Biofeedback
Volume 36, Issue 1, pp. 18–21

SPECIAL ISSUE

Heart Rate Variability Biofeedback for Major Depression

Maria Karavidas, PsyD

University of Medicine and Dentistry of New Jersey–Robert Wood Johnson Medical School, Piscataway, NJ

Keywords: heart rate variability, biofeedback, major depression, autonomic nervous system, resonant frequency

Heart rate variability for the treatment of major depression is a novel, alternative approach that can offer symptom reduction with minimal-to-no noxious side effects. The following material will illustrate some of the work being conducted at our laboratory to demonstrate the efficacy of heart rate variability. Namely, results will be presented regarding our published work on an initial open-label study and subsequent results of a small, unfinished randomized controlled trial.

Autonomic nervous system (ANS) dysfunction is thought to play a significant role in depression. Prior research indicates that individuals suffering from depression often show decreased vagal tone, increased heart rate, fatigue, sleep disturbance, and sympathetic arousal. Heart rate variability (HRV) biofeedback involves training subjects to adjust their breathing rate to a resonant frequency (RF), a breathing rate (usually slower than normal breathing) at which respiratory sinus arrhythmia (RSA) is maximized.

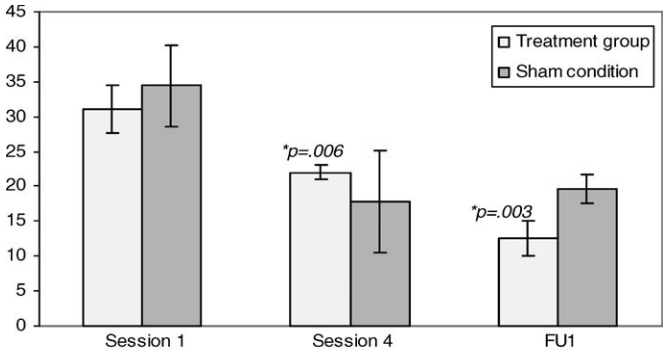
Why HRV biofeedback for depression? Colleagues from our laboratory already have demonstrated that in healthy individuals, HRV biofeedback produces a significant increase in baroreflex gain (change in heart rate for each mm Hg change in blood pressure), presumably leading to improved homeostatic control over blood pressure and other processes associated with it (Lehrer et al., 2003; Vaschillo, Lehrer, Rishe, & Konstantinov, 2002). It also appears to produce an increase in vagus nerve activity. Indirectly, through anatomical projections from the baroreceptors to the hypothalamus and limbic system and increased parasympathetic activity, this method also would be expected to increase modulation of emotionally and autonomically mediated reflexes throughout the body, resulting in reduction of depressive symptoms.

Depression and Autonomic Dysfunction

Autonomic dysfunction has been the target of numerous investigations and has been linked to generalized anxiety disorder, panic disorder, and depression. Symptoms of depression often are accompanied by ANS abnormalities, including reductions in HRV, vagus nerve activity, and baroreflex sensitivity. Animal studies exploring the specific ANS mechanisms of depression by using the chronic mild stress rodent model of depression found that exposure to chronic stress decreased HRV and elevated sympathetic tone to the heart. Successful treatment of depression with selective serotonin reuptake inhibitors (SSRIs) and cognitive-behavioral therapy (Stein et al., 2000) tends to be accompanied by an increase in HRV (Quitkin, Rabkin, Gerald, Davis, & Klein, 2000; Roose, 2003)

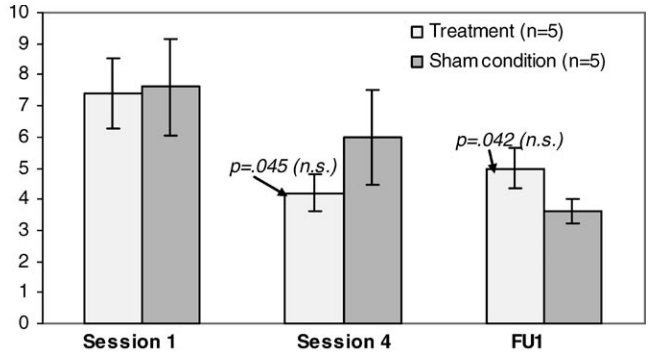
HRV Biofeedback Training for Depression

HRV biofeedback training involves slowing the breathing rate to achieve RF, the frequency at which, in each individual, amplitude of HRV is maximized and the baroreflex is stimulated, apparently causing, indirectly, modulation of limbic system activity. Resonant properties in the cardiovascular system at ~.1 Hz are produced by homeostatic effects of baroreflex activity and inertia of blood flow. As blood pressure rises, the baroreflex causes heart rate to fall. By mechanical action, this eventually causes blood pressure to fall, but only after a delay of ~5 seconds, due to several factors, including inertia of blood flowing through the system and plasticity of the blood vessels. The opposite effects occur when blood pressure falls, causing an oscillation in heart rate with a period of ~10 seconds, or .1 Hz. These resonant properties cause very large-amplitude oscillations in HR when the system is stimulated at the RF. This can be done by breathing at that frequency. Even though for most people RSA is maximized when breathing at the rate of six breaths per minute (.1 Hz), the exact RF varies from one individual to another. Lehrer et al. (2003) demonstrated that regular practice of HRV biofeedback increases baroreflex gain, both acutely and chronically, in healthy people and appears to increase vagus nerve activity. At the same time, as RSA increases, the spectral distribution of HRV shifts, with a greater percentage of total variability now residing in what is called the low frequency (LF) range, including the .1-Hz point. (The LF range includes heart rate variations of



* Significance at α = .05/4 = .0125, corresponding to a Bonferroni correction within the response, is marked by *.

Figure 1. Results of depression scale of Beck Depression Inventory-II (Beck, Brown, & Steer, 1996).



* Significance at α = .05/4 = .0125, corresponding to a Bonferroni correction within the response, is marked by *.

Figure 2. Results of neurovegetative subscale of Beck Depression Inventory-II (Beck, Brown, & Steer, 1996).

a periodicity from .05–.15 Hz [Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996].)

Thus, HRV biofeedback is based on the premise that breathing at this RF will strengthen the baroreflexes and thus improve cardiovascular and autonomic stability, and, indirectly, will reduce emotional lability. Stimulation of the vagus nerve may provide an additional pathway for therapeutic effects on depression. Vagus nerve activity appears to be diminished in depression, and direct electrical stimulation to the vagus nerve appears to help ameliorate serious depression that has been unresponsive to antidepressant medications.

Research on HRV for Major Depression

In the initial open-label study (Karavidas et al., 2007), we demonstrated effects on major depression that appeared to be stronger than those of most SSRIs, suggesting that the method may provide a method for treating depression without the side effects associated with psychopharmacological or neurosurgical interventions that lead many patients to avoid taking these medications. It appeared that HRV biofeedback stimulated the vagus nerve both during practice sessions (acutely) and between sessions (chronically) and by the fourth weekly session, specifically improved somatic symptoms of depression (Hassett et al., 2007; Karavidas et al., 2007).

These results were replicated in our uncompleted randomized control trial ($N = 10$ to date). In the randomized control trial, we demonstrated that significant mood change and symptom reductions were effected in four weekly session treatments comparing an HRV biofeedback group (slow breathing at 4.5–6.5 breaths/minute) versus a sham biofeedback condition (respiration rates between 12 and 15 breaths/minute). (Changes in overall depression rating and in vegetative/somatic symptoms of depression in the randomized control trial study are shown in Figures 1 and 2.) Additionally, in-session increases in LF HRV during biofeedback practice reflect resonance effects involving both respiratory sinus arrhythmia and baroreflex gain (Figure 3, gray bar).

Discussion

Past studies suggest that individuals with major depressive disorder (MDD) might have a dysfunctional response to emotional stress due to central nervous system abnormalities. However, although many studies link stressful life events to onset of MDD (Kendler, Kessler, Neale, Heath, & Eaves, 1993; Paykel, 1979), some depressions are clearly endogenous (i.e., with no apparent environmental trigger) (Musselman, Evans, Nemeroff, & Charles, 1998). It is likely that both the etiology and pathophysiology of depression remain elusive due to highly complex interactions between psychological and physiological factors. Without the benefit of prospective studies, it is difficult to determine whether ANS dysfunction is the cause, effect, or epiphenomenon of MDD. Because we do not know the exact mechanisms involved, the intention of the two studies mentioned above was to analyze psychophysiological data to better explore this link (e.g., baroreflex gain).

In spite of aggressive pharmacological and psychotherapy approaches, 10%–15% of patients will remain chronically depressed. The exploration of self-regulation techniques adds tremendous value to a clinician's "tool box" in both assessment and treatment of the varying components of depression that manifest differently in individuals. HRV biofeedback, as a first-line intervention for the treatment of depression, can possibly maximize treatment effects, because previous research already has demonstrated that treating somatic symptoms of depression improves depression

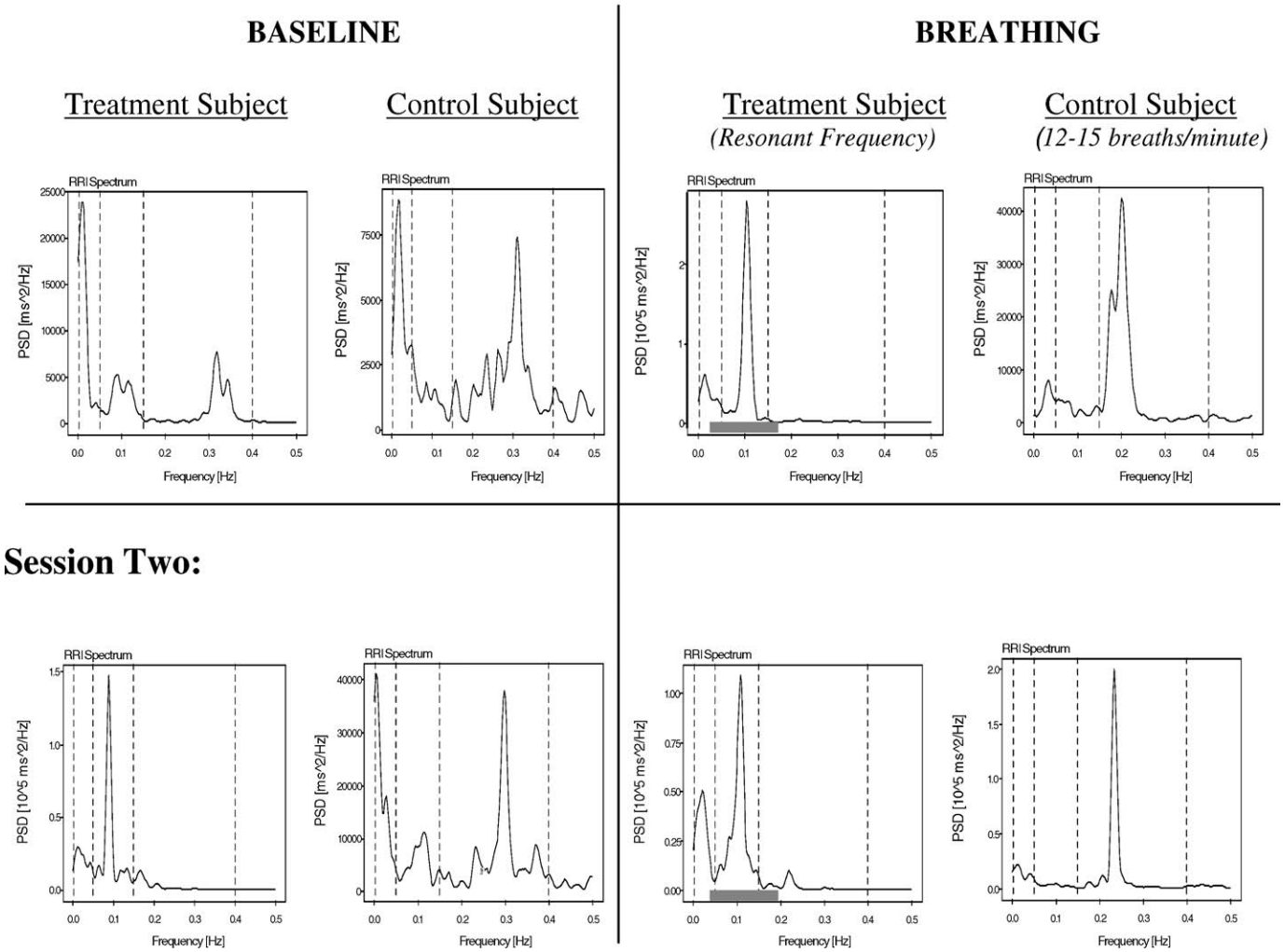


Figure 3. Low frequency (LF) data comparing a treatment and control subject.

overall (McIntyre et al., 2006). As empirically validated treatments based on self-regulation emerge, a tiered system of treatment planning might be considered. For example, a patient deemed to have substantial somatic complaints versus cognitive decrements might benefit from an initial treatment strategy involving HRV biofeedback, although other psychotherapy modalities can serve as secondary treatment options. Although psychopharmacology and psychotherapy are efficacious, underutilization is pronounced among minorities and extends to the general population (Miranda & Cooper, 2004). Given the prevalence and burden associated with MDD, it is critical to offer a variety of efficacious treatments for depression so that individuals can be presented with a variety of treatment options if the treatment currently being used is not effective. A treatment focusing on the physiological process potentially removes some of the stigma associated with depression and enables patients to become active participants, thereby returning some level of control to patients.

References

Beck, A. T., Brown, G., & Steer, R. A. (1996). *Beck Depression Inventory II manual*. San Antonio, TX: The Psychological Corporation.

Hassett, A. L., Radvanski, D. C., Vaschillo, E. G., Vaschillo, B., Sigal, L. H., Karavidas, M. K., et al. (2007). A pilot study of the efficacy of heart rate variability (HRV) biofeedback in patients with fibromyalgia. *Applied Psychophysiology and Biofeedback, 32*, 1–10.

Karavidas, M. K., Lehrer, P. M., Vaschillo, E., Vaschillo, B., Marin, H., Buyske, S., et al. (2007). Preliminary results of an open-label study of heart rate variability biofeedback for the treatment of

major depression. *Applied Psychophysiology and Biofeedback, 32*, 19–30.

Kendler, K., Kessler, R. C., Neale, M., Heath, A., & Eaves, L. (1993). The prediction of major depression in women: Toward an integrated etiological model. *American Journal of Psychiatry, 150*, 1139–1148.

Lehrer, P. M., Vaschillo, E., Vaschillo, B., Lu, S.-E., Eckberg, D. L., Edelberg, R., et al. (2003). Heart rate variability biofeedback increases baroreflex gain and peak expiratory flow. *Psychosomatic Medicine, 65*, 796–805.

McIntyre, R. S., Konarski, J. Z., Mancini, D. A., Zurowski, M., Giacobbe, P., Soczynska, J. K., et al. (2006). Improving outcomes in depression: A focus on somatic symptoms. *Journal of Psychosomatic Research, 60*, 279–282.

Miranda, J., & Cooper, L. D. (2004). Disparities in care for depression among primary care. *Journal of General Internal Medicine, 19*, 120–126.

Musselman, D. L., Evans, D. L., Nemeroff, C. B., & Charles, B. (1998). The relationship of depression to cardiovascular disease: Epidemiology, biology, and treatment. *Archives of General Psychiatry, 55*, 580–592.

Paykel, E. (1979). Causal relationships between clinical depression and life events. In J. Barrett (Ed.), *Stress and mental disorder* (pp. 17–36). New York: Raven Press.

Quitkin, F. M., Rabkin, J. G., Gerald, J., Davis, J. M., & Klein, D. F. (2000).Validity of clinical trials of antidepressants. *American Journal of Psychiatry, 157*, 327–337.

Roose, S. P. (2003). Treatment of depression in patients with heart disease. *Society of Biological Psychiatry, 54*, 262–268.

Stein, P. K., Carney, R. M., Freedland, K. E., Skala, J. A., Jaffe, A. S., Kleiger, R. E., et al. (2000). Severe depression is associated with markedly reduced heart rate variability in patients with stable coronary heart disease. *Journal of Psychosomatic Research, 48*, 493–500.

Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology (1996). Heart rate variability: Standards of measurement, physiological interpretation, and clinical use. *Circulation, 93*, 1043–1065.

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

Maria Karavidas

Correspondence: Maria Karavidas, PsyD, UMDNJ-Robert Wood Johnson Medical School, 671 Hoes Lane, Piscataway, NJ 08873, email: karavima@umdnj.edu.