COMPARISON OF ACUTE HEART RATE VARIABILITY RESPONSES TO RELAXATION ALONE VS. RELAXATION PRECEDED BY HATHA YOGA.

by

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ABSTRACT

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The objective of this study was to compare the acute heart rate variability responses to relaxation alone versus relaxation preceded by Hatha yoga. Twenty women and men (aged 18-50 years) participated in the study. Participants completed a yoga plus relaxation (YR) session and a relaxation only (R) session. The YR condition showed significant changes from baseline in HR (bpm, $p < 0.001$), RR (ms, $p < 0.001$), pNN50 (%, $p = 0.009$), LF (% $p = 0.008$) and HF (% $p = 0.035$). The R condition showed significant changes from baseline in HR (bpm, $p < 0.001$), RR (ms, $p < 0.001$), HF (ms^2, $p = 0.004$), LF (%, $p = 0.005$), HF (%, $p = 0.008$) and LF/HF (%, $p = 0.008$). There were no significant differences between conditions for the changes from baseline for any of the variables. The results demonstrate that relaxation produces favorable changes in indices of heart rate variability whether alone or preceded by a bout of Hatha yoga.

TABLE OF CONTENTS

LIST OF TABLES

I. INTRODUCTION

Regulation of the heartbeat is achieved in large part by the balance between the sympathetic and parasympathetic branches of the autonomic nervous system (ANS). Heart rate variability (HRV), the variance between R-R intervals on the electrocardiogram (ECG), can be used to assess this balance [34]. It is important to understand that the parasympathetic component of the ANS predominates over the sympathetic branch such that increased sympathetic activity is only possible with a corresponding decrease in parasympathetic tone. Increased parasympathetic tone is marked by a greater heart rate variability and creates a more electrically stable myocardium [21]. In contrast, sympathetic dominance creates an electrically unstable myocardium that is marked by decreased heart rate variability. An unstable myocardium is at greater risk for random myocite firing as is the case with premature ventricular contractions (PVC's) or more severe cardiac arrhythmias and sudden cardiac death [21]. The balance between the sympathetic and parasympathetic autonomic pathways is becoming an increasingly studied parameter because of the associations between reduced parasympathetic tone and sympathetic dominance, cardiovascular disease (CVD), and mortality [26, 32, 4]. According to Liao et al. (1997), the mechanisms mediating the association between decreased parasympathetic tone and CVD are increased myocardial oxygen consumption, ventricular excitability, coronary vasoconstriction and myocardial

ischemia, plaque fissures, macrophage-LDL oxidation, and decreased arrhythmic threshold [26]. Therefore, any means of decreasing sympathetic tone and/or increasing parasympathetic modulation may offer a cardioprotective effect and aid in the prevention or management of CVD [19, 20].

Moderate and vigorous physical activity has been shown to increase parasympathetic modulation in both healthy subjects and in those with CVD [2, 10, 15, 19, 22, 44]. Mind-body exercise, such as yoga and tai chi, are often used for stress reduction and mood elevation [17, 41] and have also been shown to increase parasympathetic tone but are less well-studied. More specifically, there are currently no studies that have investigated the effects of Hatha yoga on the ANS, acutely or chronically. Additionally, many of the studies examining the effect of physical activity on the ANS are training studies. Studies of the acute responses of the ANS to mind-body exercise are limited, but may prove to be beneficial in terms of better understanding the chronic changes seen with regular exercise [37]. Possible training adaptations may reflect the cumulative effects from acute bouts of exercise over time. Therefore, further studies to determine the most effective exercise prescriptions for increasing parasympathetic modulation are needed to better understand the responses to such interventions.

While stress reduction techniques, such as meditation and autogenic training, have been shown to improve sympathovagal balance [44], these techniques do not appear to result in ANS adaptations that allow for sustained parasympathetic modulation. Rather, they demonstrate only a temporary increase during the actual practice as seen from various stress reduction studies [21, 34, 40]. Therefore, it is not clear whether stress reduction techniques must be practiced on a daily basis to receive the increases in

parasympathetic modulation. However, as a result of their potential benefit on the ANS, stress reduction techniques are being promoted as possible therapeutic uses in the treatment of cardiovascular diseases [44].

Mind-body exercises incorporate both relaxation and light to moderate exercise. Physical yoga practices were developed to keep the body strong and healthy while also preparing the body for relaxation, allowing the body to reach a greater state of relaxation. It may be that an increase in parasympathetic modulation is in part caused by relaxation, but may also be induced by the physical movement which is consistent with studies using moderate-intensity physical activity.

It is possible that stress reduction and mind-body exercise induce increases in parasympathetic modulation by different mechanisms. Therefore, it is also possible that combining these two interventions may result in greater effect on ANS than either one by itself. To date, few studies have used a comparative research design to test multiple techniques within the same individuals. Those that have compared multiple techniques were limited to various types of meditation [36]. Furthermore, only one study has compared the effects of nonspecific relaxation techniques versus mind-body exercise on parasympathetic modulation in the same experimental group [42].

To summarize, there have been no studies investigating the effects of Hatha yoga, in its more vigorous forms, on HRV, acutely or chronically. Relaxation and mind-body exercise have been shown to acutely increase parasympathetic modulation but may act via different mechanisms. No studies have examined the acute effects of the combination of mind-body exercise with relaxation on the ANS. Therefore, the purpose of the proposed study is to compare the acute sympathovagal changes, as measured by heart rate

variability, that occur in men and women during relaxation following a Hatha yoga session and during relaxation only. Comparing the additive effects of a physical form of stress reduction to a non physical method of stress reduction may potentially lead to better therapeutic and preventative uses for mind body exercise.

It was hypothesized that yoga plus relaxation would produce greater changes in indices of HRV than relaxation alone.

II. REVIEW OF LITERATURE

THE AUTONOMIC NERVOUS SYSTEM AND REGULATION OF HEART BEAT

Regulation of the heartbeat is influenced by the balance between sympathetic and parasympathetic control. The variance between R-R intervals can be used to assess this balance [34]. Increased parasympathetic input creates a more electrically stable myocardium that is adaptable to changes in the body and sensitive to neurological commands [21], whereas a sympathetic dominance creates an electrically unstable myocardium that is at greater risk for random myocite firing as is the case with PVC's or more severe cardiac arrhythmias [21]. Greater parasympathetic activity is associated with a greater variability in heart rate but it is important to note that increased variability at rest from predominately parasympathetic influence is very different from a pathological arrhythmia.

The parasympathetic pathway begins at the medulla oblongata and innervates the heart as the vagus nerve primarily at the SA node and the AV node. The vagus nerve releases acetylcholine to which the muscarinic receptors respond and exert a slowing effect on the SA node [26]. The vagal impulses; however, are very brief because their action is prompt, needing no second messenger, and the rapid hydrolysis of acetylcholine by cholinesterase (50-100 ms) [25]. Thus, the vagus nerve is said to have beat by beat control over the SA node [1]. In healthy individuals, the heart is under parasympathetic control at rest [8, 25].

The sympathetic pathway emerges from the cervical spinal cord, innervates the heart at the base and is distributed along the myocardium. The adrenergic receptors are responsible for responding to norepinephrine resulting in increased heart rate. However, unlike the quick acting parasympathetic pathway, the sympathetic pathway has a much more gradual onset and clearance because it relies on second messengers, namely cAMP [26]. Also, the norepinephine released through one cardiac cycle is only able to make small changes compared to that of acetylcholine [26]. Furthermore, norepinephrine reuptake is slow compared to that of acetylcholine [26].

Natural rhythmic variations in heart rate exist from the ANS responses to baroreceptors, atrial receptors, and ventilation (respiratory sinus arrhythmia), as well as thermoregulation, renin-angiotensin system, as well as peripheral and central chemoreceptors [26]. It is the interaction between these two systems and the above influences that affect the variation in heart rate [43]. Therefore, the term modulation was proposed as a more accurate term than tone [47]. The greater the parasympathetic tone, the greater the beat by beat response and the greater the variation in heart rate [30].

Cardiac autonomic control can be determined by a valid, reliable, and non-invasive measure of HRV [34] to quantitatively assess cardiac autonomic activity and thus determine parasympathetic modulation [43]. HRV is a measure that can be expressed as a time domain or as a frequency domain [43]. The time domains assess various information that is obtained by the length between the R-R intervals. The most common time domain include, the square root of the mean of the sum of the squares of the differences between adjacent R-R intervals (RMSSD) and the standard deviation of the all R-R interval (SDNN) [47]. R-R interval (RRI) may also be used. Usually these are

expressed as milliseconds (ms) or milliseconds squared $(ms)^2$.

After applying a fast Fourier transformation (FFT) to the R-R interval data, frequency domains are created, which are separated as high frequency and low frequency [7]. This method, referred to as power spectral analysis, reflects the R-R intervals as an amplitude of HR fluctuations present at different frequencies The R-R interval signal is separated into sinusoidal functions of different frequencies from which a power spectrum is then created. This is done by plotting amplitude as a function of frequency. On the x-axis, LF falls between 0.04-0.15Hz and HF falls between 0.15- 0.4Hz) [42].

Parasympathetic input physiologically has a fast acting and transient effect on the heart; therefore, the parasympathetic modulation represents a high frequency domain on a power spectral analysis of heart rate variability. Therefore, high frequency power (HF) is predominately parasympathetic [1]. Conversely, because sympathetic activity has a slower acting phase and a longer lasting effect, a low frequency power is reflected on a power spectral analysis. Even though low frequency (LF) power does have minimal parasympathetic influences, it is mainly sympathetic and therefore represents sympathetic tone [4]. Total power (TP) and low to high power ratio (LF:HF) may also be used to assess frequency parameters. Frequency domains can be expressed in natural logs (ln), milliseconds (ms), or in normalized units (nu).

THE AUTONOMIC NERVOUS SYSTEM, CVD AND MORTALITY

Parasympathetic/sympathetic balance is increasingly becoming a topic of interest in the prevention and management of cardiovascular diseases. Decreased HRV indicates decreased parasympathetic modulation and increased sympathetic dominance and is associated with cardiovascular disease and mortality. Heart rate variability has been used

as a predictor of mortality not only in patients with CHD and CHF, but also in healthy controls. The Framingham Heart Study found that independent of other risk factors, persons with low HRV had greater incidences of adverse cardiac events and all-cause mortality [45]. Consequently, those who have the lowest HRV had the highest rate of sudden cardiac death. For example, the UK-Heart Study found that out of 433 patients with moderate or severe HF, those with impaired HRV had a 51.4% annual mortality rate compared to only 5.5% in those with near normal HRV values [33].

The Atherosclerosis Risk in Communities study (ARIC) was the first population-based study to examine the relationship between HRV and the development of CHD [27]. The results supported the hypothesis that a decreased cardiac autonomic function was related to the incidence of CHD. Individuals in the lowest HRV quartile had a 1.75 relative risk for incidence of CHD compared to those in the highest HRV quartile. More specifically, the time domains of overall HRV and the HF components of HRV were shown to be significant, suggesting that the imbalance of sympathetic and parasympathetic activity, especially the reduced parasympathetic activity, as shown by HRV, is associated with CHD [27]. Furthermore, HRV is depressed in those who have suffered myocardial infarction [13]. Kaufman et al. (2007) demonstrated in children significant relationships between HF, LF:HF ratio, adiposity and peak dilation of the brachial artery in response to nitroglycerin. The authors suggested that the results support the hypothesis that endothelial function may be impaired in environments of increased sympathetic activity and decreased parasympathetic activity. Furthermore, the relationship between ANS and vascular function was associated with inflammatory markers and insulin resistance, two cardiovascular risk factors. The authors noted that the

importance of this finding was that sympathetic dominance may begin in early childhood.

Marwah et al. (2007) recently proposed a new paradigm in the development of atherosclerosis. The team suggested that atherosclerosis risk factors promote a vascular sympathetic bias and that atherosclerosis may participate in the cycle of global sympathetic bias of a neurogenic origin.[31]. Litchtor et al. (1987) similarly discussed atherosclerosis as an autonomic dysfunction. In his research, monkeys that underwent sympathectomy had a resultant decrease in the progression of atherosclerosis [28]. While the proposed idea that the progression of atherosclerosis is a neurogenic cycle of sympathetic dominance, this paradigm is still very controversial and not well studied. More studies must be done to substantiate these findings.

EXERCISE AND HEART RATE VARIABILITY

With the research clearly illustrating the detrimental effects of sympathetic dominance, any means of increasing parasympathetic tone and decreasing sympathetic tone may prove to be beneficial. Exercise has been shown to do just this [41]. However, much work still needs to be done in this area. The ideal intensity, duration, and mode have yet to be determined, as well as an exercise prescription for diseased populations, old and young, male and female as variations in HRV response may exist. Both training studies and acute-response studies may help elucidate HRV changes engendered by physical activity. Longitudinal studies have shown that aerobic exercise at intensities as low as 50% VO_{2max} increase HRV parameters, although frequency remains equivocal. The literature on acute studies indicates that aerobic exercise intensities as low as 65% VO_{2max} increases HRV parameters up to 24 hours post exercise. However, there is a lack

of information in the literature on both acute and longitudinal aerobic exercise regarding intensities less than vigorous.

LONGITUDINAL EXERCISE STUDIES

The DREW study [12] investigated the effects of a six week aerobic exercise program on HRV in 373 postmenopausal women. The women were randomized into one of four groups: control (no exercise), 4kcal/kg/wk, 8 kcal/kg/wk, or 12 kcal/kg/wk. The women alternated between the recumbent bike and treadmill, working at 50% VO_{2max}; the frequency and duration depended upon their target kcal/ week.

The results showed that the women who participated in the higher activity groups (8 and 12 kcal/kg/wk groups) had significant increases in the following HRV values: rMSSD, SDNN, LF and HF. The 4kcal/kg/wk group only had significant increases in LF. The results for the 12 kcal/kg/wk group were approximately 4 ms higher than the 4kcal/kg/wk group for both the rMSSD and SDNN values. The authors suggest that there is a threshold effect regarding aerobic exercise and HRV improvements, but caution that the results of the 4kcal/kg/wk group should not be dismissed as ineffective, but rather the threshold effect may take a longer period to be fully realized.

Jurca et al. (2004) found similar increases in HRV following eight weeks of moderate intensity training in forty-nine post menopausal women [20]. Eighty-eight post menopausal women were randomly assigned to either a control group (n= 39) or exercise training group (n=49). All participants in the exercise group completed eight weeks of supervised exercise at 50% VO_{2max} on the recumbent bike or treadmill. The women exercised for an average of 44 minutes 3-4 days/week.

The results indicated that the women in the exercise intervention group had

significant ($p \leq 0.001$) increases from baseline in all averaged HRV indexes: rMSSD (+25%), SDNN (+18%), HFln (+11%), LFln (+9%),and TPln (+6%). rMSSD increased from 18.1 to 22.6 ms, SDDN (ms) increased from 26.4 to 31.2, HFln (ms^2) increased from 4.73 to 5.23, LFln (ms^2) increased from 4.72 to 5.13 and TPln (ms^2) increased from 6.40 to 6.79. Frequency domain expressed in normalized units (nu) did not show any significant increases.

The results from this study and the DREW study suggest that it is possible to elicit HRV improvements with moderate exercise. The results of these studies may suggest that exercise at moderate intensity, such as Hatha yoga [9], may also increase HRV, but longer durations or more frequent participation may be required for the suggested threshold effect to take place.

Kiviniemi et al. (2006) studied the effect of an eight week high intensity training protocol [23]. Seventeen healthy, young, male subjects participated in the study. They were asked to run or jog at 70-80% HRmax for 30-60 minutes six days/wk. The authors measured RRI (ms) and HFln (ms^2). The training resulted in significant increases ($P \leq 0.001$) in both values from baseline [23].

ACUTE EXERCISE STUDIES

Pober et al. (2004) investigated the effects of an acute bout of moderate intensity activity on HRV. Eleven healthy, young, moderately active males cycled at 65% VO_{2max} for 60 minutes. This acute bout of moderate intensity activity resulted in increases in HF by 21% one hour post exercise and 18% twenty one hours post exercise. The acute effects of moderate exercise showed a 15.4%, 9%, 27.5%, and 11.1% change in SDNN after 1, 3, 6 and 22 hours post exercise, respectively. The percent change seen in rMSSD were

646.6%, 666.9%, 359% and 123.8% after 1, 3, 6 and 22 hours post exercise, respectively. The frequency domain changes one hour after exercise showed 21%, -39.8% and -48% changes for HFnu, LFnu and LF/HF, respectively. These frequency domain percentage changes remained significant 22 hours post exercise at 17.5%, -47.1% and -59.2%, respectively. This study demonstrates that exercise at moderate intensity does produce not only an acute increase in parasympathetic modulation, but a residual effect as well.

Halliwill et al. (2006) also found similar results of increased parasympathetic outflow immediately after 60 minutes of moderate intensity (60%VO_{2max}) cycling [16]. Nine healthy young men and women cycled for 60 minutes at 60%VO_{2max}. HRV was not measured but, rather, muscle sympathetic activity, total vascular resistance and calf blood flow. The results indicated significant, ($p \leq 0.05$) decrease in muscle sympathetic activity and total vascular resistance from baseline.

Hautala et al. (2001) studied the effects of an acute bout of long duration, high intensity exercise on HRV. Ten males (average age = 36 years) who were aerobically trained participated in the study. The subjects participated in a 75km cross country ski race (approximately 4.5 hours at 87% HR_{max}). HRV was monitored continuously for 24 hours before the race and 48 hours after the race. The results showed that after the race, a parasympathetic rebound or hypervagal effect occurred. On the first day following the race, HFnu was actually slightly lower than baseline pre exercise (45.2 baseline versus 43.3 one day after and 46.3 two days after, ($p \leq 0.01$), while LFnu was higher within the first day 54.8 baseline versus 56.7 one day after and 53.7 two days after the maximal exercise bout. No significant changes were observed in the mean R-R interval one day after compared with the mean R-R interval before maximal exercise (964 vs.949ms), but

2 days after exercise the mean R-R interval (1051, $p \leq 0.001$) was longer compared with baseline and one day after values. This suggests that after prolonged high-intensity exercise, an increase in sympathetic tone and a decrease in parasympathetic modulation occurred, which was the opposite of the results seen in the other acute studies. It was not until the second day that that HF increased and LF decreased [18].

It is clear from the previous research that exercise, at a range of intensities, increases HRV. It also appears that perhaps moderate exercise at greater durations and frequencies may be safer and just as effective if not more than near maximal exercise. However, more studies are needed to make definitive conclusions.

RELAXATION AND HRV

It is known that stress induces the fight or flight response, of which an increased sympathetic drive is a key underlying component. Furthermore, stress has been associated with increased cardiovascular disease [39]. Relaxation or stress reduction have also been shown to increase parasympathetic modulation; although no known training studies have been conducted to evaluate the effects of regular daily relaxation on HRV or CVD progression. Limited evidence also suggests that relaxation techniques do not have a lasting effect on parasympathetic modulation after the acute intervention.

In a 1994 study by Sakakibara et al., autogenic relaxation training was found to increase HRV in sixteen healthy college student subjects during the relaxation intervention from an average of 30ms to 40 ms, ($P \leq 0.008$) [40]. The controls who only sat quietly and practiced no form of relaxation had no change in HRV. No HRV data was recorded after the intervention and therefore, a possible residual effect of the relaxation cannot be determined. Furthermore, the autogenic group had a decrease in feelings of

anxiety ($P \leq 0.031$), whereas the control group did not [40].

Increases in autonomic modulation have been reported after acute practice of both Chinese Chi and Kundalini meditation [36]. Peng et al. (1999) found that the subjects who practiced the meditation techniques had significantly greater changes ($p \leq .001$) in heart rate oscillations and HRV than controls who were measured during sleep and metronome-paced breathing. The heart rate oscillations and HRV continued to be measured approximately 45 minutes post intervention. However, the changes in autonomic modulation did not last past the hour of meditation. Other meditation studies have shown similar transient changes in autonomic control. These include Zen mediation by Zen monks [24] and recitation of rosary prayers and yoga mantras [5]. However, like the Sakakibara study, HRV was not measured post relaxation.

Breathing techniques are also widely used as relaxation techniques. Peng et al. (2004) measured two breathing techniques, a relaxation breath and segmented breath, both of which emphasize low respiratory rates. The results showed increases in parasympathetic tone from baseline measurements (HRV (beats/min^2) 5.3 ±4.0 and 4.7±4.1, respectively from 4.1 ±2.9) [35]. Again, while these methods increase HRV during the actual intervention, the lasting effects of relaxation on HRV do not appear to be supported.

MIND-BODY EXERCISE AND HRV

Mind-body exercise combines characteristics of relaxation, such as mindfulness and breathing, but also incorporates physical movement. The current literature on mind-body exercise is limited to meditative forms of movement, such as cyclic meditation, which is a form of meditation that is combined with simple and slow yoga postures.

Unfortunately, no research has been conducted on more vigorous forms of mind-body exercise, such as Hatha yoga or power yoga. Nevertheless, based upon the current literature, mind body exercises do increase HRV, but no residual effects have been shown, nor have longitudinal studies been conducted to determine any training effect these exercises may have on the ANS.

TAI CHI

Lu et al. (2003) investigated the acute effects of Tai Chi on HRV. Twenty Tai Chi practitioners and twenty non-practitioners participated in the study. The Tai Chi practitioners practiced a classical 40 minute Tai Chi session, while the non-practitioners served as the control group. HRV was recorded before the Tai Chi practice for baseline measurements and at thirty minutes and sixty minutes post practice.

The results showed that Tai Chi acutely increased HRV and decreased the LF:HF ratio as well as heart rate and blood pressure, suggesting that the parasympathetic control dominated after the Tai Chi practice [29]. The HFnu increased significantly from 22.8 ± 14.6 normalized units (nu) before the Tai Chi session to 28.2 ±16.1nu 30 minutes post and to 30.6 ± 18.4nu 60 minutes after Tai Chi. In contrast, the LF/HF ratio decreased significantly from 2.5 ± 2.4 before Tai Chi to 1.8 ± 1.4 30 minutes after TCC and to 2.2 ± 2.9 60 minutes after Tai Chi. The percentage increases in HFnu 30 minutes and 60 minutes after Tai Chi were 38.3 ± 63.4% and 46.7 ± 61.4%, respectively, whereas the percentage decreases in LF/HF ratio 30 minutes and 60 minutes after Tai Chi were 19.6 ± 36.8% and 16.8 ± 51.7%, respectively. No significant differences were found in time domains.

Vaananen et al. (2002) also found similar results from acute Tai Chi practice [46].

Fifteen healthy elderly men and 14 young male college students, both groups with several years of Tai Chi experience, participated in the study. Immediately after two five-minute bouts of Tai Chi practice separated by 5 minutes of rest, a significant increase ($p \leq 0.05$) in HRV occurred in both young and elderly groups from baseline values. The young group experienced a 61% increase in SDNN from 56 to 90ms and a 143% increase in Total Variance from 3133 to 7598 ms^2 occurred. LF/HF ratio did not change in either group.

YOGA

In 2005, Clay et al. examined the metabolic cost of a 30-minute Hatha yoga session and determined that the average oxygen consumption was 7.59 $ml \cdot kg^{-1} \cdot min^{-1}$ or 2.17 METs. The author concluded that Hatha yoga may not offer cardiorespiratory benefits, but does offer benefits to increase strength and flexibility [9]. Furthermore, Clay et al. stated that during more intense sequences in hatha yoga, oxygen consumption can reach up to 5 METS, which may be considered moderate intensity for some individuals.

The yoga studies in the current literature that have looked at HRV were forms of yoga of even lower intensity than the Hatha yoga Clay measured, however, increases in HRV were still elicited.

Khattab et al. (2007) investigated the acute effect of an Iygenar yoga program originally developed for cardiac patients. Eleven healthy yoga practitioners participated in the intervention and eleven healthy individuals with no relaxation experience served as the placebo group. The age range was 26-58 years. A gentle 90 minute Iyengar yoga protocol was used as the intervention. This protocol consisted of Shavasana, Supta Baddha Konasana with support (supine, bound angle pose), Purvottanasana on bench and

support (intense stretch of the front of the body, Trikonasana with a trestle (triangular standing pose), Parshvakonasana with a trestle (lateral angle standing pose), Ardha Chandrasana with a trestle (half moon standing pose, Prasarita Pardottasasana, concave back (spread legs, intensely streched), Bharadvajasana, sitting on chair, hands on trestle (twisting pose), Adho Mukha Shvanasana with ropes (downward facing dog pose), Shirshasana (headstand), Viparita Dandasana with bench (inverted stick pose), Dhanurasana with or without support (upward bow pose), Sarvangasana with chair (shoulderstand), Halasana with support (plough pose), Bhismacharyasana with support (backbend), Setubandha Sarvangasana with support (bridge pose), Viparita Karani on Setubandha Bench (inverted lake pose), and Shavasana with support. A gentle walk in the park and supine rest was used for the placebo group. A control group performing no yoga or relaxation was also included.

The yoga group increased HRV time domains during the intervention itself, while no significant changes were noted for the placebo or control groups. R-R interval was significantly higher during the yoga session compared with the placebo and the control groups, (865±119 ms; 746± 86 ms; 753± 115 ms, respectively, $p \leq 0.001$). SDNN was 86.9± 16 versus 62.9± 53.ms ($p \leq 0.001$); rMSSD was 37.3± 10 versus 30.1± 9 versus 24.1± 12 ms ($p \leq 0.01$). There were no significant changes in HRV outside the intervention time. Frequency domains were not analyzed [22].

In a similar study, Sarang et al. (2006) compared the effects of cyclic meditation (CM) and Shavasana (SR), which is a form of deep supine relaxation. Forty-two male subjects aged 18-48 years with at least 3 months CM experience participated in the study. Cyclic meditation (CM) can be described as a moving meditation. Moving meditation

combines meditation with simple yoga postures that are held for one to two minutes each. In this particular study, the participants were led through a guided meditation through the postures and instructed to focus on moving slowly with awareness and relaxation. The CM protocol consisted of five phases. Phase 1 included supine rest for two minutes thirty seconds and centered standing (Tadasana) for two minutes thirty seconds. Phase 2 included standing side bends to the right and left (Ardha Chandrasana) each for a minute and twenty seconds with a one minute and ten second centered standing (Tadasana) gap. Phase 3 included a forward bend (Uttasana) and backbend each for a minute and twenty seconds with a centered standing pose for one minute ten seconds between. Phase 4 and 5 consisted of supine relaxation. The SR consisted of supine relaxation (Shavasana) only.

The subjects were divided into two groups, with the first group practicing CM on day one and SR on day two. The second group practiced the reverse order, and then both groups switched in a crossover design. Both the CM and SR session lasted 35 minutes. The results of this study showed an increase in LF during the CM postures from baseline, whereas HF increased after CM from baseline. Shavasana (SR), showed no significant differences between pre and post measures, or during SR. During the CM phase 3, HFnu was significantly lower from baseline ($p \leq 0.001$), while LFnu was greater ($p \leq .05$) and LF/HF was significantly greater ($p \leq 0.001$). Compared to post tests, CM resulted in increased HFnu and decreased LFnu and LF/HF, whereas there were no significant changes in the control group who practiced Shavasana. R-R intervals were higher in all phases of the CM group compared to the SR group [42].

The potential cardioprotective benefits of the increasingly popular mind-body exercise of Hatha yoga are unexplored. Given the current literature, yoga and stress

reduction techniques may help to improve sympathovagal balance. The extent to which Hatha yoga is able to increase HRV has not been sufficiently addressed, nor has the HRV response been differentiated to determine how much change in parasympathetic modulation is from the relaxation experienced during yoga and how much is from the physical movement performed throughout the session. The goal of the proposed study is to determine if the yoga plus relaxation induces a greater parasympathetic response than the relaxation alone. It is the unique combination of relaxation and physical postures that spark the curiosity and devotion of its practitioners. As science begins to better understand the ANS responses to exercise and stress reduction, yoga emerges as an inviting form of practice that attracts researchers from both psychology and exercise physiology.

III. METHODS

PARTICIPANTS

Fifteen women and five men between the ages of 18-50 were recruited from Florida Atlantic University's student population and area Yoga studios to participate in the study. Each participant was asked to complete a Health History Questionnaire as well as a Physical Activity Questionnaire from which the primary inclusion criteria were identified, including participation in a Hatha yoga class at least once per week for the past two months prior to participation in the study. Exclusionary criteria included any previous history of musculoskeletal disorders that could prevent them from performing the postures, as well as any medical conditions, such as cardiovascular disease, taking medications that would affect heart rate or blood pressure, or have known cardiac arrhythmias. Individuals who engaged in regular aerobic or strength training exercise (> 3 days per week) over the prior six months were also excluded.

During the familiarization session, participants were shown images of the postures that they would be asked to perform during the yoga session. If the subject felt that he/she was not capable of performing any of the postures, they were not included in the study. All procedures were approved by the Florida Atlantic University Institutional Review Board for Human Subjects Research before initiation of the study, and each participant was advised of any possible risks before providing written informed consent.

STUDY DESIGN

This study used a randomized crossover design. Participants were initially randomly assigned to the yoga plus relaxation (YR) or the relaxation only group (R). After completion of the first laboratory session, participants subsequently switched groups, so that all subjects participated in both conditions. Participants completed the two conditions on two different days at least 48 hours apart at the same time of day. Female subjects completed both trials during the follicular phase of their menstrual cycle, because the luteal phase has been associated with increased sympathetic activity [3]. All sessions were performed in room 254 of the Education and Science building at the FAU Davie campus. Subjects were instructed not to consume any caffeine or alcohol at least 24 hours prior to testing, avoid eating at least 1 hour prior to testing and avoid strenuous activity 24 hours prior to testing.

The HRV data was measured and recorded during all sessions with a Polar RS 800 heart rate monitor (Polar Electro, Kempele, Finland), using a two way telemetry system with signal transduction via the Polar Advantage Interface receiver to a PC. Any ectopic beats were filtered by Polar, Inc. software. The time domains that were assessed were 1.) the square root of the mean of the sum of the squares of the differences between adjacent R-R intervals (RMSSD), 2.) the standard deviation of the all RR interval (SDNN), and 3.) the portion of all RR intervals having a difference of greater than 50ms (pNN50). This data was determined from the time interval between the R-R intervals. A Fourier transformation was applied to the R-R interval data to determine the frequency domains. The frequency domains that were analyzed included 1.) low frequency (LF), 2.)

high frequency (HF), 3.) total power (TP) and 4.) the low to high frequency ratio (LF:HF).

INTERVENTION PROTOCOL

Under the YR condition, subjects were led through a traditional 60 minute Hatha yoga session by watching Ganga White's, *Total Yoga* DVD. This sequence included sun salutations, standing, balance, forward bends and backward bends. The specific postures included on the DVD are: mountain pose, forward bend, lunges, cobra, plank, side plank, downward dog, upward dog, triangle pose, chair pose, warrior one, side angle pose, half moon pose, tree pose, wide leg pose, boat pose, staff pose, and Shavasana (5 min). The second part of the YR condition was relaxation, contiguous with the yoga, which included 30 minutes of Shavasana (supine) for 30 minutes while listening to the CD by Shiva Rea, *Drops of Nectar*. Specifically, Shavasana includes bringing awareness and relaxation to each part of the body and focusing on positive statements while soft music plays in the background. The relaxation condition included only the 30 minute Shavasana. Breathing frequency was not controlled as previous research by Bloomfield (2001) suggests that that controlled breathing may actually decrease parasympathetic modulation [6].

DATA ANALYSIS

A 2-way repeated-measures analysis of variance (ANOVA) was used with the effects of relaxation condition [relaxation only (R), yoga plus relaxation (YR)] and time (baseline, relaxation). Post-hoc paired t-tests were used when appropriate to probe for statistical significance. A p value <0.05 was accepted as statistically significant. The statistical analysis was performed using the Statistical Package for Social Sciences (SPSS) for Microsoft Windows (Version 17.0, 2006; SPSS, Inc., Chicago, IL).

V. RESULTS

Descriptive characteristics of the subjects, n=20 (15 female, 5 male), are presented in Table 1. Baseline values for LF, HF and LF/HF ratio were comparable with previously reported norms (42). There were no significant differences ($p > 0.05$) between the two baseline measurements for any of the dependent/HRV variables. Time and frequency domains for HRV, at baseline and at the end of relaxation for both conditions, are shown in Table 2. There were significant main effects for time for HR (bpm, $p < 0.001$), RR (ms, $p < 0.001$), pNN50 (%, $p = 0.004$) LF (%, $p < 0.001$), HF (ms^2, $p = 0.009$), HF (%, $p < 0.001$), and LF/HF ($p = 0.035$). For the YR condition, post-hoc analysis showed significant changes from baseline in HR ($p < 0.001$), RR ($p < 0.001$), pNN50 ($p = 0.009$), LF ($p = 0.008$) and HF (% $p = 0.035$). The R condition showed significant changes from baseline in HR ($p < 0.001$), RR ($p < 0.001$), HF (ms^2, $p = 0.004$), LF (%, $p = 0.005$), HF (%, $p = 0.008$) and LF/HF ($p = 0.008$). There were no significant differences between conditions for the changes from baseline for any of the variables; no significant time by condition interaction effect was detected ($p > 0.05$) for any of the HRV variables.

Table 1

Subject Characteristics (n=20)

Age (yrs)	29.15 ± 6.98
Height (cm)	163.66 ± 8.01
Weight (kg)	66.36 ± 20.76
Body Fat (%)	20.00 ± 7.72
Systolic BP (mmHg)	116.00 ± 14.48
Diastolic BP (mmHg)	74.30 ± 11.09
Non-yoga Exercise (met hrs/wk)	22.02 ± 15.23
Yoga (days/wk)	1.95 ± 1.23

Values are mean ± SD.

Table 2

Measures of heart rate variability at baseline and post-relaxation (n=20)

	YR Baseline	YR relaxation	R Baseline	R Relaxation
HR (bpm)	66.31 ± 10.16	60.64 ± 8.74*	64.77 ± 7.52	61.34 ± 7.31*
R-R (ms)	927.27 ± 156.20	1009.43 ± 146.88*	938.74 ± 113.44	990.97 ± 114.92*
RMSSD (ms)	53.77 ± 29.74	60.09 ± 27.11	54.88 ± 21.85	62.83 ± 30.15
pNN50 (%)	28.70 ± 23.39	38.29 ± 24.58*	31.15 ± 19.29	37.32 ± 23.75
LF (ms^2)	1155.85 ± 835.67	801.15 ± 511.55	1246.30 ± 1042.12	1255.30 ± 1290.02
LF (%)	45.88 ± 19.27	36.03 ± 17.92*	48.70 ± 17.64	39.19 ± 18.22*
HF (ms^2)	1409.85 ± 1476.16	1580.95 ± 1292.14	1028.15 ± 707.06	1762.45 ± 1346.09*
HF (%)	47.87 ± 20.97	56.82 ± 22.57*	45.90 ± 18.62	58.61 ± 21.56*
LF/HF (%)	1.58 ± 1.97	0.95 ± .93	1.47 ± 1.16	0.95 ± .68*
TP (ms^2)	2692.88 ± 1982.70	2546.05 ± 1306.18	2312.90 ± 1594.56	3090.74 ± 2298.12

Values are mean ± SD.

* $p \leq 0.05$; post-intervention compared to baseline.

YR Baseline= yoga plus relaxation condition baseline measures (last 5 minutes)
YR Relaxation= yoga plus relaxation condition for the relaxation phase (last 5 minutes)
R Baseline-=relaxation only condition baseline measures (last 5 minutes)
R Relaxation= relaxation only condition for the relaxation phase (last 5 minutes)

V. DISCUSSION

To the best of our knowledge, this is the first study to examine the effect of a bout of Hatha yoga prior to guided relaxation on heart rate variability, compared to relaxation alone. Briefly, the results demonstrate that 1) relaxation produces favorable changes in indices of heart rate variability whether alone or preceded by a bout of Hatha yoga and that 2) the changes were not significantly different between relaxation conditions.

Both conditions showed an increase in parasympathetic tone as seen by the increase in RR, HF%, $HFms^2$ (R group), and pNN50 (YR group), while decreasing sympathetic control as seen by the decrease in HR, LF%, and LF/HF ratio, thus shifting the cardiac autonomic balance towards a more stable cardiac environment. While there were also non-significant trends in the R condition for rMSSD ($p= 0.072$, TP ($p= 0.072$, and pNN50 ($p= 0.064$), it does not appear that under R condition there was any real difference between the two relaxation conditions.

PREVIOUS RELAXATION STUDIES

Our results indicate that relaxation does increase HRV when done alone or when preceded by Hatha yoga. The type of relaxation performed focused thoughts on relaxing each body part, as well as the internal body, and focused on feelings of connectedness and gratitude. Other forms of relaxation have also been shown to increase HRV, including various types of meditation and autogenic training.

Miu et al. (2008) demonstrated that compared to acute stress, autogenic training (a form of relaxation in which awareness is brought to the body and thoughts are focused on heaviness and warmth of each body part) produced significantly greater HF (ms^2) and RR (ms) measures of HRV, demonstrating that autogenic training has the opposite effect on HRV as stress [32]. Sarang et al. (2006) found that cyclic meditation, which is an active form of meditation consisting of holding simple postures while focusing on relaxing the body, has been shown to decrease HR, LF(nu), and LF/HF ratio from baseline, while HF(nu) increased after practicing for 20 minutes [42]. The present study had similar results with HR, LF%, LF/HF decreasing and HF% increasing for both relaxation preceded by Hatha yoga and relaxation alone, compared to baseline.

Other studies have shown that relaxation can increase parasympathetic control, however, not to the same degree as the present study. Sakakibara et al. (2007) studied the effects of autogenic training on HRV and found increases in HF (ms^2) only. HR, LF (ms^2), and LF/HF ratio did not change significantly [40]. The current study, on the other hand, showed that relaxation not only increased HF% and HF (ms^2), but also lowered HR, LF% and LF/HF ratio. The difference in HRV response may have been a function of time. The Sakakibara study only examined relaxation for 15 minutes preceded by a baseline measurement of 5 minutes, compared 30 minutes of relaxation preceded by a 20 minute baseline in the present investigation. Bernanrdi et al. (2001) demonstrated that the use of mantras and prayer are able to slow breathing, increase baroreceptor sensitivity (a marker of increased parasympathetic tone) and increase RR, compared to free talking [5]. While these findings do appear to support the findings of the present study, no measurements of HRV, aside from RR, were reported.

Contrary to the results of the present study, relaxation findings from Peng et al. (2004), showed that in two forms of meditation, relaxation response and segmented breathing, LF and LF/HF ratio increased, with an increase in HR for the segmented breathing group. There were no significant changes noted for HF in either group [35]. In the segmented breathing condition, the authors attributed respiratory sinus arrhythmia for at least some of the changes seen. The authors call the results a ‘‘meditation paradox,’’ since these so-called meditative techniques, especially the altered breathing patterns, may produce active rather than calming cardiac dynamics, associated with prominent low frequency and increases in heart rate [35]. On the contrary, the findings of the present study, which did not manipulate breathing rate, indicate that relaxation did in fact produce a parasympathetic increase as seen by the increase in HF, RR, and decrease in heart rate.

Contrary to the relaxation or mediation techniques of Peng [36] and Bernardi [5], the relaxation technique used in the present study, and those by Sarang [42], Sakakibara [40], and Miu [32], did not manipulate breathing rate. Previous research by Bloomfield (2001) showed that controlled breathing may actually decrease parasympathetic modulation [6]. Therefore, the relaxation technique used in the present study may more accurately reflect cardiac parasympathetic modulation associated with relaxation than past studies.

LIMITATIONS AND FUTURE DIRECTIONS

This study assessed changes in heart rate variability during relaxation alone versus relaxation preceded by Hatha yoga. The inclusion criteria required all participants to be practicing yoga at least one day per week, two months of yoga experience prior to the study. However, there were some individuals who had been involved on and off in yoga for years, but only practicing consistently at least two months prior to participation in the study. Likewise, there were participants who were relatively new to yoga, but met the minimum requirement of practicing consistently for two months prior to the study. Even though the inclusion criteria and the familiarization session should have been sufficient time to become comfortable with the postures performed during the yoga session, the participants' varying knowledge on yoga and understanding of the philosophy behind performing the postures may have produced different degrees of awareness of the body and receptiveness of the benefits of the postures [14] This may therefore have created some variability in the results and may be a potential limitation of the study.

Another potential limitation may have been psychological stress felt by some participants during the yoga session. The sessions were conducted one participant at a time, and for safety purposes, under the supervision of the same yoga instructor. Participants were not accustomed to practicing yoga for research purposes nor in a research setting, this may have left participants feeling nervous and under pressure to perform the postures perfectly. Given the physiological effect of emotions on heart rate variability (11, 38); this may have altered the effects of the yoga and inhibited potential parasympathetic benefits.

Future studies might strive to replicate the current study with participants who are more uniform and experienced in their knowledge of yoga. Future studies should also study relaxation and yoga separately. These should compare multiple styles and intensities of yoga to determine clearly the sole effect of Hatha yoga on heart rate variability, as well as studies on various types of relaxation. In addition to acute studies, longitudinal studies on the effects of relaxation are warranted. The majority of studies reveal that relaxation produces a transient effect on HRV, however, long term relaxation studies are lacking. It is unclear if practicing relaxation daily produces chronic changes in HRV. Finally, neither the present study nor past yoga or relaxation studies have investigated the physiological mechanisms for the increases found in heart rate variability seen with relaxation techniques. Therefore, future studies may work towards elucidating how relaxation is able to physiologically induce a parasympathetic response. In addition to physiological measures of relaxation and/or yoga, standardized questionnaires evaluating subjective feelings of relaxation may also be helpful in evaluating the benefits of such practices.

In conclusion, the results of the present study add support to past studies that have found various relaxation techniques to increase parasympathetic tone. Furthermore, it appears that performing Hatha yoga prior to engaging in deep relaxation does not produce greater changes in heart rate variability than practicing relaxation alone. These findings may have practical implications for health and well-being and provide a foundation for future studies of the benefits of mind-body exercise.

VI. APPENDIX

Appendix A: List of Abbreviations and Definitions

RMSSD	square root of the mean of the sum of the squares of the differences between adjacent R-R intervals
pNN50	The proportion of RR intervals that are greater than 50 ms.
RRI	R-R interval
TP	total power
LF	low frequency (mixed parasympathetic and sympathetic)
HF	high frequency (parasympathetic tone)
LF:HF	low to high power ratio

Appendix B: Normal Values of Standard Measures of HRV (42).

Variable	Units	Normal Values (mean±SD)
Time Domain Analysis of Nominal 24 hours		
SDNN	ms	141±39
RMSSD	ms	27±12
HRV triangular index		37±15
Spectral Analysis of Stationary Supine 5-min Recording		
Total power	ms^2	3466 ±1018
LF	ms^2	1170±416
HF	ms^2	975±203
LF	nu	54±4
HF	nu	29±3
LF/HF ratio		1.5-2.0

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