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ORIGINAL ARTICLES

Heart Rate Variability Biofeedback Increases Baroreflex Gain and Peak Expiratory Flow

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ABSTRACT

OBJECTIVE: We evaluated heart rate variability biofeedback as a method for increasing vagal baroreflex gain and improving pulmonary function among 54 healthy adults.

METHODS: We compared 10 sessions of biofeedback training with an uninstructed control. Cognitive and physiological effects were measured in four of the sessions.

RESULTS: We found acute increases in low-frequency and total spectrum heart rate variability, and in vagal baroreflex gain, correlated with slow breathing during biofeedback periods. Increased baseline baroreflex gain also occurred *across sessions* in the biofeedback group, *independent* of respiratory changes, and peak expiratory flow increased in this group, independently of cardiovascular changes. Biofeedback was accompanied by fewer adverse relaxation side effects than the control condition.

CONCLUSIONS: Heart rate variability biofeedback had strong long-term influences on resting baroreflex gain and pulmonary function. It should be examined as a method for treating cardiovascular and pulmonary diseases. Also, this study demonstrates neuroplasticity of the baroreflex.

Key Words: biofeedback, • heart rate variability, • baroreflex, • pulmonary function, • neuroplasticity.

Abbreviations: BP = blood pressure;; HF = high frequency;; HR = heart rate;; HRV = heart rate variability;; LF = low frequency;; RSA = respiratory sinus arrhythmia.

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INTRODUCTION

Biofeedback can enable people to obtain voluntary control over various physiological processes (1), and some biofeedback methods have been used widely as adjuncts to, or substitutes for, medical treatment. Typically, a biofeedback trainee views an instantaneous electronic display of a physiological function and attempts to change it. Most biofeedback methods involve teaching patients to control a *level* of a physiological function, such as

muscle tension, HR, or finger temperature. Recently we have reported using biofeedback to produce increases in heart rate *variability* (HRV). This method has been used by Russian clinicians to treat autonomic dysfunction with a variety of clinical manifestations, including anxiety and high BP (2), and we recently used it to improve airway function in asthmatic patients (3). These results tentatively suggest that the method can produce long-term changes in multiple organ systems that are affected by autonomic control. This study focuses on arterial baroreflexes and pulmonary function in a healthy population.

Arterial baroreflex responses, triggered by stretch receptors in the walls of the aortic arch and carotid artery, modulate vagus nerve traffic to the sinoatrial node, and mediate beat-by-beat HR responses to changing arterial pressures (4). Risk for cardiac events (including sudden death) in patients with heart disease is inversely related to the robustness of their baroreflex responses. La Rovere et al. (5) showed that in patients recovering from myocardial infarction, those with subnormal vagal baroreflex gains have a high risk of fatal cardiac events, especially if the patient also has low HRV. The linkage between vagal baroreflex impairment and mortality may partially reflect patients' autonomic responses to cardiac rhythm changes. Ventricular tachycardia, a rapid rhythm that commonly precedes sudden death (6), precipitously lowers arterial pressure, and increases muscle-sympathetic (7) and reduces vagal-cardiac (8) nerve activity. During ventricular tachycardia, arterial perfusion pressures recover more rapidly in patients with stronger than weaker vagal and sympathetic baroreflexes (9). In an exercise/ischemia dog model of sudden cardiac death, ventricular fibrillation occurs when baroreflexes are weak, but does not occur when they are strong (10).

Vagal mechanisms also figure importantly in asthma, because the parasympathetic nervous system plays a major role in modulating airway smooth muscle tone (11). Just as increased baroreflex responsiveness may promote successful responses to abrupt rhythm disturbances in cardiac patients, increased vagal activity can cause bronchoconstriction in asthma, and asthma exacerbations can be associated with cholinergic hyperreactivity (12). The therapeutic effects of HRV biofeedback may be through influencing the body's modulatory processes (eg, the well-known modulation of BP changes by baroreflex activity), through which vagal as well as sympathetic reflexes may be controlled.

The levels of baroreflex gain and vagal bronchoconstriction both vary over time, influenced by various neurobehavioral factors. The earliest quantitative analysis of human baroreflex function (13) documented elevated baroreflex gain during sleep. Fritsch et al. (14) reported that changes of arterial pressure lasting only seconds reset the relation between arterial pressure and vagal and sympathetic neural outflows. Systematic changes in pulmonary function also occur during behavioral laboratory tasks (12) and relaxation (15).

When people try to maximize their respiratory sinus arrhythmia (the vagally mediated HR speeding and slowing that occurs in synchrony with breathing), they spontaneously slow their breathing rates to ~0.1 Hz, about one breath every 10 seconds (16). There are previous reports that slow or deep breathing may acutely increase baroreflex gain in healthy people (17) and can even counteract the bronchoconstrictive effects of inhaled methacholine (18). A controlled trial of slow breathing in the context of yoga documented acute reductions of airway resistance among people with asthma (19). Thus, vagal cardiac and pulmonary mechanisms are linked, and there are reasons to expect that improvements in one vagal limb might spill over into the other.

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▶ **BIOFEEDBACK AND RESONANT FREQUENCY IN HRV**

For each individual there is a resonant frequency in HRV at which maximum amplitudes of HRV can be attained by biofeedback. At this frequency, we have found that HR oscillates 180° out of phase with BP and in phase (0° phase relationship) with respiration (20), such that respiratory and baroreflex effects on HRV interact, producing very high amplitudes at a single frequency, accounting for higher total variability. The resonant HRV frequency usually is ~ 0.1 Hz (6 cycles/min). At this frequency, we previously found that HR and BP oscillate 180° out of phase (20), while HR and respiration oscillate in phase with each other (0° phase relationship, with inhalation coinciding with HR accelerations and exhalation with decelerations). Thus, when people breathe at their resonant frequency, respiratory effects on HRV stimulate baroreflex effects (ie, as the individual inhales, HR rises, BP falls, and the consequent baroreflex response produces a further increase in HR, with corresponding effects during exhalation). The consequent resonance effects produce very large increases in both HRV and baroreflex gain, which can be obtained only when subjects try to increase HRV at this particular frequency (20).

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▶ **METHOD**

Participants

We recruited participants through media advertisements and personal contacts, and screened out volunteers who smoked, had a history of psychosis, mental deficiency, heart disease or arrhythmias, *chronic pulmonary disease (including asthma)*, serious neurological illness (including epilepsy), or who were taking any medication that affected the autonomic nervous system. Fifty-four people participated in the study (See [Table 1](#) for participant characteristics), and were assigned to groups using a restricted randomization procedure, balanced for age and sex: 25 to the biofeedback protocol (of whom 2 dropped out before completion), and 32 to the waiting list (of whom 1 dropped out before completion). Participants were paid \$100 for each of four testing sessions (see below) and \$50 for each of the other training sessions (biofeedback group only). This study was approved by the human research committee of UMDNJ–Robert Wood Johnson Medical School.

View this table: TABLE 1. Participant characteristics

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Physiological data were collected during 4 of the 10 treatment sessions in the biofeedback condition, and in 4 equivalently spaced sessions in the control group. Data were collected during four 5-minute periods: 1) a pretraining rest period ("Task A") in which subjects were asked to relax as deeply as possible with eyes open, and to try not to move, so as not to disturb the measuring equipment; 2) the first 5 minutes of biofeedback training ("Task B"); 3) the last 5 minutes of an approximately 30-minute biofeedback training period ("Task C"); and 4) a posttraining rest period ("Task D"), with the same instructions as for the pretest rest period. For control subjects, instructions for Tasks B and C were identical to those in Tasks A and D.

Instrumentation and Software

Physiological data were recorded on a J&J Engineering (Poulsbo, WA) I-330 DSP physiograph unit. EKG data were collected from sensors on the right arm and left leg (Lead II), digitized at the rate of 512 samples/s. Beat-to-beat BP was recorded from a Finapres unit (Ohmeda model 223), and digitized at a rate of 256 samples/s. The sensor was placed on the participant's left middle finger, and the hand was elevated on a table to approximately the level of the heart. End-tidal CO₂ was taken from a Datex 223 capnometer. The intake tube was inserted into a mouthpiece, and subjects wore nose clips and breathed through the mouth during the 5-minute testing periods. A pneumotachometer was used to record respiratory patterns from which measurements of respiratory rate and tidal volume were derived. During biofeedback sessions, strain gauges around the chest and abdomen were also used to display respiratory activity for training purposes. Spirometry was done before and after each testing session following procedures recommended by the American Thoracic Society [\(21\)](#) using a Koko pneumotach-based spirometer (PDS Instrumentation, Louisville, CO), calibrated daily with a 3-liter syringe.

HR and BP data were edited, and analyzed using the WinCPRS program (Absolute Aliens Oy, Turku, Finland), a program for general analysis of physiological data, including analysis of HR and BP variability and baroreflex gain. Spectral baroreflex gain in the LF range correlates

closely with baroreflex gain assessed directly by using phenylephrine injection to alter BP and trigger baroreflex responses BP (22). In cats, baroreceptor denervation abolishes the coherence between systolic pressure and R-R interval oscillations in this frequency range (23). We estimated baroreflex gain over coherent LF (0.04–0.15 Hz) segments from the squared coherence between pairs of measurements. In this procedure, the squared cross-spectral densities of systolic pressure and R-R intervals are divided by the product of the individual power densities. The transfer function was calculated as the cross-spectral densities divided by the power spectral densities of the systolic pressure. The modulus of the transfer function was used to estimate baroreflex gain (10, 24).

Participants also completed two self-report inventories about their experiences during the testing sessions: 1) the Relaxation Inventory (25), a factor-analytically derived scale that yields a full scale score and three dimensions of the relaxation experience, experience of physical tension, cognitive tension, and subjective assessment of relaxation; and 2) the Side Effects of Relaxation Scale (26), which assesses common adverse experiences of people undergoing various kinds of relaxation training.

Procedure for HRV Biofeedback

The details of the procedure for HRV biofeedback have been described elsewhere (27). The trainee was first taught to breathe at his/her resonant frequency, ie, the frequency at which maximum amplitudes of HRV could be generated voluntarily for each individual. The resonant frequency was determined in the first session by measuring HR oscillation amplitudes while the individual breathed for intervals of 2 minutes at each of the following frequencies: 4.5, 5, 5.5, 6, and 6.5 breaths/min. We provide a "pacing stimulus" for this purpose: a light display that moved up and down on the computer screen at the target respiratory rate. The trainee was instructed to breathe at the rate of that stimulus. The resonant frequency was determined as the respiratory frequency yielding the highest frequency power peak on a moving Fourier analysis of HR data displayed by the I-330 physiograph. Subjects were instructed to practice breathing at the resonant frequency for 20-minute periods twice daily for the next week. Throughout training, the individual was cautioned to breathe shallowly and naturally, in order to avoid hyperventilation, as can be provoked by this technique (16). Participants also were trained to breathe abdominally and to exhale through pursed lips.

At the next session, the participant was given HRV biofeedback in two forms: 1) A beat-to-beat cardiometer display superimposed on respiratory activity taken from the strain gauges. The participant was instructed to breathe approximately in phase with HR changes, with the goal of maximally increasing amplitude of RSA; 2) A moving frequency analysis of HR within the band of 0.005–0.4 Hz, updated approximately every second, reflecting the frequency of HR oscillations within the past 30 seconds. The participant was instructed to increase the spectral power peak that occurred at approximately resonant frequency. In the third session, a stand-alone analog HRV biofeedback device was provided for home practice (Cardiosignalizer KC-3, Biosvyaz Corp., St. Petersburg, Russia), which provided a light-bar display whose height was proportional to amplitude of RSA. The upper and lower limits of the display could be adjusted in order to help shape the participant's response. Participants' home practice now was assisted by the machine.

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▶ RESULTS

[Figure 1](#) shows arterial pressure and R-R interval time series (upper panels) and spectral baroreflex gain from a typical participant during 5-minute rest (left) and biofeedback periods (right). Note that biofeedback 1) lowered systolic pressure and oscillation amplitudes, 2) shortened the shortest and lengthened the longest R-R intervals, and 3) increased the average baroreflex gain (from 8.7 to 15.3 ms/mm Hg). [Figure 2](#) shows typical HR and BP variability during biofeedback, with very high amplitudes all at a single frequency, and BP oscillating 180° out of phase with HR.

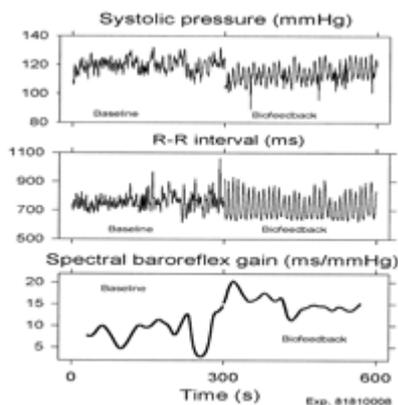


Fig. 1. Recording from one participant before and during biofeedback. In this participant, biofeedback increased systolic pressure and R-R interval oscillations, decreased mean systolic pressure, and increased baroreflex gain.

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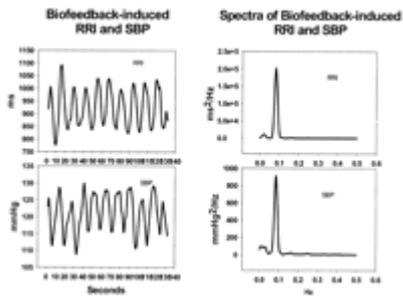


Fig. 2. Heart rate and blood pressure variability during biofeedback from a typical subject. This figure shows the typical 180° phase relationship between heart rate and blood pressure during HRV biofeedback and very high oscillation amplitudes in both measures, all at a single frequency.

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We analyzed data with mixed effects models using SAS Proc Mixed (28), with one between-groups variable (Biofeedback vs. Control) and two repeated measures [Sessions (1, 4, 7, 10) and Tasks (the 5-minute control periods at the beginning and end of each session, ie, Tasks A and D vs. the two 5-minute biofeedback periods in the middle of the sessions, ie, Tasks B and C)]. We fitted each variable with autoregressive (order of one) and compound symmetry models, and identified the better model with Akaike's Information Criteria (29) [In general, tonic measures such as R-R intervals and arterial pressures, were described better by the autoregressive model (correlations are stronger when measurements are closer in time), and dynamic measures such as baroreflex gain, were better described by compound symmetry (correlations do not depend on their closeness in time).] Baseline age, weight, height and sex were included in the model as appropriate. Our results tended to be skewed by large changes, and therefore, we normalized all data with log transformations.

We found a significant pattern of differences in baroreflex gain and HRV between the biofeedback and the control groups across sessions, although mean R-R intervals and systolic pressures were similar (Figs. 3–5 *; Table 2). During each session, baroreflex gain was significantly ($p < .0001$) higher during the two 5-minute biofeedback periods (Table 3 *) than during the two rest periods. Total R-R interval spectral power also was significantly ($p < .0001$) greater during biofeedback than during rest conditions, as was LF spectral power for both BP and R-R interval. Baroreflex gain and R-R interval spectral power did not change in the control group. Because subjects tended to breathe in the LF range, HF variability in RR-interval decreased during biofeedback tasks. The increase in baroreflex gain indicates that the increase in LF R-R variability was greater than that in LF systolic BP variability.

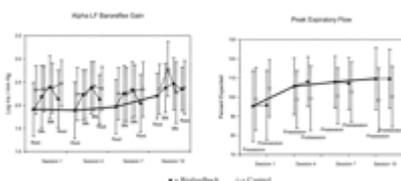


Fig. 3. Long-term physiological effects across weeks of training. Error bars represent standard deviations.

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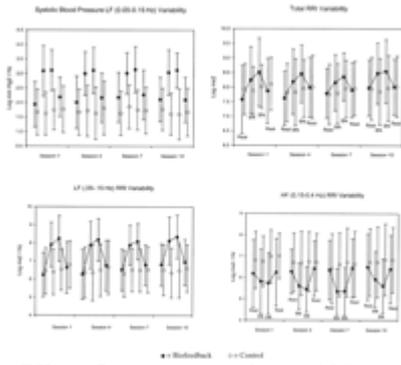


Fig. 4. Effects of biofeedback on measures of cardiovascular variability. Error bars represent standard deviations.

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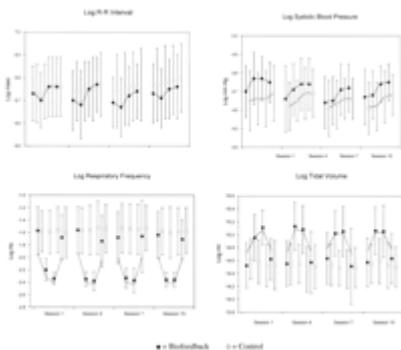


Fig. 5. Effects of biofeedback on measures of tonic physiological activity. Error bars represent standard deviations.

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View this table: **TABLE 2. Medians for cardiovascular variables**
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View this table: **TABLE 3. Results of mixed models analysis on log values**
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Biofeedback also increased baroreflex gain and R-R interval variability cumulatively during sessions. These measures were greater during the postsession rest period (Task D) than during the pre-session rest (Task A) ([Table 2](#)), and they were also greater at the end of the biofeedback training period (Task C) than at the beginning of biofeedback (Task B). *Long-term* cumulative biofeedback effects were assessed by comparing pre-session rest measures (Task A) in the first session, before any training had been given, and the last one. By the 10th weekly training session, pre-session resting baroreflex gain in the biofeedback group was significantly ($p < .003$) greater than during the first session. Baroreflex gain did not change across sessions in the control group. There were no long-term between-group differences in mean HRV.

Respiration rate slowed during biofeedback training periods to approximately six breaths/min (median = 0.1 Hz, mean = 0.092 Hz), and tidal volume increased. However, there were no significant between session differences in either measure at baseline (Task A). In order to determine whether changes in baroreflex gain were explained by changed respiratory patterns, we included tidal volume and respiration rate as factors in the mixed models analysis of baroreflex gain. The *immediate* effects of biofeedback (ie, the comparison between rest periods [Tasks A and D] and biofeedback periods [Tasks B and C]) were erased by this procedure, but the long-term baseline effects (Task A in Session 1 vs. Task A in Session 10) were not influenced by respiratory patterns ([Table 3](#) ✦). End-tidal CO₂ was not affected by the experimental procedures.

Although pulmonary function was normal in all participants, significant ($p < .0001$) increases in peak expiratory flow occurred between the first and last treatment sessions in the biofeedback group (respectively, 95.3 ± 18.5 and $109.6 \pm 16.2\%$), but no changes in the control group and no correlation between baroreflex and pulmonary effects in either group.

We found no significant between-group differences in the Relaxation Inventory, but, across sessions, subjects in the biofeedback condition reported significantly fewer negative side effects of relaxation training than subjects in the waiting list condition in Sessions 4, 7, and 10, but not in Session 1 ([Table 4](#)), indicating that regular training and/or practice of biofeedback tended to block some of the negative side effects of relaxation that might occur when people are instructed to relax without special training in how to do it.

View this table: **TABLE 4. Proportions of having one or more negative side effects commonly associated with relaxation training in each treatment group, by testing sessions**
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► DISCUSSION

Biofeedback acutely increased both HRV and baroreflex gain, and chronically increased baroreflex gain and peak expiratory flow even among healthy individuals, in whom these measures ordinarily are thought to be stable. Other interventions known to increase baroreflex gain, including β -adrenergic blockade (30) and exercise training (31), also prevent sudden death in high-risk populations. Further research may show that HRV biofeedback training may have similar salutary effects, without the side effects that medication often causes.

The acute baroreflex effects are consistent with our hypothesis that stimulation of HRV at its resonant frequency by respiratory activity involves amplification of the vagal baroreflex response, and that this "exercises" the baroreflex. Evidence for resonance in HRV includes the large and highly significant ($p < .0001$) increase in total as well as LF HRV (Tables 2–4 ***) during biofeedback, all at a single frequency. This frequency was close to 0.1 Hz, which appears to be the modal resonant frequency across individuals (20). The acute effects of biofeedback on baroreflex gain were related to respiratory frequency and tidal volume, and were probably produced by the latter. After we adjusted for changes in respiration rate by entering respiration rate as an independent variable in the mixed models analysis, we found that baroreflex gain during biofeedback periods no longer differed significantly from that during rest periods.

However, the *cumulative* changes in baroreflex gain, both within and, more importantly, *across* sessions, were *not* simple effects of slow breathing. The effects of biofeedback on baroreflex gain, both within and between sessions, remained significant, after factoring out the effects of respiration rate. Thus, although breathing at participants' resonant frequencies produced immediate baroreflex augmentation, over time (both within individual sessions and over weeks of practice) the baroreflex became intrinsically more responsive, an effect that no longer depended on breathing rate and volume. Thus, the intrinsic resting baroreflex increased.

We suggest that chronic biofeedback-induced increases in baroreflex gain, which, to our knowledge, has not previously been reported, reflects *neuroplasticity*. There are many

opportunities within the baroreflex arc for such plastic changes to occur. It is known that the neurochemical phenotype of autonomic neurons changes continuously in response to changes of neural traffic, feedback by innervated targets, and changing neurotransmitter and hormone levels (32). Also, it is known that baroreflex gain is modulated by higher centers. Electrical hypothalamic stimulation inhibits baroreflex responses (33). It seems likely that biofeedback alters central modulation of baroreflex gain.

At the same time, biofeedback appears to modulate traffic over vagal pathways involved in maintenance of airway tone. However, the lack of correlation between baroreflex and pulmonary effects suggests that the mechanisms for the two effects may be different. Similarly, none of the physiological changes were closely associated with self-reported experiences of relaxation, suggesting also that the cardiorespiratory effects cannot be explained by relaxation. The fewer relaxation side effects reported in the biofeedback condition suggest that the training procedures are less stressful than asking people to relax on demand, without special instruction.

Our peak flow results are consistent with data from preliminary studies showing that HRV biofeedback may be helpful in treating asthma (3, 16). Similarly, the baroreflex effects suggest that it may be helpful for various cardiovascular disorders linked to impaired baroreflex control, including orthostatic hypotension and perhaps other forms of BP dysregulation, and perhaps other cardiovascular diseases.

The principal limitation of our experiment is its duration. Future research should probe the possibility that the trend we identified continues.

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