Applied Psychophysiology and Biofeedback, Vol. 31, No. 2, June 2006 (© 2006)
DOI: 10.1007/s10484-006-9009-3

Characteristics of Resonance in Heart Rate Variability Stimulated by Biofeedback

Evgeny G. Vaschillo,[1,3] Bronya Vaschillo,[1] and Paul M. Lehrer[2]

Published online: 13 July 2006

As we previously reported, resonant frequency heart rate variability biofeedback increases baroreflex gain and peak expiratory flow in healthy individuals and has positive effects in treatment of asthma patients. Biofeedback readily produces large oscillations in heart rate, blood pressure, vascular tone, and pulse amplitude via paced breathing at the specific natural resonant frequency of the cardiovascular system for each individual. This paper describes how resonance properties of the cardiovascular system mediate the effects of heart rate variability biofeedback. There is evidence that resonant oscillations can train autonomic reflexes to provide therapeutic effect. The paper is based on studies described in previous papers. Here, we discuss the origin of the resonance phenomenon, describe our procedure for determining an individual's resonant frequency, and report data from 32 adult asthma patients and 24 healthy adult subjects, showing a negative relationship between resonant frequency and height, and a lower resonant frequency in men than women, but no relationship between resonant frequency and age, weight, or presence of asthma. Resonant frequency remains constant across 10 sessions of biofeedback training. It appears to be related to blood volume.

KEY WORDS: resonance; closed loop system; biofeedback; heart rate variability; baroreflex.

It is a common observation among biofeedback clinicians that heart rate variability biofeedback (HRV BFB) produces large increases in heart rate variability in almost everyone, usually within a few minutes of the beginning of training. Our previous papers (Vaschillo, 1984; Vaschillo, Lehrer, Rishe, & Konstantinov, 2002) and papers of other authors (DeBoer, Karemaker, & Strackee, 1987; Saul et al., 1991; Sleight et al., 1995; Ursino & Magosso, 2003) have shown that maximal increases in amplitude of heart rate oscillation are produced when the cardiovascular system (CVS) is rhythmically stimulated by paced breathing at a frequency of about 0.1 Hz (6 breaths per minute). These papers assume that this effect is linked to resonance properties of the cardiovascular system (CVS) resulting from activity of the heart rate (HR) baroreflex.

[1]Rutgers University, Piscataway, NJ.
[2]UMDNJ—Robert Wood Johnson Medical School, Piscataway, NJ.
[3]Address all correspondence to Evgeny G. Vaschillo, Center of Alcohol Studies, The State University of New Jersey, Rutgers, 607 Allison Road, Piscataway, NJ 08854; e-mail: Evaschil@rci.rutgers.edu.

1090-0586/06/0600-0129/1 © 2006 Springer Science+Business Media, Inc.

Resonance in Cardiovascular System

The baroreflex as any other autonomic reflex can be modeled as a "closed loop" system, i.e., control system with feedback (Ringwood & Malpas, 2001; Hammer & Saul, 2005). The baroreflex system is part of a more complex system controlling blood pressure (BP). It plays an important role in protecting the body from an acute blood pressure shifts. Due to baroreflex, increases in BP trigger HR decreases, while decreases in BP trigger HR increases. Conversely, decreases in HR produce decreases in BP through the mechanical effects of decreased blood flow, while increases in HR produce increases in BP.

The baroreflex closed loop system includes the combined effects of mechanical feed-forward from HR to BP through changes in cardiac output and feedback from BP to HR via baroreceptors (Saul et al., 1991). Arterial baroreceptors react to BP changes and trigger reflexes that increase or decrease HR to compensate BP shifts. Both HR and BP vary in the closed-loop, so that a change in either function causes a change in the other. However, HR reactions to BP shifts, as well as BP reactions to HR shifts are not instantaneous. The delay in these reactions can be caused by slow sympathetic control of baroreflex function (DeBoer et al., 1987), as well as by inertia caused by various physical parameters contributing to heart cycle and blood distribution. These parameters include the volume of circulated blood, blood viscosity, length and volume of the vasculature, etc., as well as speed of sympathetic and parasympathetic reactions, and neural transmission.

A technical control system with feedback is very stable if there is no delay between processes in a closed-loop, but it becomes an oscillatory system when the closed-loop contains a delay (Grodins, 1963; Ringwood & Malpas, 2001). Such a system exhibits the property of resonance at a particular frequency. The value of the delay defines the frequency of the resonant oscillation in the closed-loop. If the delay is $D[s]$, the frequency of resonant oscillation is $1/(2D)$ [Hz], i.e., a 5-second delay provides resonance in the closed-loop at a frequency of $1/10 = 0.1$ Hz. The baroreflex system, as closed-loop control system with feedback, manifests resonant properties because a delay between related physiological functions is always present (Halamek et al., 2003; Hammer & Saul, 2005).

We have assessed the baroreflex closed loop system using a common engineering approach (Vaschillo et al., 2002). Sine-wave oscillations in HR and BP were elicited by the HR biofeedback procedure (Lehrer, Vaschillo, & Vaschillo, 2000). The closed loop system was tested separately at each of 7 frequencies in the range of 0.01–0.15 Hz. Transfer functions between HR and BP were calculated as the ratio of amplitudes of HR and BP oscillations and phase shifts between them at each testing frequency. A phase shift of 180° was found at a frequency near 0.1 Hz for each individual, corresponding to a delay of approximately 5 s between oscillations in HR and BP. The length of the delay differed across individuals and lay in the range of 4–6.5 s for all subjects; i.e. the resonant frequency of their CVS lay in the range of 0.075–0.12 Hz.

The frequency at which the 180° phase shift occurred corresponded to the frequency at which the amplitude of HR oscillations was highest, indicating that baroreflex action contributed to the high amplitude of HR oscillations at this frequency. Thus, as in all systems with resonance characteristics, resonance characteristics of the CVS were shown to produce high-amplitude HR oscillations in response to rhythmical stimulation at the resonant frequency.

Resonant Frequency HRV Biofeedback

The resonant frequency HRV biofeedback (RF HRV BFB) procedure we devised (Lehrer et al., 2000) is based on resonance properties of the CVS. To elicit high-amplitude oscillation in autonomic functions, the procedure uses paced breathing at each individual's resonant frequency. We have found that, among healthy people, the RF HRV BFB procedure acutely increases both heart rate variability and baroreflex gain, and produces *chronic* increases in baroreflex gain and peak air flow during a forced expiratory maneuver (Lehrer et al., 2003). In a randomized controlled trial on 95 subjects, we also found decreases in airway resistance in patients with asthma, along with a decrease in asthma symptoms, a reduction in the frequency of asthma exacerbations, and a reduced need for asthma medication (Lehrer et al., 2004).

Additionally in small randomized controlled trials, Giardino, Glenny, Borson, and Chan (2003) found that the procedure improved symptoms and efficiency of pulmonary gas exchange in COPD patients; Radvanski et al., 2004 and Hasset, Radvanski, and Lehrer, (2006) found that it improved symptoms of fibromyalgia; Karavidas, 2005; Karavidas and Lehrer, 2006 found that it reduced symptoms of major depressive disorder.

We have theorized (Lehrer et al., 2003) that RF HRV BFB produces therapeutic effects because it elicits high-amplitude oscillations in cardiovascular functions, and thereby stimulates and exercises autonomic reflexes, particularly the baroreflexes, in the same way that muscular exercise trains reflexes of the somatic nervous system, and renders them more efficient. By improving the modulatory function of reflexes that control and are controlled by the sympathetic and parasympathetic systems, the balance between the two systems becomes more tightly regulated, and, where autonomic dysfunction exists, the balance is restored.

Although, theoretically, *any* rhythmical stimulation of the cardiovascular or autonomic nervous systems should induce HR oscillations, such stimulation can most easily be achieved by breathing at that frequency. Respiration is independently associated with HR oscillations, inhaling with HR increases and exhaling with HR decreases. This is known as phenomenon of respiratory sinus arrhythmia (RSA), and is controlled by various parasympathetic reflexes responsive to changes in blood chemistry and intrathoracic pressure, as well as to some endogenous CNS rhythms (Porges, 1995). However, HR oscillations do not normally occur in phase with respiration, with HR rising *simultaneously* with inhalation and decreasing *simultaneously* with exhalation. Such phase synchrony occurs only when the individual breathes at the resonant frequency of the CVS at about 0.1 Hz (Vaschillo, Vaschillo, & Lehrer, 2005) and causes high-amplitude HR oscillation.

As we show below, finding a person's resonant frequency and teaching the person to breathe at that frequency (about 6 breaths per min) requires considerable care. Normally people breathe at a rate of 12–20 breaths per min. Deliberate slow paced breathing can cause an increase in tidal volume that more than compensates for the decrease in respiration rate. It thus often produces hyperventilation and temporarily disordered cardiovascular regulation (Lehrer et al., 1997). To be beneficial, the RF HRV BFB procedure must train people not to breathe too deeply, while they breathe slowly, at the resonant frequency.

Thus, two features of resonance in the CVS (Vaschillo, Vaschillo, & Lehrer, 2004) can be used to determine an individual's resonant frequency:

1. Paced breathing at the resonant frequency elicits the highest possible amplitude of HR oscillation.
2. Respiration and HR oscillations occur in phase (i.e. HR rises simultaneously with inhalation and decreases simultaneously with exhalation) *only when the subject breathes at the resonant frequency*. HR oscillation precedes respiration (a positive phase) if the individual breathes more slowly than resonant frequency, and HR oscillation lags behind respiration (a negative phase) if the individual breathes more frequently than at resonant frequency.

The main goals of this paper are:

1. To describe a method for determining each individual's resonant frequency;
2. To discuss difficulties in determining the resonant frequency and show methods for overcoming them;
3. To show some features of resonance in the CVS, from our research studies.

For this paper we used data from two previously published studies (Lehrer et al., 2003; Lehrer et al., 2004).

METHOD

Participants and Instrumentation

Participants in this study were 24 healthy adults (16 females) (Lehrer et al., 2003) and 32 adult asthma patients (21 females) (Lehrer et al., 2004), ages 18–65. Each person received 10 sessions of HRV biofeedback during 2.5–3.5 months, one session per week. We used a J&J Engineering (Poulsbo, WA) I-330 DSP-12 physiograph to collect electrocardiogram (ECG) and respiration data, to provide HRV biofeedback, and to present a respiratory pacing stimulus to participant. ECG data was collected from sensors on right arm and left leg (Lead II), digitized at the rate of 512 samples per second. Beat-to-beat RR intervals (RRI) of the ECG signal were measured. A respiration strain gauge belt was attached around the participant's chest. An on-line display of beat-to-beat heart rate curve, a respiratory curve, and a moving Fourier analysis of heart rate provided HRV biofeedback on the computer screen. For a respiratory pacing stimulus, a bar on the computer screen moved up for inhalation and down for exhalation. To assess hyperventilation, end-tidal CO_2 was taken with a Datex (Boulder, CO) 223 capnometer.

Procedure

In the first session, participants breathed following the respiratory pacer for 2 min at each of 5 frequencies: (6.5, 6, 5.5, 5, 4.5 breaths/min or 0.108, 0.1, 0.092, 0.083, 0.075 Hz). We asked participants to breathe in natural way, not too deeply. We again reminded participants not to breathe so deeply if end-tidal CO_2 fell below 3.5%. The frequency spectrum of RRI was computed for each 2-minute record. Thus, five spectra were computed for each participant, one for each respiratory frequency. The power of

the spectral component at the respiratory frequency (respiratory component of the RRI spectrum) was measured for each of five spectra. We estimated the individual's resonant frequency as the respiratory frequency that produced the highest respiratory component in the RRI spectra.

During the following nine sessions, we adjusted the estimate of the resonant frequency based on spectral characteristics of HRV during the session. In each of these sessions, the respiratory pacer was set at the frequency found in the previous session. Each session included four five-minute training periods. In each session, we assessed end-tidal CO_2 to ensure that the study participant was not hyperventilating. If end-tidal CO_2 fell below 3.5%, we reminded the participant not to breathe so deeply.

In first of three sessions, participants were instructed to breathe following the pacer while trying to maximize HRV amplitude. For each five-minute period we evaluated the phase shift between the HR and respiration curves visually on the computer screen. If the two curves were not in phase, we reset the pacer's frequency up or down by 0.5 breaths per minute in the next training period. The pacer's frequency was increased by one step if HR oscillated ahead of respiration, and was decreased by one step if HR lagged behind respiration. The pacer's frequency was not changed if the phase between HR and respiration was close to zero degrees. At the end of each session we recorded the individual's resonant frequency as the last value of the pacer's frequency.

In sessions 4–10, we turned off the pacing stimulus after the first five minutes and instructed participants to breathe at the rate that maximized peak-to-trough amplitude in the HR curve tracing on the computer screen, and produced a zero-degree phase relationship between HR and respiration. We adjusted the estimate of the resonant frequency from the participant's performance during this period.

RESULTS

Figure 1 shows that paced breathing caused high-amplitude RRI oscillations at the respiratory frequency. RRI spectra and resonance characteristics of the participants' CVS (Figs. 2 and 3) show that: 1) When the participant breathed at or close to resonant frequency, the respiratory component was the most prominent one in the RRI spectrum; 2) RRI oscillation amplitude was very sensitive to changes in respiratory frequency; 3) The power of the respiratory component could change by a factor of more than 2, for respiratory rate changes of only 0.5 times/min; and 4) Peak amplitude HR oscillations occurred at different respiratory frequencies across individuals. The resonant frequency of participants' CVS ranged between 4.5–6.5 times/min.

Results of end-tidal carbon dioxide monitoring showed that in the first three sessions, end-tidal CO_2 frequently fell below 3.5%. This tendency gradually decreased, and disappeared by the fourth session. Data taken during biofeedback training procedures after the third session showed no changes in end-tidal carbon dioxide in comparison with data in rest conditions.

The resonant frequency determination and adjustment in all ten biofeedback sessions are illustrated in Table I. As can be seen, the resonant frequency of participants almost never varied by more than 0.5 breaths/min across 10 training sessions, nor did the mean vary across all participants, according to a one-way repeated-measures analysis of variance, $F(9,486) = 1.32$, $p = n.s.$

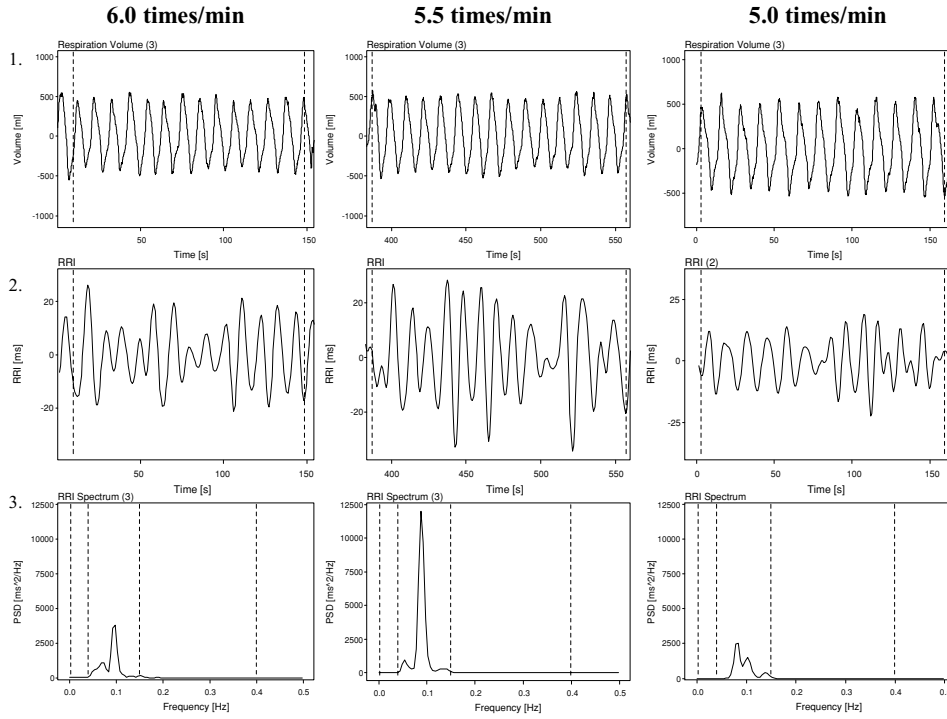

Fig. 1. Heart rate reactions to paced breathing at the resonant frequency of 5.5 times/min and at frequencies close to this frequency (recorded from one participant). 1. Current Respiration Volume. 2. Current RR interval of the ECG (RRI). 3. RRI frequency spectra. Breathing at the heart rate resonant frequency elicited high amplitude RRI oscillations at that frequency. Small shifts in breathing frequency from resonant significantly changed RRI oscillation amplitude.

The average resonant frequency across 10 sessions was calculated for each participant. The resonant frequencies had a nearly normal distribution across individuals (see Fig. 4). In 20 of the 56 participants (35.7%) resonant frequency was estimated accurately in the first session. Our assessment of resonant frequency did not change in the following nine sessions. One participant (1.8%) had a resonant frequency of 6.5/min, six (10.7%) of 6.0/min, seven (12.5%)—of 5.5/min, five (8.9%) of 5.0/min, and one (1.8%)—of 4.5/min.

The average of resonant frequency across all participants ($M \pm s.d.$) was 5.56 ± 0.41/min or 0.0926 ± 0.007 Hz. As can be seen in Tables II and III and at Fig. 5 we found a significantly lower resonant frequency among taller participants than shorter ones. Resonant frequency was lower in men than in women but the gender effect was not significant when controlled for height by covariance adjustment. We found no significant differences in resonant frequency between asthmatic and healthy participants or older vs. younger than 40 years old, and no correlation with age. Resonant frequency was not related to weight. A negative correlation between resonant frequency and height was large and significant after session 3, but was not significant for the first 3 sessions (Table IV).

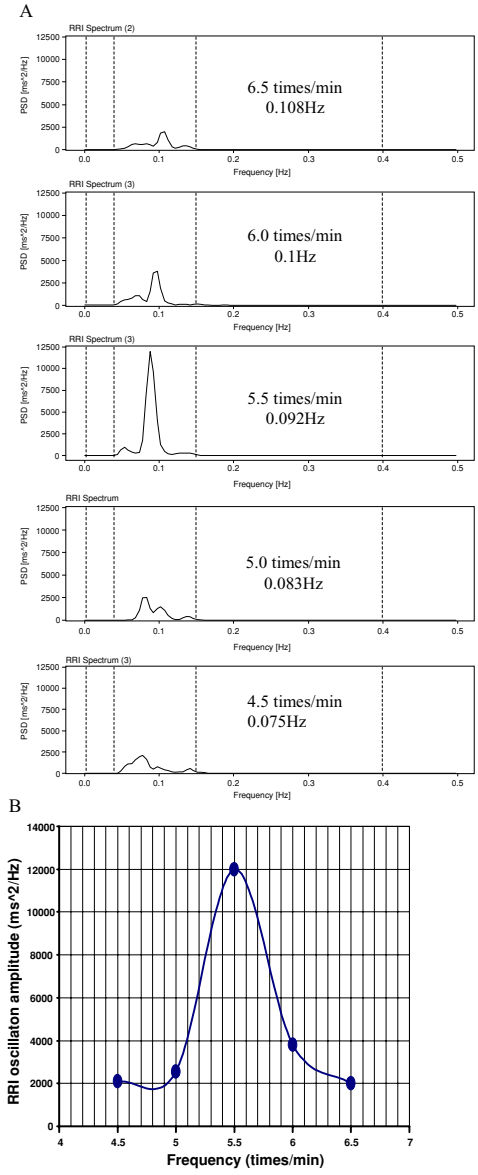

Fig. 2. Determination of heart rate resonant frequency of the participant's cardiovascular system. A. RRI frequency spectra for paced breathing at frequencies 6.5, 6.0, 5.5, 5.0, and 4.5 times/min. B. Resonance characteristic of the participant's cardiovascular system. The resonant frequency of this participant is 5.5 times/min or 0.092 Hz.

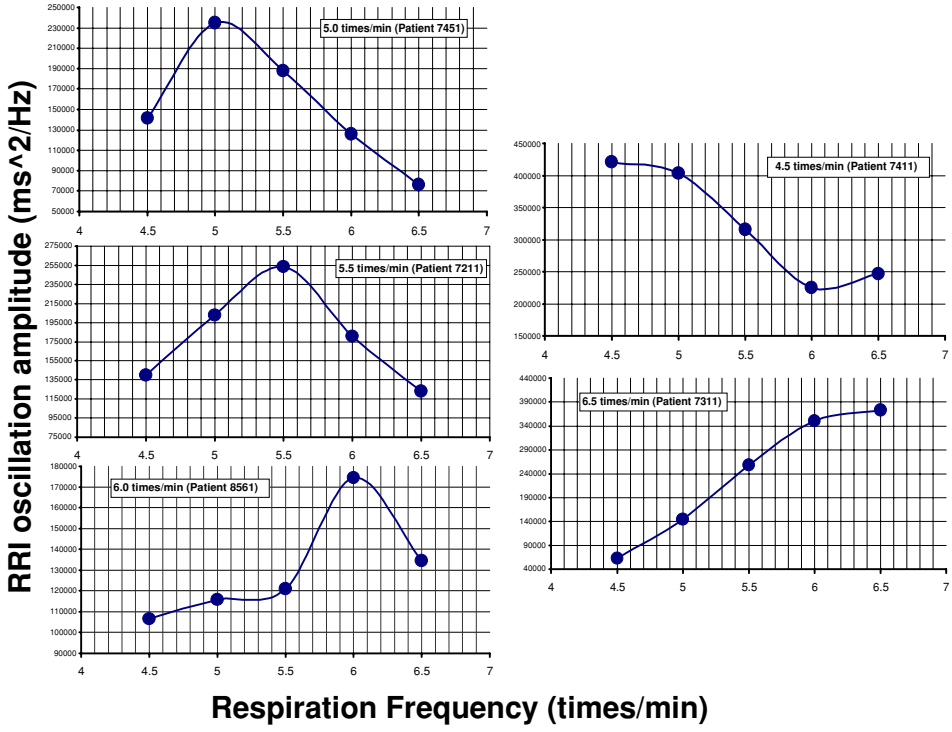

Fig. 3. Resonance characteristics of cardiovascular system of 5 participants with different resonant frequencies.

DISCUSSION

Variability in cardiovascular functions reflects adaptive regulation of the CVS (Giardino, Lehrer, & Feldman, 2000). We theorize that resonant properties underlie CVS function variability (Vaschillo, Zingerman, Konstantinov, & Menitsky, 1983; Vaschillo et al., 2002). Rhythmical physical loading (Wigertz, 1971; Tiedt, Wohlgemuth, & Wohlgemuth, 1975), gravitation tilting (Hamilton, Lindan, & Reswick, 1969), rhythmical thermal stimulation (Lindqvist, 1990), rhythmical presentation of emotional pictures (Vaschillo et al., 2005), and paced respiration all elicit high-amplitude oscillations in cardiovascular functions at resonant frequencies. HR resonance in the CVS at a frequency of $\sim$0.1 Hz has been discussed for more than 40 years. Angelone and Coulter (1964) argued that high-amplitude HR oscillation, when subjects breathed at the rate of 5–6 times/min, is a strong evidence for resonance in the CVS. Vaschillo et al. (1983) and DeBoer et al. (1987) theorized that control processes in the closed loop of the HR baroreflex provide a resonant property in CVS at $\sim$0.1 Hz.

Although rhythmical slow paced breathing can trigger HR resonance, the resonant property of the CVS is determined by its structural and physical properties, particularly by the baroreflex and other physical characteristics of the circulatory system (easily quantifiable as blood volume), but not by respiration (Vaschillo et al., 2005). The respiration can cause HR and BP oscillations due to the phenomenon of RSA. The HR baroreflex

Table I. Resonant Frequency for Each Session Among the First 10 Healthy Participants Admitted to the Study, in Cycles (Breaths/Minute)

ID number	Sess 1	Sess 2	Sess 3	Sess 4	Sess 5	Sess 6	Sess 7	Sess 8	Sess 9	Sess 10
717	6	6	6	6	5.5	5.5	6	5.5	6	6
718	5.5	5.5	5.5	6	6	5.5	6	6	6	6
721	6	5.5	5.5	5.5	5.5	5.5	5.5	5.5	6	6
724	5.5	5.5	5.5	5	5	5	5.5	5.5	5.5	5.5
725	6	6	6	6	6	6	6	6	5.5	5.5
726	5.5	5.5	5.5	5.5	5.5	6	5.5	5.5	6.5	6
728	5	5	5	5	5	5	5.5	5.5	5.5	5.5
731	6	6	6.5	6.5	6.5	6.5	6	6	6.5	6.5
745	5.5	5.5	5.5	5.5	5.5	5.5	5.5	5.5	5.5	5.5
755	4.5	5	5	5	5	5	5	5	4.5	4.5

closed-loop amplifies these oscillations if their frequency matches the resonant frequency. The closed-loop links HR and BP oscillations. Each heart beat output elicits acute BP increase, due to increase in the quantity of blood circulating during each pulse. More blood circulates at higher HR, causing an increase in BP, which in turn decreases HR through baroreflex. However, inertia and plasticity of the vasculature produce a delay in these effects, which accounts for 0.1 Hz resonance in the CVS. Delay shifts phase between HR and BP oscillations. The resonance occurs if phase shift is 180°. This delay can depend on the volume of circulated blood, blood viscosity, length and volume of the vasculature, etc., as well as speed of sympathetic and parasympathetic reactions, and neural transmission.

These relationships are consistent with our finding that the resonant frequency negatively and high significantly correlates with height, is related to gender, and does not

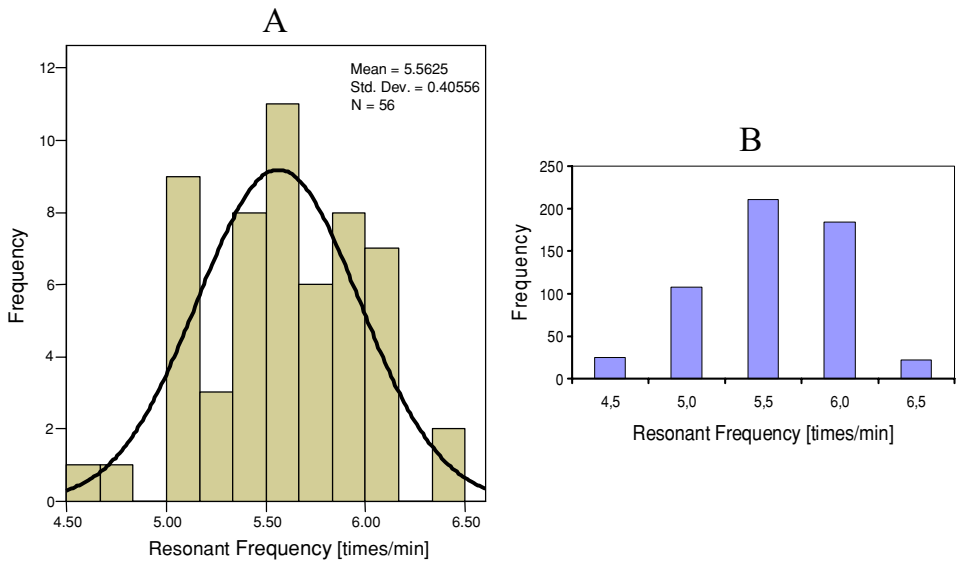

Fig. 4. Distribution histograms of the resonant frequencies among 56 participants. A. Distribution of the resonant frequencies averaged across 10 sessions for each participant. B. Distribution of the resonant frequencies determined in each from 10 training sessions for each participant.

Table II. Averaged Resonant Frequency Across all Participants, by Diagnosis, Gender, and Age, in Cycles (Breaths/Minute)

Groups	*N*	Means (Hz)	Std. dev. (Hz)	T-test
Total	56	5.56 (0.0926)	0.41 (0.007)	
Females	37	5.66 (0.0943)	0.43 (0.0072)	$t = 4.07$; $p < 0.0001$
Males	19	5.21 (0.0868)	0.38 (0.0063)	
Asthma	32	5.55 (0.0925)	0.39 (0.0065)	$t = 0.35$; $p = 0.73$
Healthy	24	5.50 (0.0916)	0.57 (0.0096)	
Age 40 and less	35	5.54 (0.0923)	0.44 (0.0073)	$t = 0.45$; $p = 0.61$
Age more then 40	21	5.52 (0.0920)	0.50 (0.0083)	

correlate with weights and ages. The lower resonant frequency in taller people is consistent with the greater volume of vasculature and mass of circulated blood in taller individuals. With greater mass, inertia in the CVS is greater, so the delay in the baroreflex system is longer and the resonant frequency is lower. Lack of correlation between resonant frequency and weight may reflect a relative lack of blood supply in adipose tissue. The lower resonant frequency among men than in women is consistent with greater blood volume in men (Mier, Domenick, Turner, & Wilmore, 1996; Shevde et al., 2002). The lack of correlation with age is not consistent with findings of age-related decreases in blood volume (Ito et al., 2001; London, Guerin, Laurent, London, & Safar, 1985). Perhaps the stability of resonant frequency with age is due to other aging factors that compensate for decrease in blood volume: e.g., increase of arterial stiffness with age and less efficient neural regulation.

Our attempt to find each subject's resonant frequency in the first few sessions was slightly inaccurate, probably because paced breathing in the low frequency range was not familiar to participants. Participants often breathed too deeply, causing hyperventilation, and sometimes anxiety. This, in turn, could have decreased HRV amplitude by depressing parasympathetic activity, causing errors in our comparing HRV amplitude at one respiration rate with amplitude at other rates. We apparently determined participants' resonant frequencies more accurately using biofeedback in later sessions than was possible using paced respiration in the first three sessions. Perhaps unfamiliarity with the procedure in the first three sessions also may have added error variance, from emotionally induced distortions in respiration and heart rate variability. The cumulative inaccuracies may account for the lack of correlation between resonant frequency and height in the first three sessions, despite high correlations in subsequent sessions. Small session-to-session variations to individual resonant frequency probably also reflected measurement error.

Our data confirm that the CVS has the property of resonance at a frequency near 0.1 Hz, and that this frequency is specific to each individual. Due to this property, HRV biofeedback

Table III. Correlations of Age, Weight, and Height of Participants ($n = 56$) with their Resonant Frequencies Averaged Across 10 Sessions

Variable	*r*	*p*
Height	−0.55	< 0.0001
Weight	0.02	*n.s*
Age	0.01	*n.s*

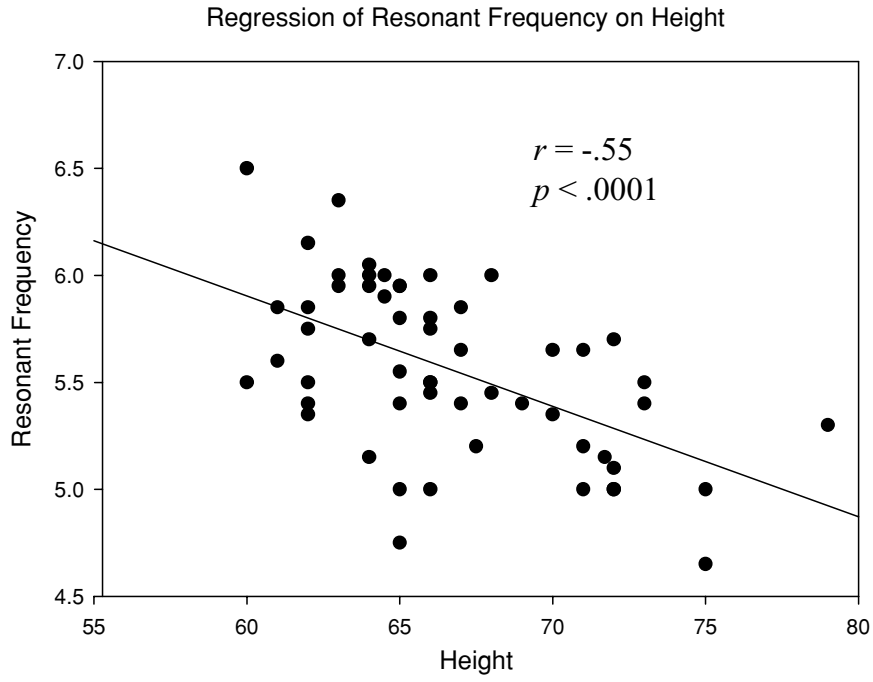

Fig. 5. Correlation between resonant frequency value and height of the participants.

elicits high-amplitude oscillations in CVS functions. Lehrer et al. (2003) have theorized that these oscillations train reflexes that modulate autonomic reactivity, thus providing the therapeutic effects of the HRV biofeedback. Figures 2 and 3 show that amplitude of HR oscillation is sometimes very sensitive to small changes in breathing rate around 0.1 Hz. Therefore we hypothesize that HRV biofeedback should produce the strongest salutary clinical effects if the biofeedback procedure leads the individual to breathe at his or her exact resonant frequency.

Table IV. Correlation of Height of Participants ($n = 55$) with their Resonant Frequencies Determined in each Training Session

Session no	r	p
1	−0.26	*n.s*
2	−0.24	*n.s*
3	−0.30	*n.s*
4	−0.61	<0.0005
5	−0.63	<0.0001
6	−0.57	<0.001
7	−0.50	<0.005
8	−0.61	<0.0005
9	−0.48	<0.006
10	−0.59	<0.0005

Additionally our data showed that 1) at least three training sessions are necessary to teach a person to breathe at resonant frequency without hyperventilation and, as a corollary, 2) if paced breathing is used to determine resonant frequency, the accuracy of estimation cannot be provided in the first training session. Training in slow breathing is necessary before this can be done.

However, as a caveat, we point out that it has not yet been proven whether precise measurement of resonant frequency is *essential* for the various salutary clinical affects of HRV biofeedback. Indeed, several papers (e.g., Herbs, Gevirtz, & Jacobs, 1993; McCraty, Atkinson, & Tomasino, 2003) have found strong therapeutic effects even when HRV biofeedback was performed without specifically determining the personal resonant frequency. It is possible that, despite the findings described in this paper, even standardized paced breathing at 5.5 or 6 times/minute for *all* people may produce clinical effects that are just as strong, without the need for biofeedback equipment, or that a estimate of resonant frequency in a single session is sufficient, followed by assignments to breathe at that frequency at home, without biofeedback instrumentation. This must be determined in future research.

ACKNOWLEDGMENTS

This work was supported by Grant #R01 HL58805 from the Heart, Lung and Blood Institute, National Institutes of Health and by Grant # R01 AA0 15248–01 from NIDA and NIAAA. The authors are indebted to Dwain Eckberg for his assistance in this research.

REFERENCES

Angelone, A., & Coulter, N. A. Jr. (1964). Respiratory sinus arrhythmia: A frequency depended phenomenon. *Journal of Applied Physiology*, *19,* 479–82.

Chernigovskaya, N. V., Vaschillo, E. G., Rusanovsky, B. B., & Kashkarova, O. E. (1990). Instrumental autotraining of mechanisms for cardiovascular function regulation in treatment of neurotics. *The SS Korsakov's Journal of Neuropathology and Psychiatry*, *90,* 24–28.

Cooke, W. H., Cox, J. F., Diedrich, A. M., Taylor, J. A., Beightol, L. A., Ames, J. E. 4th, Hoag, J. B., Seidel, H., & Eckberg, D. L. (1998). Controlled breathing protocols probe human autonomic cardiovascular rhythms. *American Journal of Physiology*, *274*(2 Pt 2), H709–18.

DeBoer, R. W., Karemaker, J. M., & Strackee, J. (1987). Hemodynamic fluctuations and baroreflex sensitivity in humans: A beat-to-beat model. *American Journal of Physiology*, *253 (Heart and Circulatory Physiology, 22*), H680–H689.

Giardino, N., Lehrer, P. M., & Feldman, J. (2000). The role of oscillations in self-regulation: their contribution to homeostasis. In D. Kenney & F. J. McGuigan (Ed.), *Stress and health: Research and clinical applications* (pp. 27–52). Harwood Publishers.

Giardino, H. D., Glenny, R. W., Borson, S., & Chan, L. (2003). Respiratory sinus arrhythmia is associated with efficiency of pulmonary gas exchange in healthy humans. *American Journal of Physiology (Heart and Circulatory Physiology, 284)*, H1585–H1591.

Giardino, N. D., Chan, L., & Borson, S. (2004). Combined heart rate variability and pulse oximetry biofeedback for chronic obstructive pulmonary disease: preliminary findings. *Applied Psychophysiology and Biofeedback*, *29,* 121–133.

Grodins, F. S. (1963). *Control theory and biological systems*. New York: Columbia University Press.

Halamek, J., Kara, T., Jurak, P., Soucek, M., Francis, D. P., Davies, L. C., Shen, W. K., Coats, A. J., Novak, M., Novakova, Z., Panovsky, R., Toman, J., Sumbera, J., & Somers, V. K. (2003). Variability of phase shift between blood pressure and heart rate fluctuations: a marker of short-term circulation control. *Circulation*, *22,108*(3), 292–307.

Hamilton, L. L., Lindan, O., & Reswick, J. B. (1969). Dynamic effects of sinusoidal tilting on heart rate of healthy and paralyzed persons. *Journal of Applied Physiology*, *27*(3), 378–384.

Hammer, P. E., & Saul, J. P. (2005). Resonance in a mathematical model of baroreflex control: arterial blood pressure waves accompanying postural stress. *American Journal of Physiology Regulatory, Integrative and Comparative Physiology*, *288*(6), R1637–1648.

Herbs, D., Gevirtz, R. N., & Jacobs, D. (1993). The effect of heart rate pattern biofeedback for the treatment of essential hypertension. *Biofeedback and Self-Regulation*, *19,* 281 (abstract).

Hasset, A., Radvanski, D., & Lehrer, P. (2006). Heart rate variability biofeedback as a treatment for fibromyalgia. Paper presented at the annual meeting of the International Society for Advancement of Respiratory Psychophysiology, Princeton, NJ, October 17–19. *Biological Psychology*, *72,* 232–233 (abstract).

Ito, T., Takamata, A., Yaegashi, K., Itoh, T., Yoshida, T., Kawabata, T., Kimura, M., & Morimoto, T. (2001). Role of blood volume in the age-associated decline in peak oxygen uptake in humans. *Japanese Journal of Physiology*, *51,* 607–612.

Karavidas, M. (2005). Heart variability biofeedback in the treatment of major depressive disorder. *Applied Psychophysiology and Biofeedback*, *30*(4), 397–423.

Karavidas, M., & Lehrer, P. (2006). A pilot study of heart rate variability (HRV) Biofeedback as a treatment for major depression. Paper presented at the annual meeting of the International Society for Advancement of Respiratory Psychophysiology, Princeton, NJ, October 17–19. *Biological Psychology*, *72,* 234–235.

Legramante, J. M., Raimondi, G., Massaro, M., Cassarino, S., Peruzzi, G., & Iellamo, F. (1999). Investigating feed-forward neural regulation of circulation from analysis of spontaneous arterial pressure and heart rate fluctuations. *Circulation*, *99,* 1760–1766.

Lehrer, P. M., Carr, R. E., Smetankine, A., Vaschillo, E., Peper, E., Porges, S., Edelberg, R., Hamer, R., & Hochron, S. (1997). Respiratory sinus arrhythmia vs neck/trapezius EMG and incentive inspirometry biofeedback for asthma: a pilot study. *Applied Psychophysiology and Biofeedback*, *22,* 95–109.

Lehrer, P. M., Vaschillo, E., & Vaschillo, B. (2000). Resonant frequency biofeedback training to increase cardiac variability: rationale and manual for training. *Applied Psychophysiology and Biofeedback*, *25,* 177–191.

Lehrer, P. M., Vaschillo, E., Vaschillo, B., Lu, S. E., Eckberg, D. L., Edelberg, R., Shih, W. J., Lin, Y., Kuusela, T. A., Tahvanainen, K. U. O., & Hamer, R. (2003). Heart rate variability biofeedback increases baroreflex gain and peak expiratory flow. *Psychosomatic Medicine*, *65,* 796–805.

Lehrer, P., Vaschillo, E., Vaschillo, B., Lu, S., Scardella, A., Siddique, M., & Habib, R. (2004). Biofeedback Treatment for Asthma. *Chest*, *126,* 352–361.

Lindqvist, A. (1990). Noninvasive methods to study autonomic nervous control of circulation. *Acta Physiologica Scandinavica Supplementum*, *588,* 1–107.

London, G. M., Guerin, A. P., Laurent, S., London, A. M., & Safar, M. E. (1985). Cardiopulmonary blood volume and plasma renin activity in man: preliminary report. *Journal of Hypertension-Supplement*, *3,* S121–S123.

McCraty, R., Atkinson, M., & Tomasino, D. (2003). Impact of a workplace stress reduction program on blood pressure and emotional health in hypertensive employees. *The Journal of Complementary and Alternative Medicine*, *9,* 355–369.

Mier, C. M., Domenick, M. A., Turner, N. S., & Wilmore, J. H. (1996). Changes in stroke volume and maximal aerobic capacity with increased blood volume in men women. *Journal of Applied Physiology*, *80,* 1180–1186.

Porges, S. W. (1995) Orienting in a defensive world: mammalian modifications of our evolutionary heritage. A polyvagal theory. *Psychophysiology*, *32,* 301–318.

Radvanski, D., Vaschillo, E., Vaschillo, B., Hassett, A., Lehrer, P., & Sigal, L. (2004) Heart Rate Variability Biofeedback for Fibromyalgia Treatment. *Applied Psychophysiology and Biofeedback*, *29,* 308.

Ringwood, J. V., & Malpas, S. C. (2001). Slow oscillations in blood pressure via a nonlinear feedback model. *American Journal of Physiology. Regulatory, Integrative and Comparative Physiology 280*(4), R1105–R1115.

Saul, J. P., Berger, R. D., Albrecht, P., Stein, S. P., Chen, M. H., Cohen, R. J. (1991). Transfer function analysis of the circulation: unique insights into cardiovascular regulation. *American Journal of Physiology*, *261*(4 Pt 2), H1231–1245.

Shevde, K., Pagala, M., Tyagaraj, C., Udeh, C., Punjala, M., Arora, S., & Elfaham, A. (2002). Preoperative blood volume deficit influences blood transfusion requirements in females and males undergoing coronary bypass graft surgery. *Journal of Clinical Anesthesia*, *14,* 512–517.

Sleight, P., La Rovere, M., Mortara, A., Pinna, G., Maestri, R., Leuzzi, S., Bianchini, B., Tavazzi, L., & Bernardi, L. (1995). Physiology and pathophysiology of heart rate and blood pressure variability in humans: is power spectral analysis largely an index of baroreflex gain? *Clinical Science*, *88,* 103–109.

Tiedt, N., Wohlgemuth, B., & Wohlgemuth, P. (1975). Dynamic characteristics of heart-rate responses to sine-function work-load patterns in man. *Pflugers Archiv*, *355*(2), 175–187.

Ursino, M., & Magosso, E. (2003). Role of short-term cardiovascular regulation in heart period variability: a modeling study. *American Journal of Physiology—Heart and Circulatory Physiology*, *284,* H1479–H1493.

Vashchillo, E. G., Zingerman, A. M., Konstantinov, M. A., & Menitsky, D. N. (1983) Research of the resonance characteristics for cardiovascular system. *Human Physiology*, *9,* 257–265.

Vaschillo, E. G. (1984). *Dynamics of slow-wave cardiac rhythm structure as an index of functional state of an operant*. Unpublished Doctoral dissertation. Saint Petersburg, Russia: Institute of Experimental Medicine.

Vaschillo, E., Lehrer, P., Rishe, N., & Konstantinov, M. (2002). Heart rate variability biofeedback as a method for assessing baroreflex function: a preliminary study of resonance in the cardiovascular system. *Applied Psychophysiology and Biofeedback*, *27,* 1–27.

Vaschillo, E., Vaschillo, B., & Lehrer, P. (2004). Heartbeat synchronizes with respiratory rhythm only under specific circumstances. *Chest*, *126*(4), 1385–1406.

Vaschillo, E., Vaschillo, B., Lehrer, P., Bates, M. E., Ray, S., Udo, T., & Pandina, R. (2005). Using Heart Rate Variability to evaluate response to drug-related stimuli: A new approach. *Psychophysiology*, *42,* Suppl. 1, 125.

Vaschillo, E., Vaschillo, B., Bates, M. E., Lehrer, P., Pandina, R., Ray, S., & Udo, T. (2005). Heart rate resonance features do not depend on respiration. *Biological Psychology*, in press.

Wigertz, O. (1971). Dynamics of respiratory and circulatory adaptation to muscular exercise in man. A systems analysis approach. *Acta Physiologica Scandinavica Supplementum*, *363,* 1–32.