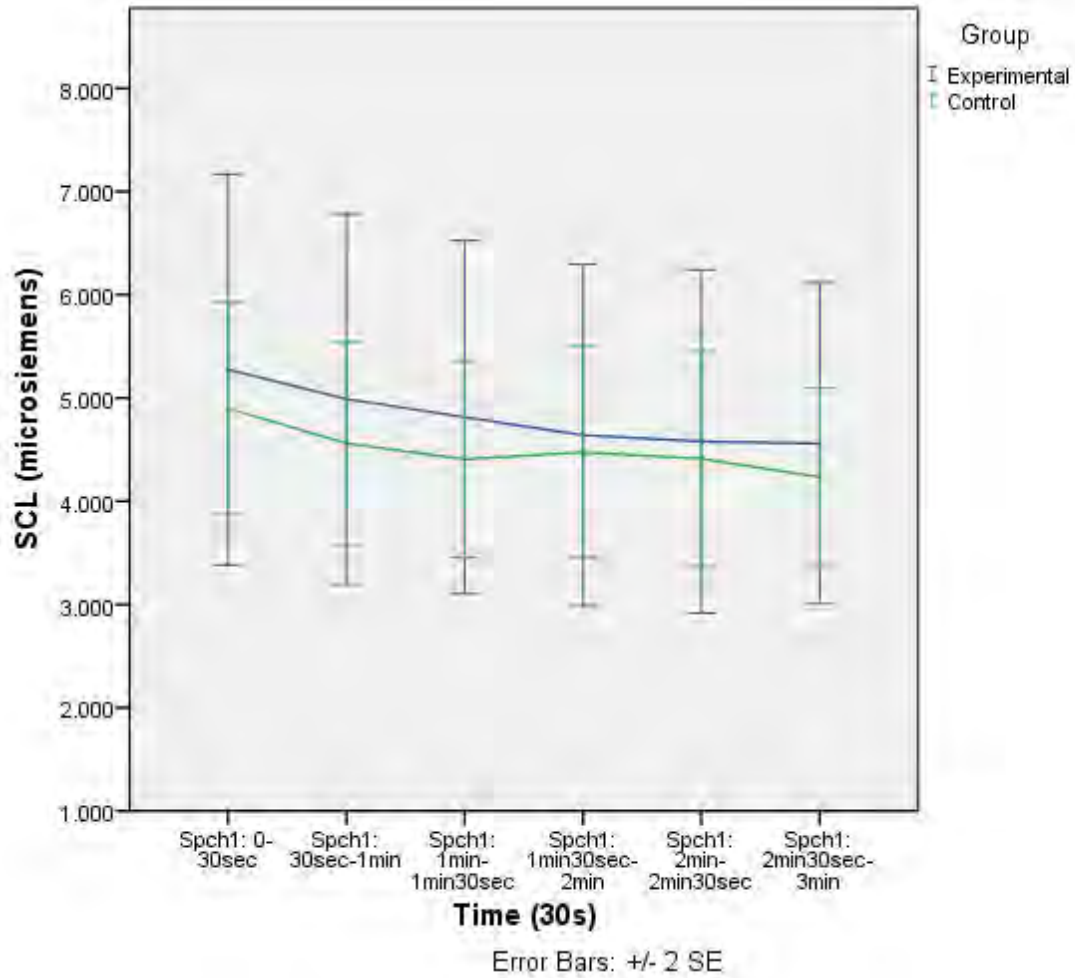


Figure 7. SCL during speech 1. There was significant decrease in mean SCL across time, but there were no group by time interactions, $F(5,175)=14.638, p=.001$; $F(5,175)=.837, p=.525$.

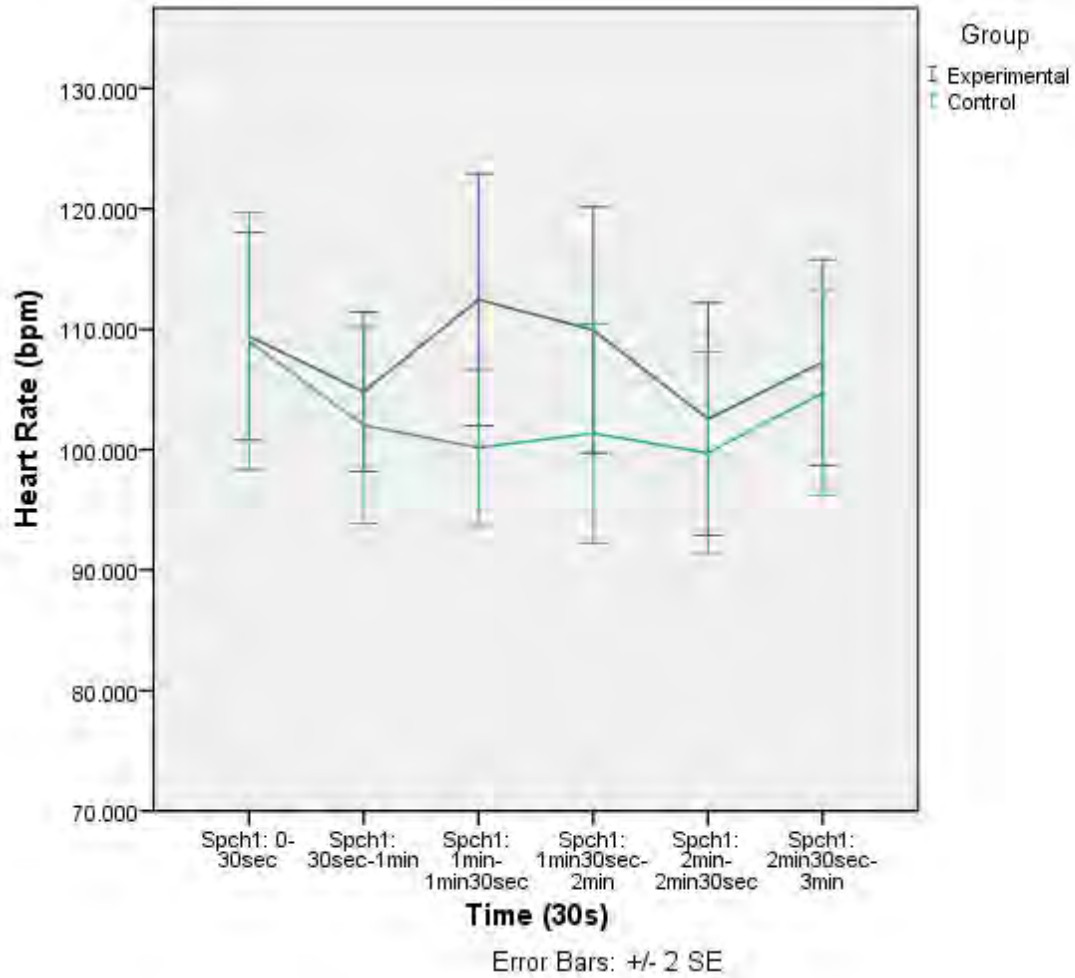


Figure 8. HR during speech 1. There were no differences in mean HR across time or any time by group interactions during speech 1, $F(5,75)=1.633, p=.153$; $F(5,175)=1.114, p=.355$.

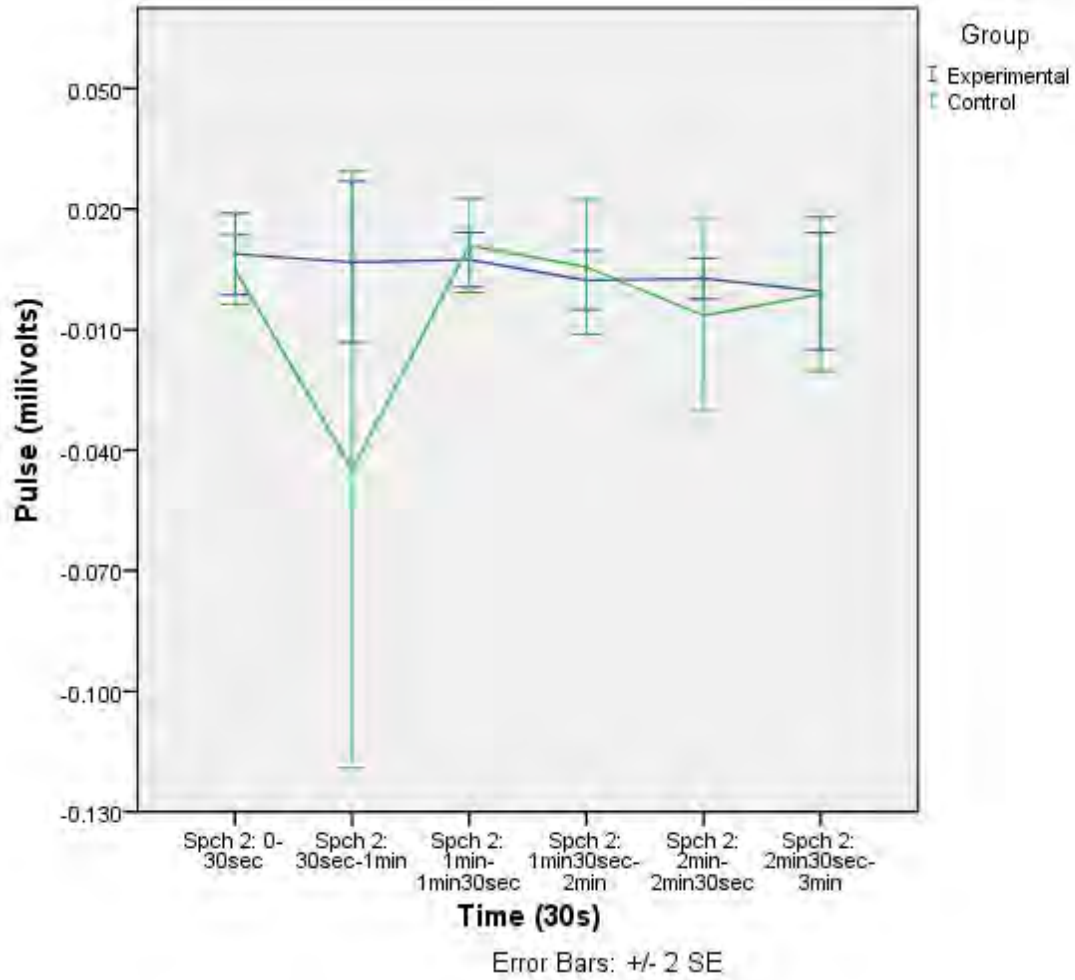


Figure 9. Pulse during speech 2. There were no significant differences across time or any time by group interactions in mean pulse measurements, $F(5,175)=1.360$, $p=.242$; $F(5,175)=1.409$, $p=.202$.

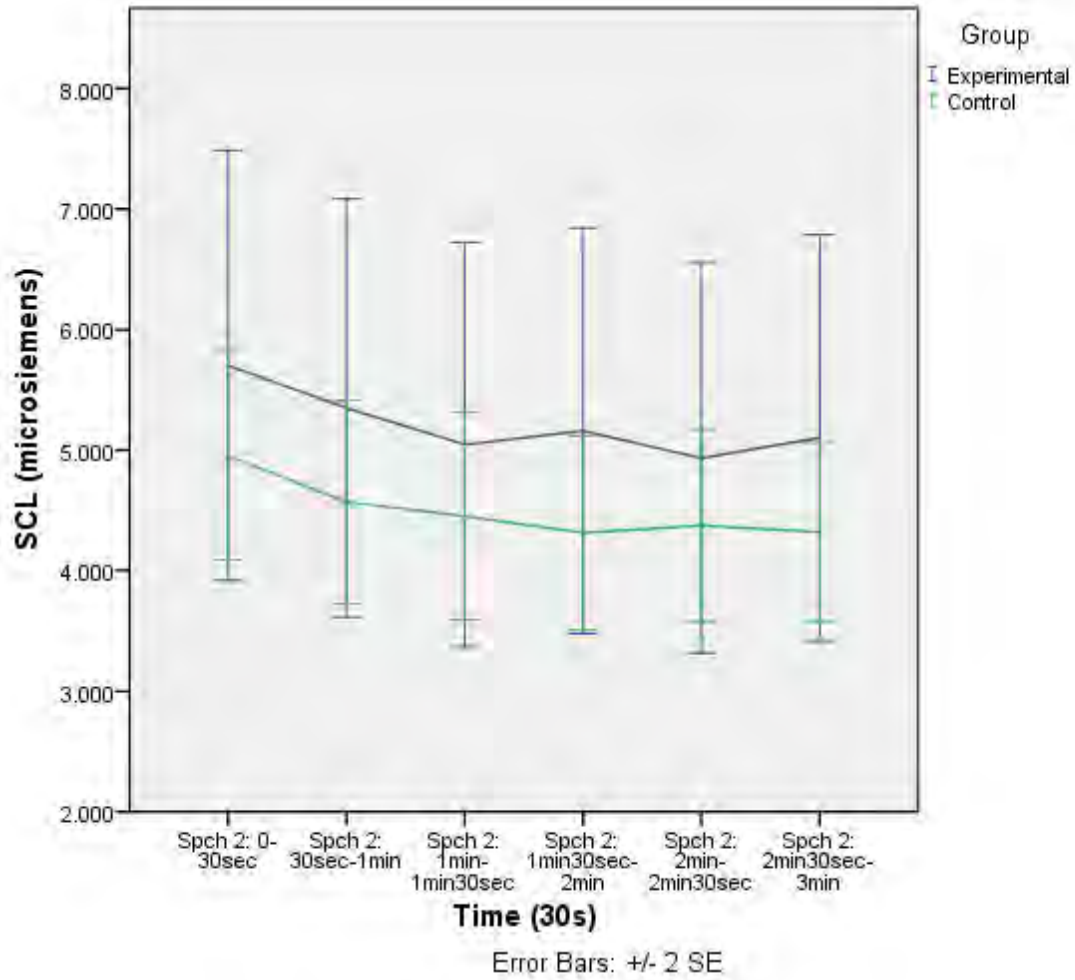


Figure 10. SCL during speech 2. There was a significant decrease in mean SCL across time, but there were no time by group interactions, $F(5,175)=14.137, p=.001$; $F(5,175)=.725, p=.605$.

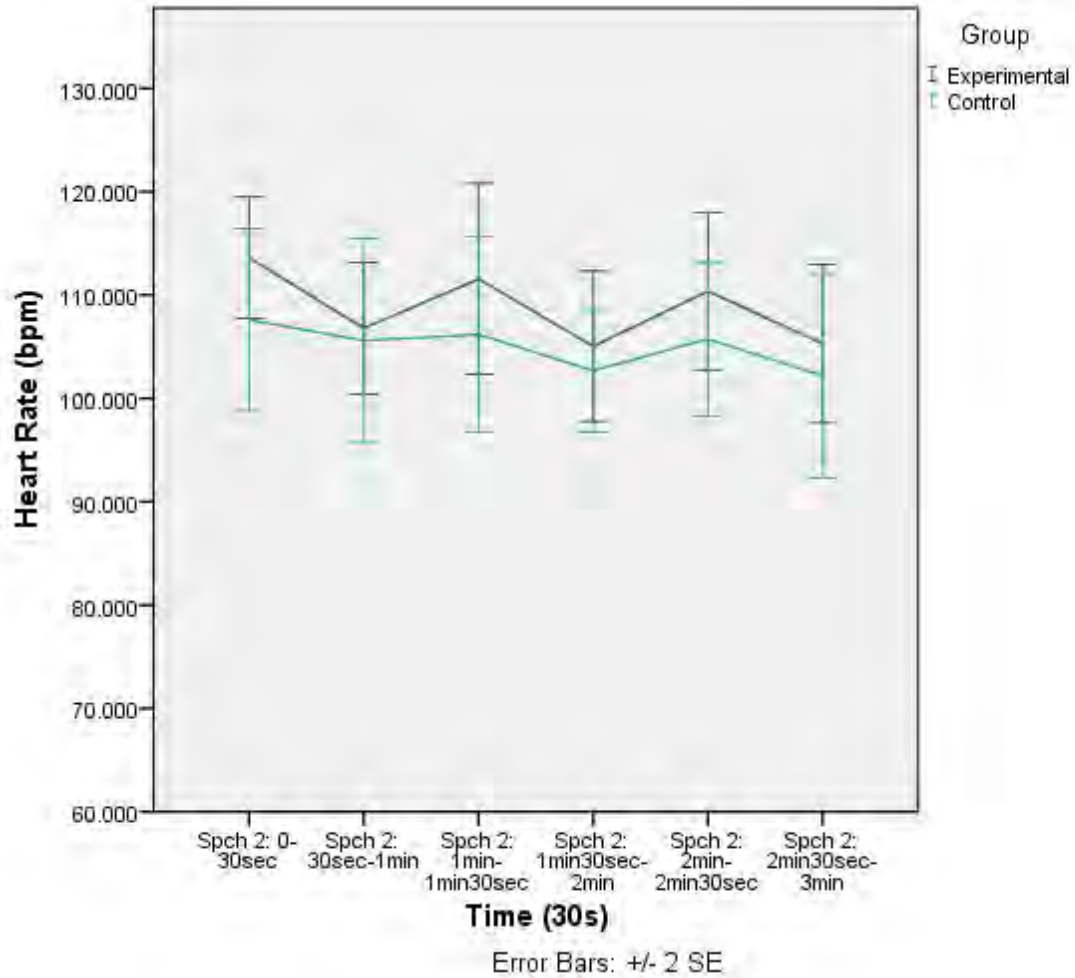


Figure 11. HR during speech 2. There were no differences across time or any time by group interactions in mean HR measurements, $F(5,175)=1.730, p=.130$; $F(5,175)=.197, p=.963$.

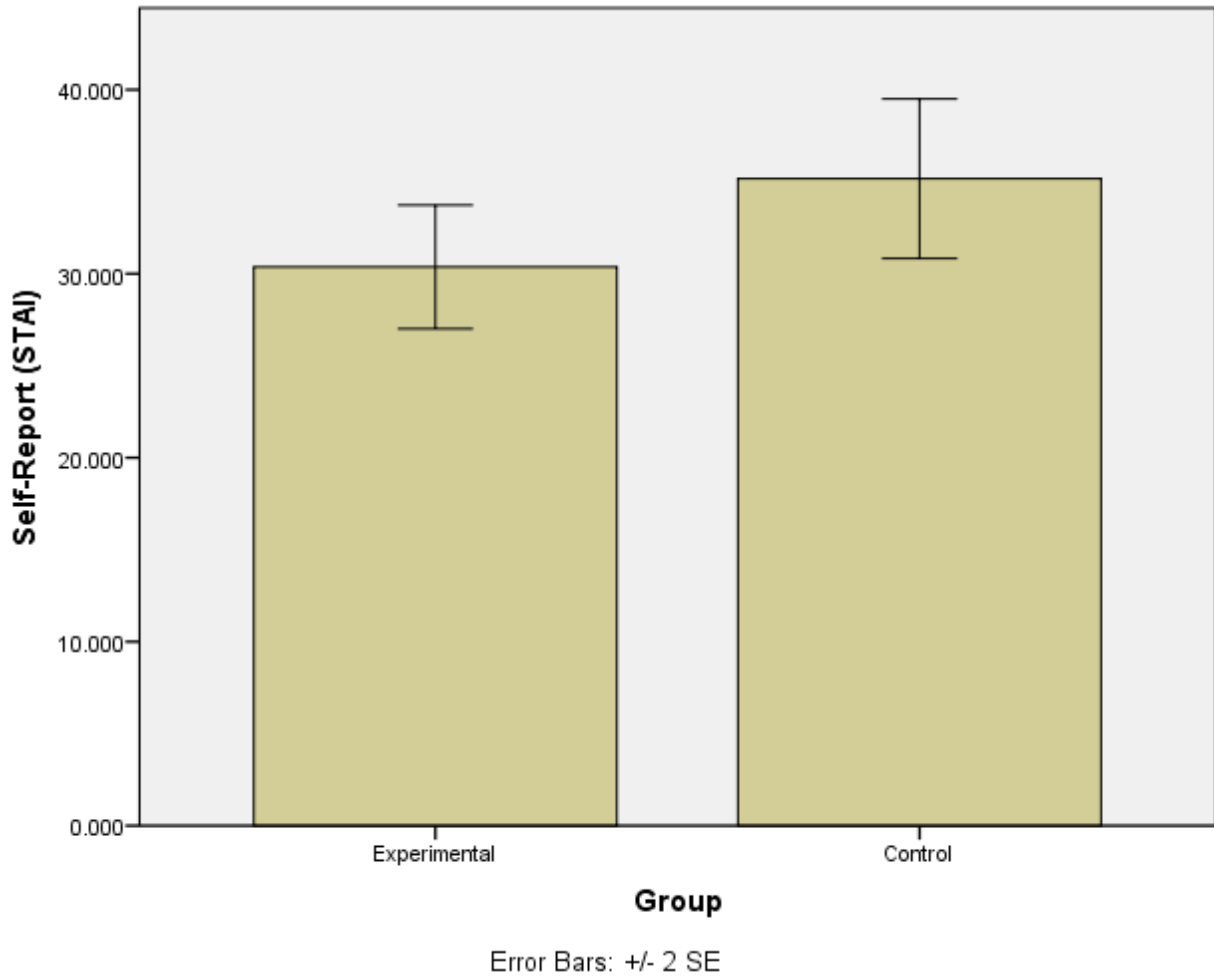


Figure 12. Post-stress manipulation differences in state anxiety by groups. This figure illustrates that there was a trend toward significance in mean state anxiety scores between groups in which the experimental group reported lower state anxiety than controls, $t(35)=-1.762, p=.087$.

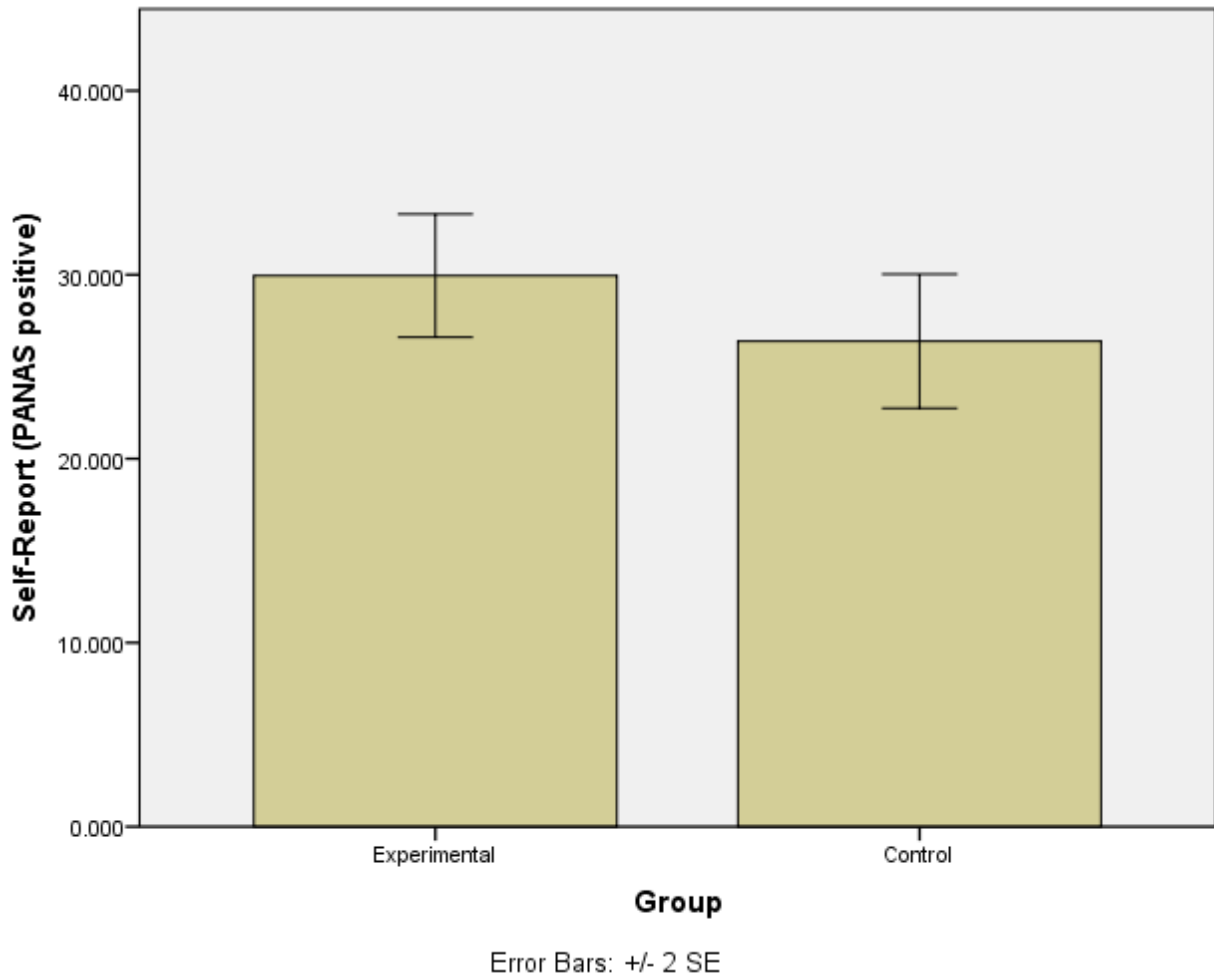


Figure 13. Post-stress manipulation positive affect scores by group. This figure illustrates that there were no significant differences in mean positive affect scores between group, $t(35)=1.441$, $p=.159$.

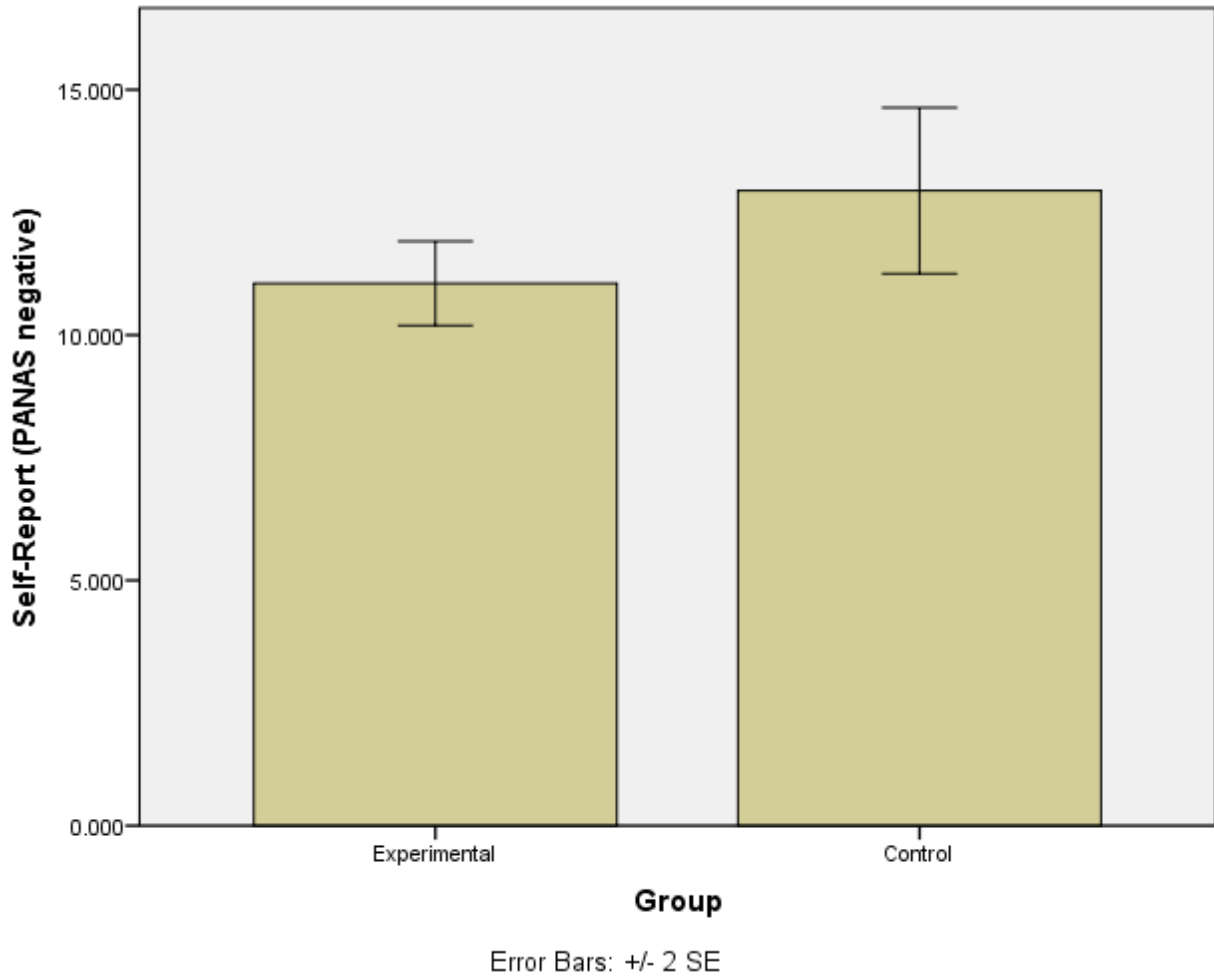


Figure 14. Post-stress manipulation negative affect scores by group. This figure illustrates that there was a significant difference in mean negative affect scores between groups in which the experimental group reported lower negative affect than controls, $t(35)=-2.027$, $p=.050$.

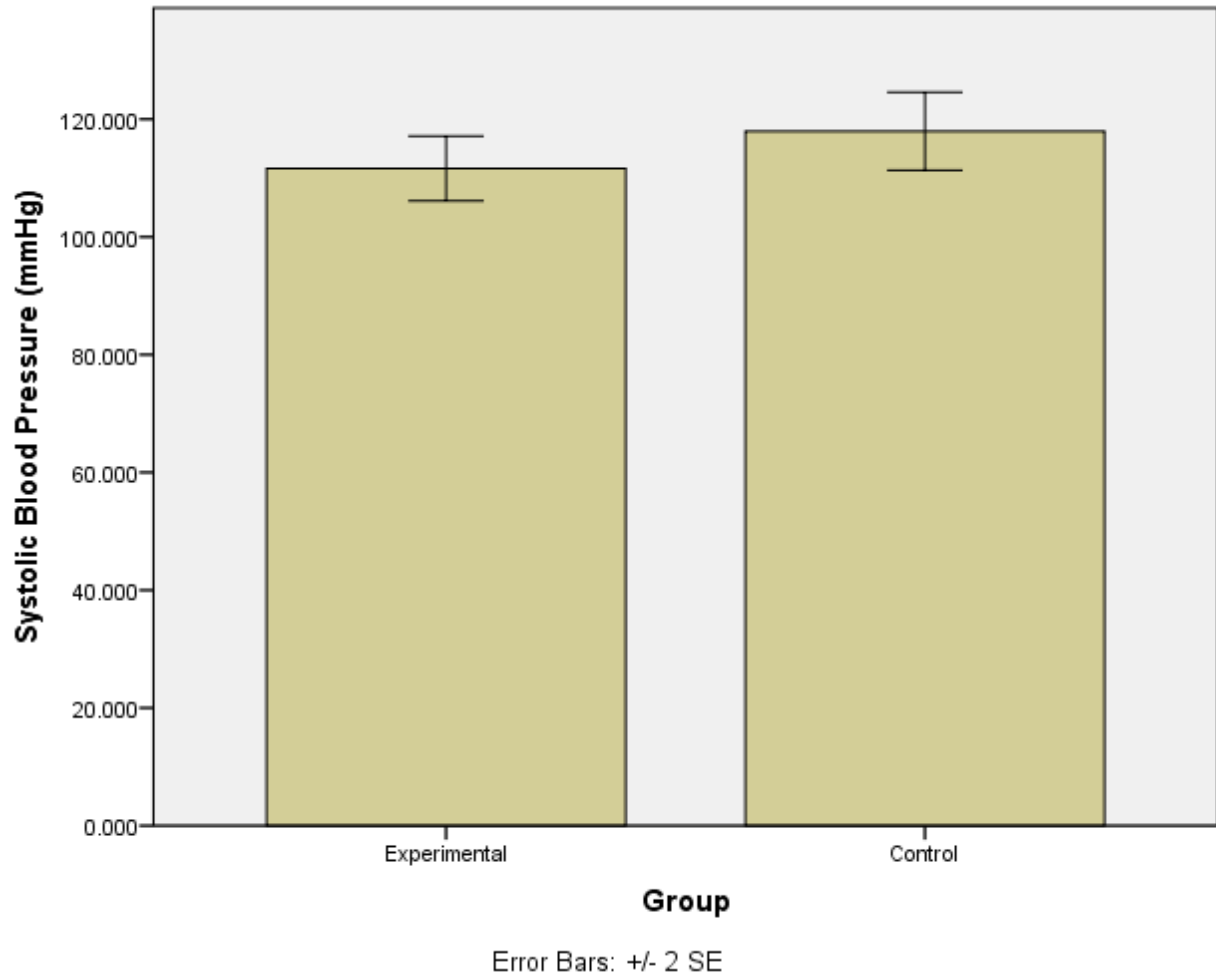


Figure 15. Post-stress manipulation systolic BP differences between groups. This figure illustrates that there were no significant differences in mean systolic BP between groups, $t(34)=-1.472, p=.150$.

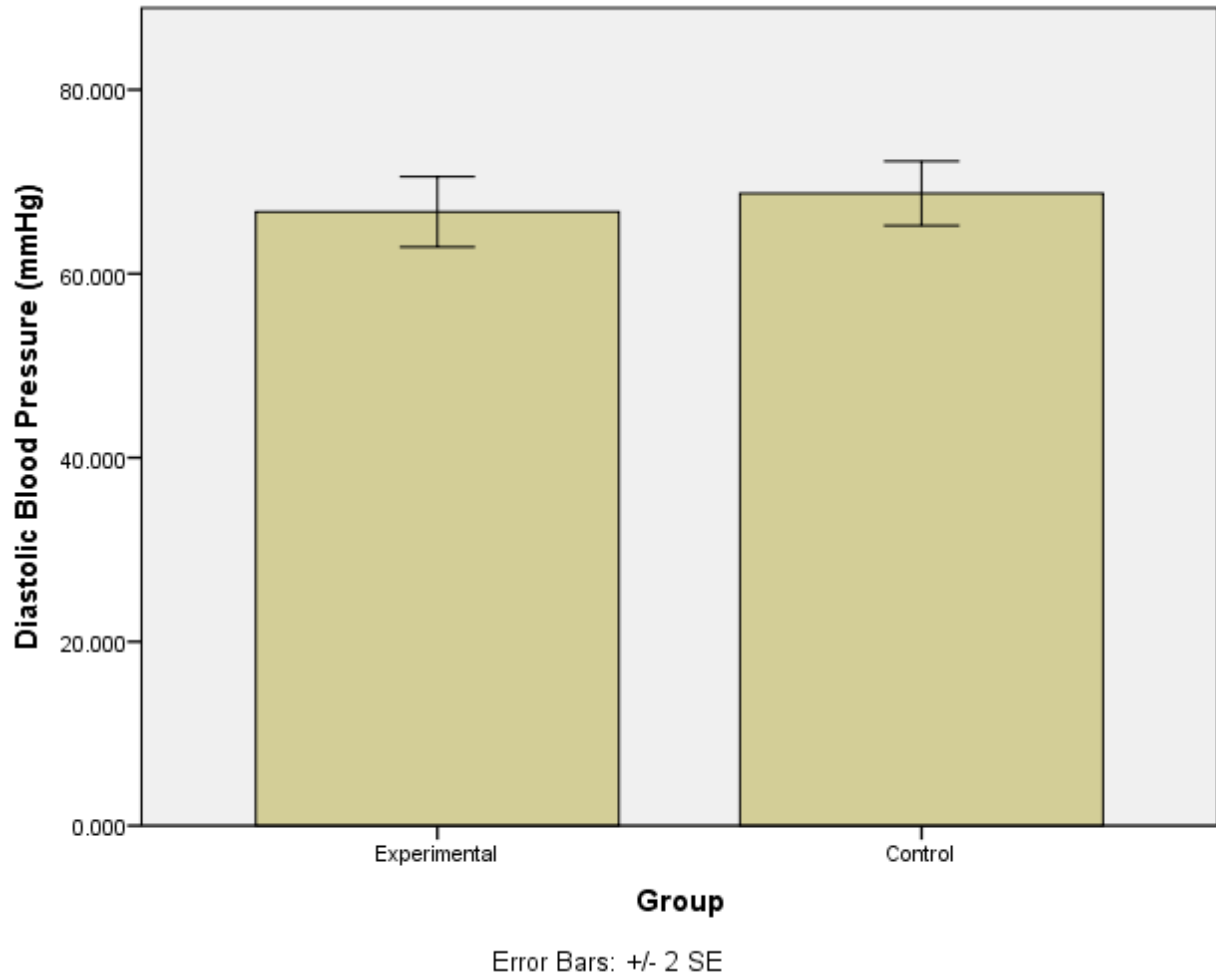


Figure 16. Post-stress manipulation diastolic BP differences between groups. There were no significant differences in mean diastolic BP between groups, $t(34)=-.772$, $p=.445$.

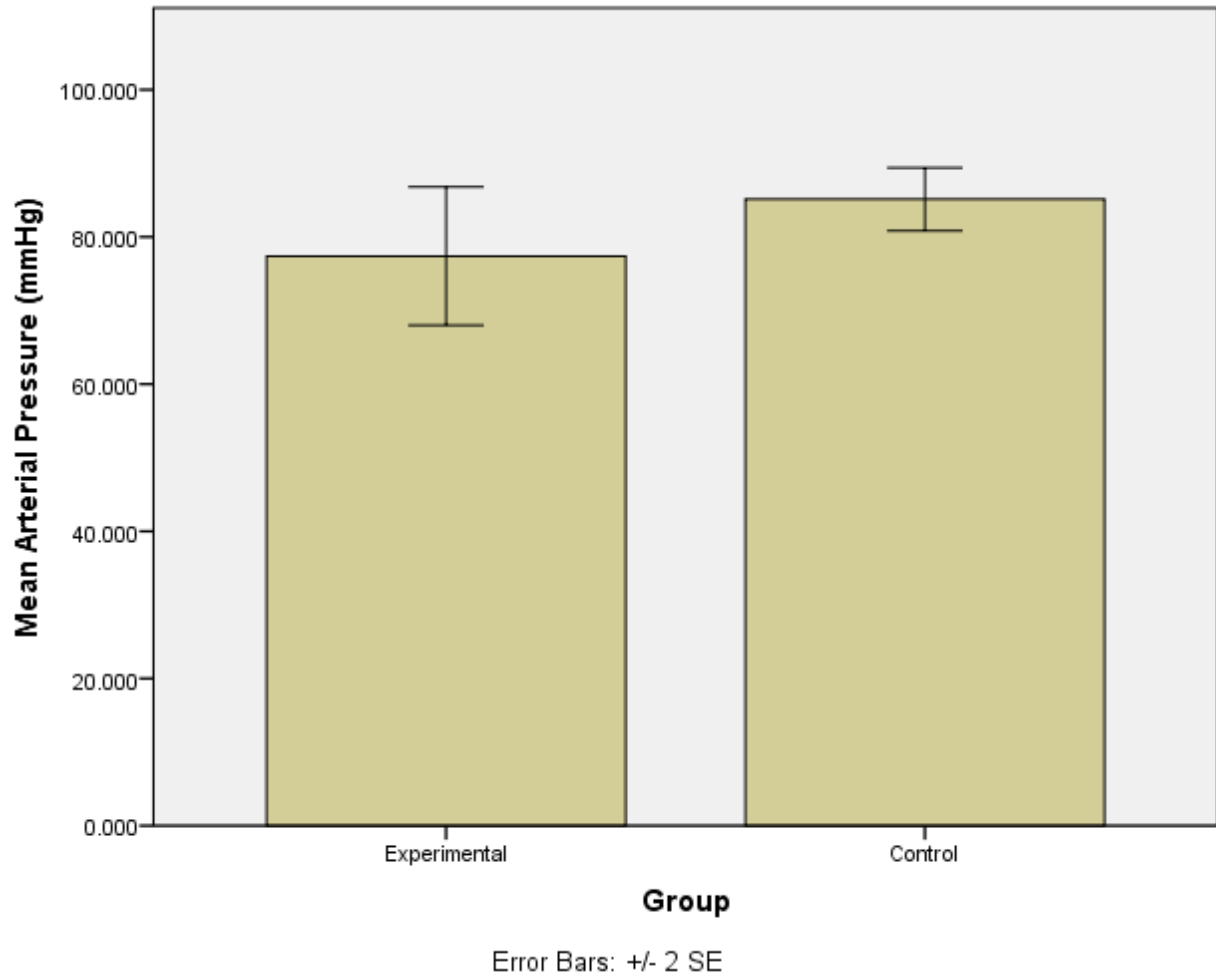


Figure 17. Post-stress manipulation mean arterial pressure by group. This figure illustrates that there were no significant differences in mean arterial pressures between groups, $t(35)=-1.473$, $p=.150$.

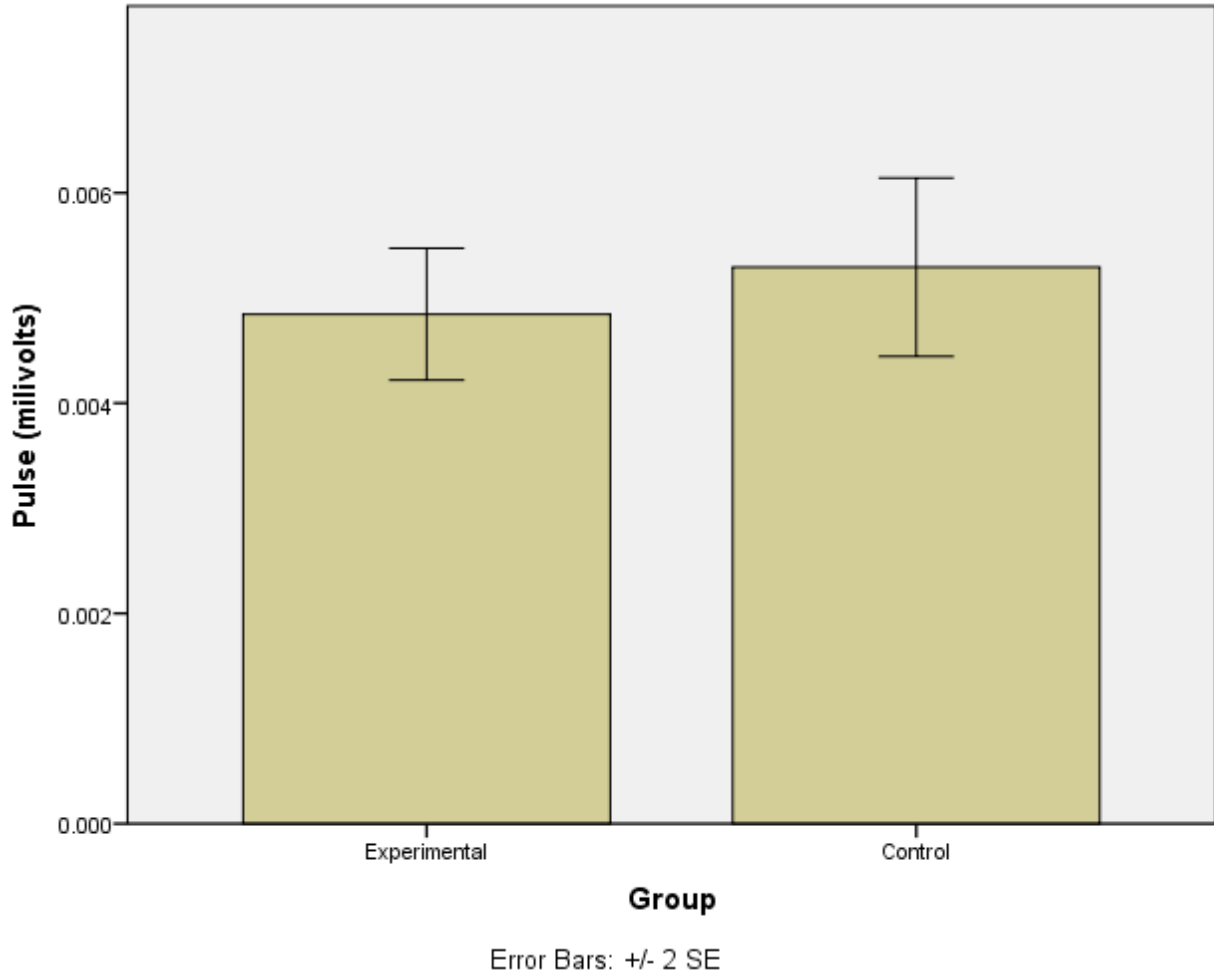


Figure 18. Post-stress manipulation grand mean pulse by groups. This figure illustrates that there are no differences between group in mean pulse measurements, $t(35)=-.855, p=.398$.

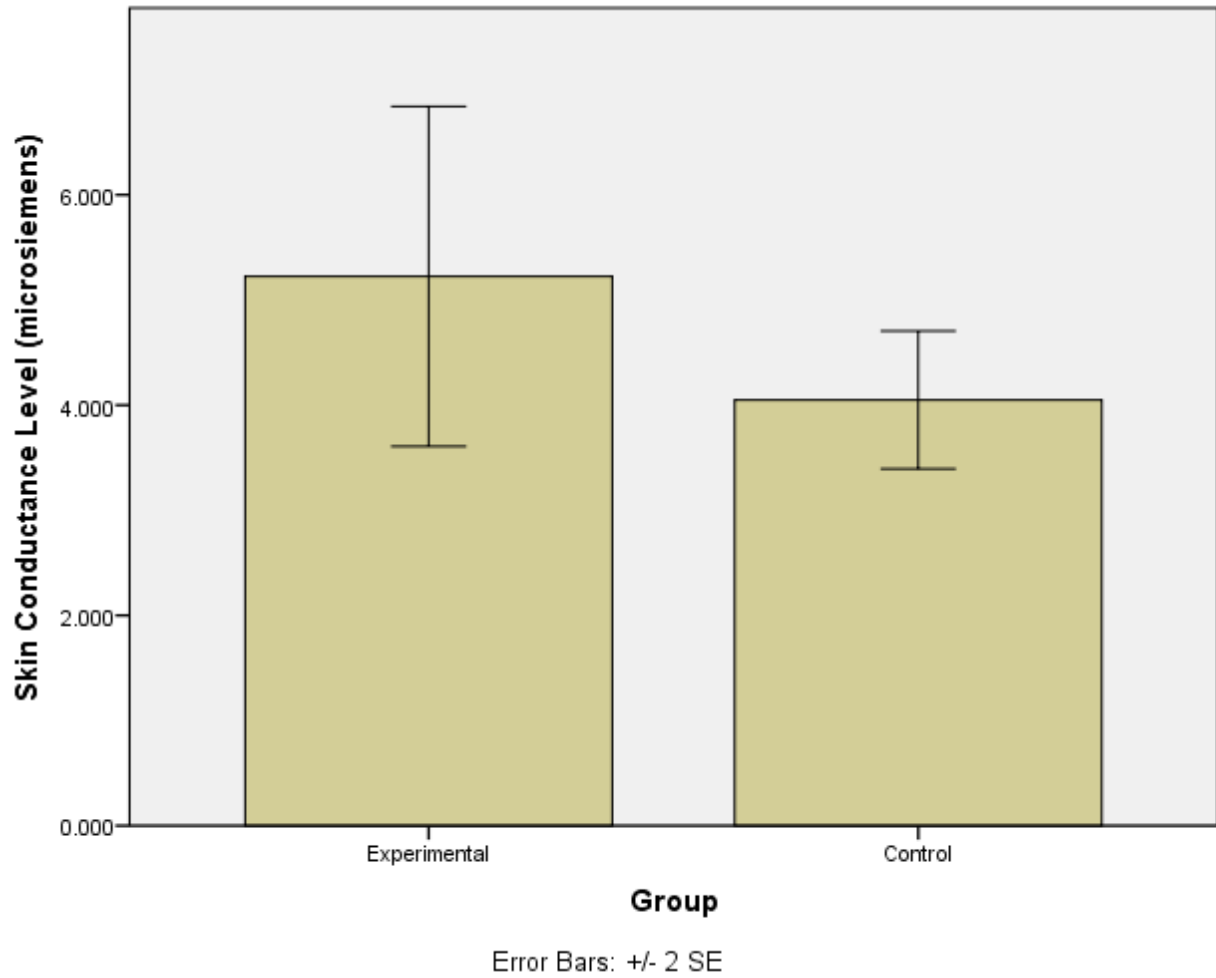


Figure 19. Post stress manipulation grand mean SCL by groups. This figure illustrates that there were no group differences in mean SCL, $t(35)=1.320$, $p=.195$.

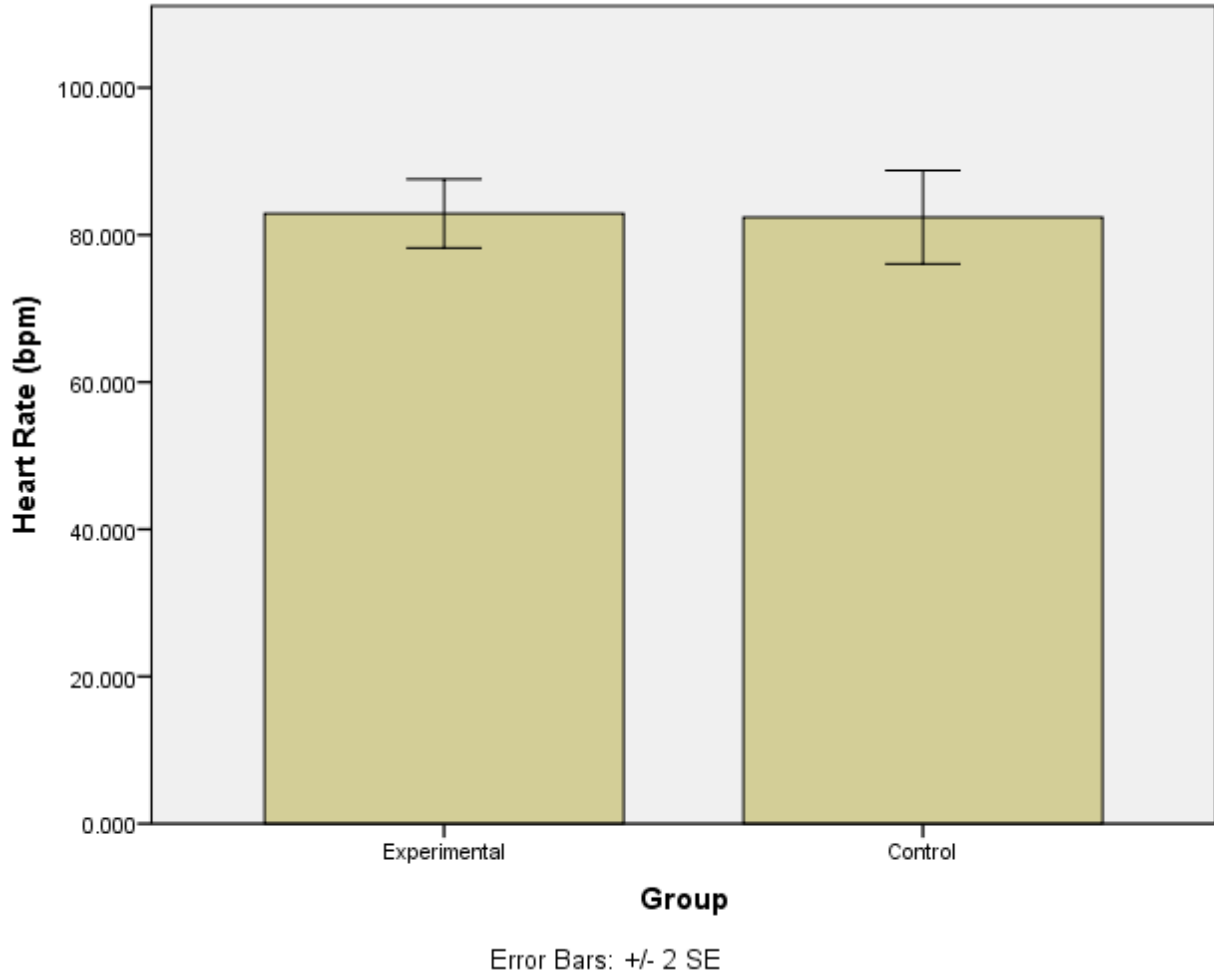


Figure 20. Post stress manipulation grand mean HR by groups. This figure illustrates that there are no group differences in mean HR, $t(35)=.127, p=.900$.

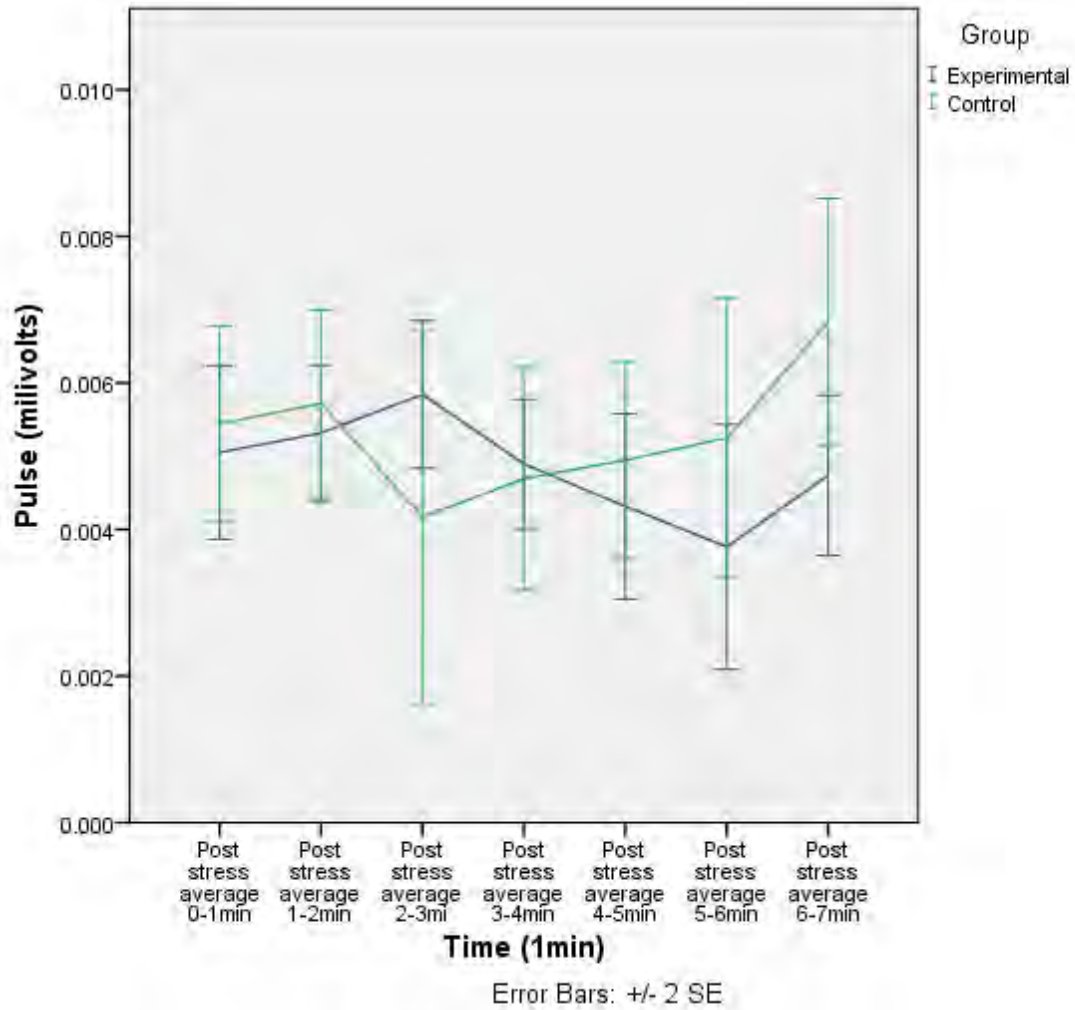


Figure 21. Pulse during post-stress manipulation. There were no significant differences across time or any time by group interactions, $F(6,210)=.979, p=.440$; $F(6,210)=1.606, p=.147$.

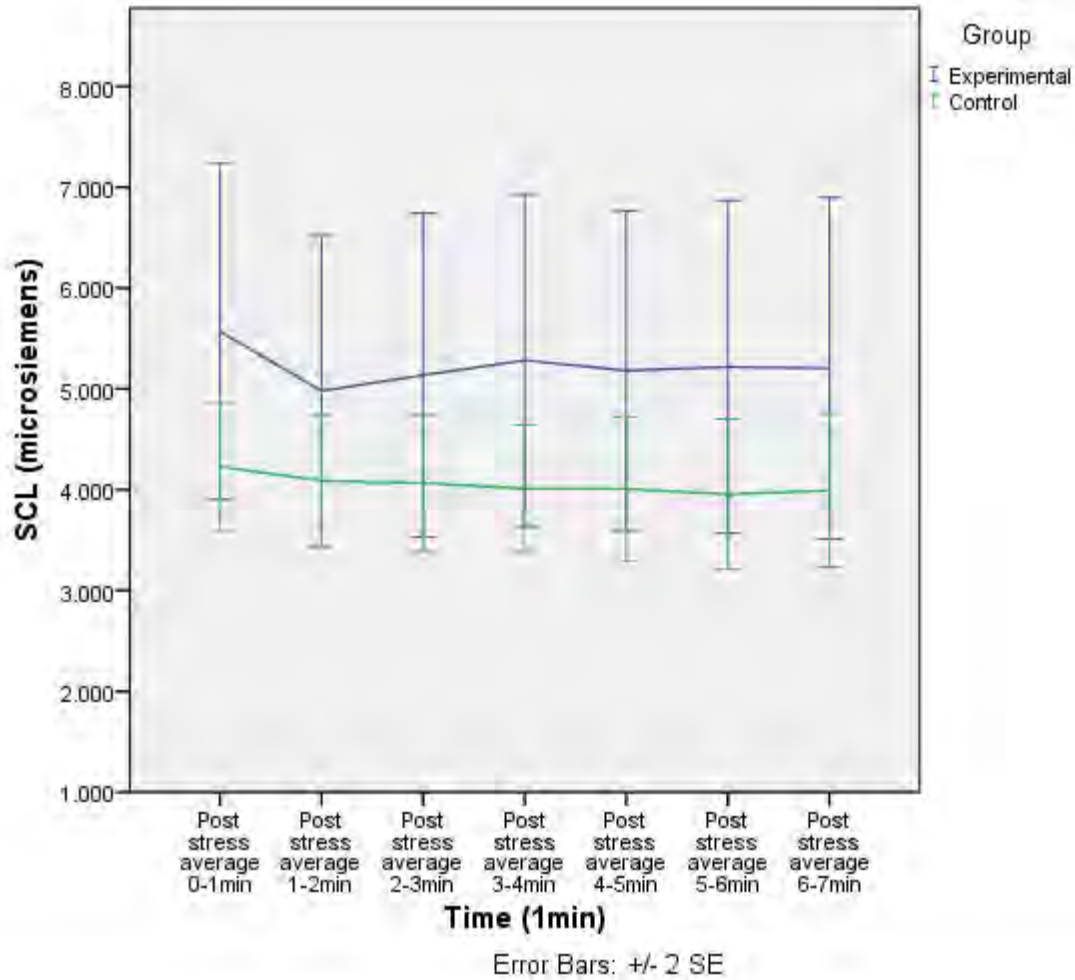


Figure 22. SCL during post-stress manipulation. There was a significant difference across time, but there were no time by group interactions in mean SCL between groups, $F(6,210)=2.466$, $p=.025$; $F(6,210)=.991$, $p=.432$.

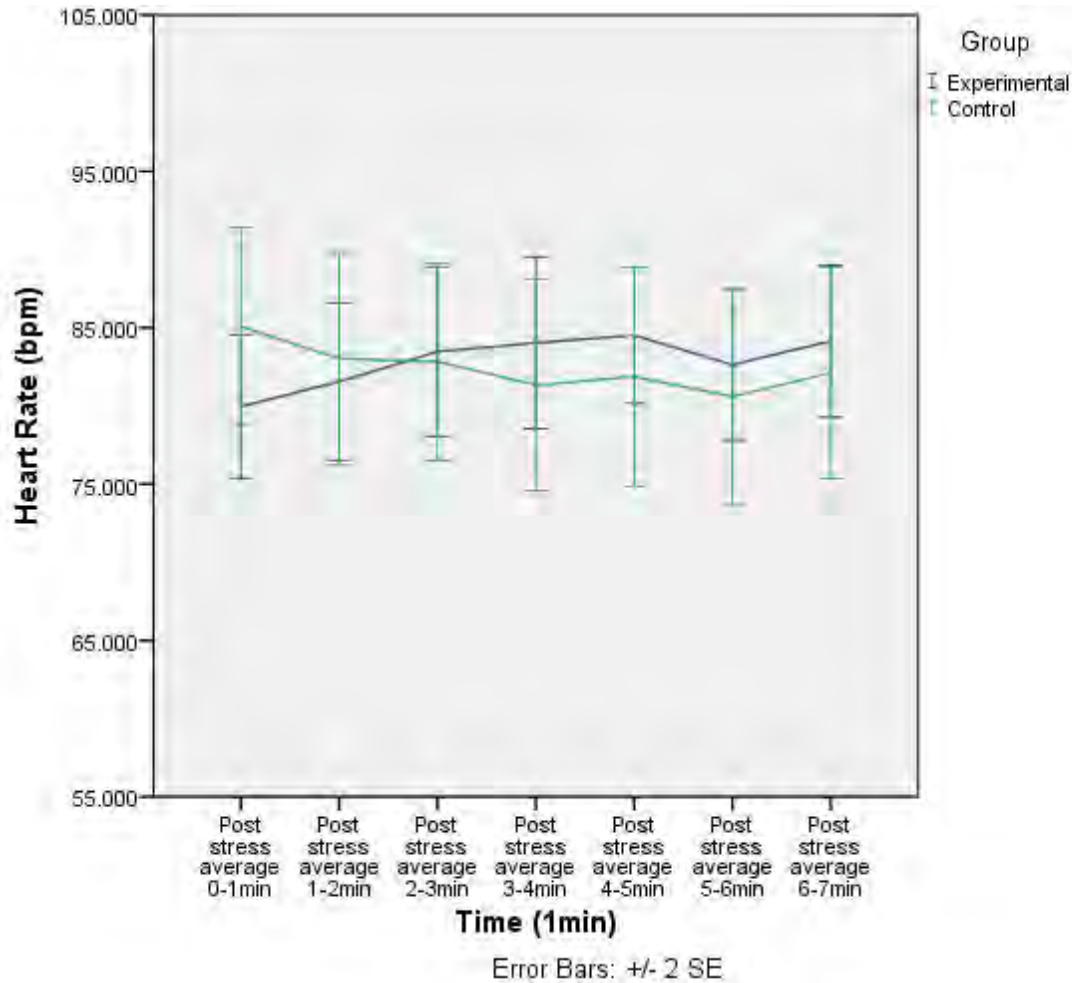


Figure 23. HR during post-stress manipulation. There were no differences across time, but there was a significant time by group interaction in mean HR, $F(6,210)=.617, p=.717$; $F(6,210)=3.797, p=.001$. Post hoc analyses revealed a marginal change in both experimental and control group between time points 1 and 3, $t(17)=1.899, p=.075$; $t(18)=-1.908, p=.072$. The means indicated that the control group's HR decreased from 85.02 (SD=13.47) to 82.83 (SD=13.39) while the experimental group's mean HR increased from 79.97 (SD=9.99) to 83.47 (SD=11.83) suggesting that these changes can account for most of the time by group interaction.

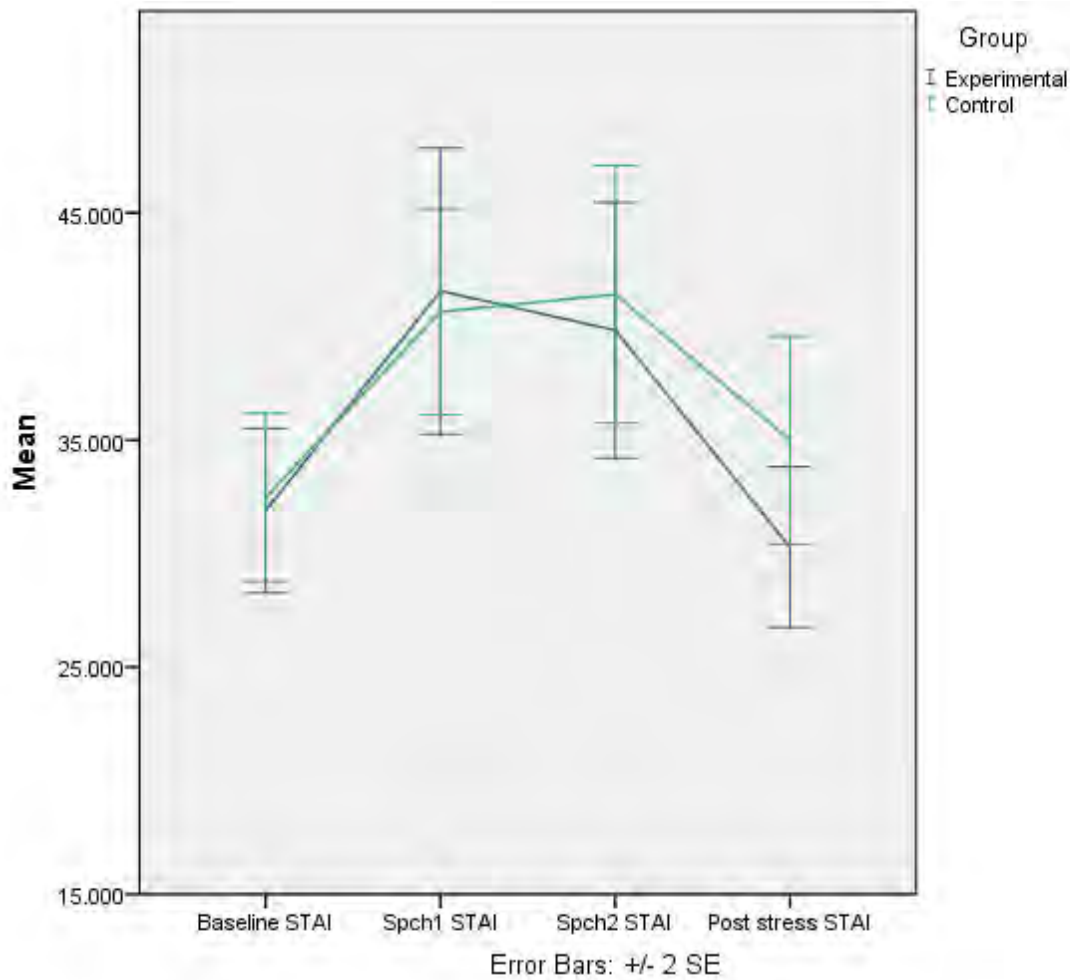


Figure 24. State anxiety change over time. State anxiety scores significantly changed across the course of the experiment, $F(3,99)=23.702, p=.001$. Post hoc analysis indicated that state anxiety increased from baseline to speech 1, did not change between the speeches, and decreased from speech 2 to post-stress, $t(34)=-6.121, p<.05$; $t(34)=.500, p>.05$; $t(36)=5.450, p<.05$. Further analyses indicated no change directly from baseline to post stress manipulation, $p>.05$.

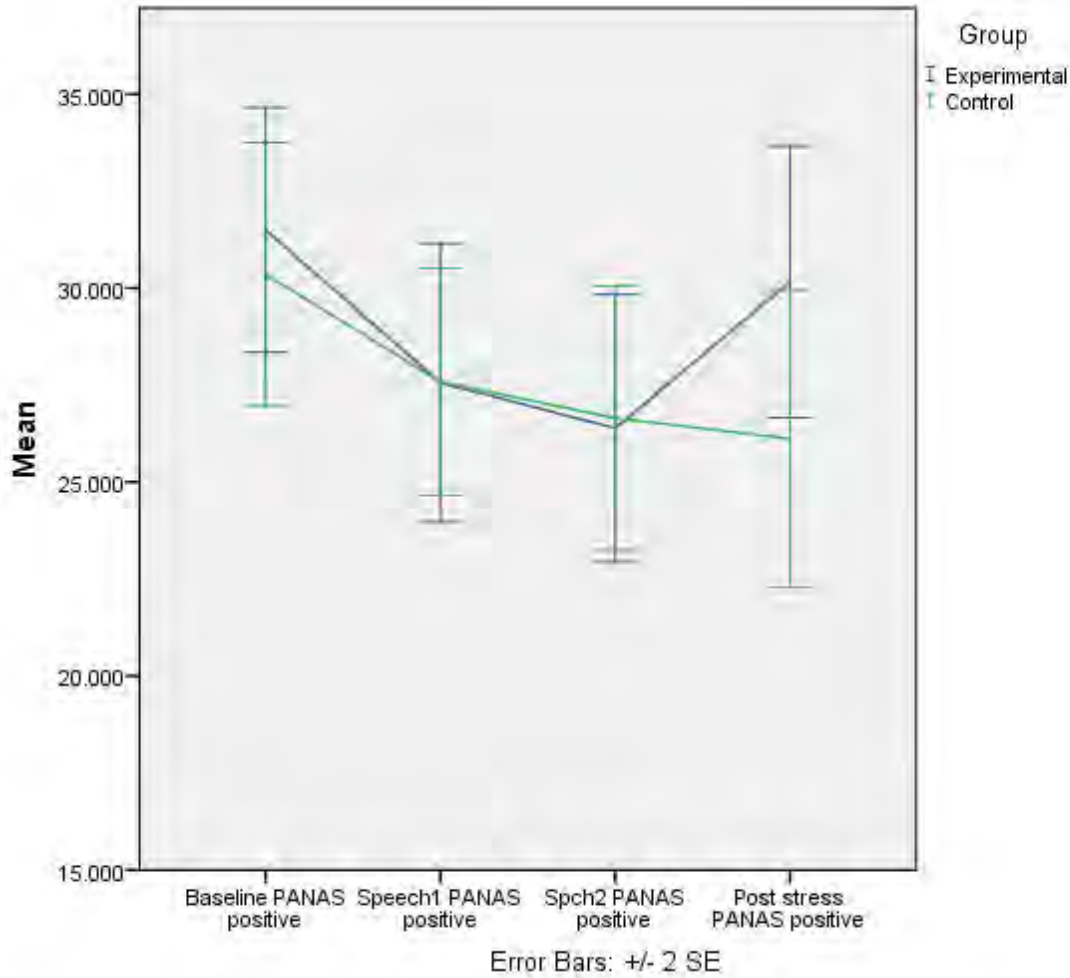


Figure 25. Positive affect change over time. Positive affect scores significantly changed across the entire experiment, $F(3,99)=10.326$, $p=.001$. Post hoc analysis also indicated that positive affect decreased from baseline to speech 1, did not change between speeches, and increased from speech 2 to post-stress, $t(34)=4.9$, $p<.05$; $t(34)=1.708$, $p>.05$, $t(36)=-1.957$, $p<.05$. Further analyses indicated that positive affect was significantly decreased directly from baseline to post-stress manipulation, $p<.05$.

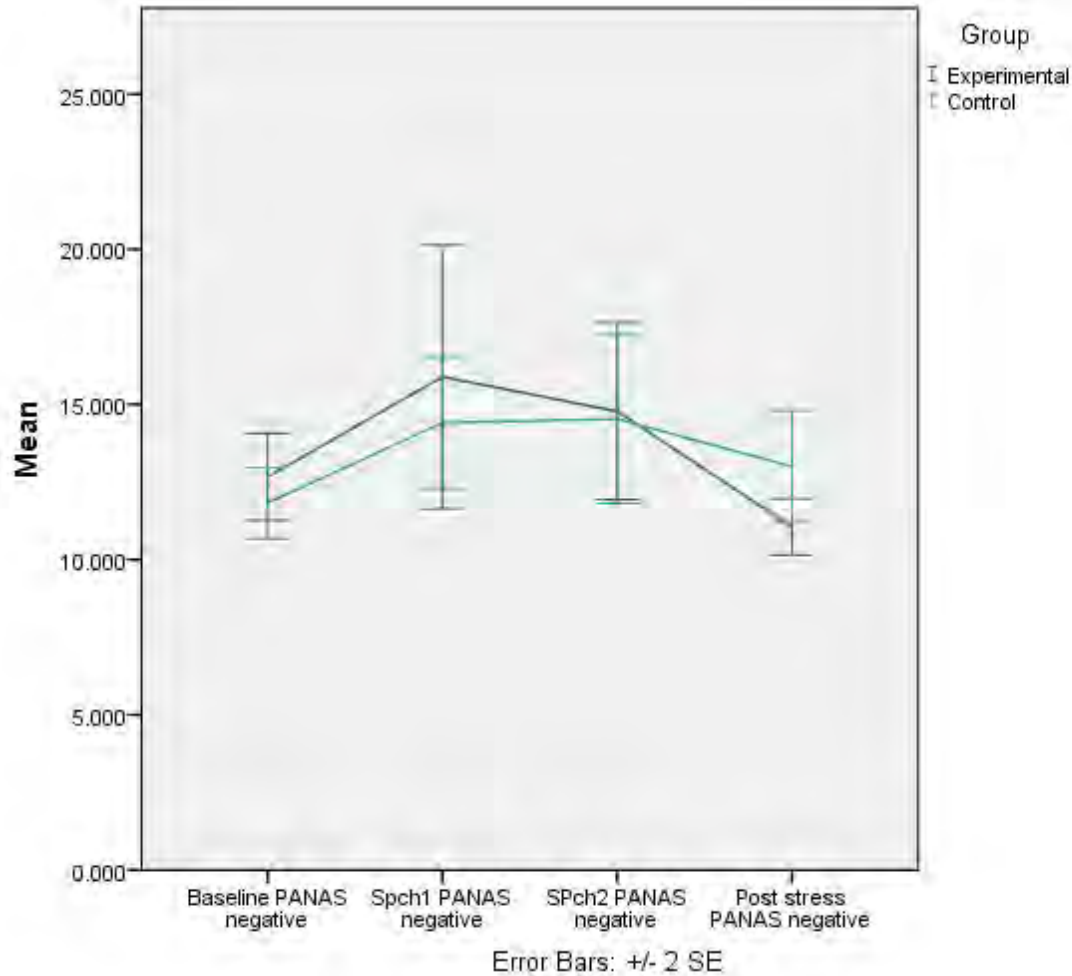


Figure 26. Negative affect change over time. Negative affect scores significantly changed over the course of the experiment, $F(3,99)=6.819$, $p=.001$. Post hoc analysis indicated that negative affect increased from baseline to speech 1, did not change between speeches, and decreased from speech 2 to post-stress, $t(34)=-2.639$, $p<.05$; $t(34)=.849$, $p>.05$; $t(36)=3.334$, $p<.05$. Further analyses indicated a time by group interaction directly from baseline to post-stress manipulation in which the experimental groups negative affect decreased while the control group's increased, $p<.05$.

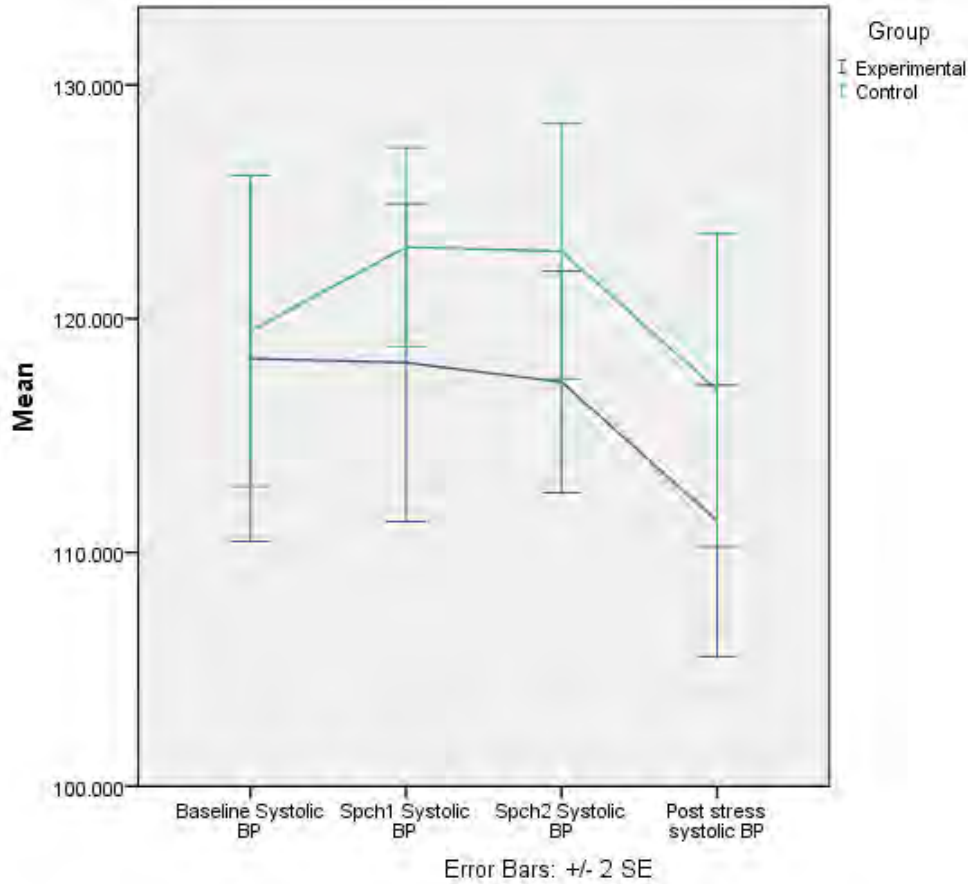


Figure 27. Systolic BP change over time. Systolic BP significantly changed over the course of the experiment, $F(3,96)=6.435$, $p=.001$. Post hoc analysis indicated that there was no significant change from baseline to speech 1, no change from speech 1 to speech 2, but a decrease from speech 2 to post-stress, $t(33)=-1.023$, $p>.05$; $t(33)=.312$, $p>.05$; $t(35)=4.113$, $p<.05$. Further analyses indicated the systolic BP significantly decreased directly from baseline to post stress manipulation, $p<.05$.

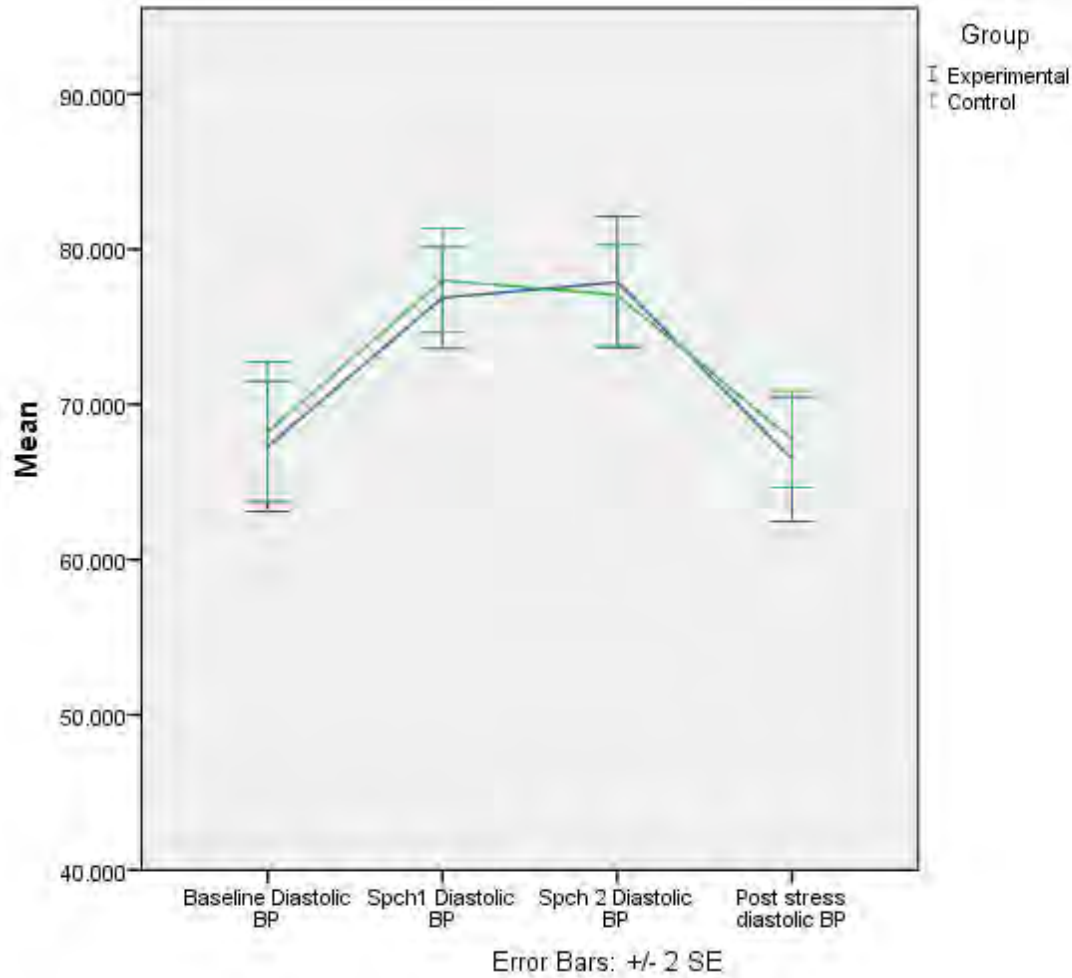


Figure 28. Diastolic BP change over time. Diastolic BP significantly changed over the course of the experiment, $F(3,96)=76.013$, $p=.001$. Post hoc analysis indicated that diastolic BP significantly increased from baseline to speech 1, did not change between the speeches, and significantly decreased from speech 2 to post-stress, $t(33)=-10.015$, $p<.05$; $t(33)=-.035$, $p>.05$; $t(35)=11.925$, $p<.05$. Further analyses indicated no change directly from baseline to post stress manipulation, $p>.05$.

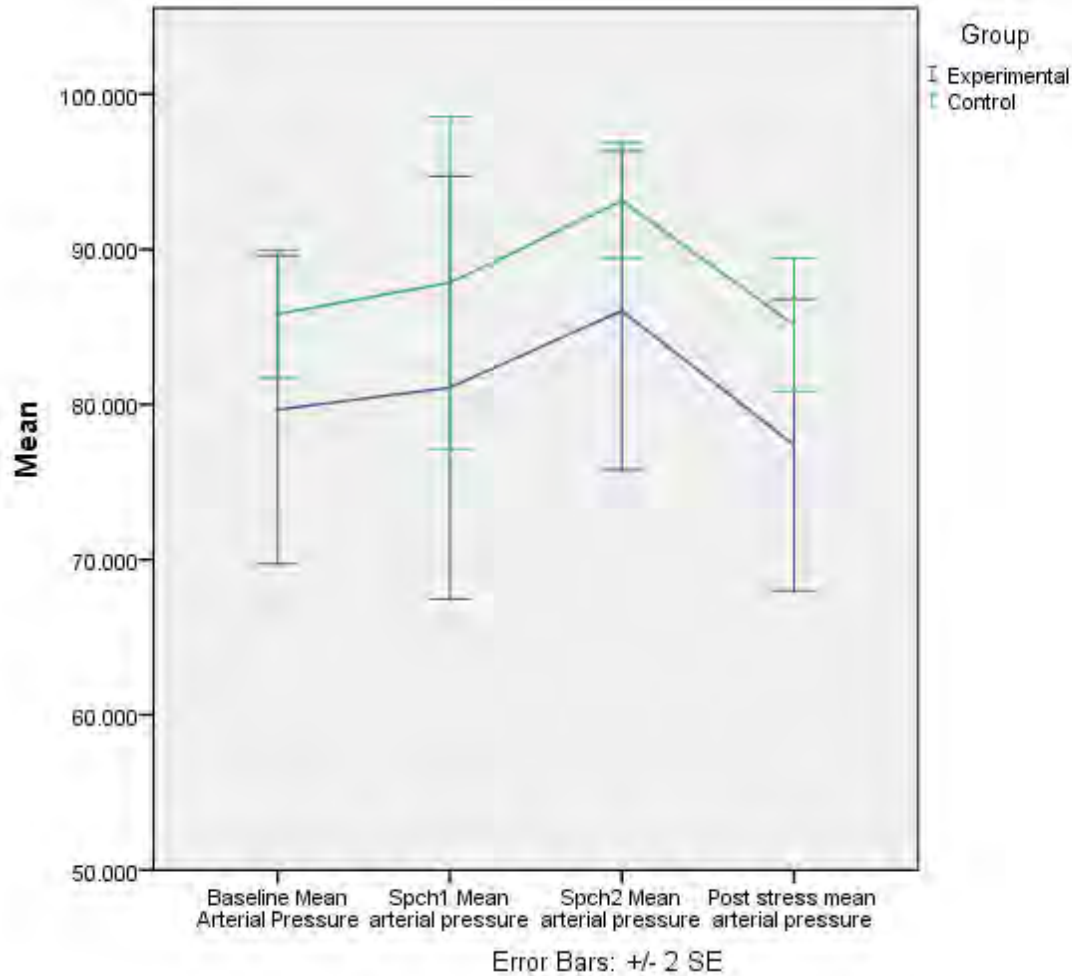


Figure 29. Mean arterial pressure change over time. Mean arterial pressure significantly changed across the course of the experiment, $F(3,105)=3.393$, $p=.021$. Post hoc analyses indicated that there was no significant change from baseline to speech 1, no change from speech 1 to speech 2, but a decrease from speech 2 to post-stress, $t(36)=-.467$, $p>.05$; $t(36)=-1.362$, $p>.05$; $t(36)=11.282$, $p<.05$. Further analyses indicated no change directly from baseline to post stress manipulation, $p>.05$.

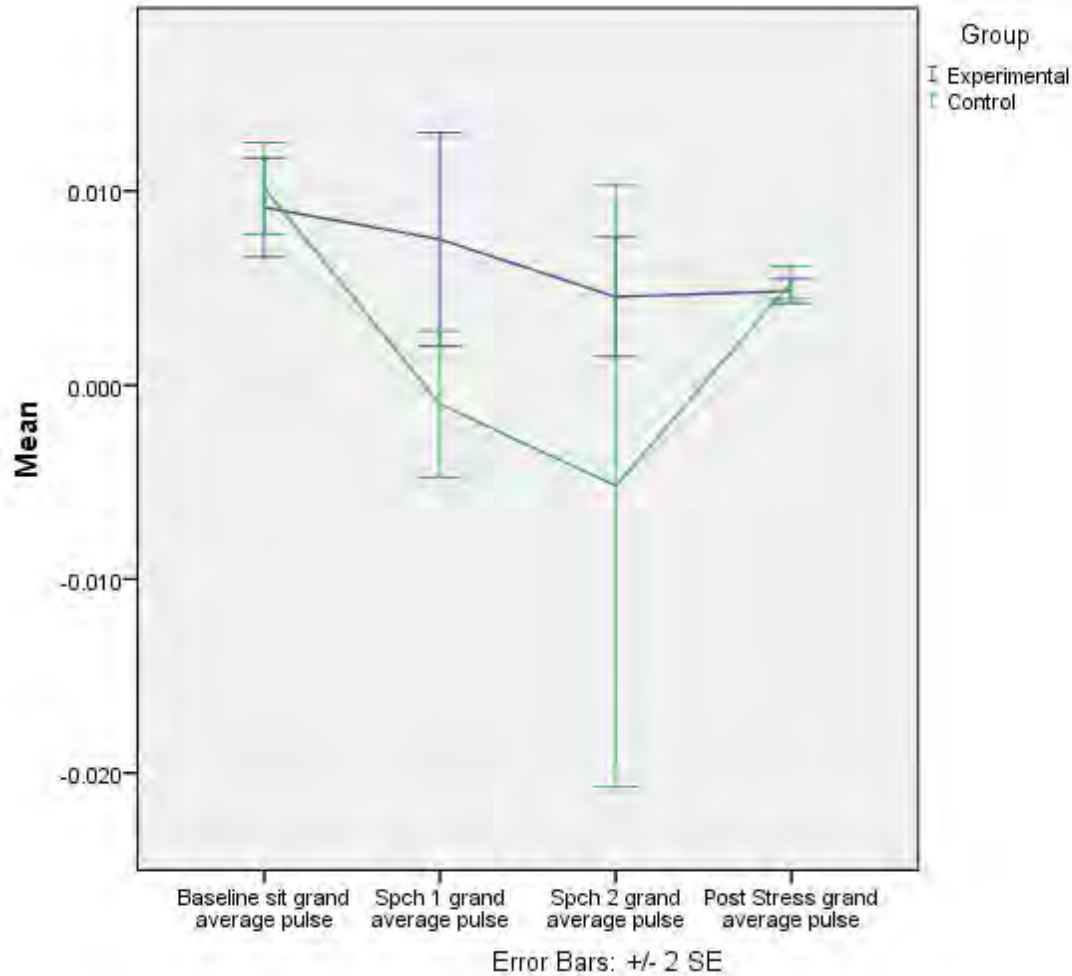


Figure 30. Pulse change over time. Pulse significantly changed over the course of the experiment, $F(3,99)=3.725, p=.014$. Post hoc analysis indicated that pulse significantly decreased from baseline to speech 1 and between speeches, but did not change from speech 2 to post-stress, $t(36)=-9.455, p<.05$; $t(36)=9.480, p<.05$; $t(36)=-1.354, p>.05$. These results may have been affected by an outlier. Further analyses indicated a significant decrease directly from baseline to post-stress manipulation, $p<.05$.

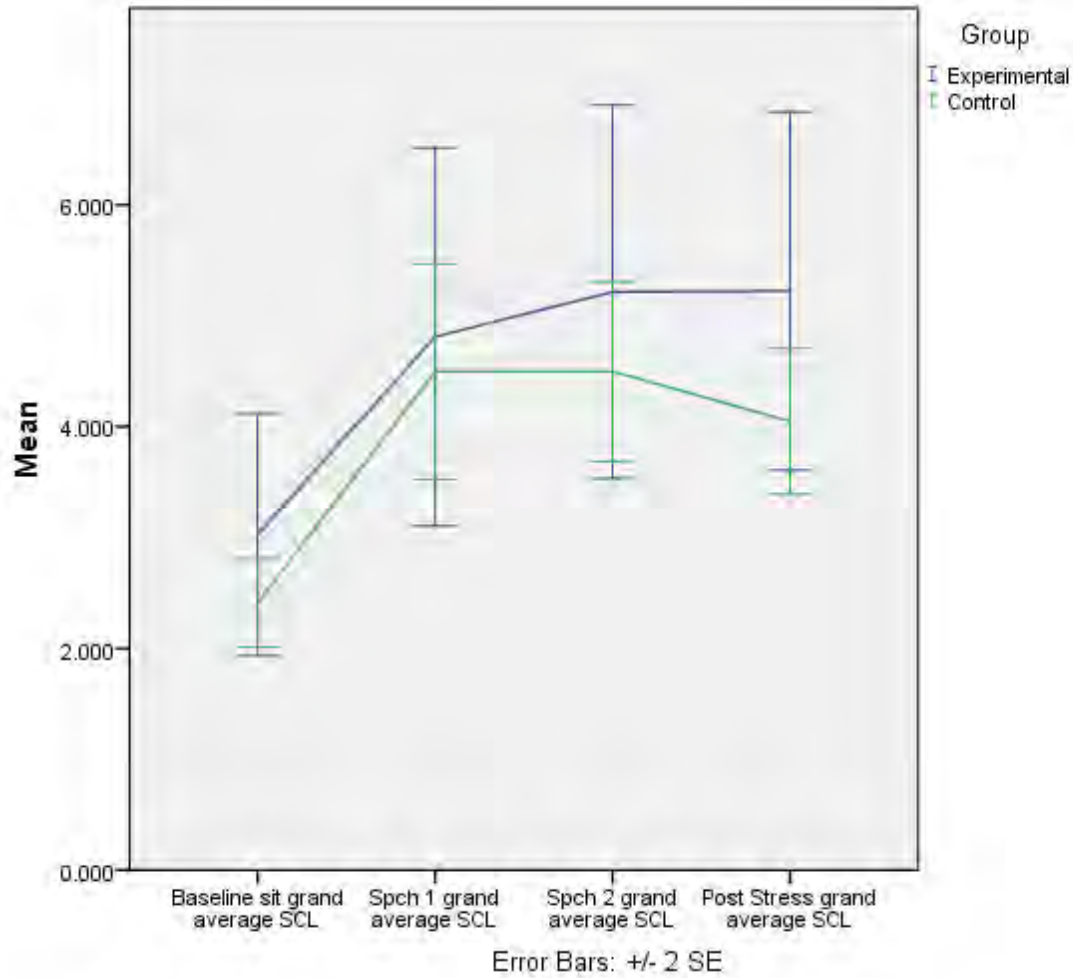


Figure 31. SCL change over time. SCL significantly changed over the course of the experiment, $F(3,105)=42.985, p=.001$. Post hoc analysis revealed that SCL significantly increased from baseline to speech 1, did not change between speeches and from speech 2 to post-stress, $t(36)=-6.531, p<.05$; $t(36)=-1.926, p>.05$; $t(36)=1.246, p>.05$. Further analyses indicated a significant increase directly from baseline to post-stress manipulation, $p<.05$.

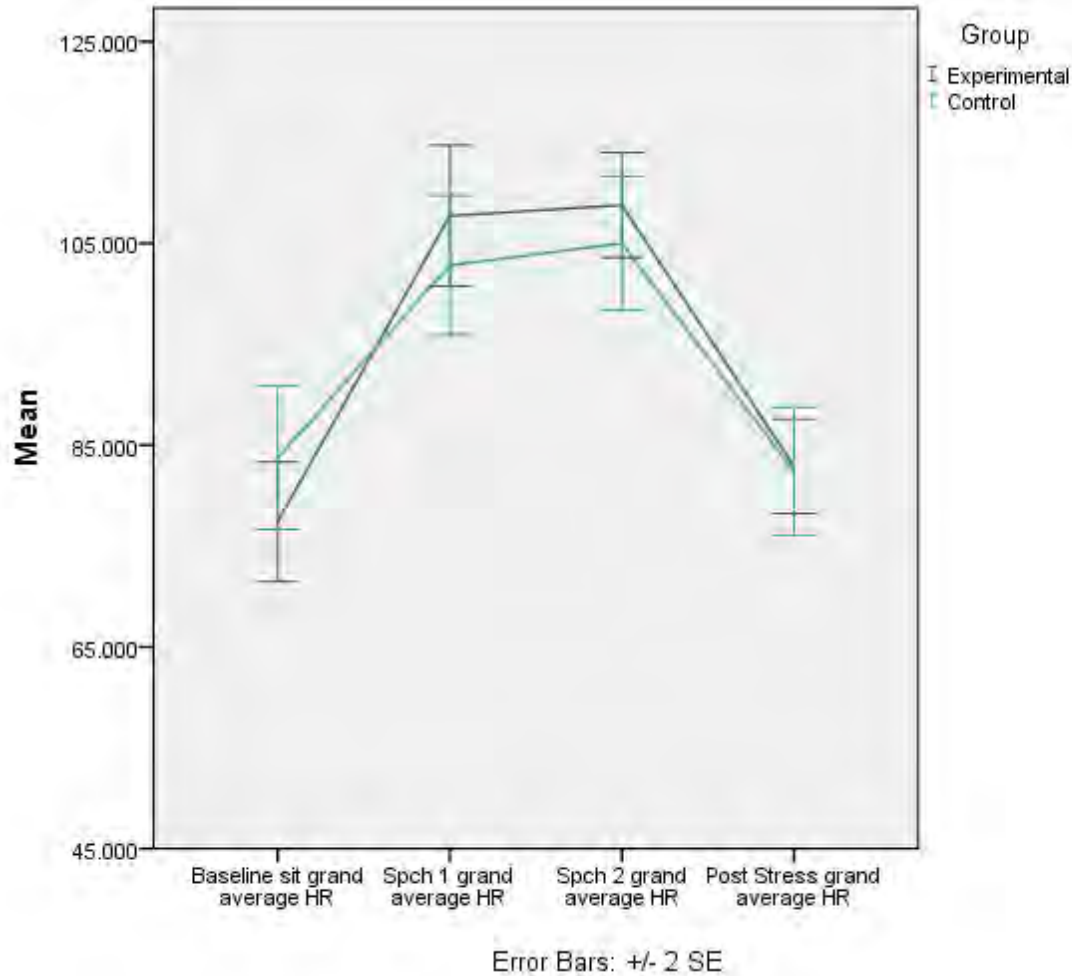


Figure 32. HR change over time. HR significantly changed over the course of the experiment, $F(3,105)=89.770, p=.001$. Post-hoc analyses indicated that HR significantly increased from baseline to speech 1, did not change between speeches, and significantly decreased from speech 2 to baseline, $t(36)=-9.558, p<.05$; $t(36)=-1.139, p>.05$; $t(36)=10.420, p<.05$. Further analyses indicated a significant time by group interaction in which the experimental group's HR increased directly from baseline to post-stress manipulation, whereas the control group's decreased, $p<.05$.

Appendix A

Baseline to Post-Stress Manipulation

Self-reported affect. A paired samples t-test was used to assess changes in self-report measuring from baseline to following the post-stress manipulation. Analyses revealed that there was a significant difference between mean positive affect scores from baseline to post-stress manipulation, $t(36)=3.241$, $p=.003$. Mean positive affect baselines scores were 31.03 (SD=6.58), while mean positive affect scores following post-stress manipulation were 28.22 (SD=7.62). There was no significant difference from baseline to post-stress manipulation in mean negative affect scores, $t(36)=.526$, $p=.602$. There were also no significant differences in mean state anxiety scores from baseline to post-stress manipulation, $t(36)=-.130$, $p=.897$.

Repeated measures ANOVA was used on self-report measures to assess change over time by group from baseline to post-stress manipulation. Between-subjects analyses revealed no differences in mean state anxiety by group, $F(1,35)=1.399$, $p=.245$. Within-subjects analyses revealed no change over time, but there was a marginally significant time by group interaction indicating that groups did not change in the same direction, $F(1,35)=.032$, $p=.859$; $F(1,35)=2.884$, $p=.098$. State anxiety scores for the experimental group at baseline was 32.21 (SD=7.58), and decreased to 30.37 (SD=7.31) following the post-stress manipulation. The mean state anxiety score for the control group at baseline was 32.89 (SD=7.61), and increased to 35.17 (SD=9.19) following post-stress manipulation. Between subjects analyses revealed no significant difference between groups in negative affect scores, $F(1,35)=.512$, $p=.479$. Within-subject analyses revealed no significant change across time, but there was a significant time by group interaction in mean negative affect score between group, $F(1,35)=.253$, $p=.618$; $F(1,35)=10.205$, $p=.003$. The mean score for the experimental group at baseline was 12.58 (SD=2.91), and

decreased to 11.05 (SD=1.87) following post-stress manipulation. The mean negative affect score for the control group at baseline was 11.83 (SD=2.31) which increased to 12.94 (SD=3.56) following post-stress manipulation. Between-subjects analyses revealed no significant difference in mean positive affect between groups from baseline to post-stress manipulation, $F(1,35)=1.114, p=.299$. Within-subjects analyses revealed a significant change over time, but there were no time by group interactions, $F(1,35)=11.114, p=.002$; $F(1,35)=2.201, p=.147$.

Blood Pressure. Paired samples t-tests were used on cardiovascular variables to analyze differences from baseline to following the post-stress manipulation phase. Analyses revealed a significant difference in mean systolic BP from baseline to post-stress manipulation, $t(35)=2.610, p=.013$. Mean systolic BP at baseline was 118.83 mmHg (SD=14.46), while mean systolic BP following post-stress manipulation was 114.78 (SD=13.12). There was also a trend toward significance from baseline to post-stress manipulation in mean arterial pressure, $t(36)=1.792, p=.082$. The average mean arterial pressure during baseline was 82.67 (SD=16.72), while the average mean arterial pressure following post-stress manipulation was 81.15 (SD=16.24). However, there was no significant difference in mean diastolic BP from baseline to post-stress manipulation, $t(35)=-.325, p=.747$.

A repeated measures ANOVA was used to assess changes in time from baseline to post-stress manipulation by group. Between-subjects analyses revealed no differences between group in mean systolic BP, $F(1,34)=.923, p=.344$. Within-subjects analyses of systolic BP revealed that there was a significant change across time, but there was no time by group interaction, $F(1,34)=7.008, p=.012$; $F(1,34)=2, p=.166$. Between-subjects analyses of diastolic BP revealed no differences between groups from baseline to post-stress, $F(1,34)=.477, p=.494$. Within-subjects analyses revealed no changes across time, and no time by group interactions,

$F(1,34)=.103, p=.751; F(1,34)=.042, p=.840$. Between-subjects analyses on mean arterial pressure indicated no differences between groups from baseline to post-stress, $F(1,35)=1.720, p=.198$. Within-subject analyses on mean arterial pressure revealed a trend towards change over time from baseline to post-stress manipulation, but there were no time by group interactions $F(1,35)=3.106, p=.087; F(1,35)=.867, p=.358$.

Physiological measures. Paired samples t-tests were used on grand means created from the sitting baseline and post-stress manipulation phase for pulse, SCL, and HR. Analyses revealed a significant difference in pulse from baseline to post-stress manipulation, $t(36)=4.709, p=.001$. The mean pulse measurement during baseline was .009 milivolts (SD=.005), while mean pulse measurement during post-stress manipulation was .005 milivolts (SD=.002). Analyses also indicated a significant difference in mean SCL from baseline to post-stress manipulation, $t(36)=-9.623, p=.001$. The mean SCL during baseline was 2.72 microsiemens (SD=1.81), while mean SCL during the post-stress manipulation was 4.65 microsiemens (SD=2.73). There were no significant differences in mean HR from baseline to post-stress manipulation, $t(36)=-1.506, p=.141$.

Repeated measures ANOVA was used to grand averages created from the sitting baseline and the post-stress manipulation for pulse, SCL, and HR. Between-subjects analyses revealed no differences between group in pulse, $F(1,35)=.756, p=.390$. Within-subjects analyses revealed a significant difference across time in mean pulse, but there were no time by group interactions, $F(1,35)=21.658, p=.001; F(1,35)=.076, p=.785$. Between-subjects analyses revealed no differences in mean SCL from baseline to post-stress, $F(1,35)=1.501, p=.229$. There was a significant change across time for mean SCL, but there were no time by group interactions, $F(1,35)=94.368, p=.001; F(1,35)=2.002, p=.166$. Between-subjects analyses revealed no

differences in mean HR from baseline to post-stress, $F(1,35)=.519, p=.476$. Within-subjects analyses revealed no change over time, but there was a significant time by group interaction in mean HR between groups from baseline to post-stress manipulation, $F(1,35)=2.398, p=.130$; $F(1,35)=6.542, p=.015$. Mean HR of the experimental group at baseline was 77.41 bpm (SD=12.93), and increased to 82.90 bpm (SD=10.17) during post-stress manipulation. Mean HR of the control group at baseline was 83.75 bpm (SD=12.08), and decreased to 82.40 bpm (SD=13.45).

Appendix B

Speech 1 to Post Stress Manipulation

Self-reported affect. A repeated measures ANOVAs was used to assess the interaction of change over time by group from speech 1 to post-post stress manipulation measurements. Between subjects effect reveal no significant differences in mean state anxiety scores between groups, $F(1,33)=.387, p=.538$. Within-subject analysis indicated a significant change across time in mean state anxiety scores, and a trend toward a significant time by group interaction, $F(1,33)=29.564, p=.001$ $F(1,33)=3.272, p=.080$. Specifically, the experimental group's mean state anxiety decreased from 41.55 (SD=13.37) following speech 1 to 30.28 (SD=7.51) following post-stress manipulation. The control group's mean state anxiety scores only decreased from 40.65 (SD=9.35) following speech 1 to 35 (SD=9.45) following post-stress manipulation. Between subjects effects indicate no group difference in mean positive affect scores, $F(1,33)=.777, p=.385$. Within-subject analyses indicated no significant change over time in positive affect scores, but there was a time by group interaction, $F(1,33)=.369, p=.548$; $F(1,33)=4.729, p=.037$. Specifically, mean positive affect scores of the experimental group increased from 27.56 (SD=7.63) following speech 1 to 30.17 (SD=7.45) following post-stress manipulation. Whereas mean positive affect scores of the control group decreased from 27.6 (SD=6.03) following speech 1 to 26.12 (SD=7.87) following post-stress manipulation. Between subjects effects reveal no significant differences in mean negative affect scores, $F(1,33)=.025, p=.875$. Within-subjects analyses indicated a significant change over time, but there was no significant group by time interaction in negative affect scores, $F(1,33)=7.789, p=.009$; $F(1,33)=2.338, p=.136$. However, the means indicate that the experimental groups negative affect scores decreased from 15.89 (SD=9.02) following speech 1 to 11.06 (SD=1.92) following

post-stress manipulation. The control group's mean negative affect scores only decreased from 14.41 ($SD=4.37$) following speech 1 to 13 ($SD=3.69$) following post-stress manipulation.

Blood pressure. Repeated measures ANOVA were also used on all physiological measures to assess changes from speech 1 to post stress manipulation. Between subjects analyses also revealed no significant differences between groups in mean systolic BP, $F(1,32)=1.794$, $p=.190$. Within-subjects analyses indicated a significant change across time in systolic BP measurements, but there was no time by group interactions, $F(1,32)=17.276$, $p=.001$; $F(1,32)=.044$, $p=.836$. Between subjects analyses confirm no differences by group in mean diastolic BP, $F(1,32)=.281$, $p=.599$. Furthermore, within-subjects analyses indicated a significant change across time in mean diastolic BP, but no time by group interactions, $F(1,32)=134.023$, $p=.001$; $F(1,32)=.010$, $p=.922$. Between-subjects effects revealed no differences between groups in mean arterial pressure, $F(1,35)=1.473$, $p=.233$.

Physiological measures. Between-subjects analyses revealed a significant difference between groups in pulse from baseline to speech 1, $F(1,35)=5.224$, $p=.028$. Between subjects effects indicated group differences in SCL, $F(1,35)=.647$, $p=.427$. Within-subjects analyses of SCL from speech 1 to post stress manipulation reveal no significant change across time, but there was a time by group interaction, $F(1,35)=.008$, $p=.931$; $F(1,35)=4.979$, $p=.032$. Descriptive statistics reveal that the mean SCL for the experimental group increased from 4.81 ($SD=3.72$) following speech 1 to 5.22 ($SD=3.52$) following post-stress manipulation. Whereas the control group's mean SCL decreased from 4.5 ($SD=2.06$) following speech 1 to 4.05 ($SD=1.39$) following post-stress manipulation. Between subjects effects confirm no group differences in mean HR, $F(1,35)=.505$, $p=.482$. Lastly, within subjects effects on mean HR revealed a

significant difference across time, but there were no time by group interactions, $F(1,35)=96.650$, $p=.001$; $F(1,35)=.918$, $p=.345$.

Appendix C

Speech 2 to Post Stress Manipulation

Self-reported affect. A repeated measures ANOVA was used to assess changes in time in self-reported measures from speech 2 to post-stress manipulation. Between subjects effects confirm no group differences in mean state anxiety scores, $F(1,35)=1.172, p=.286$. Within subjects analyses reveal a significant change across time in mean state anxiety scores, but this change were no time by group interactions, $F(1,35)=29.553, p=.001$; $F(1,35)=1.242, p=.273$. Between-subjects effects revealed no differences between groups, $F(1,35)=.370, p=.547$. Further, within subjects analyses of positive affect scores from speech 2 to post-stress manipulation revealed a significant change across time, but no significant time by group interaction, $F(1,35)=4.272, p=.046$; $F(1,35)=8.265, p=.007$. Specifically, the mean positive affect score of the experimental increased from 26.21 (SD=7.15) following speech 2 to 29.95 (SD=7.3) following post stress manipulation. However, the mean positive affect score for the control group only increased from 27 (SD=7) following speech 2 to 26.39 (SD=7.72) following post-stress manipulation. Between subjects effects confirmed no group differences in mean negative affect scores, $F(1,35)=.330, p=.570$. Lastly, within subjects analyses of mean negative affect score from speech 2 to post-stress manipulation revealed a significant difference across time, but no time by group interaction, $F(1,35)=11.224, p=.002$; $F(1,35)=2.253, p=.142$.

Blood pressure. A repeated measures ANOVA was used on all physiological measures to assess differences in change over time from speech 2 to post-stress manipulation. Between subjects analyses reveal a trend towards group differences in mean systolic BP, $F(1,34)=3.177, p=.084$. Within-subjects analysis of systolic BP reveal a significant change across time, but no time by group interaction, $F(1,34)=16.449, p=.001, F(1,34)=.032, p=.858$. Between subjects effects confirm no group differences in diastolic BP by group from speech 2 to post-stress

manipulation, $F(1,34)=.174, p=.679$. Within subjects effects of diastolic BP reveal a significant change across time, but no time by group interaction, $F(1,34)=143.628, p=.001$; $F(1,34)=1.350, p=.253$. Between subjects effects confirm no group differences in mean arterial pressure, $F(1,35)=1.907, p=.176$. Within-subjects effects of mean arterial pressure indicate a significant change across time, but no time by group interaction, $F(1,35)=124.072, p=.001$; $F(1,35)=.189, p=.667$.

Physiological measures. Between subjects effects revealed no group differences in mean pulse from speech 2 to post-stress, $F(1,35)=1.435, p=.239$. Between subjects effects revealed no group differences in mean SCL, $F(1,35)=1.093, p=.303$. Between subjects effects confirm no group differences in mean HR, $F(1,35)=.420, p=.521$. Lastly, within subjects analyses on mean HR indicate a significant difference from speech 2 to post-stress manipulation, but no time by group interaction, $F(1,35)=106.562, p=.001$; $F(1,35)=.490, p=.488$.