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SPECIAL ISSUE

How Does Heart Rate Variability Biofeedback Work? Resonance, the Baroreflex, and Other Mechanisms

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Heart rate variability biofeedback is known to have multiple effects on the cardiovascular system, the respiratory system, and emotional reactivity. This paper reviews the origins of work on heart rate variability biofeedback, and mechanisms for its various effects, including direct effects on the baroreflex system and gas exchange efficiency, as well as indirect effects on emotional reactivity and possibly inflammatory activity. Resonance in the cardiovascular system is explained, as well as ways that heart rate variability biofeedback stimulates these resonance effects, through interactions between respiratory sinus arrhythmia and the baroreflex system. Relationships of these mechanisms to various clinical applications of heart rate variability biofeedback are explored, as are future extensions of biofeedback to the vascular tone baroreflex.

The Origins of Heart Rate Variability Biofeedback

Heart rate variability (HRV) biofeedback appears to have profound effects across systems: cardiovascular, respiratory, behavioral, and gastrointestinal. It also improves athletic performance. Has it become the snake oil of the biofeedback profession, or are there underlying mechanisms for its cross-system effects? For over 20 years, inspired by and, more recently in collaboration with, the Russian-American physiologist and engineer Evgeny Vaschillo, I have been involved in studying these mechanisms. The most direct effects are on the cardiovascular system, but some pathways to other effects are known and there are viable hypotheses about mechanisms underlying other effects.

In Vaschillo's original studies, carried out in St. Petersburg with the Russian space program, he investigated resonance effects in the cardiovascular (CV) system, and effects of stimulating the system at particular frequencies in each individual. When the CV system is rhythmically stimulated at the specific frequencies with these naturally occurring amplitude peaks, very large oscillations in heart rate (HR) and blood pressure (BP) take place at these frequencies (Vashchillo, Konstantinov, and Menitskii, 1984; Vashchillo, Zingerman, Konstantinov, and Menitskii, 1983).

Vaschillo systematically studied this phenomenon. Working with cosmonauts, he asked study participants to replicate with HR a pattern of very high amplitude oscillations displayed as a pattern on an oscilloscope. Participants in the study were not told how to do this, nor were they even told that the goal was to affect HR. They only were told to make the HR biofeedback tracing replicate the stimulus pattern on the oscilloscope. In order to perform the task as instructed, the individuals had to achieve a fluctuation of 60 beats per minute with each HR oscillation. Vaschillo varied the frequency of oscillations from periods of 7 to 100 seconds. For each individual, he found a single frequency where the amplitude of HR oscillations was highest, approaching the goal of a 60 beats/min fluctuation amplitude. The range of these frequencies was between 4.5 and 6.5 times per minute, differing among individuals. I helped Vaschillo to present his findings in English, and, after some struggle on my part understanding the scientific principles and the Russian language and scientific style, a paper was eventually published in *Applied Psychophysiology and Biofeedback* describing this work (Vaschillo, Lehrer, Rishe, and Konstantinov, 2002), after rejections from various physiology journals. Objections to the paper varied, but one sticks in my mind: a categorical statement that the reviewers did not believe that HR oscillations could possibly reach the amplitude of 60 beats per minute in normal healthy individuals. They insinuated that the data were faked, the measuring equipment or calculation method was inaccurate, or that Vaschillo was studying "some kind of Yogis."

The effects were resonance effects. When participants voluntarily produced maximal-amplitude heart rate oscillations, they were triggering resonance in the cardiovascular system. This was analogous to putting a speaker in front

of a microphone, where only a resonance-frequency squeal can be heard, blotting out all other sound frequencies, the "Larsen" effect (Weaver and Lobkis, 2006). A single high-amplitude oscillation, triggered either by noise (stochastic resonance) or by pulsatile stimulation close to the resonance frequency, can obscure the effect of otherwise meaningful perturbations. Loss of variable-frequency information can also affect regulation of physiological processes.

What underlies these resonance properties? Vaschillo performed a calculation called a "transfer function analysis," a method well known by engineers, where he looked at amplitude and phase characteristics of signal pairs at each stimulation frequency. Among the pairs he looked at were: (a) the stimulus oscillation paired individually with HR, BP, and respiration (i.e., the tracing of inhalations and exhalations); (b) HR vs. BP; and (c) HR vs. breathing. He found some very interesting relationships at the specific frequency where participants produced the maximum amplitude heart rate oscillations (Vaschillo et al., 2002), and at no other frequency:

1. Heart rate and breathing were completely in phase with each other, such that heart rate rose and fell precisely at the same time as participants inhaled or exhaled (a 0° phase relationship).
2. Heart rate and blood pressure were completely (180°) *out* of phase with each other.

Physiology of the Resonance Frequency

To understand why these relationships produced the effects that Vaschillo found, a digression is necessary to describe two underlying physiological processes stimulated in his experiment, respiratory sinus arrhythmia and the baroreflex.

Respiratory Sinus Arrhythmia (RSA)

RSA is the fluctuation in heart rate corresponding to breathing, which modulates HR through the vagus nerve, the major parasympathetic nerve innervating the cardiovascular system. RSA is systematically enhanced during relaxed states, and is systematically decreased during stress and various disease states. Vaschillo's work contributed greatly to our understanding of the phase and amplitude relations between RSA and breathing. Later work in Japan by Hayano and his colleagues found that the phase relationships between RSA and breathing had profound effects on respiratory gas exchange (Hayno, Yasuma, Okada, Mukai, & Fujinami, 1996; Yasuma & Hayano, 2004). They artificially ventilated dogs whose cardiac nerves had been severed, and artificially produced HR:respiratory phase relationships of 0°, 90° (the phase relationship between RSA and breathing during normal resting respiration), and 180° (Yasuma and Hayano, 2004). They found that gas exchange was most efficient at 0°, and least efficient at 180°. Gas exchange most efficiently occurred when HR was greatest at exactly the time when the lung was maximally oxygenated, during inhalation, where the greatest airway blood flow corresponded to the greatest airway oxygen content. Similarly, since RSA is controlled by the vagus nerve, it is not surprising that efficiency of gas exchange in humans is associated with increased vagus nerve activity under conditions of hypoxia (Ito et al., 2006). Because this phase relationship occurs only when individuals breathe at a specific frequency (averaging 5.5 times/minute across individuals) (Vaschillo, Vaschillo, and Lehrer, 2004), breathing at this rate should produce the same effect. This was, in fact, found earlier by Bernardi and his colleagues in Italy. They found better oxygenation and a smaller tendency to hyperventilate among individuals given a hypoxic ventilatory challenge, where participants rebreathed their exhaled air (Bernardi, Gabutti, Porta, and Spicuzza, 2001). They also found better oxygen saturation among heart failure patients performing this maneuver (Bernardi et al., 1998).

Note also that, when individuals try to maximize the amplitude of HR oscillations, they almost invariably do this by breathing at their resonance frequency. Thus, these large-magnitude increases in heart rate oscillations represent, at least in part, increases in RSA amplitude.

The Baroreflexes

The baroreflexes are important mechanisms for controlling BP. The stem "baro-" comes from the Greek word for "pressure." The baroreceptors are, indirectly, pressure receptors, located in the large blood vessels of the neck: the carotid artery and the aorta. Actually, they are stretch receptors. When blood pressure increases, these arteries stretch, thus triggering the baroreceptors. When BP increases, the baroreflexes cause HR to decrease. When BP decreases, the baroreflexes cause HR to increase. The changes in HR, by mechanical action (i.e., changes in amount of blood flowing through a tube of approximately the same size), consequently produce changes in BP. That is, BP drops after HR decreases and rises after HR increases. However, because of inertia in the blood supply, there is a delay of several seconds between changes in HR and changes in BP. Thus, the baroreflex effects on HR represent a "negative feedback" system with a constant delay. *All* such systems have characteristics of oscillation and resonance (Grodins, 1963). The length of the delay

determines the resonance frequency of the system. For the HR component of the baroreflex system, the delay is approximately 5 seconds, leading to a resonance frequency of 10 seconds (5 seconds in each direction, up and down). Although increases in BP cause an almost immediate baroreflex-induced decrease in HR, the consequent mechanical decrease in BP takes about 5 seconds.

When people breathe at the frequency producing maximal-amplitude increases in RSA, they simultaneously produce maximal increases in gain of the baroreflex (amount of change in HR per unit change in BP), such that the HR oscillations represent a combination of RSA and HR oscillations caused by the baroreflex. Indeed, in our opinion, a large component in the beneficial effects of HRV biofeedback occurs because it stimulates the baroreflex while magnifying the gas exchange effects of RSA. The size of the baroreflex effects can be calculated as baroreflex gain: that is, the amplitude of the decrease in HR that occurs after each increase in BP, and vice versa for decreases. In our own work, we have proven that practicing HRV biofeedback produces immediate large-amplitude increases in the gain of the baroreflex, while regular practice of HRV biofeedback over a period of several months produces an increase in resting baroreflex gain (i.e., when individuals are not practicing HRV biofeedback). This apparently reflects a characteristic of neuroplasticity in the baroreflex (Lehrer et al., 2003). The large amplitude of the increases in RSA and the baroreflex are due to resonance characteristics of the baroreflex system, as described above. Regular stimulation of the baroreflex by HRV biofeedback apparently increases the efficiency of that reflex. As in other aspects of neurobiological function, "practice makes perfect." (My violin teacher taught me that a long time ago.)

Research on HRV Biofeedback and the Baroreflexes

Vaschillo and I also examined the relationship between resonance frequency and various personal characteristics: age, height, weight, gender, and, since we were studying asthma, whether the individual did or did not have this condition (Lehrer et al., 2006). Of these variables, the only ones that correlated with resonance frequency were height and gender. Taller people and men had slower resonance frequencies. These effects were independent of each other. We surmised, then, that blood volume was the predictor of resonance frequency. Men have more blood volume than women, and taller people have more blood volume than shorter people. Greater blood volume would explain greater inertia in the blood supply, and thus a greater delay between changes in HR and consequent changes in BP. Informal observations by a pregnant colleague of mine appear to confirm this theory. As pregnancy progressed, accompanied by an increase in blood volume, her resonance frequency slowed. Several months after birth, resonance frequency returned to its previous level.

Other evidence for the influence of resonance characteristics in producing large-amplitude HR oscillations comes from studies where rhythmical stimulation is applied to the system at resonance frequency by methods *other* than breathing. We found that very large HRV fluctuations are produced by rhythmical muscle tension release cycles carried out at 0.1 Hz (6/min), which approximates the resonance frequency of the human baroreflex loop (Lehrer, Vaschillo, Trost, and France, 2009). Similar effects can be produced by rhythmical presentation of emotion-inducing pictures (Vaschillo et al., 2008) at this frequency. When rhythmical muscle tension/relaxation cycles were performed at other frequencies, the HR effects were much smaller (Vaschillo, Vaschillo, Pandina, and Bates, 2011).

Mechanisms for Clinical Usefulness of HRV Biofeedback

Given its effects on the baroreflex, it would not be too much of a leap to predict that HRV biofeedback should also show some beneficial effects for treating high blood pressure. The baroreflex tends to be depressed in hypertension, as it is in some other forms of cardiovascular disease, where either neural or structural events affect either the baroreflex itself or the ability of the heart muscle to respond to it. Therefore, it is not surprising that training of the baroreflex should help bring blood pressure down among people with hypertension. Several studies have found modest, but clinically significant, effects using HRV biofeedback for treating hypertension (McCraty, Atkinson, and Tomasino, 2003; Nolan et al., 2010; Reineke, 2008). A similar biofeedback method that involves slowing of respiration (but not assessment of specific resonance frequency) also appears to have good effects (Gavish, 2010; Gavish et al., 2011), and a device doing this kind of training has been FDA approved for treating high blood pressure. Incidentally, low blood pressure also might be treatable with this method, although no studies have been reported on applications to this problem. Such an effect has been reported only anecdotally by Russian practitioners (Chernigovskaya, Vaschillo, Petrash, & Rusanovsky, 1990).

Some preliminary data also suggest that the HRV biofeedback training methods may help in treating chronic

heart failure (Moravec and McKee, 2011; Swanson et al., 2009). Here the mechanism is less certain: greater neurocardiac efficiency, better oxygenation, and exercise of the heart muscle all are possible mechanisms. Moravec and McKee (2013, this issue) discuss their findings and their current hypotheses about mechanisms for HRV biofeedback also appears to help in treating anxiety and depression, perhaps from an overlap of brain stem control mechanisms for the baroreflexes in the nucleus tractus solitarius (Di Fede, Parati, Pagani, Shank, and Scheuer, 2006; Rogers, Rybak, and Schwaber, 2000; Shank and Scheuer, 2003; Walker, Easton, and Gale, 1999), which lies in the center of various limbic pathways. Preliminary findings have shown that HRV biofeedback provides better quality of life among patients with emphysema (Giardino, Chan, and Borson, 2004), perhaps because of improved gas exchange when heart rate oscillations occur in phase with breathing during HRV biofeedback.

Other theoretical mechanisms for HRV biofeedback effects are possible, although still speculative at this point. A possibly important one could be a decrease in inflammation. Stimulation of the vagus nerve, as happens when RSA increases, is known to produce decreases in inflammatory activity (Tracey, 2002). Inflammation of the blood vessels has been linked to heart disease (Kalogeropoulos, Georgiopoulou, and Butler, 2012; Tousoulis et al., 2011), so future research may find that HRV biofeedback could improve cardiovascular health by reducing such inflammation. Effects on asthma may have a more complicated causal pathway. However, in one study we demonstrated that HRV biofeedback had 100% effectiveness in preventing significant asthma exacerbations, while it allowed study participants to take less asthma medication, with better pulmonary function and fewer symptoms (Lehrer et al., 2004). Beneficial effects on asthma also were demonstrated in two smaller and less well controlled studies (Lehrer et al., 1997; Lehrer, Smetankin, and Potapova, 2000). The mechanism for these effects requires further research. The effects may stem from decreases in inflammation, from decreases in autonomic reactivity, or even from mechanical stretching of the airways during deep breathing, which is known to reduce bronchoconstriction (Jackson, Murphy, Rassulo, Celli, and Ingram, 2004; Jensen, Atileh, Suki, Ingenito, and Lutchen, 2001). Although no anti-inflammatory effects of HRV biofeedback have yet been found, HRV biofeedback has been found to partially reverse depression of autonomic function that occurs during experimentally induced inflammation (Lehrer et al., 2010).

Looking Forward to the Future: Stimulation of the Vascular Tone Baroreflex

Before closing, it is important to mention a whole new dimension of baroreflex research that is just beginning. It is known that the effects of the baroreflexes are not restricted to effects on heart rate. They also affect vascular tone. When BP increases, baroreflexes make the blood vessels dilate. When blood pressure falls, the baroreflexes make the blood the blood vessels constrict. This negative feedback loop also has a delay in it, but the delay is longer than for the HR loop. The baroreflexes cause almost instantaneous changes in HR, but effects on the blood vessels is more gradual. Vaschillo has collected some data suggesting that the resonance frequency of the vascular tone loop is around 0.03 Hz, or less than two times per minute (Vaschillo, Vaschillo, Buckman, Pandina, and Bates, 2012; Vaschillo et al., 2011). Pulse transit time can be considered to be an indirect measure of vascular tone (Vaschillo et al., 2012). We have just analyzed pulse transit time data from a small group of subjects given one session of HRV biofeedback. At a rest period after the training session, we found an increase in pulse transit time oscillations in the very low frequency range, .005–.05 Hz. Had we found that stimulation of the baroreflex produced a carryover effect of increased baroreflex gain in the vascular tone baroreflex loop? We are now investigating this possibility.

What would the implications be of looking at the vascular tone biofeedback loop? Most theories of hypertension conclude that the problem is due more to vascular problems than neurocardiac. Could systematic stimulation of the vascular tone baroreflex produce even more powerful antihypertensive effects than stimulation of the heart rate baroreflex? Could stimulation of this reflex even be harmful? We do not yet know the answers to these questions.

Conclusion

In summary, we know that HRV biofeedback systematically stimulates and strengthens the baroreflex, should increase gas exchange efficiency, and improves a variety of cardiorespiratory disorders. Although we are not yet certain that the effects on health are directly mediated by baroreflex effects, this is certainly a very viable theory for some HRV biofeedback applications, with solid indirect support. Studies of mechanism as well as effects of vascular tone baroreflex stimulation definitely deserve scientific attention. Other mechanisms could include improved gas exchange efficiency, mechanical effects of slow breathing, and possible anti-inflammatory effects.

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