Slow Yogic Breathing and Long-Term Cardiac Autonomic Adaptations: A Pilot Study

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Abstract

Objectives: To examine whether long-term practice of yogic breathing alters cardiac autonomic control.

Design: Age–sex matched, cross-sectional, physiologic pilot study.

Settings/Location: Spaulding Rehabilitation Hospital, Cardiovascular Laboratory, Cambridge, MA.

Participants: Twenty-six (26) long-term yoga practitioners and 26 age- and sex-matched controls, free of cardiovascular disease.

Outcome: Cardiac vagal outflow as assessed by respiratory sinus arrhythmia (RSA).

Results: During unpaced (spontaneous) breathing, yoga practitioners exhibited augmented RSA compared to controls (yoga 364.8 ± 75.3 vs. 194.7 ± 46.0 ms²Hz⁻¹, $p=0.03$). However, during paced breathing at 0.25 Hz (15 breaths/min), which accounts for inter- and intravariability in breath rate, RSA did not differ between groups (yoga 224.8 ± 48.4 vs. 271.3 ± 59.7 ms²Hz⁻¹, $p=0.98$). Furthermore, the relationship between age and RSA, such that RSA declines with age, did not qualitatively differ between groups.

Conclusions: Long-term practice of yogic slow breathing does not appear to augment cardiac vagal control nor prevent known age-related declines.

Keywords: yoga, autonomic, vagal, heart rate variability, respiratory sinus arrhythmia

Introduction

REGULATED BREATHING IS fundamental to all mind–body therapies and is considered an essential pathway to establish the mind–body connection. Across multiple meditative traditions, breath control is considered vital to steadying the mind and thus essential to well-being. Of the hundreds of mind–body breathing practices described,^{1–8} the majority focus on slowing the breath. This is achieved through conscious regulation (yoga, tai chi, Zen Buddhist) or reflexive deceleration (Transcendental Meditation, Vipassana, relaxation response).⁸

Numerous studies have suggested that mind–body practices acutely impact cardiovascular regulation,^{9–17} as evidenced by the amplification of respiratory sinus arrhythmia (RSA) (i.e., heart period variations in synchrony with the respiratory cycle)

with slowing of the respiratory rate. These findings suggest that modulation of autonomic activity may be one critical common denominator underlying the physiologic mechanisms driving the potential therapeutic effects of mind–body therapies.

However, whether years of repetitive practice of slow breathing causes sustained alterations in autonomic activity remains unknown; the vast majority of cross-sectional studies assessing the impact of slow meditative breathing on heart rate variability were limited to either practitioners^{13,16,17} or healthy nonpractitioners only,^{9–12,14,15} and the vast majority of randomized control trials were of relatively short duration (i.e., intervention ≤ 12 weeks) and did not account for variation in respiratory rate between the intervention and control groups.^{18–24}

We sought to determine if advanced yoga practitioners, defined by at least 5 years of regular practice of yoga that

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routinely incorporated slow breathing techniques, exhibit adaptations in cardiac vagal efferent outflow, as evidenced by augmented RSA compared to age- and sex-matched controls. By comparing long-term practitioners to non-practitioners (i.e., those without substantive practice with slow meditative breathing), we could discern whether long-term practice of yogic slow breathing imparts long-term alterations in cardiovascular control.

Materials and Methods

All procedures were approved by the Institutional Review Board at Spaulding Rehabilitation Hospital and conformed to the Declaration of Helsinki, with the Beth Israel Deaconess Medical Center Institutional Review Board ceding review. All participants provided written informed consent.

Study participants

Twenty-six (26) men and women with at least 5 years of regular practice in of a slow, yogic breathing technique (long-term practitioner) were recruited from the greater Boston area and local yoga studios. All participants were aged 20–55 years, with a body-mass index 18.5 to <30 kg/m², and a normal resting electrocardiogram (EKG). Telephone screening was conducted to evaluate criterion for long-term practitioners. This was defined as a minimum of 5 consecutive years of consistent practice in yoga, a self-reported strong agreement with the statement “Breathing is one of the fundamental aspects of my practice,” rate the importance of slow breathing to their practice of 4 or 5 on a 5-point Likert scale (1=not at all important to 5=very important), and regularly incorporate at least one slow breathing technique into their practice. Nonpractitioners ($n=26$) were recruited from the community and were defined as a person without any prior experience with yoga, Tai Chi, *qigong*, or meditation (including, but not limited to, Transcendental Meditation, Zen meditation, Buddhist meditation, “relaxation response,” or mindfulness meditation). Exclusion criteria included the following: (1) previously diagnosed hypertension, current antihypertensive medication use or high blood pressure (systolic ≥ 140 or diastolic ≥ 90 mm Hg) at either study visit, (2) self-reported prevalent cardiovascular or pulmonary disease, (3) history of diabetes, (4) weight change >10 lbs within past 6 months, (5) self-reported tobacco or illicit substance use, (6) consumption of >10 alcoholic drinks/week for women or >15 alcoholic drinks/week for men, (7) use of antipsychotics, stimulants, or anxiolytics, (8) pregnancy, and (9) presence of a condition prohibiting ability to consent or perform protocol.

Measurements

A standard EKG (DASH 2000; General Electric) and beat-by-beat photoplethysmographic arterial pressure (Portapres; Finapres Medical Systems) were recorded continuously. Oscillometric brachial pressures (DASH 2000; General Electric) were taken every 3 min in the contralateral arm to ensure photoplethysmographic finger pressure calibration throughout the protocol. Respiratory depth and frequency were monitored and recorded using a respiratory transducer band around the mid chest. All signals were digitized and stored at 1000 Hz (PowerLab; ADInstruments).

Protocol

All studies were conducted in the morning after participants fasted overnight and refrained from strenuous exercise for >48 h, as well as from alcohol and caffeine for 24 h. Premenopausal women were studied during the follicular phase of their menstrual cycle. All participants were supine throughout the protocol. Baseline (unpaced breathing) recordings were obtained for 5 min. Subsequently, participants performed paced breathing through audio instruction at 0.25 Hz (15 breaths/min) for 5 min, followed by a 5-min recovery period. Although it has limitations,²⁵ phasic respiratory modulation of vagal outflow results in RSA, which is proportional to the mean level of cardiac vagal outflow. Controlled paced breathing at 15 breaths/min allows for accurate quantification of RSA amplitude by limiting variations in respiration (e.g., rate; volume; duty cycle) that strongly influence RSA.²⁶ Practitioners then practiced a self-selected slow-breathing yoga technique that they regularly incorporated into their practice, to allow for assessment of adaptations to a long-term pranayama practice. Yogic slow breathing was performed for 10 min, to ensure sufficient number of respiratory cycles recorded during slow breathing.

Data analysis

Baseline mean heart rate and blood pressure (oscillometric brachial blood pressure) were derived. R-R intervals were determined from the EKG recordings using a peak detection algorithm custom written in MATLAB (version 7.4; MathWorks) and were visually inspected for artifacts and errors. R-R interval rather than heart rate was used as it most linearly represents changes in parasympathetic chronotropic effect.^{27–29} RSA values were derived from power spectral analyses calculated with fast-Fourier transforms based on the Welch algorithm of averaging periodograms (MatLab) of the R-R interval time series.³⁰ Because of the difference in the length of the different breathing periods, all R-R interval time series were divided into five segments of equal length overlapped by fifty percent. While spectral resolution will be lower in shorter time series, the confidence in the estimates will be equivalent. RSA was assessed as the average R-R interval power over the band that corresponded to 80% of the respiratory fluctuations. Mean, systolic, and diastolic pressures were obtained from the continuous beat-by-beat blood pressure waveform over the data collection periods. To assess arterial pressure variability and R-R intervals, we used spectral characterization as described above.

Statistical analysis

Group differences (yoga practitioners vs. matched controls) in R-R power and systolic and diastolic pressure during unpaced and paced respiration were compared with unpaired *t* tests. Among yoga practitioners, differences in assessments during separate respiratory patterns (i.e., unpaced, 0.25 Hz, yoga) were compared using paired *t* tests. We also assessed the correlation between respiratory frequency and RSA, as well as the relationship between age and RSA, using Pearson's rank correlation coefficient.

TABLE 1. CARDIORESPIRATORY CHARACTERISTICS DURING UNPACED, PACED (0.25 Hz), AND YOGA BREATHING, BY GROUP

	Baseline (unpaced)		Paced breathing (0.25 Hz)		Yoga breathing	
	Control	Practitioner	Control	Practitioner	Control	Practitioner
Breathing frequency, Hz	0.20 ± .02	0.15 ± 0.02*	0.25 ± 0.00	0.25 ± 0.00	N/A	0.06 ± 0.01
Respiratory frequency, range, Hz	0.06–0.41	0.06–0.28	N/A	N/A	N/A	0.02–0.12
Respiratory frequency standard deviation, Hz	0.06 ± 0.01	0.05 ± 0.01	0.02 ± 0.00	0.03 ± 0.01	N/A	0.03 ± 0.00
R-R interval, ms	1051.1 ± 25.2	1022.3 ± 16.4	1002.5 ± 28.8	991.1 ± 15.1	N/A	1001.09 ± 13.64
Systolic blood pressure, mmHg	110.3 ± 2.6	106.6 ± 2.5	109.6 ± 2.0	108.4 ± 2.7	N/A	109.75 ± 2.44
Diastolic blood pressure, mmHg	66.5 ± 1.8	64.3 ± 1.6	64.8 ± 2.0	65.0 ± 1.7	N/A	65.18 ± 1.30

All data are presented as mean ± SE, except when indicated.

* $p < 0.05$.

N/A, nonapplicable.

Values are presented as mean ± SE, and differences were considered statistically significant when $p < 0.05$.

Results

Overall, our sample had a mean age of 36.3 ± 2.1 (range 22–55) years, was about 25% male, and had normal body-mass index. Practitioners ($n = 26$) reported an average 9.5 ± 1.1 (range 5–25 years) years of practice, encompassing a range of self-reported styles (hatha [7], vinyasa [7], kundalini [2], asthanga [1], iyengar [1], multiple/mixed [10]). By design, there were no group differences in age or sex, although the control group did have a modestly higher

body-mass index (25.3 ± 0.9 vs. 23.2 ± 0.6 kg/m², $p < 0.05$). Group cardiorespiratory characteristics during each breathing segment are shown in Table 1. R-R interval, systolic, and diastolic pressures did not differ between groups during either unpaced or paced breathing. During unpaced breathing, yoga practitioners had a slower mean respiratory rate compared with controls (9.0 ± 1.2 vs. 12.0 ± 1.2 breaths/min, $p = 0.03$), and RSA (i.e., R-R interval power) was, on average, about 50% higher in yoga practitioners compared to controls (364.8 ± 75.3 vs. 194.7 ± 46.0 ms²Hz⁻¹, $p = 0.03$). During paced breathing at 0.25 Hz (15 breaths/min), there were no differences in RSA between groups (yoga 224.8 ± 48.4 vs. control 271.3 ± 59.7 ms²Hz⁻¹, $p = 0.98$). Average respiratory

FIG. 1. Average power spectrums of respiration and R-R intervals by breathing type.

FIG. 2. Average respiratory and R-R interval power spectrums by yoga breathing style.

and R-R interval power spectrums by breathing type (paced, unpaced, yoga) are depicted in Figure 1.

For yogic slow breathing, 11/26 practitioners reported performing ujjayi breathing, six performed segmented breathing, three performed three parts breathing, and five

described other styles. During yogic breathing, practitioners achieved an average breath rate of about 3–4 breaths/min, over 10 min. While this rate was slower than during unpaced breathing, RSA did not differ between yoga and unpaced breathing (yoga 282.34 ± 34.9 vs. unpaced breathing 364.8 ± 75.3 ms² Hz⁻¹, $p=0.33$) among practitioners. Of note, among the nine yoga practitioners with a baseline (unpaced) breathing rate of 0.20 Hz or higher (mean 0.24 ± 0.01 Hz), during yoga breathing, breathing rate decreased to a mean 0.06 ± 0.01 Hz, and RSA increased on average by 20%. Figure 2 depicts the average respiratory and R-R interval power spectrum among practitioners by type of yogic breathing (ujjayi vs. other yoga).

In general, there was a relationship between RSA and breath rate (Fig. 3). Across the entire sample, during unpaced breathing, we found a correlation between RSA and mean breath rate $r=-0.44$, $p=0.001$. However, during yoga breathing, there was a modest nonsignificant correlation between RSA and breath rate ($r=-0.33$, $p=0.10$). Across all participants during paced breathing, we found a correlation between RSA and age ($r=-0.38$, $p<0.01$ (Fig. 4)), such that RSA was lesser among older participants. There was no substantive qualitative difference in the correlation between RSA and age between practitioners and controls (yoga $r=-0.43$ vs. control $r=-0.34$). The relationship between RSA and age remained during yoga breathing (-0.54 , $p<0.01$).

Discussion

We found that although RSA was higher in practitioners compared to age–sex matched controls at rest, RSA did not differ between groups, when controlling breathing frequency—a critical determinant of inter- and within individual variability in RSA. Among yoga practitioners, RSA was higher during yogic breathing than during paced breathing, but did not differ compared to unpaced breathing. In addition, long-term practice of slow yogic breathing did not appear to alter age-related declines in RSA during paced breathing. Hence, our findings suggest that after controlling for respiratory variation among individuals, long-term practice of yoga-derived slow breathing does not impart

FIG. 3. Respiratory sinus arrhythmia versus breath rate for all individual participants during unpaced breathing and for practitioners during yogic breathing.

FIG. 4. Respiratory sinus arrhythmia versus age during paced breathing.

sustained effects on vagal efferent outflow, nor reduce age-related declines in vagal outflow.

Human physiology researchers have explored the relationship between respiration and the cardiovascular system for hundreds of years.^{31–33} Sustained and appropriate tissue perfusion and transport of oxygen and carbon dioxide to and from cells demand continuous and complex interplay among the

FIG. 5. Representative example of R-R interval power spectrum in an individual during unpaced breathing and yoga breathing.

respiratory, cardiac, and vascular systems. It has long been known that respiration directly and indirectly influences fluctuations in heart period and blood pressure. These fluctuations are not merely an incidental consequence of breathing, but represent the interplay of numerous feed-forward and feedback systems. Respiratory-related cardiovascular oscillations may derive from changes in intrathoracic pressures,^{34–36} stimulation of arterial^{37,38} and/or cardiopulmonary baroreceptors,^{35,39} activity of pulmonary afferent stretch receptors,⁴⁰ and inspiratory motoneurons.^{41,42} Respiratory patterns not only determine the magnitude of these periodicities^{43,44} but are also a critical determinant of cardiopulmonary entrainment—the synchronization of multiple oscillators to the same frequency.^{45,46} For instance, at “normal” resting respiratory frequencies, RSA typically occurs at higher frequencies, above ~ 0.15 Hz (or ~ 9 breaths per minute), and is a primary peak in the R-R interval power spectrum (Fig. 5, unpaced breathing, representative data).^{43,47–50} When respiratory rates approach the resonant frequency (~ 0.10 Hz, or six cycles per minute), heart period and blood pressure oscillations are enhanced and this is reflected by a single large dominant peak in the power spectra (Fig. 5, yoga breathing).^{43,45} Therefore, observations that yoga and similar meditative techniques acutely augment RSA can be simply explained by their characteristic reduction in breathing frequency.⁸ For instance, Peng et al. demonstrated that the relaxation response and segmented breathing produced high-amplitude, low-frequency oscillations in heart period in Kundalini yoga practitioners compared to baseline respiration. These high-amplitude low-frequency oscillations were tightly coupled (i.e., entrained) to the slow respiratory rate.¹³ Similarly, during yoga nidra, heart rate variability indices show increased power compared to unpaced breathing in healthy volunteers with at least 2 months of hatha yoga practice.¹⁴ Our results describing large RSA during yogic breathing are consistent with these and numerous other studies.^{20,51,52} Although these findings suggest that slow-breathing meditative practices impart specific physiologic effects by creating a marked RSA, our study supports that breathing frequency, rather than subtype of breathing (yoga vs. unpaced), more strongly determines RSA. Furthermore, despite the plethora of evidence demonstrating the acute influence of slow breathing on RSA, evidence for a longer term influence of the practice of slow breathing on vagal outflow remains sparse.

In the past few years, several small randomized trials^{18,19,21–24,53} have investigated changes in various heart rate variability indices following a yoga intervention (intervention ranges 1 week to 6 months). While the vast majority of studies have demonstrated increases in HRV indices from baseline to end-intervention, the findings are difficult to interpret as breathing rate was not measured and group variations in respiratory rate were not considered. Given the large influence of breathing rate on RSA, it is therefore impossible to discern whether these group differences in RSA were due to alterations in vagal efferent flow, or merely represent changes in resting breathing rates during the testing periods. Our findings do not support that long-term yoga practice imparts changes on vagal efferent flow, as assessed by RSA during paced breathing. Measuring RSA during paced breathing avoids differences in respiratory rate that influence cardiovascular fluctuations⁴³ and, hence, allows for more meaningful comparisons of vagal efferent flow between different groups compared to RSA quantification during uncontrolled breathing. However, although beyond the scope of this work, it is possible that long-term practice of slow pranayama may lead to a lower resting breathing rate, which may therefore increase RSA at rest. We also did not find that long-term practice of yoga modifies known age-related declines in RSA, as the correlation between age and RSA was similar between practitioners and nonpractitioners. Taken together, our results suggest that while RSA is acutely impacted by yoga, it does not alter longer term vagal efferent outflow.

We also found that RSA did not differ between unpaced breathing and very low-frequency yoga breathing among practitioners. However, the data actually fit with some of our previous research.⁵⁴ Taylor and Studinger examined healthy participants who performed ramped frequency breathing between 0.05–0.25 Hz with constant alveolar ventilation and found that very slow breathing, around 0.05 Hz, reduces RSA, with maximal RSA occurring at resonant frequencies averaging around 0.08 Hz.⁵⁴ Hence, the lack of a significant difference in RSA during yoga breathing, compared to unpaced breathing, can be attributed to the large proportion of yoga practitioners whose resting breathing frequency was at or below 0.10 Hz. These individuals further reduced their breathing frequencies during yoga breathing, likely moving away from their resonant frequency and thereby reducing RSA. Whether this reduction in RSA occurring at very low breathing frequencies truly reflects a reduction in vagal outflow is difficult to discern, and previous studies suggest that RSA can plateau during states of very high vagal outflow.⁵⁵

Limitations

Our study had a number of strengths, including age–sex matched controls and careful assessment of respiratory frequency. This study also had certain limitations. Although we matched on age and sex, we were not able to concurrently adjust for other potential confounders, which could impact our findings. Although we had participants who were long-term yoga practitioners, our sample size precluded us from further exploring the potential differential influences of various yoga styles on our outcomes. Furthermore, as our main interest was yogic-slow breathing, as practiced in the community, we did not have a scripted yoga breathing

pattern, nor did we control for variations in duty cycle or fluctuations of carbon dioxide, which may influence RSA.^{50,56} Finally, we studied participants who were healthy, nonsmokers, and free of cardiovascular disease, and thus, our healthy sample may bias some of our results.

Conclusions

Our preliminary results suggest that long-term practice of yoga-derived slow breathing does not impact long-term alterations, nor effect known age-related decline, in cardiac efferent vagal outflow, as measured by RSA during paced breathing. During unpaced breathing, yoga practitioners demonstrated augmented RSA that was due to a lower breathing rate, and therefore, long-term practice of slow yogic breathing may have an impact on RSA during natural/unpaced breathing. Whether long-term practice of yoga reduces spontaneous breathing rates and whether or not this may impact other aspects of long-term autonomic control remain to be explored.

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Author Disclosure Statement

All authors state no competing financial interests exist.

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