# Vagal Mediation of Low-Frequency Heart Rate Variability During Slow Yogic Breathing

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## ABSTRACT

**Objective:** Changes in heart rate variability (HRV) associated with breathing (respiratory sinus arrhythmia) are known to be parasympathetically (vagally) mediated when the breathing rate is within the typical frequency range (9–24 breaths per minute [bpm]; high-frequency HRV). Slow yogic breathing occurs at rates below this range and increases low-frequency HRV power, which may additionally reflect a significant sympathetic component. Yogic breathing techniques are hypothesized to confer health benefits by increasing cardiac vagal control, but increases in low-frequency HRV power cannot unambiguously distinguish sympathetic from parasympathetic contributions. The aim of this study was to investigate the autonomic origins of changes in low-frequency HRV power due to slow-paced breathing.

**Methods:** Six healthy young adults completed slow-paced breathing with a cadence derived from yogic breathing patterns. The paced breathing took place under conditions of sympathetic blockade, parasympathetic (vagal) blockade, and placebo. HRV spectral power was compared under 11 breathing rates during each session, in counterbalanced order with frequencies spanning the low-frequency range (4–9 bpm).

**Results:** HRV power across the low-frequency range (4–9 bpm) was nearly eliminated ($p = .016$) by parasympathetic blockade (mean (SD) spectral power at breathing frequency = 4.1 (2.1)) compared with placebo (69.5 (8.1)). In contrast, spectral power during sympathetic blockade 70.2 (9.1) and placebo (69.5 (8.1)) was statistically indistinguishable ($p = .671$).

**Conclusions:** These findings clarify the interpretation of changes in HRV that occur during slow-paced breathing by showing that changes in low-frequency power under these conditions are almost entirely vagally mediated. Slow-paced breathing is an effective tool for cardiac vagal activation.

**Key words:** heart rate variability, yoga, paced breathing, autonomic blockade, vagal tone, sympathetic-parasympathetic balance.

## INTRODUCTION

Mounting evidence suggests that yogic breathing practices or pranayama has a wide variety of health benefits and may constitute a cost-effective, noninvasive primary therapy or adjunct to standard therapies (1–3). Yogic breathing techniques have been shown to reduce subjective measures of stress, anxiety, and depression (4,5), while producing objective reductions in hypertension and inflammatory markers as well as increasing cardiac autonomic control (6–9). Whereas an association between yogic breathing and health is reasonably clear, the underlying mechanisms are not well understood. Increases in vagal (parasympathetic) control are thought to underlie at least some of these cognitive, experiential, and physiological benefits (3,10). More generally, decreased vagal control is associated with increased risk of heart disease, depression, and anxiety in healthy populations and vagus nerve stimulation has been shown to be effective in treatment-resistant epilepsy and depression with improved long-term outcomes over standard treatments (11–13). Therefore, definitively determining whether yogic breathing practices promote cardiac vagal (versus sympathetic) control would be an important advance.

The sympathetic and parasympathetic nervous systems exhibit reciprocal activity as well as coactivation, producing checks and balances to neural control of physiologic processes (14). One manifestation of this reciprocal control at the cardiac level is the phenomenon of respiratory sinus arrhythmia (RSA), consisting of changes in the cardiac interbeat interval that covary with the inspiratory and expiratory breathing pattern. Sudarshan Kriya Yoga (SKY) ujjayi breathing, one popular, but not well studied, variant of yogic breathing, has been hypothesized to enhance vagal activity by lengthening the postinspiratory period in relation to the postexpiratory period through breath holding and prolonged expiration (15). The mechanisms behind RSA are likely the result of complex interactions between respiratory-driven baroreceptor and chemoreceptor activation, although these interactions are widely accepted to be vagally mediated for respiratory rates in the adult range (9–24 breaths per minute [bpm]) as evidenced

**bpm** = breaths per minute, **FFT** = fast Fourier transform, **GLM** = generalized linear model, **HF** = high frequency, **HRV** = heart rate variability, **LF** = low frequency, **RSA** = respiratory sinus arrhythmia, **SKY** = Sudarshan Kriya yoga, **VLF** = very low frequency

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by obliteration of RSA with parasympathetic blockade or vagotomy (16–19).

Heart rate variability (HRV) can be analyzed in the frequency domain, most commonly by fast Fourier transform or autoregressive analysis. The observed power spectral density peaks can be categorized into high (HF), low (LF), and very low (VLF) frequencies, with typical ranges of 0.15 to 0.40, 0.04 to 0.15, and 0.003 to 0.04 Hz, respectively (20). Previous pharmacological blockade studies have provided broad empirical support for the inference of vagal control from HF HRV (a periodicity in the change in interbeat intervals corresponding to 9–24 bpm) (21,22). The degree to which the LF oscillations represent sympathetic activity is not as clear, however (23). Physiological studies have recorded changes in LF power associated with tilt table manipulation (24) and vasodilatory challenge (25), strongly supporting a link between LF power and sympathetic nervous system activity under these conditions (26). However, previous pharmacologic autonomic blockade studies of resting participants have shown that parasympathetic blockade nearly eliminates HF power but also greatly reduces LF power. Pomeranz et al. (27) concluded that LF and HF power were entirely mediated by parasympathetic activity for resting participants. The origin of VLF oscillations is unclear but may represent complex metabolic processes (renin-angiotensin system activity, thermoregulation) (22,28).

Due to systematic differences in connectivity and neurotransmitter synaptic clearance, changes in sympathetic modulation occur on a slower timescale (seconds) than vagal modulation (milliseconds) (29,30). These physiological constraints are thought to restrict sympathetic activity to the LF HRV range where increases in power would reflect an adaptive transient sympathetic response to physiological or mental stress. Because oscillations driven by vagal activity are not restricted to the HF HRV range and extend into the LF range (17), the relative contributions of sympathetic and parasympathetic mechanisms to LF power in the context of yogic breathing remain to be elucidated.

Understanding the significance of changes in HRV power becomes especially difficult when the breathing rate falls within the LF range, i.e., less than 9 bpm, superimposing the frequency distribution of RSA power onto the LF HRV range (23). Whereas previous work has clearly demonstrated that resting state LF HRV primarily reflects parasympathetic activity (22,27), sudden increases in LF power in response to physiological or mental challenge are still generally associated with sympathetic activation (23,26). This ambiguity limits the interpretation of changes in LF HRV during many common activities such as singing, reading aloud, or quietly talking through one's thoughts during problem solving. These everyday occurrences can push the effective respiratory rate down to frequencies corresponding to the LF HRV range (31–33). In addition, controlled calming breathing techniques used in various mindfulness, meditation, and yoga practices often employ a breathing rate of 4 to 6 bpm with some variants as low as 1 to 2 bpm (18,34). Some highly conditioned athletes have been observed to have spontaneous breathing rates less than 9 bpm as well with correspondingly elevated LF HRV power (35). Given our present state of knowledge, the autonomic basis of such increases in LF power remains unclear, leaving their interpretation with respect to yoga ambiguous. Although it is possible that these breathing techniques or physical conditioning produce increases in vagal control, the current state of the literature also leaves open the possibility that they may paradoxically foster increased sympathetic tone. Additional empirical investigation of the origins of HRV changes is needed to definitively interpret the LF HRV power change under varying physiological and cognitive conditions.

The present study sought to clarify the autonomic effects of slow yoga breathing by elucidating the differential sympathetic and parasympathetic contributions to LF power during slow breathing rates. If, during slow-paced breathing, HRV spectral power in the LF range exclusively reflects sympathetic activity, then parasympathetic blockade should not alter HRV power, whereas sympathetic blockade should significantly reduce LF power. Because evidence of the association between slow yoga breathing and reduced heart rate and blood pressure (36), we hypothesized that increases in LF power due to slow yoga breathing would be vagally mediated.

## MATERIALS AND METHODS

### Participants

This study was performed in six healthy, normotensive, nonobese, adults (2 women and 4 men between 18 and 25 years of age, mean age = 22). Participants were recruited from the general community and did not have any previous expertise in yogic breathing. Participants taking any medications except birth control were excluded. The participants provided written informed consent, and the study protocol was approved by the institutional review board of the University of Arizona. Participants performed paced breathing at differing rates, while receiving muscarinic-cholinergic blockade, β-adrenergic blockade, or placebo on three separate days. All data were collected for a 3-month period and testing times for each participant were between 8:00 AM and 11:00 AM and testing sessions were at least 48 hours apart and took place within an 8-day span.

### Protocol

All testing was completed with participants reclining at 60 degrees with legs elevated. An intravenous line was placed in an antecubital vein upon arrival. ECG, respiration (from a thoracic transducer belt), and noninvasive arterial blood pressure were recorded throughout the session. After 10 to 20 minutes of rest following instrumentation, glycopyrrolate (6-μg/kg bolus followed by 0.05-μg/kg per minute infusion), esmolol (1-mg/kg bolus followed by 0.4-mg/kg per min infusion), or normal saline (10-ml bolus followed by 50-ml/h infusion) was administered for muscarinic-cholinergic blockade, β-adrenergic blockade, or placebo, respectively (37). These pharmacologic agents were chosen for their rapid onset and cessation of effect with limited central nervous system penetration (37–39). The short half-life affords greater experimental control and decreased need for postexperiment monitoring. After 5 minutes of infusion time, participants began the controlled breathing protocol. Participants completed 11 trials consisting of 60 seconds of paced breathing at each rate between 4.0 and 9.0 bpm (in 0.5-bpm increments) with 4 minutes of rest between each trial during which participants breathed normally. Thus, all paced breathing rates fell within the commonly cited LF HRV range of 0.04 to 0.15 Hz (2.4–9.0 bpm). Breathing cadence, based on the SKY ujjayi method (19), was the same for all breathing rates, consisting of four counts of inspiration, four counts of breath holding, six counts of expiration, and two counts of breath holding per respiration cycle. For all trials, breathing was guided by a computer-generated image of a balloon inflating and deflating at the prescribed rate and cadence. Breathing rate orders and drug condition were pseudorandomized and counterbalanced within and across participants.

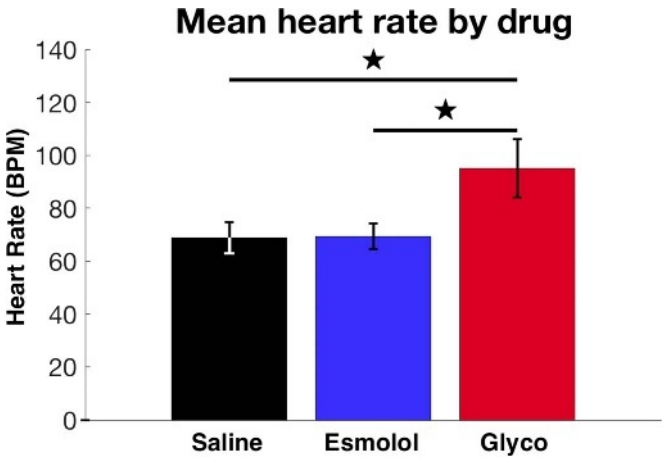


**FIGURE 1.** Mean heart rate across all participants for each drug condition (error bars show the standard error of the mean). Color image is available only in online version (www.psychosomaticmedicine.org).

## Data Collection

Simultaneous recordings of ECG with respiration band (BIOPAC Systems Inc, Goleta, CA) and plethysmographic arterial pressure (Finapres NOVA; Ohmeda, Englewood, CO) were obtained and synchronized. ECG, respirations, and blood pressure were digitized at a sampling frequency of 500 Hz. ECG was processed offline to derive interbeat intervals using automated beat detection software (QRSTool (40)) verified by manual visual inspection. HRV was derived using a version of CMetX (41), implemented in Matlab. Each 1-minute paced breathing event was divided into two 30-second epochs to increase the reliability of analysis. For each epoch, the interbeat interval series was transformed to a time-series sampled at 10 Hz, tapered with a Hamming Window, and a 2000-point (200-second) fast Fourier transform (FFT) was used to extract spectral power with 0.005-Hz resolution. Respiratory waveform peak and trough timings were extracted using a custom Matlab script in conjunction with visual inspection to confirm participants' adherence to the paced breathing patterns.

## Statistical Analysis

Variability across all HRV frequencies and at specific bands corresponding to breathing frequencies was extracted and natural log transformed to satisfy normality assumptions for statistical analysis. A repeated-measures general linear model was performed (SPSS Version 23) to assess the main effect of drug (saline, esmolol, glycopyrrolate), breathing rate (11 rates from 4 bpm to 9 bpm in 0.5-bpm increments) and their interaction. In each case, the dependent variable was the spectral power at the breathing frequency. As manipulation checks, the impact of drug on heart rate was compared in a drug by breathing rate repeated-measures general linear model, and the fidelity of breathing rate was assessed by correlating the intended breathing rate to the frequency of maximum power in the spectral series from the FFT.

# RESULTS

Autonomic Blockade – Validity Checks as expected, there was a main effect of drug ($F(2,10) = 38.9$, $p < .001$) (Fig. 1) and pairwise comparison revealed that at rest sympathetic blockade (esmolol) did not lower heart rate relative to placebo ($p = .81$), whereas parasympathetic blockade (glycopyrrolate) significantly elevated heart rate relative to placebo and esmolol ($p < .001$). Fidelity to the paced breathing rate was verified by correlating the intended breathing rate to the frequency of the maximum power from a FFT of the measured breathing rate ($r = .94$, $p < .001$).

## HRV Power Across Low-Frequency Breathing Rates

Across all breathing rates, a main effect of autonomic blockade condition was observed (Wilks' $\lambda = .181$, approximate $F(2,4) = 9.03$, $p < .033$) (Fig. 2). Parasympathetic blockade by glycopyrrolate greatly reduced HRV at every breathing rate ($p = .016$ versus

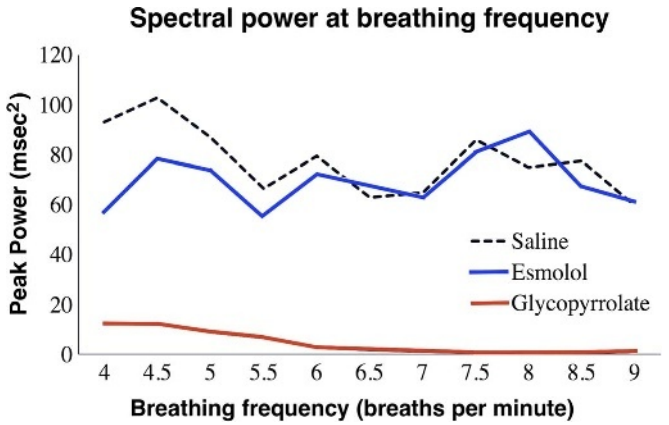


**FIGURE 2.** Mean peak HRV power at each of the 11 breathing rates for each drug condition. Color image is available only in online version (www.psychosomaticmedicine.org).

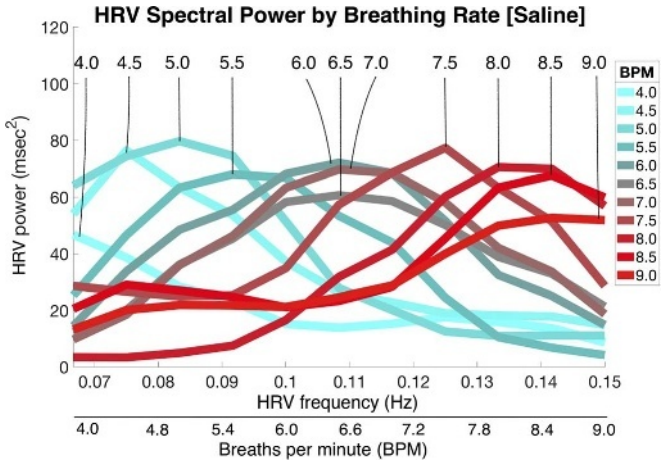


**FIGURE 3.** HRV power spectrum for each breathing rate for the placebo condition with each curve labeled with its breathing rate. Note that peak HRV spectral power in each case corresponds to the breathing rate frequency in breaths per minute and has been labeled as such for clarity. Color image is available only in online version (www.psychosomaticmedicine.org).

saline) (Fig. 2). Esmolol had an indistinguishable effect on HRV compared with saline ($p$ = .671 versus saline) when measured across all breathing rates (Fig. 2). There was no main effect of breathing rate or a rate by drug interaction effect. An exploratory pairwise comparison revealed a slight decline with esmolol relative to saline for the slowest breathing rate (at 4.0 bpm, $p$ = .004) (Fig. 2). The visual trend of HRV away from zero at the slowest breathing rates in the glycopyrrolate condition was not statistically significant, although the power to detect this small difference was insufficient given the small sample size.

The full range of spectral power is presented in Figures 3 to 5. For the placebo condition (Fig. 3), HRV spectral power for each breathing rate is maximal at the frequency of that breathing rate. This pattern is preserved during sympathetic blockade (Fig. 4) and virtually eliminated with parasympathetic blockade (Fig. 5).

## DISCUSSION

The results of this study reveal that for participants at rest, respiration-driven changes in HRV power (RSA) are vagally mediated. This is true for not only the higher-frequency bands that are thought to reflect exclusively parasympathetic influence, as shown in previous studies, but also when the respiratory rate is in the lower-frequency range that can reflect both sympathetic and parasympathetic influences. For only the very lowest rates near the VLF range (<0.08 Hz, <5 bpm), a small but significant influence of the sympathetic branch on HRV was observed, indexed by a small decrease in HRV power at these breathing rates when sympathetic influence was blocked by esmolol, and also indexed to a lesser extent by a small amount of power in these frequency bands under parasympathetic blockade by glycopyrrolate. Although sympathetic activation may drive changes in LF power

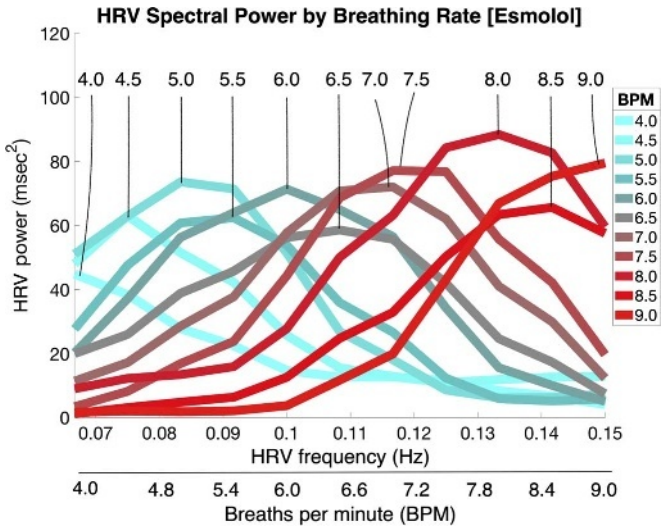


**FIGURE 4.** HRV power spectrum for each breathing rate for the sympathetic blockade condition with each curve labeled with its breathing rate. Note that peak HRV spectral power in each case corresponds to the breathing rate frequency and has been labeled as such for clarity. Color image is available only in online version (www.psychosomaticmedicine.org).

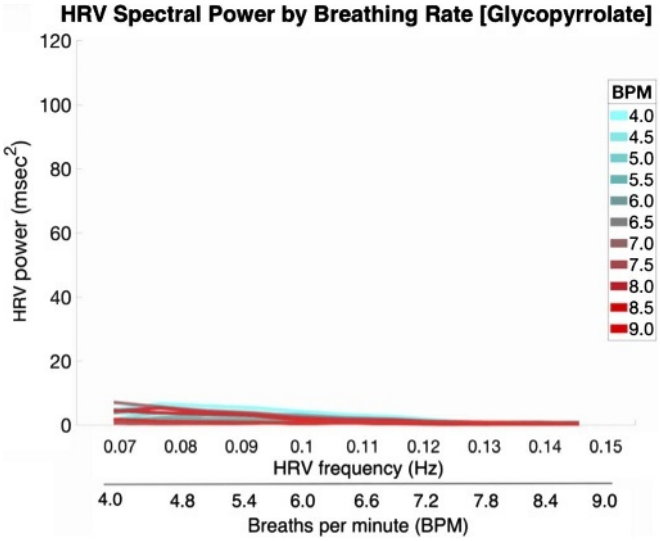


**FIGURE 5.** HRV power spectrum for each breathing rate for the parasympathetic blockade condition. Color image is available only in online version (www.psychosomaticmedicine.org).

under physiologic stress challenge, the results of the present study reveal that this interpretation does not apply to resting individuals performing slow-paced breathing, where the effect on LF power is almost exclusively parasympathetically driven. This finding supports a vagal interpretation for changes in RSA observed during the practice of yogic breathing or other mind-body disciplines that use slow calming breathing.

The SKY ujjayi technique uses a fixed ratio of 4-4-6-2 (4 counts inspiration, 4 counts postinspiratory hold, 6 counts expiration, and 2 counts postexpiratory hold), maintaining a postinspiratory (10 counts of hold plus expiration) to postexpiratory (6 counts of hold plus inspiration) ratio of 5:3 (15). Vagal activity is enhanced during the postinspiratory period and inhibited during the postexpiratory period (42). Although increases in RSA during slow yogic breathing seem to reflect increases in cardiac vagal control, the relative effectiveness of different postinspiratory to postexpiratory ratios was not examined in this study design. Further work is also needed to compare changes in HRV power between paced yogic breathing and sinusoidal breathing patterns. Identification of the optimal rate and breathing cadence to maximize RSA is an important next step in designing a personalized breathing intervention that can maximize vagal control for any given individual. In addition, further studies are needed to better understand the relationship between short- and long-term changes brought about by breathing interventions.

Resonance frequency biofeedback has been previously used to maximize HRV through controlled breathing, although the autonomic interpretation of the large increases in RSA power in the LF HRV range seen with low breathing rates has been uncertain (16,43). Our findings suggest that RSA increases seen with this technique represent bolstered vagal activity even when a participant's respiratory rate drops into the low-frequency range. Song and Lehrer (43) found that peak HRV power was strongly rate dependent with an optimal paced breathing rate of 4 bpm. We did not observe this strong rate dependency, but it is important to note that we incorporated a yogic breathing cadence with a postinspiratory to postexpiratory ratio of 5:3, whereas their study used a 1:1 ratio. Bertisch et al. (19) have also reported that RSA is primarily rate dependent, suggesting that the large and significant increases in RSA noted in yoga practitioners is simply due to their low baseline respiratory rate. Unfortunately, their report did not measure or control for differences in breathing pattern, making it difficult to draw inferences about yogic breathing. The observation from our study that RSA remains relatively steady across many breathing rates when a yogic-derived pattern is used suggests that varying inspiratory and expiratory ratios away from a symmetric sinusoidal breathing pattern may further optimize RSA over frequency changes alone. Yoga and other mind-body practices (meditation, tai-chi) incorporate many other distinct components in addition to using a controlled breathing cadence (19). Vocalizations, diaphragmatic breathing, and resistive breathing techniques (tracheal, nasal, or oral) are common to breathing practices. These additional components may further accentuate the enhancement of vagal activity brought about by increasing the postinspiratory to postexpiratory ratio. Indeed, tracheal stimulation has been shown to further increase vagal outflow during the postinspiratory period in animal studies (42). Traditional bodily movements and postures are perhaps the most visible element of yogic practice and may offer distinct health benefits as well. In addition to physical conditioning and strengthening, these postural changes provide a challenge to the autonomic control of blood volume distribution through changes in peripheral vascular tone and cardiac output. Finally, the mindfulness component of yogic practice may further affect brain areas involved in regulation of cardiac vagal control, increasing top-down cognitive control over impulses generated by fight-or-flight stress responses (44).

## Limitations and Future Directions

Although this study has a relatively small sample size, the pharmacological manipulations produced large effects, and given the invasive procedure, it was prudent to use a small sample for this demonstration. Future work with larger samples might examine the extent to which the conclusion that yogic-breathing effects are vagally mediated generalizes across individual differences in

age, sex, racial and ethnic status, socioeconomic demographics, and stress-related psychopathology such as anxiety disorders or depression. In addition, this study was not designed to assess the magnitude of RSA change in relation to a baseline immediately before each breathing trial or whether RSA enhancement during paced breathing produced clinically relevant and enduring changes in RSA or associated variables of interests such as mood and perceived stress.

The results of this study help improve the interpretability of HRV changes brought about by interventions that employ low-frequency paced breathing to improve health. An important question to be addressed in future research is how to vary the rate and cadence to optimize the vagal effects of paced breathing for each individual. Portable, noninvasive, and widely accessible technology with the ability to capture ECG signals continues to advance in conjunction with improvements in processing power, battery size, and societal attitudes toward technology. ECG signals can be collected via direct skin contact by a host of wearable bands and watches (45). Continued improvements in ambulatory monitoring technology increase the scope of possibility for real-time, personalized, breathing-based interventions to maximize their effects on vagal control. An improved ability to reliably capture HRV changes combined with a clearer understanding of what these changes indicate broadens the scope of possibility for monitoring and diagnosing disease processes related to autonomic imbalance and creating noninvasive interventions tailored to the health needs of an individual.

In conclusion, when assessing autonomic influence in healthy volunteers at rest, HRV changes due to breathing rates at 6 bpm and greater can be inferred to exclusively reflect changes in parasympathetic control, and those between 4 and 6 bpm can be inferred to reflect predominantly parasympathetic control. These findings raise the possibility that mind-body techniques that use slow breathing at rest exert their breathing-related relaxation effects through vagal mechanisms.

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