A Randomized Controlled Trial of Ujjayi Breath and Paced Breath has no Effect on Cardiovascular Reactivity to Mental Stress in Young Adults



Maggie A. Colicchio

April 3, 2017

Submitted to Allegheny College Neuroscience Department in partial requirement for Bachelor of Science degree in Neuroscience

Acknowledgments

I would like to thank Professor Sarah Conklin and Professor Jeffrey Hollerman, my research supervisors, for their patient guidance, enthusiastic encouragement and useful critiques of this research work. I would also like to extend my thanks to the participants of this study, who took the time out of their busy schedules to take part in this study.

Finally, I wish to thank my mom and closest friends from school and home for their endless support and encouragement throughout my study.

It has been a truly challenging, but fulfilling project.

Table of Contents

Abstract 4
A Randomized Controlled Trial of Ujjayi Breath and Paced Breath has no Effect on Cardiovascular Reactivity to Mental Stress in Young Adults 5
The Autonomic Nervous System 6
Respiration 8
Pranayama 11
Mechanism of Pranayama 14
Methods 17
Participants 17
Breathing Task 17
Measures 19
Results 21
Discussion 22
Conclusions and Future Research 28
References 29
Appendix 40

Abstract

The cardiovascular reactivity hypothesis proposed hyperactivity of the sympathetic nervous system to acute stress as a nontraditional, psychosocial risk factor for the development of cardiovascular disease - the leading cause of mortality in the United States. Pramayana has been observed to increase parasympathetic activity, in turn decreasing sympathetic activity, reducing the risk for CVD. The present study compared cardiovascular reactivity to a mental stressor in participants who practiced the pranayama techniques of Ujjayi breathing (n=15) or paced breathing (n=15), to normal breathing (n=15). Heart rate, systolic blood pressure and diastolic blood pressure were recorded while participants performed a validated stress task. No significant differences of cardiovascular reactivity were observed between groups, in opposition to what was predicted. Unexpectedly, the subjective measures of the stress task demonstrated that the Ujjayi breathing and paced breathing groups perceived the stress task as significantly more difficult, stressful and negative, as well as rated their performance as being more poor when compared to the control group. These differences of subjective measures did not accord with the physiology between groups. This suggested that there is some stress relieving benefit to regulating breath because there were no differences between the conditions on any cardiovascular parameter during the mental stress task, even though the two breathing groups rated their perceptions of the task as significantly more difficult, stressful, and negatively impactful. This study supported the notion that pranayama has the potential to reduce cardiovascular reactivity in response to a mental stressor, if the novelty of the breathing practices is eliminated through practice.

A Randomized Controlled Trial of Ujjayi Breath and Paced Breath has no Effect on Cardiovascular Reactivity to Mental Stress in Young Adults

Cardiovascular disease (CVD) is the leading cause of mortality in the United States (Hoyert & Xu, 2012). Several epidemiological studies have determined tobacco use, diabetes, hypertension and hyperlipidemia as leading risk factors for CVD (Lim et al., 2012). However, only 50% of deaths associated with heart disease are linked to these traditional risk factors (Iskandrian & Canto, 2003; Khot et al., 2003). This has peaked interest in nontraditional risk factors of heart disease.

The cardiovascular reactivity hypothesis proposed hyperactivity of the sympathetic nervous system to acute stress as a nontraditional, psychosocial risk factor for the development of cardiovascular disease (Julius, 1993; Light, 1981; Lucini, Norbiato, Clerici & Pagani, 2002; Manuck, Kasprowicz, & Muldoon, 1990). In response to acute stress, the sympathetic nervous system is activated causing increases in oxygen consumption, respiration, blood pressure and cardiac output (Chrousos & Gold, 1992). According to the reactivity hypothesis, if overactivation of the sympathetic nervous system is sustained due to chronic stress, changes in cognitive function, immune system function and hormones are likely to result, increasing susceptibility to disease and poor overall health (Kyrou & Tsigos, 2009). The autonomic dysfunction that stress is proposed to facilitate within the body has been studied with animal and laboratory models (Brunner et al., 2002; Pagani et al., 1991), but research is limited on the effect of human stress on autonomic function (Lucini, Norbiato, Clerici & Pagani, 2002; Mezzacappa Sibolboro, 2001).

The significance of exaggerated reactivity was exhibited in the work of Carroll et al. (2012), that demonstrated increased risk of mortality over the course of 16 years for people with high cardiovascular reactivity (CVR) to mental stress. Steptoe et al. (2016) found that heightened cardiovascular and inflammatory reactivity in response to mental stress also was associated with risk of hypertension. This

suggested that any cognitive or behavioral treatment that could be associated with lowering CVR to mental stress could be protective (Carroll et al., 2012). A study completed at the Harvard Medical School observed that heart attack survivors who were unable to control their anger doubled their risk for future heart attacks, compared to heart attack survivors who were able to control their anger in daily situations (Frentzel-Beyme & Grossarth-Maticek, 2001; Mittleman et al., 1995). Importantly, people who were able to practice self regulatory abilities were observed overtime to be more likely to be living without a chronic disease, compared to those who displayed poor self regulation abilities (Frentzel-Beyme & Grossarth-Maticek, 2001). Therefore, if autonomic function could be regulated under voluntary control, exaggerated sympathetic activity due to mental stress could be quelled and possibly reduce risk of CVD. The objective of this present study was to examine the effects of voluntary methods of regulating respiration through yogic breathing, on cardiovascular reactivity to mental stress.

The Autonomic Nervous System

The autonomic nervous system (ANS) is comprised of the sympathetic and parasympathetic nervous systems (Kreibig, 2010). Through innervation of most of the major internal organs and bodily system, these two branches of the ANS function as complementary systems to promote physiological homeostasis in the face of changing circumstances (Poh et al., 2014). Specifically, the ANS innervates the heart and blood vessels - to control blood pressure and flow, and the bronchi of the lungs - to meet the oxygen demands of the body. The sympathetic division has preganglionic axons that emerge from the lumbar and thoracic segments of the spinal cord. Preganglionic axons of the parasympathetic nervous system emerge from the brain stem and the sacral segments of the spinal cord (Bear, Connors & Paradiso, 2007). The sympathetic nervous system preganglionic axons synapse on neurons in the ganglia of the sympathetic chain, next to the spinal column within the abdominal cavity. This causes innervation on, in or to target organs from neurons that are found in the ganglia of the sympathetic chain. The sympathetic

nervous system tends to be more active during perceived and real crisis (Bear, Connors & Paradiso, 2007; Fox, 2016).

The parasympathetic nervous system's preganglionic axons travel farther than the sympathetic axons to the parasympathetic ganglia, which are located next to, on, or in the target organs that the parasympathetic division innervate. The parasympathetic fibers that are of the vagus nerve contribute to some of the largest parasympathetic effects within the body. The fibers of the vagus nerve synapse in the ganglia near the heart, lungs, gastrointestinal tract, liver, and pancreas (Fox, 2016). Therefore, the parasympathetic nervous system is more active during processes of digestion, growth, immune responses and energy storage. Both divisions of the ANS innervate target organs (e.g., glands, smooth muscle, cardiac muscle) and generally act to oppose each other (Bear, Connors & Paradiso, 2007).

Chronic stress has been found to increase sympathetic activity and cause sustained elevations in blood pressure, which facilitates plaque buildup inside arteries secondary to microvascular injury from the shear stress of elevated pressure (Kaplan, Petterson, Manuck & Olsson, 1991). Increased sympathetic activity also supports and can cause inflammation (Lewthwaite, Owen, Coates, Henderson, & Steptoe, 2002), high cortisol levels (Chrousos, 1995) and other unhealthy behaviors (Everly & Lating, 2002). It is hypothesised that acute stress could cause short, but lasting increases in arterial pressure and heart rate through impairment of endothelial function - the balance of vasoconstriction and vasodilation of blood vessels (Gokce et al., 2002), and reduce the threshold for arrhythmia and even sudden death (Ghiadoni et al., 2000; Rozanski, Blumenthal, Davidson, Saab, & Kubzansky, 2005; Schwartz et al., 2003; Yeung et al., 1991). Acute stress also has been proposed to reduce baroreflex performance - the ability to maintain the body's relatively constant blood pressure (Lucini, Norbiato, Clerici & Pagani, 2002; Mezzacappa Sibolboro, 2001), which caused impairment of this vital autonomic protective mechanism of the body (La Rovere, Biffer, Marcus, Mortara & Schwartz, 1998).

In the laboratory, cardiac autonomic function can be assessed in response to a stressful stimuli by introducing a task that creates cognitive or social stress (Hagins, Haden & Daly, 2013). The degree to which the body responds physiologically to the stress task compared to a pre-stress baseline is defined as stress reactivity. Elevated stress reactivity has been observed in individuals with hypertension (Carroll, Ring, Hunt, Ford & MacIntyre, 2003; Carroll et al., 2001) and atherosclerosis (Barnett, Spence, Manuck & Jennings, 1997; Jennings et al., 2004). The parasympathetic nervous system has the ability to reverse the effects of sympathetic activity (stress reactivity) through the 'relaxation response', which decreases oxygen consumption, blood pressure, heart rate and respiration rate (Wallace, Benson, & Wilson, 1971). If autonomic function can be regulated and immune responses and energy storage can be preserved in the body through voluntary effort to increase parasympathetic activity during acute stress, medical conditions that are associated with overactivity of the sympathetic nervous system during acute stress, can be eliminated or decreased.

Respiration

Respiration includes three functions - ventilation, gas exchange in the lungs and oxygen utilization by tissues of the body. The mechanical process that moves air into and out of the lungs is pulmonary ventilation, or breathing. Pulmonary ventilation is composed of two phases, inspiration and expiration. Both phases are accomplished by the continuous increasing and decreasing volumes of the thorax and lungs. During inspiration, the diaphragm contracts and the external costal muscles increase the thoracic and lung volumes. This lowers intrapulmonary pressure, allowing air to flow into the lungs. During expiration, the diaphragm relaxes and the external intercostal muscles relax. This decreases lung volume and increases pulmonary pressure, causing air to be pushed out of the body (Fox, 2016).

Inspiration and expiration occurs by the contraction and relaxation of the respiratory skeletal muscles. The movement of these muscles is in response to activity of motor neurons in the spinal cord that innervate the respiratory muscles. These motor neurons are controlled by two pathways, one that

controls voluntary breathing and another pathway that controls involuntary breathing. The pathway that contains neurons descending from the respiratory control centers in the cerebral cortex of the brain control voluntary breathing and the neurons descending from respiratory control centers in the medulla oblongata control involuntary breathing (Bear, Connors & Paradiso, 2007; Fox, 2016).

Reflexes in the brain stem regulate respiration patterns by determining the depth of breath, duration of the phases of the breathing cycle, and lung volume at which inspiration begins (Remmers, 1976). Voluntary control of respiration can alter how the nervous system functions, including heart rate variability (Lehrer, Sasaki & Saito, 1999; Sovik, 2000), the sensitivity of the baroreflex and overall excitation of the central nervous system (Carbon, Wübbeler, Trahms, & Curio, 2000).

Functioning of the heart and respiratory system (cardiorespiratory function), brain and muscles depend on oxygen consumption. As mentioned above, oxygen enters the body through the process of respiration. During intensive exercise, oxygen consumption is maximal and during rest, it is at its minimal level. Oxygen consumption influences how much oxygen is delivered to tissues, the metabolic response the body has to activity and cardiorespiratory function (Bonnet & Arand, 2003; McArdle, Katch, & Katch, 2010). Oxygen consumption increases with psychological and physiological activity such as, exercise and REM sleep, and pathology (Epel, 2009; Jumpertz et al., 2011; Olshansky & Rattan, 2005; Ruggiero et al., 2008). Specifically, congestive heart failure (Poehlman, Scheffers, Gottlieb, Fisher, & Vaitekevicius, 1994), diabetes (Huang, Kormas, Steinbeck, Loughnan & Caterson, 2004) and hypertension (Kunz, Schorr, Klaus & Sharma, 2000; Snodgrass, Leonard, Sorensen, Tarskaia, & Mosher, 2008) are pathological conditions associated with increased oxygen consumption. Interestingly and importantly, increased oxygen consumption has also been linked to anxiety and activities that evoke psychological stress, such as, video gaming and cognitive challenge in the laboratory (Balanos, 2009; Carroll, Phillips & Balanos, 2009; Van Diest et al., 2006).

Variations in breathing patterns have been associated with psychopathology and emotional states (Boiten, Frijda, & Wientjes, 1994; Boiten, 1998), indicating that emotions are involved in the regulation of breath (Wientjes & Grossman, 1998). During labor, women are instructed to steady their breath and to focus on their breath to relieve some of the pain from labor. Slow, patterned breathing has been shown to relieve pain during labor and to also help promote a more positive perception of labor pain (Chuntharapat, Petpichetchian, & Hatthakit, 2008). Slowed breathing has also been shown to enhance mood and stress tolerance in individuals suffering from depression, posttraumatic stress disorder (Brown & Gerbarg, 2005) and even epilepsy (Streeter, Gerbarg, Saper, Ciraulo, & Brown, 2012). Therefore, respiration rate has the potential to serve as a possible physiological parameter that can be voluntarily regulated, in order to assist in fighting harmful health effects of environmental stressors.

Respiration has been shown to facilitate a balance of sympathetic activity and parasympathetic activity (Berntson et al., 1997; Berntson, Cacioppo, & Quigley, 1993; Grossman & Kolla, 1993; Porges, 1995; Wenger & Cullen, 1972). Oxygen consumption increases during stress because sympathetic action increases both heart rate and blood pressure (Lambert & Lambert, 2011;Licht et al., 2010; Vaccarino & Bremner, 2005). However, the parasympathetic nervous system works to reverse the stress response created by the sympathetic nervous system in order to restore the body back in its homeostatic range via decreased oxygen consumption, which enhances vagal activity (Dusek & Benson, 2009).

Reduction in sympathetic nervous system activation has been demonstrated through decreased heart rate observed during slow respiration (Harris, Katkin, Lick, & Habberfield, 1976; McCaul, Solomon, & Holmes, 1979). Because of the beneficial health outcomes, simplicity and cost effectiveness of slowed and controlled breathing, it is used often within the health field as part of intervention methods to combat negative physiological effects that psychological disorders have on the body (Brown, Gerbarg & Muench, 2013; R. Jerath, V. Jerath, Edry, & Barnes, 2015; S. Zope & R. Zope, 2013). Specifically, yogic breathing techniques that involve the voluntary control of retention and respiration rate, could play

a role in reducing oxygen consumption during experiences of psychological stress (Tyagi, & Cohen, 2013), indicating that controlling breathing rate is a behavior that is beneficial to health (Fried, 1993; Timmons & Ley, 1994). Currently, there is a lack of controlled research on voluntary regulation of breath and how its practice influences physiological activity- especially during mental stress.

Pranayama

Ashtanga Yoga, a classical form of Indian yoga, is comprised of eight limbs intended to benefit physical and psychological health. The 3rd of the eight limbs is the practice of yogic breathing - pranayama (Hiriyanna, 2002). Prana meaning "vital force or breath" and asana meaning "to prolong" (Telles, Joseph, Venkatesh, & Desiraju, 1993). Yogis viewed breathing regulation as a way to steady mental force (Muktibodhananda, 2002) and balance the body (Chuntharapat, Petpichetchian, & Hatthakit, 2008), in order to return the body to its homeostatic state (McEwen, 2007). Performing the practice of yogic breathing has been observed to contribute to improvements in blood pressure, immunity (Kochupillai et al., 2005), glucose metabolism and cognitive functioning (S. Zope & R. Zope, 2013). This is in accord with the idea that continuous practice of yogic breathing assists in reaching optimal human performance.

Stress can cause both physiological and psychological changes, resulting in evident diminution in human performance. Within the human body, the cardiovascular system, the autonomic system and metabolic functioning operate under physiological reserves. Pranayama has been observed to have preservation and restoration effects on the compensatory mechanisms that maintain physiological functions within the body (K. Carter & Carter, 2016). Specifically, by practicing pranayama, autonomic nervous system functions such as heart rate, cardiac vagal tone (Fokkema, 1999; Lehrer et al., 1999; Sovik, 2000) and central nervous system excitation (Carbon et al., 2000) have demonstrated the effect yogic breathing has on cardiovascular parameters.

Reduced respiratory chemoreceptor sensitivity, which accounts for the variation of breathing rate in response to oxygen and carbon dioxide in the body (Spicuzza, Gabutti, Porta, Montano, & Bernardi, 2000) and excitation of the baroreflex, one of the body's mechanisms to maintain blood pressure within homeostatic range (Carbon et al., 2000), has also demonstrated how yogic breathing increases vagal stimulation and effects the functioning of the nervous system. Specifically, through alteration of the functionality of the parts of the autonomic nervous system, lower heart rates and larger decreases in blood pressure have resulted from pranayama when compared to simply breathing with awareness (Telles, Singh & Balkrishna, 2011; Raghuraj & Telles, 2003). Therefore, suggesting pranayama practice as a beneficial addition to treatment for CVD, along with imbalances within the autonomic nervous system (Pal, Velkumary, & Madanmohanet, 2004), stress related and psychological disorders (Brown, & Gerbarg, 2005).

Pranayama has been observed to increase parasympathetic nervous system activity (Pilkington, Kirkwood, Rampes, & Richardson, 2005) within healthy participants (Dhungel, Malhotra, Sarkar, & Prajapati., 2008; Subbalakshmi, Saxena, Urmimala, & Urban, 2005), participants with asthma (Cooper et al., 2003) and irregular heart beats (Ravindra & Madanmohan, 2006). Innes, Bourguignon, and Taylor (2005) have proposed the mechanism of action behind these benefits to be vagal stimulation. Increased risk of cardiovascular disease and death after myocardial infarction have been associated with decreased vagal stimulation (La Rovere et al., 1998), indicating vagal modulation as vital for cardiovascular recovery from psychological stress (Mezzacappa et al., 2001). Because pranayama shifts the function of the nervous system to be more parasympathetic through increased vagal activation (Pilkington et al., 2005) and therefore decreased heart rate, it helps the body to only appropriately activate the sympathetic nervous system. This results in preservation and restoration of the "physiological reserve" that is construed as the compensatory mechanisms assessable to maintain physiological function, to protect

against development of diseases (Poh et al., 2014). This, suggests a mechanistic explanation why yoga has been found to be an effective stress management technique (Kirkwood et al., 2005).

There are many forms of pranayama, which all include three specific phases - inhalation (*puraka*), retention (*kumbhaka*), and exhalation (*rechaka*). The duration of these phases can be long or brief (Jerath, 2006). Decreased oxygen consumption, heart rate and blood pressure have been observed as positive, immediate effects of slow, deep, pranayama (Jerath, 2006; Madanmohan et al., 2005; Telles & Desiraju, 1991; Upadhyay-Dhungel, Malhotra, Sarkar, & Prajapati, 2008). Jerath et al. (2006) and Ankad, Herur, Patil, Shashikala, & Chinagudi (2011) proposed that pranayama resets the nervous system over time. Increased oxygen consumption is associated with brief breath retention and decreased oxygen consumption has been associated with long breath retention, indicating different effects on autonomic activity (Telles & Desiraju, 1991). Pranayamas have been shown to reduce symptoms of anxiety and stress (Brown & Gerbarg, 2005), which lead to pranayama techniques being used with the goal of reducing arousal through decreasing sympathetic activity and prompting vagal nerve activity (Telles et al., 2013; Van Dixhoorn, 1998).

For example, there is high frequency yogic breathing, referred to as Bhastrika or Bellows Breath, which has been associated with increases in cardiac output and respiration (Telles, Singh & Balkrishna, 2011). Bhastrika Breath, also referred to as fire breath, is the technique of intensely, inhaling and exhaling quickly. This breathing technique has shown to cause excitement of the central nervous system and activation of the sympathetic nervous system, hence its effect on cardiac output and respiration (Roldan & Dostalek, 1983; Roldan & Dostalek, 1985). The moderate sympathetic activation that Bhastrika Breath causes has shown to increase the capacity of the body's stress response without depleting the body (Brown & Gerbarg, 2005), indicating the effect of yogic breathing on the preservation and restoration of the body's compensatory mechanism to maintain physiological functioning.

Divergently, Ujjayi or victorious breath, involves longer retention of breath. This pranayama technique decreases airflow and focuses on the slight contraction of the laryngeal muscles and partial closure of the glottis, located in the pharynx. This causes mild airway resistance and thus, a sensation of pressure in the back of the throat during inspiration and expiration (Brown & Gerbarg, 2005; Jovanov, 2005; S. Zope and R. Zope, 2013). This airway resistance stimulates somatosensory vagal nerves that begin in the pharynx and lung tissue (Ruan, Ho & Kou, 2003). When these vagal afferents are activated, this parasympathetic sensory information is sent to the brain and causes the respiratory centers within the medulla that affect involuntary respiration, to prolong the phases of inspiration and expiration (Calabrese, Perrault, Dinh, Eberhard, & Benchetrit, 2000). Moderate airway resistance has shown to increase stimulation of the parabrachial nucleus (PBN), located at the junction of the midbrain and pons in the brain stem. The PBN receives nerve input from the vagus nerve innervation that is within the chest wall stretch receptors (Gozal et al., 1995). This is a proposed reason why Ujjayi breathing or pranayama in general, creates a shift in autonomic function as it affects the pulmonary stretch receptors within the lungs that facilitate parasympathetic activity.

Mechanism of Pranayama

It has been frequently observed that pranayama techniques have the ability to reduce heart rate and blood pressure at rest and in response to environmental stressors. Several theories have been proposed to address the possible autonomic nervous system modulation, which occurs when slow pranayamic breathing is practiced. The nature of the practice of pranayama is slow and deep breathing. This produced an overall decrease in the sympathetic pathways regulating cardiovascular systems via activation of autonomic and central nervous system mechanisms, as well as hemodynamic and contraction and relaxation adjustments of the heart (Audimulapu, 2016).

Autonomic functions such as breathing pattern and heart rate are influenced by inhibitory vagal nerve impulses in the lungs (Schelegle & GreenSchelegle, 2001). Pranayamic breathing increased the

amount of air inhaled during respiration, which caused the inhibition of slowly adapting pulmonary stretch receptor (SAR) activity within the lungs (Audimulapu, 2016; Matsumoto et al., 2000). Inhibitory currents controlled stimulation of neural tissues (Cuttle, Rusznak, Wong, Owens, & Forsythe, 2001) and created synchronized activity between networks within the brain (Westbrook, Kandel, Schwartz, & Jessell, 2000), such as the hypothalamus, brainstem (Newberg, & Iversen, 2003), heart, lungs and limbic system (Bhimani, Kulkarni, Kowale, & Salvi, 2011), which reduced sympathetic tone in the blood vessels of skeletal muscles. Thus, increased parasympathetic activity (Busek & Kemlink, 2005; Lutz, Greischar, Rawlings, Ricard, & Davidson, 2004) resulted in generalized vasodilatation and reduction in peripheral resistance, decreasing blood pressure and heart rate (Audimulapu, 2016).

Pranayama also stretched connective tissue around the lungs which caused widespread hyperpolarization. Hyperpolarization regulated the excitability of neurons within the heart, lungs, and limbic system and prevented unsynchronized neural activity (Bhimani et al., 2011). Through synchronization between brain and cardiorespiratory function (Siegelbaum, & Robinson, 2003), the occurrence of decreased metabolic activity indicated a shift in autonomic function to increased parasympathetic activation (Audimulapu, 2016). However, the specific mechanism that was the cause of the autonomic nervous system shift remains unknown (Jerath, 2006).

Therefore, it was plausible to believe that pranayama techniques could reduce cardiovascular reactivity to a mental stress task. If pranayama was practiced, a shift in autonomic function to more parasympathetic activity should have been observed through physiological responses, such as decreased heart rate and blood pressure. Ujjayi pranayama, compared to paced breath and unregulated breath, was believed to lead to indications of whether decreased airflow and focus on the slight contraction of the laryngeal muscles and partial closure of the glottis increased parasympathetic activity, thus causing a reduction of cardiovascular reactivity, more than simply paced breath to elongate exhalation when mental stress was induced.

The current study compared Ujjayi breathing and paced breathing, to unregulated breathing during a mental stress task to examine if pranayama contributed to decreased cardiovascular reactivity. It was hypothesized that practices of pranayama techniques - Ujjayi breathing and paced breathing- would decrease cardiovascular reactivity, compared to unregulated, normal breathing, indicating an increase of parasympathetic activity.

Larger decreases in heart rate, systolic blood pressure and diastolic blood pressure were predicted to be observed in the Ujjayi breathing treatment group, when compared to the paced breathing treatment group and control group because of the element of concentration and manipulation of air flow that is specific to that pranayama technique. Concentration should have caused greater overall relaxation and therefore, a larger decrease in heart rate, systolic blood pressure and diastolic blood pressure in response to a cognitive stressor. It was expected that there would be no changes in heart rate, systolic blood pressure and diastolic blood pressure measurements and therefore, no change in cardiovascular reactivity observed in the unregulated breathing group.

The Perceived Stress Survey (PSS) was used to assess stress before the testing session in the pranayama conditions and control group and then also to assess stress after the testing session in only the pranayama conditions. Overall, the (PSS) scores were predicted to not differ across conditions before the testing session, to indicate that all participants were experiencing the same amount of stress. Specifically, the lowest PSS scores were to be observed in the Ujjayi breathing, when compared to the paced breathing group after the testing session because of the element of concentration and manipulation of air flow specific to this pranayama technique.

Methods

Participants

Young adult, college undergraduates ranging from ages 18-22 years of both sexes were recruited. Exclusion criteria included current or history of respiratory or cardiovascular illnesses, experience with pranayama practices and habitual smoking defined as smoking more than 5 days a week, which was obtained through a questionnaire on Allegheny's SONA website. All participants were instructed to complete the Perceived Stress Survey (PSS) -a self-report, 14-question measure used to assess their perception of the degree of stressfulness before the start of the lab testing. Then, the participants were randomly assigned to one of three testing groups: (i) Ujjayi breathing, (ii) paced breathing, and (iii) unregulated, normal breathing (control). Each participant was instructed on how to perform the specific breathing technique through short instruction videos. The control group did not have any exposure to these videos or any other instruction on how to breathe. Informed written consent was obtained from all participants.

Breathing Task

Participants were randomly assigned to one of the three possible sessions, (i) Ujjayi breathing with a 1:2, inhalations-exhalations ratio ; (ii) paced breathing with a 1:2 inhalations-exhalations ratio, and (iii) natural, unregulated breathing. All testing took place during a single lab session for each participant.

Participants were tested during one time trial, during which they were seated in an upright position on a chair in front of a computer screen. Blood pressure, heart rate and respiration pattern data were continuously collected throughout each trial (Critikon Dinamap Vital Signs Monitor 1846 SX, Critikon, Inc., Tampa, FL). The participants who were randomly assigned to Ujjayi breathing were first instructed to watch a 45 second video that showed how to perform the breathing technique. A second

video was also shown to this group that instructed each participant on how to pace their breath. This video consisted of a moving circle that expanded in and out. Participants were instructed to inhale as the circle expanded and to exhale as the circle contracted. This video played throughout the entirety of the time trials of this group to serve as a breathing pacer. The paced breathing group only viewed this breath pacer video, which was also played throughout the entirety of that group's time trials to serve as a breath pacer.

Participants in both group 1 and 2 participated in three main phases: (i) "Baseline" (10 min), (ii) "Stress" (10 min), and (iii) "Recovery" (10 min). Depending on what testing group the participant was randomly assigned, the baseline phase involved 10 minutes of either paced, Ujjayi breathing or general, paced breathing. The stress phase included The Paced Auditory Serial Recognition Task (PASAT), a simple yet demanding math arithmetic task (Grownwall, 1977; Ring, Burns, & Carroll, 2002; Winzer et al., 1999) for a 10 minute phase. During this, participants continued to breath as the video/s instructed, following the visual breath pacer. Finally, the recovery phase took place for 10 minutes during which participants also continued breathing as the video/s instructed, while still following the visual breath pacer. After the recovery phase was complete, each participant in the pranayama conditions were asked to complete the Perceived Stress Survey (PSS) for a second time. They also completed the Post-PASAT survey, which is specific to the PASAT. Participants completed a survey that was created to indicate how the breathing techniques were percepted, as well.

The participants who served as controls (unregulated breathing) did not have any specific instructions other than just to sit during the baseline phase, verbally answer the math questions to their best ability during the stress phase and to rest during the recovery phase. Nothing was communicated to the control group about how to breathe. They were also asked to complete the Post-PASAT survey after the end of the testing session.

Blood pressure and heart rate were measured every 3 minutes throughout the entire time trial using an automated blood pressure cuff (Critikon Dinamap Vital Signs Monitor 1846 SX, Critikon, Inc.,

Tampa, FL) in all three groups. A respiration strain gauge monitor (iWorx Systems Inc., Dover, NH) was used to record breathing patterns continuously throughout the entire length of the lab session in groups 1 and 2 only.

Measures

During testing, the participants in groups 1 and 2 wore an RM-204 Respiration Monitor (iWorx Systems Inc., Dover, NH) that measured respiration. This monitor was used to control for physical effort while performing the breathing techniques. Blood pressures and heart rates were measured using an automated blood pressure cuff (Critikon Dinamap Vital Signs Monitor 1846 SX, Critikon, Inc., Tampa, FL) in all three groups. Cardiovascular reactivity was calculated by subtracting baseline phase means (HR, resp rate, SBP, DBP) from stress phase means.

Systolic blood pressure (SBP) and diastolic blood pressure (DBP) were recorded using an automated brachial cuff (Critikon Dinamap Vital Signs Monitor 1846 SX, Critikon, Inc., Tampa, FL) for every 3 minutes during each 10 minute phase in all three groups. Heart Rate was measured every 3 minutes for each 10 minutes phase as well, using the automated brachial cuff (Critikon Dinamap Vital Signs Monitor 1846 SX, Critikon, Inc., Tampa, FL) in all three groups.

Respiration was continuously monitored throughout the entire testing session in groups 1 and 2 only, as mentioned before by the RM-204 Respiration Monitor (iWorx Systems Inc., Dover, NH). This monitor was placed around each participant's chest, directly below the sternum. The RM-204 Respiration Monitor was a transducer which will be used to measure the depth and frequency of breathing in the participants. Labscribe software was used to be able to generate depictions of respiration patterns (iWorx Systems Inc., Dover, NH). This monitor was used to control for physical effort while performing the breathing techniques through the visual depictions Labscribe software created. Graphs of respiration patterns showed the breathing patterns of all participants. This served as evidence that breathing was controlled through these yogic breathing techniques.

The PASAT creates acute mental stress that has shown high reliability (Willemsen et al., 1998). There are two forms of this test, 3" PASAT and 2" PASAT. Each form consists of a set of single digit numbers which are dictated through an audio recording every 3 seconds for the 3" PASAT or every 2 seconds for the 2" PASAT. Each new number presented is to be added to the number before it. The results of the test are based on the number of correctly summed numbers out of 60 (Gronwall, 1977; Ring, Burns, & Carroll, 2002; Winzer et al., 1999).

The Perceived Stress Survey (PSS) is a self-report, 14 question measure designed to assess an individual's perception of the degree of stressfulness during a given situation. The PSS contained seven positively worded 'stress' items (e.g., In the past month, how often have you felt nervous or stressed?) and seven negatively worded 'contra-stress' items (e.g., In the past month, how often have you felt that things were going your way?). Each item was rated on a 5-point rating scale of occurrence of these statements over the past month (0=never, 1=almost never, 2=sometimes, 3-fairly often, 4=very often) (Chiu et al., 2016).

The Post-PASAT survey is a 14 item questionnaire that indicated each participant's perception of the task using seven positively worded items (e.g., How stressful did you find the task?) and seven negatively worded items (e.g., I had a sense of control over this task). Each item was rated on a 6-point rating scale (e.g., 0=not stressful, 6=extremely stressful).

The perception of breathing techniques survey that was created and used to indicate how the breathing techniques were perceived by the participants in the present study. This survey included three positively worded items (e.g. How stressful did you find the breathing) and two negatively worded items (e.g., How well do you think you performed the breathing?). Each item was rated on a 6-point rating scale (e.g., 0= not at all difficult, 6= extremely difficult).

Results

Table 1 shows participant demographic, stress and PASAT performance data. There were no differences between group in age. The percentage of participants in each testing condition did not differ by gender, $\chi^2(2, N = 45) = 5.2$, $p = .08$.

As can be seen, there were unexpected significant differences between the paced breathing group and control group on PSS score (*ps* < .05). The paced breathing group scored higher on the PSS before the start of the testing session. There was no difference in preliminary PSS score between the Ujjayi breathing group and control group, nor in the post-testing PSS score between the Ujjayi breathing and paced breathing group. There were also unexpected significant differences between the Ujjayi breathing group and control group in PASAT score (*ps* < .05). The Ujjayi breathing group performed worse on the PASAT. There was no difference between the paced breathing group and control group in PASAT score.

Table 2 shows participant respiration rates (breaths/minute) during each phase and subjective measures of breathing difficulty, stressfulness, performance, embarrassment, and enjoyment. As can be seen, participants within both breathing conditions were compliant with the task of following the breath pacer to regulate their breath to 6 breaths/minute. Independent samples t-test revealed no significant group differences between the two breathing groups on any measure of subjective breathing difficulty, stressfulness, performance, embarrassment or enjoyment. As indicated, the means and standard deviations of these characteristics are displayed in Table 2.

As expected, a main effect of phase was observed for systolic blood pressure ($F(2, 84)= 80$), $p <.001$, $\acute{\eta}_p^2 = .66$), diastolic blood pressure ($F(2, 84)= 111$), $p <.001$, $\acute{\eta}_p^2 = .73$) and heart rate ($F(1.7, 71) = 26$, $p <.001$, $\acute{\eta}_p^2 = .38$), indicating stress phase means were higher than both baseline (all *ps* < .05) and recovery phase means (all *ps* < .05).

In order to test the primary study hypothesis that practicing Ujjayi breathing and paced breathing

will decrease cardiovascular reactivity during a mental stress task, a mixed-design analysis of variance was conducted. Condition served as a between subjects independent variable (Ujjayi, paced, control), while cardiovascular measurements (systolic blood pressure, diastolic blood pressure and heart rate) obtained during each task phase (baseline, stress, recovery), served as dependent variables. The phase by condition interaction was not significant (all *ps* > .05) for all three cardiovascular measures, indicating no support for the study hypothesis (Figure 1).

A one way analysis of variance did reveal significant main effects of testing condition on measures of subjective PASAT difficulty ($F(2,42)= 3.4$, $p=.04$), stressfulness ($F(2, 42)= 3.8$, $p=.03$), performance ($F(2, 42)= 6.2$, $p= .004$), negativity of experience ($F(2, 42)= 3.6$, **p**= .04) and negativity of impact ($F(2, 41)= 4.0$, $p=.03$). A Tukey post hoc test revealed that the subjective PASAT difficulty score was significantly higher in the paced breathing testing group, compared to the control testing group. A Tukey post hoc test revealed that the subjective PASAT stressfulness, negativity of experience and negativity of impact scores were significantly higher in the Ujjayi breathing testing group compared to the control testing group. A Tukey post hoc test revealed that the subjective PASAT performance score was significantly lower in the Ujjayi breathing testing group, compared to the control testing group (Figure 2). Group differences in subjective measures of PASAT difficulty, stressfulness, performance and negativity of impact and experience were unexpected. All other subjective PASAT items were not different between conditions (all *ps* > .05).

Discussion

The present study aimed to examine the effects Ujjayi pranayama and slow paced breathing, on cardiovascular reactivity to mental stress. Contrary to expectations, Ujjayi breathing and paced breathing did not significantly influence systolic blood pressure, diastolic blood pressure, or heart rate compared to unregulated breathing during a mental stressor in the laboratory. These findings are not consistent with other studies that tested the effects of Ujjayi breathing on blood pressure and heart rate. Lathadevi, Uma

Maheswari, & Nagashree (2012) reported 6-weeks of Ujjayi pranayama as a way to significantly decrease heart rate and blood pressure, which was also in accordance with other studies (Pramanik et al., 2009; Pramanik, Pudasaini & Prajapati, 2010). Lathadevi, Uma Maheswari, & Nagashree (2012) suggested that Ujjayi pranayama decreases airflow and focuses on the slight contraction of the laryngeal muscles and partial closure of the glottis, located in the pharynx. It is suggested that Ujjayi breathing causes mild airway resistance and thus, a sensation of pressure in the back of the throat during inspiration and expiration (Brown & Gerbarg, 2005; Jovanov, 2005; Ruan, Ho & Kou, 2003; S. Zope and R. Zope, 2013). The airway resistance stimulates somatosensory vagal nerves that begin in the pharynx and lung tissue. When vagal afferents are activated, this parasympathetic sensory information is sent to the brain and causes the respiratory centers within the brain that affect involuntary respiration, to prolong the phases of inspiration and expiration (Calabrese et al., 2000). Moderate airway resistance has shown to increase stimulation of the parabrachial nucleus (PBN), located at the junction of the midbrain and pons in the brain stem. The PBN receives nerve input from the vagus nerve innervation that is within the chest wall stretch receptors (Gozal et al., 1995). This is a proposed reason why Ujjayi breathing or pranayama in general, creates a shift in autonomic function as it affects the pulmonary stretch receptors within the lungs.

However, it is possible that because Ujjayi breathing reduces airflow and increases intrathoracic pressure during expiration due to the slight contraction of the glottis muscles, Ujjayi breathing has the potential to increase intensity of vagal activity (Brown & Gerbang, 2005; Ruan, Ho & Kou, 2003). Oxygen absorption could also be enhanced more because of the increase in expiratory intrathoracic pressure during Ujjayi breathing, compared to slow breathing. This could cause blood pressure to rise more than during slow breathing, enhancing baroreflex sensitivity (Telles & Naveen, 2008). However, increased intrathoracic pressure causes more effort needed to exhale and this potentially could stimulate

sympathetic activity. This could be a reason as to why Ujjayi pranayama did not significantly lower cardiovascular reactivity in the Ujjayi breathing group as was hypothesized in the present study.

The findings from the present study are partially consistent with what Tomar and Singh (2011) found when they investigated the effect of Ujjayi breathing on resting blood pressure, resting heart rate, resting pulse rate, resting respiratory rate, vital capacity, and breath holding time over an 8-week period. There was no effect of Ujjayi breathing observed in resting respiratory rate or blood pressure. However, there was a significant decrease in resting heart rate and pulse rate over an 8-week period of practicing Ujjayi breathing, indicating that 8-weeks may have been too short of a time period to observe significant effects on resting blood pressure and the other physiological parameters. This may be why the present study did not show significant decreases in heart rate reactivity and blood pressure reactivity after the Ujjayi breathing and paced breathing testing sessions - the duration of the task was too short to change physiology significantly and the introduction of novel breathing practices could have affected the results of the present study. Other studies have demonstrated that the length of time spent engaged in yoga programs that include Ujjayi pranayama, yoga postures and meditation has an effect on the degree of benefit that medical patients with anxiety disorders (Miller, Fletcher & Kabat-Zinn, 1995), medical students with examination anxiety, (Malathi & Damodaran, 1999) and caregivers of patients with dementia (Waelde, Thompson, & Gallagher- Thompson, 2004) receive. In addition, Cowen and Adams (2007) observed that Ujjayi breathing was difficult for beginners to maintain throughout entire ashtanga posture series and that this breathing style could cause increased body temperature and perspiration. This could explain why higher heart rates are observed sometimes during this pranayama technique (Cowen & Adams, 2007).

However, the present study did find that Ujjayi breathing and paced breathing were associated with participants responding significantly different to five of the subjective Post-PASAT measures. This

demonstrates participant's perception of the mental stress task to be less positive than expected in the Ujjayi breathing and paced breathing conditions, when compared to the unregulated breathing condition.

The participants within the paced breathing group reported the mental stress task to be significantly more difficult than the control group. The participants in the Ujjayi breathing group reported the mental stress task to be significantly more stressful and experienced significantly more negativity from the experience than the control group. The Ujjayi breathing group also rated their perceived performance as significantly lower than the control group. Therefore, what has been found to be "relaxing" breathing techniques (Sathidevi, Uma Maheswari & Nagashree, 2012) were found to be perceived as more difficult, stressful and negative. These results coincide with a study that was done by Busch et al. (2012) that compared different ways of pacing breathing. Busch et al. (2012) reported that subjects who were required to maintain full concentration during a testing session, in order to try to match their respiration curve to the ideal respiration curve presented on monitors, demonstrated more sympathetic activity. These subjects were compared to subjects who were only instructed to breath in accord to verbal directions and to direct their attention to their breathing, not any visual guide. These findings could be explained by how the attentional demands of the respiratory feedback task maintained arousal. While this procedure of pacing breath with a visual guide may help to get the breath where is thought to be ideal for human health, this approach may influence heart rate variability during an emotional or cognitive task because of the duel attention required while consciously following a breath cue, as well as a task. This dual attention phenomenon has been observed to significantly increase task difficulty (Pashler, 1994). Increases in sympathetic nervous system activity have also been observed during a cognitive task, influencing cardiorespiratory coupling during a cognitive task (Zhang et al., 2010). Cappo and Holmes (1984) demonstrated this proposed phenomenon when sympathetic arousal was increased during a breathing exercise that was accomplished by consistent attentiveness. In accordance with this finding, paced respiration in combination with an attention tracing condition caused no reduction

in stress responses (Holmes, McCaul, & Solomon, 1978) or skin conductance responses. However, breathing without concentration on a pacing tone caused greater reduction of skin conductance responses (Clark, & Hirschman, 1990). The increased effort and demands on attention required among novices during pranayama may explain why Ujjayi breathing and paced breathing in the present study caused the stress task to be perceived as more difficult, stressful and negative.

However, these differences of subjective measures did not accord with the physiology between groups. As mentioned above, there were no significant differences in systolic blood pressure, diastolic blood pressure or heart rate between the groups, as was predicted. This suggests that there is some stress relieving benefit to regulating breath because there were no differences between the conditions on any cardiovascular parameter during the mental stress task, even though the two breathing groups rated their perceptions of the task as significantly more difficult, stressful, and negatively impactful.

The present study also predicted testing groups would not differ between preliminary PSS scores and only the Ujjayi breathing group would have significantly lower post-testing PSS scores, when compared to the paced breathing group. Unexpectedly there were significant differences in preliminary PSS scores between conditions, indicating the testing groups as beginning the testing session with different levels of perceived stress. Because the pranayama groups had higher preliminary stress scores, it could be why the pranayama groups perceived the stress task as significantly more stressful, difficult, and negative and rated their performance as worse than the the control group. These unexpected perceptions of the stress task observed in the pranayama groups could also have resulted from the previously mentioned phenomenon of dual attention, which has been observed to significantly increase task difficulty (Pashler, 1994), while causing increases in sympathetic nervous system activity (Zhang et al., 2010).

Limitations of this study include a small sample size of mostly female participants, within a specific age range. This lack of generalizability causes replication of this study to be questionable in

males and other ages. This study also only consisted of individuals who had no experience with pranayama. Replication of this study may not be possible with people who are extensively trained in yogic breathing because it would be expected that habitual practice of paced breathing and Ujjayi breathing would allow participants to perform these pranayamas with less effort. This would cause less cardiovascular reactivity in response to a mental stressor and more positive perception of the breathing and stress task. In this study, the effects of Ujjayi breathing and paced breathing were only tested during one laboratory session. The long-term effects of these yogic breathing techniques are of high interest because of the previous reports of Ujjayi breathing and paced breathing decreasing heart rate and blood pressure after several weeks of practice (Tomar & Singh, 2011). This study did had a control group. However, it would have been beneficial to measure the respiration patterns of the control group, like the respiration of the Ujjayi and paced breathing groups. This would have provided a comparison of how many breaths per minute a person would naturally breath during a resting and a stressful period. The present study also only obtained preliminary perceived stress, using PSS scores, across all three conditions. Post-testing PSS scores were only obtained in the Ujjayi breathing and paced breathing groups. It would have been beneficial to have preliminary *and* post-testing PSS scores for all three conditions, in order to observe if the perceived stress of the pranayama groups was different compared to the unregulated breathing group after the testing session.

Future studies that capture the long term effects of Ujjayi breathing and paced breathing could clarify if the novelty of pranayama effects its physiological benefits. The present study only consisted of participants who had no previous knowledge of pranayama and only thirty minutes of practicing Ujjayi breathing and paced breathing, during one laboratory session. A continuation of this project could include an acclimation, or "training" period of Ujjayi breathing and paced breathing. This would reduce the novelty of the pranayama techniques, potentially leading to observable physiological benefits. Including an acclimation period may also improve perceptions of the breathing techniques, allowing participants to

perceive the breathing as a relaxing activity, which could lead to significant decreases in cardiovascular reactivity during a mental stress task, as was predicted in this present study. Future work could also consist of reducing attentional demands during the breathing techniques. This could consist of not displaying the breath pacer on a monitor, but verbal dictation of the pacer, in order to test if the degree of attentional demand is what affects the physiological benefits of pranayama.

Conclusions and Future Research

Participants who were inexperienced with pranayama, showed no significant differences in cardiovascular reactivity during a mental stress task when practicing Ujjayi breathing or paced breathing in a laboratory setting, when compared to participants who breathed normally. However, participants who were in the pranayama groups reported the stress task as significantly more difficult, stressful, negative and perceived their performance as significantly worse than the control group. Because there were no physiological differences across groups, the differences of the subjective measures of the PASAT observed between groups indicated pranayama as possibly beneficial for human health. Further studies are needed in order to establish if regular practice of pranayama techniques, such as, Ujjayi breathing and paced breathing, reduces the novelty of the breath. Thus, potentially improving subjectivity of the pranayama techniques and the physiological benefits during mental stress.

References

Ankad, R. B., Herur, A., Patil, S., Shashikala, G. V., & Chinagudi, S. (2011). Effect of short-term pranayama and meditation on cardiovascular functions in healthy individuals. *Heart Views : The Official Journal of the Gulf Heart Association, 12*(2),58-62.

Audimulapu, S. (2016). Study of pranayama on heart rate and blood pressure. *Indian Journal of Research, 5*(9), 325-327.

Balanos, G. M., Phillips, A. C., Michael, P., Frenneaux, M.P., McIntyre, D., Lykidis, C.,...Carroll, D. (2010). Metabolically exaggerated cardiac reactions to acute psychological stress: the effects of resting blood pressure status and possible underlying mechanisms. *Biol Psychiatry*, *85*, 104–111.

Barnett, P., Spence, J., Manuck, S., & Jennings, J. (1997). Psychological stress and the progression of carotid artery disease. *Journal of Hypertension, 15*(1), 49-55.

Bear, M. F., Connors, B. W., & Paradiso, M. A. (2007). *Neuroscience: exploring the brain* (3rd ed.). Philadelphia, PA: Lippincott Williams & Wilkins.

Berntson, G. G., Bigger, T., Jr., Eckberg, D. L., Grossman, P., Kaufmann, P. G., Malik, M.,...Van Der Molen, M. W. (1997) Heart rate variability: Origins, methods, and interpretive caveats. *Psychophysiology*, *34*, 48-623.

Berntson, G. G., Cacioppo, J. T., & Quigley, K. S. (1993). Respiratory sinus arrhythmia: Autonomic origins, physiological mechanisms, and psychophysiological implications. *Psychophysiology, 30*, 96-183.

Bhimani, N. T., Kulkarni, N. B., Kowale, A., & Salvi, S. (2011). Effect of pranayama on stress and cardiovascular autonomic function. *Indian Journal of Physiology and Pharmacology, 55*(4), 370.

Boiten, F. A., Frijda, N. H., & Wientjes, C. J. E. (1994). Emotions and respiratory patterns : Review and critical analysis. *International Journal of Psychophysiology, 17*, 28-103.

Boiten, F. A. (1998). The effects of emotional behaviour on components of the respiratory cycle. *Biological Psychology*, *49*, 29-51.

Bonnet, M., & Arand, D. (2003). Insomnia, metabolic rate and sleep restoration. *Journal of Internal Medicine, 254*(1), 23-31.

Brown, R., & Gerbarg, P. (2005). Sudarshan Kriya Yogic breathing in the treatment of stress, anxiety, and depression: part II–clinical applications and guidelines. *J Altern Complement Med*, *11*(4),711–717.

Brown, R., Gerbarg, P., & Muench, F. (2013). Breathing practices for treatment of psychiatric and

stress-related medical conditions. *Psychiatric Clinics of North America, 36*(1), 121-121.

Brunner, E., Hemingway, H., Walker, B., Page, M., Clarke, P., Juneja, M., . . . Marmot, M. (2002). Adrenocortical, autonomic, and inflammatory causes of the metabolic syndrome - nested case-control study. *Circulation, 106*(21), 2659-2665.

Busch, V., Magerl, W., Kern, U., Haas, J., Hajak, G., & Eichhammer, P. (2012). The effect of deep and slow breathing on pain perception, autonomic activity, and mood Processing—An experimental study. *Pain Medicine, 13*(2), 215-228.

Busek, P., & Kemlink, D. (2005). The influence of the respiratory cycle on the EEG. *Physiol Res*, *54*, 33-327.

Calabrese, P., Perrault, H., Dinh, T., Eberhard, A., & Benchetrit, G. (2000). Cardiorespiratory interactions during resistive load breathing. *American Journal of Physiology-Regulatory Integrative and Comparative Physiology, 279*(6), R2208-R2213.

Cappo, B. M., & Holmes, D. S. (1984). The utility of prolonged respiratory exhalation for reducing physiological and psychological arousal in non-threatening and threatening situations. *Journal of Psychosomatic Research, 28*(4), 265.

Carbon, M., Wübbeler, G., Trahms, L., & Curio, G. (2000). Hyperventilation-induced human cerebral magnetic fields non-invasively monitored by multichannel 'direct current' magnetoencephalography. *Neuroscience Letters, 287*(3), 227-230.

Carroll, D., Ginty, A. T., Der, G., Hunt, K., Benzeval, M., & Phillips, A. C. (2012). Increased blood pressure reactions to acute mental stress are associated with 16‐year cardiovascular disease mortality. *Psychophysiology, 49*(10), 1444-1448.

Carroll, D., Phillips A.C., & Balanos G.M. (2009). Metabolically exaggerated cardiac reactions to acute psychological stress revisited. *Psychophysiology*, *46*, 270–275.

Carroll, D., Ring, C., Hunt, K., Ford, G., & MacIntyre, S. (2003). Blood pressure reactions to stress and the prediction of future blood pressure: Effects of sex, age, and socioeconomic position.*Psychosomatic Medicine, 65*(6), 1058-1064.

Carroll, D., Smith, G., Shipley, M., Steptoe, A., Brunner, E., & Marmot, M. (2001). Blood pressure reactions to acute psychological stress and future blood pressure status: A 10-year follow-up of men in the whitehall II study. *Psychosomatic Medicine, 63*(5), 737-743.

Carter, K. S., & Carter, 3., Robert. (2016). Breath-based meditation: A mechanism to restore the physiological and cognitive reserves for optimal human performance. *World Journal of Clinical Cases, 4*(4), 99.

Chiu, Y., Lu, F., Lin, J., Nien, C., Hsu, Y., & Liu, H. (2016). Psychometric properties of the perceived stress scale (PSS): Measurement invariance between athletes and non-athletes and construct validity. *Peerj, 4*, e2790.

Chrousos, G. (1995). Seminars in medicine of the beth-israel-hospital, boston - the hypothalamic-pituitary-adrenal axis and immune-mediated inflammation. *New England Journal of Medicine, 332*(20), 1351-1362.

Chrousos, G.P., & Gold, P.W. (1992). The concepts of stress and stress system disorders. Overview of physical and behavioral homeostasis. *JAMA*, *267*(9), 52-1244.

Chuntharapat, S., Petpichetchian, W., & Hatthakit, U. (2008). Yoga during pregnancy: Effects on maternal comfort, labor pain and birth outcomes. *Complementary Therapies in Clinical Practice, 14*(2), 105-115.

Clark, M. E., & Hirschman, R. (1990). Effects of paced respiration on anxiety reduction in a clinical population. *Biofeedback and Self-Regulation, 15*(3), 273-284.

Cooper, S., Oborne, J., Newton, S., Harrison, V., Thompson, C.J., Lewis, S. & Tattersfield A. (2003). Effect of two breathing exercises (Buteyko and pranayama) in asthma: a randomised controlled trial. *Thorax*. 64-75.

Cowen, V. S., & Adams, T. B. (2007). Heart rate in yoga asana practice: A comparison of styles. *Journal of Bodywork & Movement Therapies, 11*(1), 91-95.

Cuttle, M. F., Rusznák, Z., Wong, A. Y. C., Owens, S., & Forsythe, I. D. (2001). Modulation of a presynaptic hyperpolarization – activated cationic current (Ih) at an excitatory synaptic terminal in the rat auditory brainstem. *J Physiol, 534* (3), 733–744.

Dhungel, K.U., Malhotra, V., Sarkar, D. & Prajapati, R. (2008). Effect of alternate nostril breathing exercise on cardiorespiratory functions. *Nepal Med Coll J. 10*:25-27.

Dusek, J. A., & Benson, H. (2009). *Mind-body medicine: A model of the comparative clinical impact of the acute stress and relaxation responses*. United States.

Epel, E.S.(2009). Psychological and metabolic stress: a recipe for accelerated cellular aging? Hormones, 8, 7–22.

Everly, G. S., 1950. (1989). *A clinical guide to the treatment of the human stress response*. United States: *Experiment HS-5: Breathing techniques and heart rate.* (2006). United States: iWorx Systems Inc.

Fokkema, D.S. (1999). The psychobiology of strained breathing and its cardiovascular implication: A functional system review. *Psychophysiology*, *36*,164- 175.

Fox, S.T. (2016) Human Physiology. New York, NY: McGraw-Hill Education.

Frentzel-Beyme, R., & Grossarth-Maticek, R. (2001). The interaction between risk factors and self-regulation in the development of chronic diseases. *International Journal of Hygiene and Environmental Health, 204*(1), 81-88.

Fried, R. (1993). *The Psychology and Physiology of Breathing in Behavioral Medicine, Clinical Psychology, and Psychiatry.*

Ghiadoni, L., Donald, A., Cropley, M., Mullen, M., Oakley, G., Taylor, M., . . . Deanfield, J. (2000). Mental stress induces transient endothelial dysfunction in humans. *Circulation, 102*(20), 2473-2478.

Gokce, N., Keaney, J., Hunter, L., Watkins, M., Menzoian, J., & Vita, J. (2002). Risk stratification for postoperative cardiovascular events via noninvasive assessment of endothelial function - A prospective study. *Circulation, 105*(13), 1567-1572.

Gozal, D., Omidvar. O., Kirlew, K.A., Hathout, G.M., Hamilton, R., Lufkin, R.B.,...Harper, R.M. (1995). Identification of human brain regions underlying responses to resistive inspiratory loading with functional magnetic resonance imaging. *Proc Natl Acad Sci USE*, *92*: 6607-6611.

Gronwall, D.M.A. (1977). Paced auditory serial-addition task: A measure of recovery from concussion. *Perceptual and Motor Skills*, *44*, 367-373.

Grossman, P., & Kollai, M. (1993). Respiratory sinus arrhythmia, cardiac vagal tone, and respirations: Within- and between-individual relations. *Psychophysiology, 30*, 486-95.

Hagins, M., Haden, S., & Daly, L. (2013). A randomized controlled trial on the effects of yoga on stress reactivity in 6th grade students. *Evidence-Based Complementary and Alternative Medicine*, 9-607134.

Harris, V. A., Katkin, E. S., Lick, J. R., & Habberfield, T. (1976). Paced respiration as a technique for the modification of autonomic response to stress. *Psychophysiology*, *13*, 386-91.

Hiriyanna, M. (2002). *Essentials of Indian philosophy*, Varanasi, India: Banarasidas

Holmes, D. S., McCaul, K. D., & Solomon, S. (1978). Control of respiration as a means of controlling responses to threat. *Journal of Personality and Social Psychology, 36*(2), 198-204.

Hoyert, D. L., & Xu, J. (2012). Deaths: preliminary data for 2011. *National Vital Statistics Reports: From the Centers for Disease Control and Prevention, National Center for Health Statistics, National Vital Statistics System, 61*(6), 1.

Huang, K.C., Kormas, N., Steinbeck, K., Loughnan, G. & Caterson, I.D. (2004). Resting metabolic rate in severely obese diabetic and nondiabetic subjects. *Obesity, 12*, 840–845.

Innes, K., Bourguignon, C., & Taylor, A. (2005). Risk indices associated with the insulin resistance syndrome, cardiovascular disease, and possible protection with yoga: A systematic review .*Journal of the American Board of Family Practice, 18*(6), 491-519.

Iskandrian, A. E., & Canto, J. G. (2003). Major risk factors for cardiovascular disease: Debunking the "only 50%" myth. *Jama, 290*(7), 947-949.

Jennings, J., Kamarck, T., Everson-Rose, S., Kaplan, G., Manuck, S., & Salonen, J. (2004). Exaggerated blood pressure responses during mental stress are prospectively related to enhanced carotid atherosclerosis in middle-aged finnish men. *Circulation, 110*(15), 2198-2203.

Jerath, R., Jerath, V., Edry, J. W., & Barnes, V. A. (2006). Physiology of long pranayamic breathing: Neural respiratory elements may provide a mechanism that explains how slow deep breathing shifts the autonomic nervous system. *Medical Hypotheses, 67*(3),566-571.

Jovanov, E. (2005). Spectral analysis of heart rate variability during very slow yogic breathing. *Engineering in Medicine and Biology Society*, *3*, 2467–2470.

Julius, S. (1993). Sympathetic hyperactivity and coronary risk in hypertension. *Hypertension, 21*(6), 886-893.

Jumpertz, R., Hanson, R.L., Sievers, M.L., Bennett, P.H., Nelson, R.G. & Krakoff, J. (2011). Higher energy expenditure in humans predicts natural mortality. *J Clin Endocrinol Metab, 96*, E972–E976.

Kaplan, J., Pettersson, K., Manuck, S., & Olsson, G. (1991). Role of sympathoadrenal medullary activation in the initiation and progression of atherosclerosis. *Circulation, 84*(6), 23-32.

Khot, M. B., Khot, U. N., Ohman, E. M., Brener, S. J., Sapp, S. K., Bajzer, C. T.,...Topol, E. J. (2003). Prevalence of conventional risk factors in patients with coronary heart disease. *Jama, 290*(7), 898-904.

Kirkwood, G. (2005). Yoga for anxiety: A systematic review of the research evidence commentary.*British Journal of Sports Medicine, 39*(12), 884-891.

Kochupillai, V., Kumar, P., Singh, D., Aggarwal, D., Bhardwaj, N., Bhutani, M., & Das, S. N. (2005). Effect of rhythmic breathing (sudarshan kriya and pranayam) on immune functions and tobacco addiction. *Annals of the New York Academy of Sciences, 1056*(1), 242-252.

Kreibig, S. D. (2010). Autonomic nervous system activity in emotion: A review. *Biological Psychology, 84*(3), 394-421.

Kunz, I., Schorr, U., Klaus, S., & Sharma, A.M. (2000). Resting metabolic rate and substrate use in obesity hypertension. *Hypertension*, *36*, 26–32.

Kyrou I. & Tsigos C. (2009). Stress hormones: physiological stress and regulation of metabolism. *Curr Opin Pharmacol, 9*, 787–793.

Lambert, E. A., & Lambert, G. W. (2011). Stress and its role in sympathetic nervous system activation in hypertension and the metabolic syndrome. *Current Hypertension Reports, 13*(3), 244-248.

La Rovere, M.T., Biffer, J.T., Marcus, F.l., Mortara, A. & Schwartz, P.J. (1998). Baroreflex sensitivity and heart-rate variability in prediction of total cardiac mortality after myocardial infarction.

Lancet, *351*: 478-484.

Lathadevi, G.V., Uma Maheswari, T. & Nagashree, R. (2012). Modulation of cardiovascular response after ujjayi pranayama and shavasana training in normal human volunteers. *Journal of Clinical and Diagnostic Research*, *6*(4), 571-573.

Lehrer, P., Sasaki, Y., & Saito, Y. (1999). Zazen and cardiac variability. *Psychosomatic Medicine, 61*(6), 812-821.

Lewthwaite, J., Owen, N., Coates, A., Henderson, B., & Steptoe, A. (2002). Circulating human heat shock protein 60 in the plasma of british civil servants - relationship to physiological and psychosocial stress. *Circulation, 106*(2), 196-201.

Licht, C., Vreeburg, S., Dortland, A., Giltay, E., Hoogendijk, W., DeRijk, R., . . . Penninx, B. (2010). Increased sympathetic and decreased parasympathetic activity rather than changes in hypothalamic-pituitary-adrenal axis activity is associated with metabolic abnormalities. *Journal of Clinical Endocrinology & Metabolism, 95*(5), 2458-2466.

Light, K. C. (1981). Cardiovascular responses to effortful active coping: Implications for the role of stress in hypertension development. *Psychophysiology*, *18*, 216-225.

Lim, S. S., Vos, T., Flaxman, A. D., Danaei, G., Shibuya, K., Adair-Rohani, H. (2012). A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions, 1990–2010: A systematic analysis for the global burden of disease study 2010. *The Lancet. 380*(9859), 2224-2260.

Lucini, D., Norbiato, G., Clerici, M., & Pagani, M. (2002). Hemodynamic and autonomic adjustments to real life stress conditions in humans. *Hypertension, 39*(1), 184-188.

Lutz, A., Greischar, L. L., Rawlings, N. B., Ricard, M., & Davidson, R. J. (2004). Long-term meditators self-induce high-amplitude gamma synchrony during mental practice. *Proc Natl Acad Sci USA*, *101*(46), 16369–16373.

Madanmohan, Navasakthi, Rajajeyakumar, M., Prabhakar, P., Bhattacharjee, M., & Balachander, J. (2014). Effect of single pranayam technique on cardiovascular parameters and mental chronometry among medical students. *International Journal of Physiology, 2*(1), 54-58.

Malathi, A., & Damodaran, A. (1999). Stress due to exams in medical students--role of yoga. *Indian Journal of Physiology and Pharmacology, 43*(2), 218.

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

Matsumoto S., Ikeda M., Nishikawa T., Tanimoto T., Yoshida S. & Saiki C. (2000). Inhibitory mechanism of slowly adapting pulmonary stretch receptors after release from hyperinflation in anesthetized rabbits. *Life Sci, 67*(12), 1423–1433.

McArdle, W. D., Katch, F. I., & Katch, V. L. (2010). *Exercise physiology: Nutrition, energy, and human performance* (7th ed.). Baltimore, MD: Lippincott Williams & Wilkins.

McCaul, K. D., Solomon, S., & Holmes, D. (1979). Effects of paced respiration and expectations on physiological and psychological responses to threat. *Journal of Personality and Social Psychology, 37*, 564-71.

McEwen, B.S. (2007). Physiology and neurobiology of stress and adaption:central role of the brain, *Physiol Rev, 87*, 873-904.

Mezzacappa, E., Kelsey, R., Katkin, E., & Sloan, R. (2001). Vagal rebound and recovery from psychological stress. *Psychosomatic Medicine, 63*(4), 650-657.

Miller, J. J., Fletcher, K., & Kabat-Zinn, J. (1995). Three-year follow-up and clinical implications of a mindfulness meditation-based stress reduction intervention in the treatment of anxiety disorders. *General Hospital Psychiatry, 17*(3), 192-200.

Mittleman, M. A., Maclure, M., Sherwood, J. B., Mulry, R. P., Tofler, G. H., Jacobs, S. C., . . . Muller, J. E. (1995). Triggering of acute myocardial infarction onset by episodes of anger. determinants of myocardial infarction onset study investigators. *Circulation, 92*(7), 1720.

Muktibodhananda, S. (2002). *Hatha Yoga Pradipika*. Bihar, India: Yoga Publications Trust.

Newberg, N., & Iversen, J. (2003). The neural basis of the complex mental task of meditation: neurotransmitter and neurochemical considerations. *Med Hypotheses*, *61*(2),282–291.

Olshansky, S.J., & Rattan, S. (2005).What determines longevity: metabolic rate or stability. *Discov Med*., *5*, 359–362.

Pagani, M., Mazzuero, G., Ferrari, A., Liberati, D., Cerutti, S., Vaitl, D., . . . Malliani, A. (1991). sympathovagal interaction during mental stress - a study using spectral-analysis of heart-rate-variability in healthy control subjects and patients with a prior myocardial-infarction. *Circulation, 83*(4), 43-51.

Pal, G., Velkumary, S., & Madanmohan. (2004). Effect of short-term practice of breathing exercises on autonomic functions in normal human volunteers. *Indian Journal of Medical Research, 120*(2), 115-121.

Pashler, H. (1994). Dual-task interference in simple tasks: Data and theory. *Psychological Bulletin, 116*(2), 220-244.

Pilkington, K., Kirkwood, G., Rampes, H., & Richardson, J. (2005). Yoga for depression: The research evidence. *Journal of Affective Disorders, 89*(1), 13-24.

Poehlman, E. T., Scheffers, J., Gottlieb, S. S., Fisher, M. L., & Vaitekevicius, P. (1994). Increased resting metabolic rate in patients with congestive heart failure. *Annals of Internal Medicine, 121*(11), 860.

Poh, P. Y. S., Carter, R., Hinojosa-Laborde, C., Mulligan, J., Grudic, G. Z., & Convertino, V. A. (2014). Respiratory pump contributes to increased physiological reserve for compensation during simulated haemorrhage: Respiration and compensatory reserve. *Experimental Physiology, 99*(10), 1421-1426.

Porges, S. W. (1995). Orienting in a defensive world: Mammalian modifications of our evolutionary heritage. A polyvagal theory. *Psychophysiology*, *32*, 30-18.

Pramanik, T., Pudasaini, B., & Prajapati, R. (2010). Immediate effect of the slow pace breathing exercise, bhramari pranayama on the blood pressure and the heart rate. *Nepal Med Coll J*, *12*(3), 7-154.

Pramanik, T., Sharma, H. O., Mishra, S., Mishra, A., Prajapati, R., & Singh, S. (2009). Immediate effect of slow pace bhastrika pranayama on blood pressure and heart rate. *Journal of Alternative and Complementary Medicine (New York, N.Y.), 15*(3), 293.

Raghuraj, P., & Telles, S. (2003). Effect of yoga-based and forced uninostril breathing on the autonomic nervous system. *Perceptual and Motor Skills, 96*(1), 79-80.

Ravindra, P.N. & Madanmohan, P.P. (2006). Effect of pranayam (yogic breathing) and shavasan (relaxation training) on the frequency of benign ventricular ectopics in two patients with palpitations. *Int J Cardiol*, *108*, 124-125.

Remmers, J. E. (1976). Analysis of ventilatory response. *Chest, 70 (Suppl. 1)*, 134-7.

Ring, C., Burns, V., & Carroll, D.. (2002). Shifting hemodynamics of blood pressure control during prolonged mental stress. *Psychophysiology*, *39*, 585–590.

Roldan, E., & Dostalek, C. (1983). Description of an EEG pattern evoked in central-parietal areas by the Hathayogic exercise Agnisara. *Act Nerv Super (Praha)*, *25*, 6-241.

Roldan, E., & Dostalek, C. (1985). EEG patterns suggestive of shifted levels of excitation affected by hathayogic exercises. *Act Nerv Super (Praha), 27*, 8-81.

Rozanski, A., Blumenthal, J., Davidson, K., Saab, P., & Kubzansky, L. (2005). The epidemiology, pathophysiology and management of psychosocial risk factors in cardiac practice - the emerging field of behavioral cardiology. *Journal of the American College of Cardiology, 45*(5), 637-651.

Ruan T., Ho C. Y., & Kou Y. R. (2003). Afferent vagal pathways mediating respiratory reflexes evoked by ROS in the lungs of anesthetized rats. *Journal of Applied Physiology*, *94*(5),1987–1998.

Ruggiero, C., Metter, E.J., Melenovsky, V., Cherubini, A., Najjar, S.S.,Ble, A.,...& Ferrucci, L. (2008). High basal metabolic rate is a risk factor for mortality: the Baltimore Longitudinal Study of Aging. *J Gerontol A Biol Sci Med Sci*., *63*, 698–706.

Sathidevi, G.V., Uma Maheswari, T., & Nagashree, R. (2012).Modulation of Cardiovascular Response After Ujjayi Pranayama and Shavasana Training in Normal Human Volunteers. *Journal of Clinical and Diagnostic Research, 6*(4), 571-574.

Schelegle, E., & GreenSchelegle, J. (2001). An overview of the anatomy and physiology of slowly adapting pulmonary stretch receptors. *Respir Physiol, 125*(1–2), 17–31.

Schwartz, A., Gerin, W., Davidson, K., Pickering, T., Brosschot, J., Thayer, J., . . . Linden, W. (2003). Toward a causal model of cardiovascular responses to stress and the development of cardiovascular disease. *Psychosomatic Medicine, 65*(1), 22-35.

Siegelbaum, R., & Robinson, S. (2003). Hyperpolarization activated cation current: from molecules to physiological function. *Annu Rev Physiol*, 65.

Snodgrass, J.J., Leonard, W.R., Sorensen, M.V., Tarskaia, L.A., & Mosher, M.J. (2008) The influence of basal metabolic rate on blood pressure among indigenous Siberians. *Am J Phys Anthropol*, *137*, 145–155.

Sovik, R. (2000). The science of breathing--the yogic view. *Progress in Brain Research, 122*, 491.

Spicuzza, L,. Gabutti, A., Porta, C., Montano, N. & Bernardi, L. (2000). Yoga and chemoreflex response to hypoxia and hypercapnia. *Lancet, 356*, 1495-1496.

Steptoe, A., Kivimäki, M., Lowe, G., Rumley, A., & Hamer, M. (2016). Blood pressure and fibrinogen responses to mental stress as predictors of incident hypertension over an 8-year period. *Annals of Behavioral Medicine, 50*(6), 898-906.

Streeter, C. C., Gerbarg, P. L., Saper, R. B., Ciraulo, D. A., & Brown, R. P. (2012). Effects of yoga on the autonomic nervous system, gamma-aminobutyric-acid, and allostasis in epilepsy, depression, and post-traumatic stress disorder. *Medical Hypotheses, 78*(5), 571.

Subbalakshmi, N.K., Saxena, S.K., Urmimala, D., & Urban, J.A. (2005). Immediate effect of Nadi-shodhan Pranayama on some selected parameters of cardiovascular, pulmonary and higher functions of brain. *Thai J Physiological Sciences, 18*,10-16.

Telles, S., & Naveen, K. V. (2008). Voluntary breath regulation in yoga: Its relevance and physiological effects. *Biofeedback, 36*(2), 70.

Telles, S., Singh, N., & Balkrishna, A. (2011). Heart rate variability changes during high frequency yoga breathing and breath awareness. *BioPsychoSocial Medicine, 5*(1), 4.

Telles, S., Yadav, A., Kumar, N., Sharma, S., Visweswaraiah, N., & Balkrishna, A. (2013). Blood pressure and purdue pegboard scores in individuals with hypertension after alternate nostril breathing, breath awareness, and no intervention. *Medical Science Monitor, 19*, 61-66.

Telles, S. & Desiraju, T. (1991). Oxygen consumption during pranayamic type of very slow-rate breathing. *Indian J Med Res*, *94*, 357–363.

Telles, S., Joseph, C., Venkatesh, S., & Desiraju, T. (1993). Alterations of auditory middle latency evoked potentials during yogic consciously regulated breathing and attentive state of mind. *Int J Psychophysiol*, *14*(3), 98-189.

Timmons, B. H., & Ley, R. (Eds.) (1994). *Behavioral and Psychological Approaches to Breathing Disorders.*

Tomar, R. & Singh, N. (2011). Effect of ujjayi pranayama on selected physiological variables. *Ovidius University Annals, Series Physical Education & Sport/Sci*, *11*(1), 140.

Tyagi, A., & Cohen, M. (2013). *Oxygen consumption changes with yoga practices: A systematic review*. Los Angeles, CA: SAGE Publications.

Upadhyay Dhungel, K., Malhotra, V., Sarkar, D., & Prajapati, R. (2008). Effect of alternate nostril breathing exercise on cardiorespiratory functions. *Nepal Medical College Journal : NMCJ, 10*(1), 25.

Vaccarino, V., Goldberg, J., Rooks, C., Shah, A., Veledar, E., Faber, T., . . . Bremner, J. (2013). Post-traumatic stress disorder and incidence of coronary heart disease. *Journal of the American College of Cardiology, 62*(11).

Van Diest, I., Thayer, J. F., Vandeputte, B., Van de Woestijne, Karel P, & Van den Bergh, O. (2006). Anxiety and respiratory variability. *Physiology & Behavior, 89*(2), 189-195.

Van Dixhoorn, J. (1998). Cardiorespiratory effects of breathing and relaxation instruction in myocardial infarction patients. *Biological Psychology, 49*(1), 123-135.

Waelde, L. C., Thompson, L., & Gallagher‐Thompson, D. (2004). A pilot study of a yoga and meditation intervention for dementia caregiver stress. *Journal of Clinical Psychology, 60*(6), 677-687.

Wallace, R.K., Benson, H., & Wilson, A.F. (1971). A wakeful hypometabolic physiologic state. *Am J Physiol*, *221*(3), 795–9.

Wenger, M. A., & Cullen, T. D. (1972). Studies of autonomic balance in children and adults. In N.S. Greenfield & R. A. Sternbach (Eds.), *Handbook of Psychophysiology*, 69-535.

Westbrook, G. L., Kandel, E. R, Schwartz, J. H. & Jessell, T. M. (Eds.). (2000). Principles of neuroscience, McGraw-Hill, New York.

Wientjes, C. J., & Grossman, P. (1998). Respiratory psychophysiology as a discipline: Introduction to the special issue [Editorial]. *Biological Psychology*, *49*, 1-8.

Willemsen, C., Ring, Carroll, D., Evans, P., Clow, A., & Hucklebridge, F. (1998). Secretory immunoglobulin A and cardiovascular reactions to mental arithmetic and cold pressor. *Psychophysiology*, *35*, 252–259.

Winzer, A., Ring, C., Carroll, D., Willemsen, G., Drayson, M. & Kendall, M. (1999), Secretory immunoglobulin A and cardiovascular reactions to mental arithmetic, cold pressor, and exercise: Effects of beta-adrenergic blockade. Psychophysiology, *36*, 591–601.

Yeung, A. C., Vekshtein, V. I., Krantz, D. S., Vita, J. A., Ryan, T. J., Ganz, P.,...Selwyn, A. P. (1991). The effect of atherosclerosis on the vasomotor response of coronary arteries to mental stress. *The New England Journal of Medicine, 325*(22), 1551-1556.

Zhang, J., Yu, X., & Xie, D. (2010). Effects of mental tasks on the cardiorespiratory synchronization. *Respiratory Physiology & Neurobiology, 170*(1), 91-95.

Zope, S. A., & Zope, R. A. (2013). Sudarshan kriya yoga: Breathing for health. *International Journal of Yoga, 6*(1), 4-10.

Appendix

Table 1
Demographic, Clinical Characteristics and PASAT Scores of Eligible Recruited Sample

	Group		
	Ujjayi Breathing (*n*=15)	Paced Breathing (*n*=15)	Control (*n*=15)
Variable	M(SD)	M(SD)	M(SD)
Age	19.6(1.24)	19.9(0.961)	20.4(1.18)
Gender	80.0	80.0	46.7
Preliminary PSS Score	17.8(5.10)	20.3(6.31)	14.6(6.30)
Post-Testing PSS Score	18.0(5.69)	21.4(6.70)	
PASAT Score	923(37.4)	930(35.0)	955(22.4)

Note. Gender is expressed in percent female. To access PASAT score, M derived from *n*=14 in Ujjayi group and *n*=14 in paced breathing group because 2 participants were excluded due to lack of compliance during task. Significant differences were observed between the Ujjayi breathing group and the control (*ps* < 0.05) for PASAT score. Significant differences were observed between the paced breathing group and control (*ps* < 0.05) for PSS score.

Table 2
Respiration Rate (Breaths/Min) During Each Phase and Subjective Measures of Breathing

	Group	
	Ujjayi Breathing (*n*=15)	Paced Breathing (*n*=15)
Variable	M(SD)	M(SD)
Resting Breaths/Min, M (SD)	6.33(1.03)	6.01(0.128)
Stress Breaths/Min, M (SD)	13.0(6.67)	16.0(5.65)
Recovery Breaths/Min, M (SD)	6.06(0.398)	6.08(0.113)
How difficult did you find the breathing?	2.60(1.30)	2.33(1.95)
How stressful did you find the breathing?	2.73(1.83)	2.27(1.49)
How well you think you performed the breathing?	2.60(1.18)	3.27(1.33)
How embarrassing did you find the breathing?	1.00(1.51)	0.867(1.55)
How enjoyable did you find the breathing?	2.67(1.45)	2.93(1.91)

Note. The maximum score is 6 for each subjective measure. No significant differences were observed (all *ps* >.05).

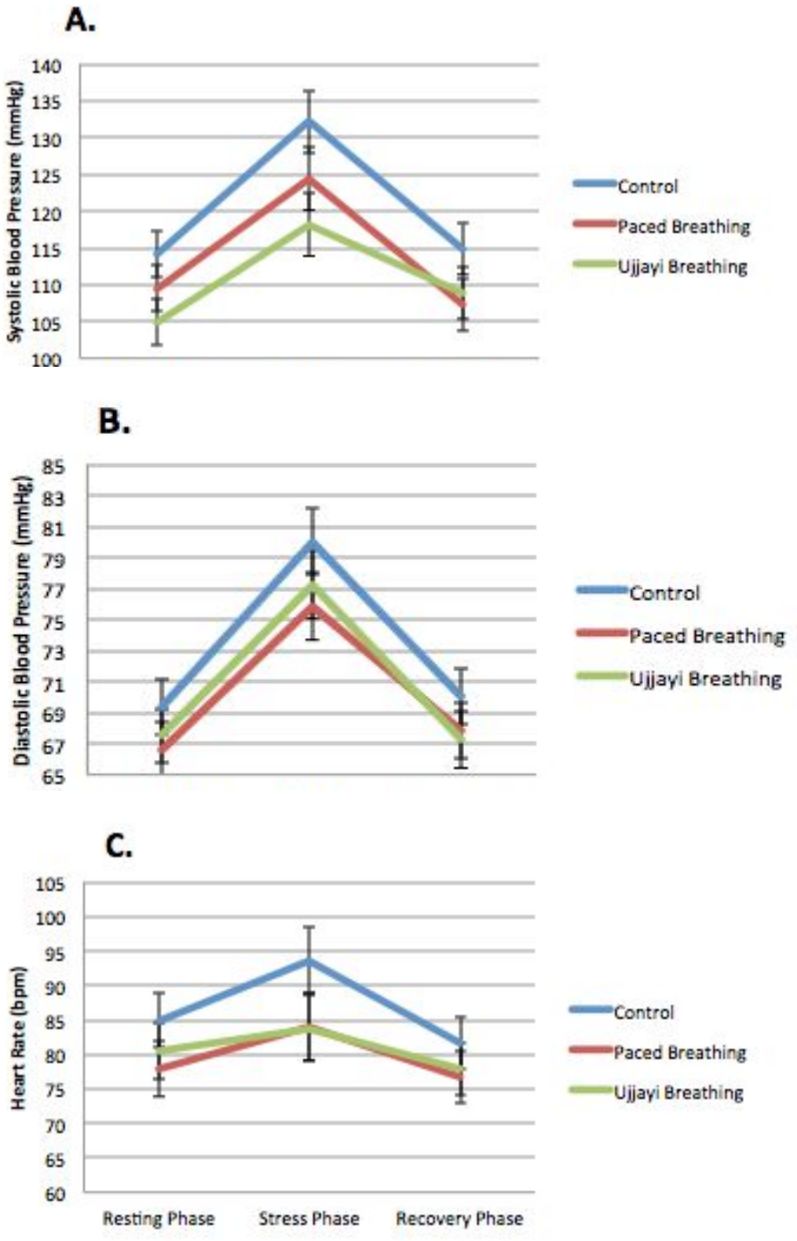


Figure 1. A. Systolic blood pressure, B. Diastolic blood pressure, and C. Heart rate reactivity to mental stress across testing three consecutive sessions in paced breathing (n = 15), Ujjayi breathing (n = 15), and unregulated breathing (n=15) conditioned groups. Error bars represent standard error of the mean.

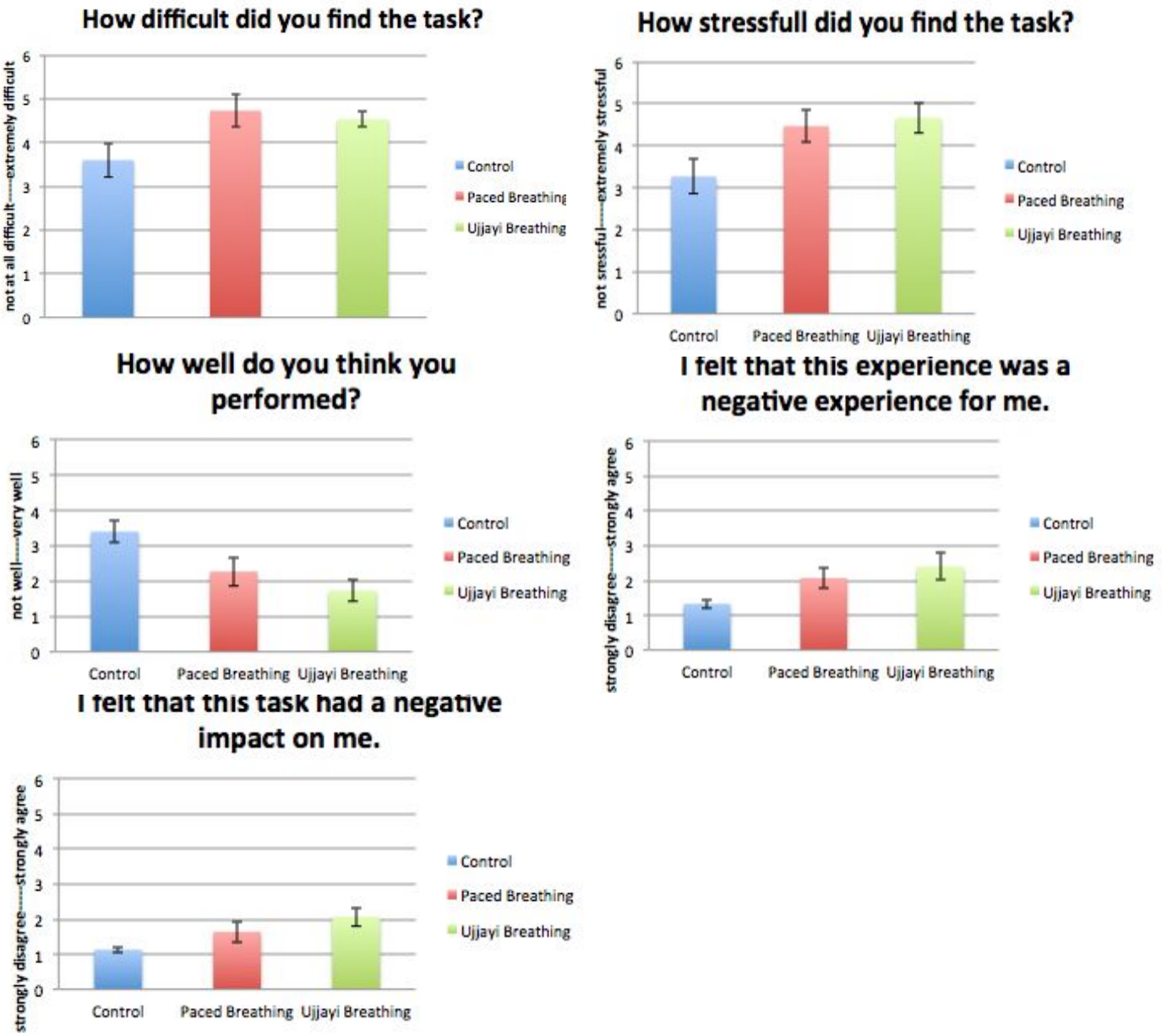


Figure 2. Subjective PASAT measures of difficulty, stressfulness, performance, and negativity of impact and experience in paced breathing (n = 15), Ujjayi breathing (n = 15), and unregulated breathing (n=15) conditioned groups. Error bars represent standard error of the mean.