Effectiveness of *emWave* Biofeedback in Improving Heart Rate Variability Reactivity to and Recovery from Stress

Amanda Whited · Kevin T. Larkin ·
Matthew Whited

Abstract The current study examined the efficacy of heart rate variability (HRV) biofeedback using *emWave*, a publicly available biofeedback device, to determine whether training affected physiological tone and stress responses. Twenty-seven individuals aged 18–30 years were randomized to a treatment or no-treatment control group. Treatment participants underwent 4–8 sessions of *emWave* intervention, and all participants attended pre-treatment and post-treatment assessment sessions during which acute stressors were administered. Physiological data were collected at rest, during stress, and following stress. *emWave* treatment did not confer changes in tonic measures of HRV or in HRV recovery following stress. However, treatment participants exhibited higher parasympathetic responses (i.e., pNN50) during stress presentations at the post-treatment session than their control counterparts. No treatment effects were evident on self-reported measures of stress, psychological symptoms, or affect. Overall, results from the current study suggest that the *emWave* may confer some limited treatment effects by increasing HRV during exposure to stress. Additional development and testing of the *emWave* treatment protocol is necessary before it can be recommended for regular use in clinical settings, including the determination of what physiological changes are clinically meaningful during HRV biofeedback training.

Introduction

Reduced heart rate variability (HRV) is associated with numerous physical and mental health disorders (e.g., Agelink et al. 2002; Correia et al. 2006; La Rovere et al. 1998; Schroeder et al. 2003; Watkinset al. 2002). Consequently, it has been suggested that interventions aimed at increasing HRV may prove beneficial for treating a broad range of stress-related conditions. HRV biofeedback is one intervention that has shown some promise in treating several physical and mental health conditions (for review, see Wheat and Larkin 2010).

Although HRV biofeedback is a relatively new treatment strategy, several studies lend support to the proposition that it is effective in improving HRV. Most studies of this type have implemented a manualized HRV biofeedback protocol developed by Lehrer et al. (2000) wherein the practice is believed to exercise baroreflexes and improve HRV, thereby conveying beneficial health effects. Investigations of HRV biofeedback show that increases in low frequency HRV occur during biofeedback (Hassett et al. 2007; Karavidas et al. 2007; Lehrer et al. 1997, 2003, 2004, 2006). Alternative modalities of biofeedback have recently appeared that are functionally similar in methods and/or objectives as the initial protocol devised by Lehrer (e.g., Amon and Campbell 2008; Heilman et al. 2008; McCraty et al. 2003; Muench 2008). The potential for these newer modalities to be utilized clinically for issues such as anxiety, stress management, or hypertension, etc., is great, especially considering their affordability. The programs also target lay persons and various health professionals as

A. Whited (✉) · M. Whited
Department of Psychology, East Carolina University, East 5th Street, Greenville, NC 27858, USA
e-mail: WhitedAm@ecu.edu

K. T. Larkin
West Virginia University, Morgantown, WV, USA

consumers and therefore are user friendly. However, there is a paucity of research dedicated to discerning whether biofeedback treatment with these instruments confers any improvement in HRV.

The *emWave* (formerly the *Freeze Framer*, *HeartMath Institute*, Boulder, CO) is one such product that currently is marketed for use as biofeedback treatment and is available to the public for purchase on the worldwide web. Whereas the protocol utilized in traditional HRV biofeedback studies emphasizes breathing at the resonance frequency to produce a ~ 0.1 Hz peak, treatment with the *emWave* focuses on the induction of positive emotional states that are associated with "psychophysiological coherence," as termed by its creators at the *HeartMath Institute* (McCraty et al. 2006). Although the biofeedback methods differ, it is clear that *emWave* training embodies biofeedback session goals that functionally are similar to those implemented in historical HRV biofeedback research. In fact, the visual biofeedback of HRV provided with the *emWave* software is strikingly similar to that which is provided with software utilized by HRV biofeedback researchers (e.g., Lehrer et al. 2003). Further, McCraty et al. directly equate their concept of coherence with what "[Lehrer] calls 'resonance'" (p. 23), and calculation of coherence utilizes information derived during power spectral density analysis of HRV. In brief, the coherence ratio is the proportion of the waveform immediately surrounding the peak of the spectral density analysis to the remaining components of the waveform. High coherence ratios indicate that one's heart rhythm occurs consistently within the frequency band that surrounds the peak waveform. In contrast, low coherence ratios are depicted by a broader display of frequencies in the spectral waveform.

It may be reasonably expected then that coherence training conducted using the *emWave* biofeedback device should produce improvement in HRV, if the training was effective. However, no study has been conducted to validate the *emWave* as a tool for improving HRV via biofeedback. The vast majority of intervention studies conducted using the *emWave* or *Freeze Framer* programs have employed full use of the emotional management techniques that accompany these devices without employing HRV biofeedback among all participants. Investigations that have included biofeedback showed that treatment resulted in increased workplace satisfaction, positive affect, positive attitudes, and peacefulness, as well as reductions in areas such as stress, symptoms of anxiety, symptoms of depression, social inhibition, negative affect, anger, fatigue, sleeplessness, and blood pressure (Barrios-Choplin et al. 1999; Climov 2008; McCraty et al. 1999, 2000, 2003a, b). However; of the studies incorporating biofeedback, only two studies have assessed HRV as an outcome. The first study failed to find significant differences in HRV following treatment (McCraty et al. 2003a), although it is unclear the

extent to which each participant received HRV biofeedback training. Results of the second study indicated that middle school students who received the full emotional management treatment exhibited increased HRV during recovery from a stressor following treatment compared to students in a control group (McCraty et al. 1999). However, biofeedback was not systematically implemented for all participants in the treatment group.

A major limitation of the biofeedback literature in general, which includes the *emWave* literature, is the tendency for investigators to focus on altering tonic physiological levels rather than phasic physiologic responses to various types of stress. If indeed biofeedback is intended to make coherence/resonance easier to achieve within the context of stress (McCraty et al. 2006), studies of its efficacy need to employ both tonic and phasic measures of HRV as outcome variables. Despite the conception that improvements in HRV may serve to buffer the effects of stress on the cardiovascular system, only two biofeedback studies have examined whether HRV biofeedback treatment resulted in improved phasic HRV responses to stress; one traditional HRV biofeedback study and one *emWave* study (McCraty et al. 1999; Nolan et al. 2005).

In the traditional HRV biofeedback study (Nolan et al. 2005), patients with coronary heart disease who received traditional HRV biofeedback demonstrated increases in high frequency HRV in contrast to those in a control condition, indicating improved vagal recovery from stress. However, interpretation of this finding is complicated by the fact that participants received additional intervention components beyond HRV that could have conveyed treatment effects (e.g., autogenic relaxation training). The *emWave* biofeedback study that implemented a stressor protocol for measuring treatment outcomes involved middle-school student participants who were enrolled in a course in emotional competence ("Heart Smarts") (McCraty et al. 1999). As mentioned above, only a portion of these participants elected to engage in HRV biofeedback training as part of the *emWave* program. Further, only a subset of participants was selected to participate in a stress recovery protocol following treatment. No analyses were performed to separate those who did and did not utilize the biofeedback component, so it cannot be determined whether results were attributed to biofeedback. Given the limited research investigating the effect of HRV biofeedback on phasic physiological responses to stress, and the limitations of the two studies that have been performed (McCraty et al. 1999; Nolan et al. 2005), the need to determine whether biofeedback alone improves reactivity to and recovery from stressors is evident.

The current study aimed to address whether biofeedback treatment with the *emWave* effectuated increases in tonic HRV and/or improved phasic HRV profiles (i.e., reactivity
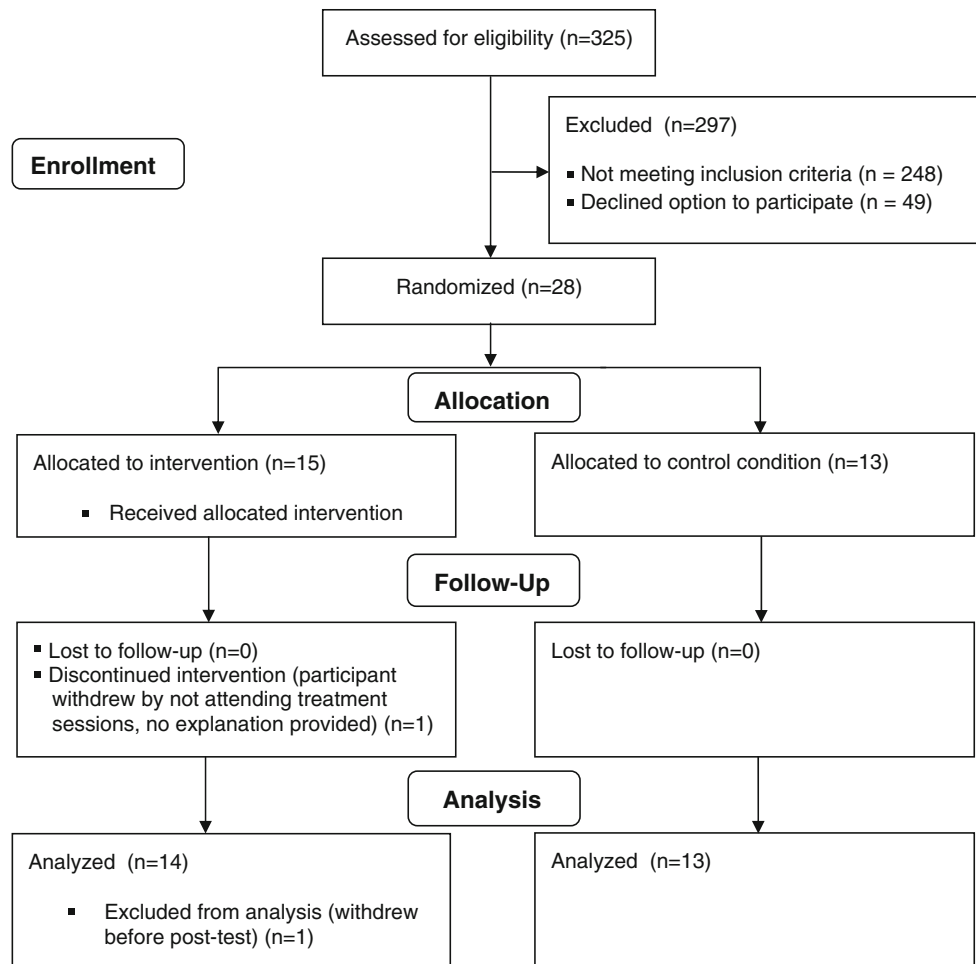
Fig. 1 Summary of participant flow through the research protocol

to and recovery from stress). Although used in full or in part of several empirical endeavors, standalone biofeedback treatment with the *emWave* has not been subject to analytical scrutiny regarding its effects on HRV. Further, although a few attempts have been made to discern whether HRV biofeedback treatment resulted in improved cardiovascular responses to a stressor, the attempts were unsystematic and limited by methodological weaknesses. If treatment with the *emWave* can systematically be shown to provide improved tonic and/or phasic HRV outcomes, a valuable clinical resource that is both affordable and easily utilized may emerge for implementation in the future with a reliable evidence base.

Methods

Participants

A total of 28 undergraduate and graduate student participants at a large state university were recruited via

psychology classes and screened using an online data collection system. Participants were excluded if they smoked or used smokeless tobacco, had chronic major health problems (e.g., heart disease, cancer), or if they were on drug regimens that directly influenced heart rate and/or blood pressure (e.g., beta blockers). Participants received extra credit and/or cash for their participation, which varied based on the length of their participation (e.g., six sessions vs. eight sessions), nature of their participation (i.e., treatment vs. control group), and student status (undergraduate vs. graduate). All participants received equal compensation for pre- and post-treatment assessment visits (i.e., $15). Participants assigned to the treatment group also received cash for each treatment session attended (i.e., $5). Undergraduate students received both cash and extra credit, whereas graduate students only received cash due to insufficient course opportunities for earning extra credit.

Of an original pool of 325 participants completing online screening, 77 respondents met inclusion criteria and were contacted via e-mail to participate in the lab-based portion of the study. Twenty-eight participants initiated

participation in the study, and 27 (96.4 %) completed the study. A flow diagram in Fig. 1 based on CONSORT 2010 recommendations (Schulz et al. 2010) provides information regarding recruitment of the study sample. This study was approved by the relevant Institutional Review Board, and informed consent was obtained from all participants.

Measures

Demographics

Participants completed a demographics questionnaire including items related to participant characteristics (e.g., age, sex), health behaviors (e.g., exercise, smoking), health problems (e.g., chronic illness), and medications known to have cardiovascular effects that could confound HRV data (e.g., beta-blockers, statins).

Heart Rate Variability

HRV was measured using HR data gathered with a *Polar* (Lake Success, New York) RS800CX heart rate monitor. Data obtained from the Polar monitor have been shown to correlate highly and significantly with ECG-derived measures of HR during rest periods and during performance of stressful tasks (Goodie et al. 2000) and have been utilized successfully in several of our prior studies (Whited and Larkin 2009; Whited et al. 2010). This device functions by detecting heartbeats from a sensor strapped around the participants' chest and transmitting to a wristwatch in an adjoined room. Via a USB device, the wristwatch was wirelessly connected to a computer where data collection was monitored real-time by the experimenter.

Self-Report Instruments

Two self-report measures of psychological symptoms and distress [Brief Symptom Inventory (BSI) and Perceived Stress Scale (PSS)] were included for purposes of comparing results relevant to these outcomes with previous studies implementing *emWave* training. The BSI (Derogatis and Melisaratos 1983) provided a global measure of distress (i.e., Global Severity Index) and nine specific subscale scores (i.e., Somatization, Obsessive–compulsive, Interpersonal Sensitivity, Depression, Anxiety, Hostility, Phobic Anxiety, Paranoid Ideation, Psychoticism), and the PSS (Cohen et al. 1983) measured stress. Sufficient alpha coefficient reliabilities were observed for these scales in this study. On average, Cronbach's alpha for pre-treatment subscales of the BSI in the current study was 0.72, and for post-treatment subscales was 0.80. Cronbach's alpha for the Global Severity Index and the PSS at the pre-treatment

session was 0.95 and 0.88, respectively, and 0.96 and 0.89 at the post-treatment session, respectively.

Additionally, state affect was measured to supplement measures of physiological indices of reactivity and recovery during pre- and post-treatment reactivity sessions using the multiple adjective affect checklist-revised (MAACL-R). The MAACL-R contains five subscales: Anxiety, Depression, Hostility, Positive Affect, and Sensation Seeking. Participants indicated which of 66 adjectives described how they felt at baseline and during each stressor. As the MAACL-R was administered three times (i.e., rest, reactivity, recovery) during the two assessment sessions (i.e., pre-treatment and post-treatment), internal consistency was computed six times for each subscale for the current study. Average Kuder-Richardson values were: Anxiety = 0.49, Depression = 0.58, Hostility = 0.67, Positive Affect = 0.69, and Sensation Seeking = 0.47. Low internal consistency values were likely reflective of the small sample and low variance due to many or all participants endorsing few, or sometimes no, subscale items at one or more points of measurement.

Procedure

Participants were randomly assigned to treatment or control groups using a list of odd or even numbers from a random number table. After randomization, participants attended a lab session during which the pre-treatment stress reactivity assessment occurred. Following the pre-treatment assessment, participants completed activities corresponding to their group assignment. When such activities were completed, as outlined below, a post-treatment assessment occurred that mirrored the pre-treatment assessment. Participants randomized to the control condition were told that following their completion of the post-treatment assessment, they would be given the opportunity to receive the study treatment at no cost to them. However, no control participants chose to pursue treatment when it was offered at that juncture of their participation in the study.

Stress Reactivity Protocol

During their first visit to the lab, participants completed self-report measures prior to physiological assessment. Participants then equipped themselves with the *Polar* monitor and were instructed to remain seated in a chair and to remain relatively still with feet flat on the floor while HR data were collected during a 15-min rest period. Data from the final 5 min of the rest period were used in analyses. Following completion of the rest period, but before the first stressor, participants completed the first MAACL-R.

Participants then underwent a stress reactivity protocol. Specifically, a mental arithmetic task required that the participant count aloud and backward from 9,000 in increments of seven. Participants were encouraged to continue from the last place they remembered if they lost track of counting during the task. The task was 5 min in length. Following the mental arithmetic task, there was a 5-min recovery period during which they completed the second MAACL-R. After the recovery period, participants engaged in an isometric hand-grip task (*Lafayette Instruments* hand dynamometer, Model 78010, Lafayette, USA). The investigator assessed participants' maximum grip strength, and the participants were instructed to maintain 30 % of that grip strength for 3 min. Again, another 5-min recovery period followed, and participants completed the third and final MAACL-R.

Treatment Protocol

The *emWave* PC Stress Relief System (*HeartMath*, LLC, Boulder, Colorado) provided HRV biofeedback treatment. The *emWave* includes a fingertip plethysmograph that transfers heart rate data to a software program through which biofeedback is provided. Data from the sensor is transformed to display participants' heart rate in real time. In addition, graphs depicting the HRV power spectrum or pulse wave were displayed. The third biofeedback component involves coherence. There are three bars of varying colors, and each responds to low, medium, or high coherence levels. This visual biofeedback provided information to the participant to assist in physiological and emotional self-regulation while they implemented the quick coherence technique taught with *emWave* (see below).

Following their initial assessment, participants in the treatment group worked with the first author to customize a schedule for biofeedback training. HRV, HRV biofeedback, and how HRV is related to health were explained at the first biofeedback visit. In addition, the *emWave*'s "quick coherence technique" was reviewed and practiced during the first treatment visit. The technique, as described by *HeartMath*, involves attending to the physical area of the heart ("heart focus"), breathing deeply ("heart breathing"), and producing positive emotion ("heart feeling"). A more detailed description of these steps can be found elsewhere (e.g., Culbert et al. 2007). All treatment participants wore the *Polar* heart rate monitor during each entire biofeedback training session to allow for assessment of physiological change across training. Because individuals progressed in exhibiting adequate coherence levels at different paces, total sessions completed varied from 4 to 8 sessions between participants. Guidelines provided by *HeartMath* in their Practitioner's Guide (Culbert et al. 2007) suggest that four to six sessions are typically recommended, so a minimum for four completed sessions prior to completion of the post-test assessment was imposed. Sessions were 32 min in length, and they occurred approximately weekly. Days between the initial and follow-up visits ranged from 28 to 84 days. Control participants were yoked to treatment participants to assure equivalence of length of time between pre- and post-treatment visits.

Participants practiced attaining more moderate and/or high coherence, relative to low coherence, as treatment progressed. Participants remained in treatment until they achieved <50 % low coherence (as suggested in the *emWave* practitioner's guide) and evidenced a distinguishable peak within the low frequency range on the power spectrum within a training session. The latter criterion is not typically a part of *emWave* treatment; however, LF peaks typically occur during biofeedback in traditional HRV biofeedback studies. Further, this criterion should naturally be reached if coherence indeed is reflective of a peak around 0.1 Hz in the power spectrum. The above listed criteria had to be met first at the *emWave*'s low level of difficulty, and then again at the medium level of difficulty. If participants met those criteria within four sessions, they then completed the post-treatment session. For participants who did not meet these criteria by the conclusion of the fourth session, up to four additional sessions were conducted before completing the post-treatment session.

Throughout treatment, participants were instructed to practice the techniques learned with the *emWave* program. They were instructed to do so for 10 min each day at a time when they were relatively calm and in an environment with as little distraction as possible. They also were instructed to practice the techniques when perceiving stress throughout the day. Participants were provided logs to record practicing the technique. The experimenter reviewed the logs with participants at each visit prior to beginning biofeedback so any questions or concerns the participants had could be addressed.

Post Treatment Assessment Session

The stressor protocol administered during the initial assessment was repeated as a separate and final lab session. Using the quick coherence technique alters physiology and would obscure the physiological response normally expected during stress reactivity and recovery. Therefore, treatment participants were instructed not to use the techniques learned during their biofeedback sessions. The first author monitored the heart rate waveform on the computer real-time during the assessment as well, as a distinct waveform is detectable when participants use the quick coherence technique, to detect whether participants were adhering to these instructions.

Table 1 Summary of demographic information for study participants

		Treatment	Control	Overall
Age (in years)	Range	18–30	19–29	18–30
	Mean	22.29	23.15	22.54
	SD	3.6	4.06	3.82
Gender N (%)	Male	2 (14.3 %)	2 (15.4 %)	4 (14.8 %)
	Female	12 (85.7 %)	11 (84.6 %)	23 (85.2 %)
Race N (%)	White	14 (100 %)	11 (84.6 %)	26 (92.8 %)
	Black	0	1 (7.7 %)	1 (3.6 %)
	Other	0	1 (7.7 %)	1 (3.6 %)
Ethnicity N (%)	Non-Hispanic	14 (100 %)	11 (84.6 %)	26 (92.9 %)
	Hispanic	0	2 (15.4 %)	2 (7.1 %)
Weight (in pounds)	Range	102–227.4	96–239.4	96–239.4
	Mean	146.83	156.19	151.5
	SD	34.93	39.42	36.07
Height (in inches)	Range	59–69	58.75–73.75	58.75–73.75
	Mean	64.59	65.4	64.96
	SD	3.01	4.48	3.67
BMI	Range	18.21–37.13	19.55–36.94	18.21–37.13
	Mean	24.76	25.53	25.13
	SD	5.77	5.42	5.51
Parent income N (%)	<24999	1 (7.1 %)	2 (15.4 %)	3 (11.1 %)
	25–34999	0	2 (15.4 %)	2 (7.4 %)
	35–49999	3 (21.4 %)	4 (30.7 %)	7 (25.9 %)
	50–74999	6 (42.9 %)	1 (7.7 %)	7 (25.9 %)
	75–99999	1 (7.1 %)	2 (15.4 %)	3 (11.1 %)
	100–149999	0	1 (7.7 %)	1 (3.6 %)
	150+	1 (7.1 %)	1 (7.7 %)	2 (7.4 %)
	Missing	2 (14.4 %)	0	2 (7.4 %)
Student status N (%)	Undergraduate	6 (42.9 %)	6 (46.2 %)	12 (42.9 %)
	Graduate	8 (57.1 %)	7 (53.8 %)	16 (57.1 %)
Number of treatment	4	10 (71.4 %)	–	–
	5	2 (14.3 %)		
Sessions N (%)	6	0		
	7	0		
	8	2 (14.3 %)		

Note. Groups did not differ significantly on any demographic variable

Results

Preliminary Analyses

Data Preparation

Heart rate data for each period of pre- and post-treatment assessment sessions (i.e., Initial Rest Period; Mental Arithmetic Task; Mental Arithmetic Recovery; Handgrip Task; Handgrip Recovery) were imported to *Kubios* HRV Analysis Software v2.0 (Biosignal Analysis and Medical Imaging Group, Kuopio, Finland) for preparation and analysis of HRV. Within the *Kubios* program, R–R interval data were visually inspected for aberrant or missing data.

Low-level artifact correction was employed in these cases using the artifact correction feature of *Kubios,* which applies an interpolation method to correct erroneous data. Following screening and error correction of the physiological data, LF and HF HRV values were logarithmically transformed to correct for skewness observed among HRV measures (e.g., Kuo et al. 1999; Pinna et al. 2007).

Sample Characteristics

No significant differences between participants in the treatment and no-treatment control groups were evident on any demographic characteristic (all $ps > 0.05$) (see Table 1). Age was related to several measures of HRV and

was therefore incorporated as a covariate in all main analyses involving physiological data. HRV was not related to sex or BMI.

Reactivity to the Stressor Tasks

Repeated measures ANOVAs comparing rest and task periods showed that both tasks elicited significant heart rate reactions at pre- ($M_{mental\ arithmetic} = +7.1$ bpm; $M_{handgrip} = +9.4$ bpm) and post-treatment sessions ($M_{mental\ arithmetic} = +5.0$ bpm; $M_{handgrip} = +7.3$ bpm). To determine whether significant differences existed in reactivity between the two stressor tasks (i.e., mental arithmetic; hand-grip), 2 (Group) × 2 (Task) ANCOVAs were performed on measures of HRV during task presentation at pre-treatment, covarying respective pre-task resting values. No Group × Task interactions were significant for any HRV measure (all $ps > 0.05$), nor were any main effects for Task significant (all $ps > 0.05$). Comparable analyses were conducted on HRV reactivity values at post-treatment with similar results. HRV data therefore were averaged across the two tasks at both pre- and post-treatment phases for conducting the primary study analyses.

Home Practice

Thirteen of 14 treatment participants completed home practice logs in between treatment sessions. One participant did not submit logs, but reported practicing the technique in between training sessions. For the remaining 13 participants, practice of the quick coherence technique reportedly occurred during both calm and stressful times during their daily lives, as instructed. The average number of minutes practiced during times of calm and of stress per week was reported to be 44.3 (SD = 24.48; range 9–89) and 47.4 (SD = 37.13; range 7–110) min, respectively. Overall, the average amount of recorded homework practice was 98.5 (SD = 60.79 range 17–197) min per week. The average number of *emWave* coherence points earned during each session and the average number of minutes practiced per week were not significantly correlated ($r = 0.19$, $p > 0.05$).

Primary Study Analyses

Mixed factors 2 (Group: Treatment, Control) × 2 (Time: Pre- vs. Post-treatment) analyses of covariance (ANCOVAs) were performed to test for differences in tonic HRV, phasic HRV reactivity to stress, and HRV recovery from stress before and after treatment. Age was used as a covariate for these analyses along with pre-task rest period parameters to control for potential differences in resting

levels of the observed parameters for reactivity and recovery analyses. Dependent variables included LF and HF HRV, as well as RMSSD, pNN50, and SDNN. To assess for differences in self-report measures of affect from pre- to post-treatment, comparable 2 × 2 ANCOVAs were conducted, with BSI scales, PSS, and MAACL-R data serving as dependent variables.

Physiological Outcomes

Tonic Measures of Heart Rate Variability

Results of the ANCOVAs on tonic measures of HRV (measured from the initial rest period of the pre- and post-treatment assessment sessions) revealed neither significant main effects for Time, nor Group × Time interactions (see Table 2). This indicated that treatment using the *emWave* protocol did not influence tonic HRV measures in study participants. However, there were significant main effects for Group for both LF HRV and SDNN. At rest, the treatment group exhibited higher LF HRV ($EMM = 7.20$ $_{log}ms^2$, $SE = 0.21$) and SDNN ($EMM = 69.59$ $_{log}ms^2$, $SE = 4.16$) than the control group (LF HRV, $EMM = 6.40$ $_{log}ms^2$, $SE = 0.22$; SDNN, $EMM = 51.27$ $_{log}ms^2$, $SE = 4.31$). Despite randomized group assignment, participants in the treatment group evidenced higher levels of autonomic stability at rest than control participants, a phenomenon that was evident both prior to and following the treatment phases. Based upon these findings, resting measures of HRV were incorporated as covariates in all subsequent analyses of HRV reactivity to and recovery from stress.

Phasic Heart Rate Variability Reactivity to Stress

Using aggregated reactivity data across both tasks (i.e., mental arithmetic and handgrip), Group × Time ANCOVAs were performed on LF HRV, HF HRV, SDNN, RMSSD, and pNN50, covarying age and corresponding resting levels of HRV. A significant Group × Time interaction was evident for pNN50. The treatment and control groups did not differ regarding HRV reactivity to stress during the pre-treatment session, $F(1, 23) = 0.60$, $p > 0.05$, but a significant group difference in pNN50 stress reactivity emerged at the post-treatment session, $F(1, 23) = 6.25$, $p < 0.05$, $\eta_p^2 = 0.21$ (See Table 3; Fig. 2). Specifically, the treatment group exhibited higher pNN50 stress reactivity ($EMM = 25.58$ %, $SE = 2.38$) than the control group ($EMM = 16.98$ %, $SE = 2.47$) at the post-test session. No significant main effects or interactions were observed for LF HRV, HF HRV, SDNN, or RMSSD reactivity (all $ps > 0.05$).

Table 2 ANCOVA for effects of group and session on tonic measures of HRV, controlling for age

	Pre-treatment		Post-treatment		F		
	EM mean	SE	EM mean	SE	Group	Session	Group × session
HF HRV					3.02^{ns}	0.00^{ns}	0.02^{ns}
Treatment	6.61	0.30	6.83	0.20			
Control	6.16	0.29	6.32	0.36			
LF HRV					7.10^{*}	0.18^{ns}	0.00^{ns}
Treatment	7.11	0.21	7.25	0.26			
Control	6.35	0.27	6.51	0.32			
SDNN					9.29^{**}	0.44^{ns}	0.38^{ns}
Treatment	69.28	5.20	68.12	6.18			
Control	50.29	3.44	54.16	6.10			
pNN50					0.71^{ns}	0.06^{ns}	1.83^{ns}
Treatment	25.07	4.62	22.64	4.00			
Control	16.70	4.43	24.31	6.59			
RMSSD					1.48^{ns}	0.01^{ns}	0.08^{ns}
Treatment	47.33	5.38	46.65	4.32			
Control	36.19	4.46	44.87	7.98			

$* p < 0.05.$ $** p < 0.01$

Heart Rate Variability Recovery from Stress

Analyses on all measures of HRV recovery following exposure to stressor tasks yielded no main effects or interactions for LF HRV, HF HRV, SDNN, RMSSD, and pNN50 ($ps > 0.05$; see Table 3).

Exploratory Analyses of Treatment Responders

Two participants progressed less consistently and expeditiously than the other 12 participants in the treatment group, raising the possibility that their gains may have been different in quality or quantity than the remaining participants who engaged with the *emWave* treatment. The main analyses explained above were repeated excluding these two participants to explore whether their inclusion influenced study findings.

The interaction between Group and Time for pNN50 reactivity remained significant, and comparable interactions reached statistical significance in the exploratory analyses for LF HRV, SDNN, and RMSSD (see Table 4). Follow-up analyses evidenced that, among participants who received and responded favorably to treatment, LF ($\eta_p^2 = 0.58$), SDNN ($\eta_p^2 = 0.50$), pNN50 ($\eta_p^2 = 0.78$), and RMSSD ($\eta_p^2 = 0.61$) were higher during stress at the time of the post-treatment session relative to the pre-treatment session ($ps < 0.05$). The control group showed no significant changes in LF HRV, SDNN, pNN50 or RMSSD in response to stress from pre- to post-treatment sessions (all $ps > 0.05$). The interaction for HF HRV was not significant. Finally, consistent with the main analyses including all study participants, there were no apparent treatment effects on HRV recovery following stress when conducting these analyses using only treatment responders.

Self-Report Outcomes

No significant Group × Time interactions, which would indicate a differential effect of treatment versus control status, were observed for MAACL-R (reactivity to or recovery from stress), PSS, or BSI scores. A main effect of group was present for the BSI Anxiety subscale, with treatment participants reporting higher levels of anxiety in general $F(1, 25) = 4.28$, $p < 0.05$, $\eta_p^2 = 0.15$. The groups did not differ on any other self-report outcomes. There were several main effects of Time across the following measures, all indicative of a regression toward the mean pattern across both treatment and control groups: MAACL-R sensation seeking (during mental arithmetic only), depression, and anxiety; PSS; BSI global severity index, anxiety, and somatization (all $ps < 0.05$).

Discussion

This study was designed to empirically address gaps in the HRV biofeedback literature, specifically via employment of biofeedback using the *emWave* system. Although it may be anticipated that improvement in HRV would be associated with increased parasympathetic tone as well as reduced sympathetic nervous system response to stress, and improved coherence through biofeedback training with the *emWave*, no studies to date have been designed exclusively to examine this phenomena.

Table 3 Group × Session ANCOVAs on HRV measures during and following stress, controlling for resting HRV and participant age

	Stress reactivity							Stress recovery						
	Pre-treatment		Post-treatment		F			Pre-treatment		Post-treatment		F		
	EM mean	SE	EM mean	SE	Group	Session	Group × Session	EM mean	SE	EM mean	SE	Group	Session	Group × Session
HF HRV					0.16^{ns}	1.07^{ns}	2.09^{ns}					2.77^{ns}	2.38^{ns}	0.30^{ns}
Treatment	6.47	0.16	6.83	0.21				7.01	0.16	7.07	0.11			
Control	6.25	0.11	6.28	0.13				6.39	0.09	6.67	0.12			
LF HRV					3.18^{ns}	4.11^{ns}	3.80^{ns}					3.36^{ns}	0.01^{ns}	1.58^{ns}
Treatment	7.21	0.16	6.68	0.14				7.50	0.11	7.46	0.13			
Control	6.71	0.12	6.78	0.16				6.62	0.13	6.82	0.22			
SDNN					0.70^{ns}	3.38^{ns}	3.24^{ns}					0.31^{ns}	0.48^{ns}	1.86^{ns}
Treatment	65.34	5.03	75.89	4.09				82.61	3.96	79.59	2.98			
Control	57.51	2.46	59.21	3.53				58.06	3.98	67.41	3.53			
pNN50					2.51^{ns}	9.65^{**}	7.43^{*}					3.08^{ns}	0.04^{ns}	0.01^{ns}
Treatment	18.09	2.37	25.12	2.44				30.87	2.51	28.76	2.29			
Control	13.26	1.66	17.47	2.42				18.92	1.21	25.56	1.75			
RMSSD					1.84^{ns}	6.35^{*}	3.58^{ns}					2.36^{ns}	0.05^{ns}	0.06^{ns}
Treatment	40.40	3.18	50.49	3.99				57.45	4.90	55.27	3.60			
Control	34.30	2.03	40.44	2.52				40.43	1.54	49.67	2.19			

$* \ p < 0.05.$ $** \ p < 0.01$
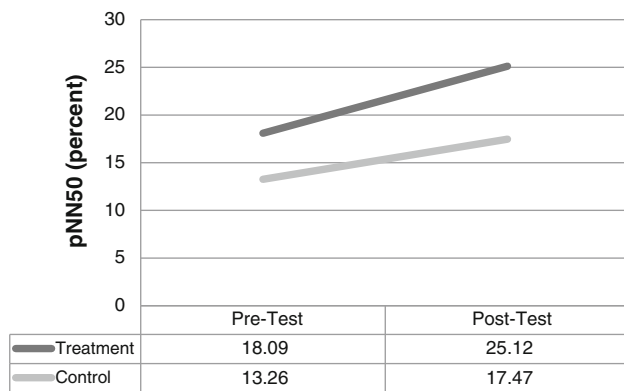
Fig. 2 Interaction between Group and Session for pNN50 (percent consecutive normal-to-normal beats differing >50 ms) during stress, $p < 0.05$

	Pre-Test	Post-Test
Treatment	18.09	25.12
Control	13.26	17.47

Effect of *emWave* Treatment on Tonic, or Resting, Levels of HRV

In the current study, participants receiving *emWave* treatment did not evidence significant change in resting LF, HF, SDNN, pNN50, or RMSSD measures of HRV from the pre-treatment to post-treatment periods. One previous *emWave* study examined changes in physiology following treatment and found that individuals receiving *emWave* treatment showed an increased ratio of LF HRV to HF HRV (McCraty et al. 2003a), a somewhat controversial measure of sympathovagal balance (Berntson et al. 1997; Reyes del Paso et al. 2013). However, this change was not contrasted against a control group so it is unclear whether this effect was due to treatment or some unrelated factor

(e.g., time) (McCraty et al. 2003a). Also, like findings in the current study, no changes were observed in resting LF HRV, HF HRV, SDNN, or RMSSD in their study. Therefore, the current results are in accordance with the finding that *emWave* treatment does not appear to change resting levels of HRV. As the current study included a control group and evidenced similar results to the study conducted by McCraty and colleagues, it does not appear that tonic HRV is affected by *emWave* treatment.

The current results also are largely consistent with the traditional HRV biofeedback literature. Limited evidence has shown possible change in tonic measures of HRV (e.g., Hallman et al. 2011; Zucker et al. 2009), but the vast majority of research have failed to find significant changes in resting HRV (e.g., Hallman et al. 2011; Hassett et al. 2007; Karavidas et al. 2007; Lehrer et al. 2003, 2004; Swanson et al. 2009). Therefore, absence of change in tonic measures of HRV following training with *emWave* biofeedback in this study in combination with available evidence suggests that HRV biofeedback does not reliably confer changes in resting HRV.

Effect of *emWave* Treatment on Phasic HRV

Absence of changes in tonic measures of HRV may be considered evidence against the effectiveness of HRV biofeedback in general and in *emWave* treatment specifically, but caution must be exercised in interpreting findings in this way. Although changes in tonic HRV would certainly be beneficial and lend credence to the efficacy of HRV biofeedback, such changes may not be needed in order to confer therapeutic clinical advantages. For

Table 4 Exploratory Group × session ANCOVAs on HRV measures during stress, controlling for resting HRV and participant age, and after excluding select treatment participants

	Pre-test		Post-test		F		
	EM mean	SE	EM mean	SE	Group	Session	Group × session
HF HRV					1.63[ns]	1.67[ns]	2.83[ns]
Treatment	6.54	0.10	6.94	0.20			
Control	6.25	0.11	6.28	0.13			
LF HRV					5.24*	5.36*	5.01*
Treatment	7.28	0.16	7.83	0.13			
Control	6.71	0.12	6.78	0.16			
SDNN					1.67[ns]	5.00*	5.01*
Treatment	65.69	4.45	78.19	4.40			
Control	57.51	2.46	59.21	3.53			
pNN50					2.76[ns]	14.50**	11.64**
Treatment	17.89	2.00	26.33	2.68			
Control	13.26	1.66	17.47	2.42			
RMSSD					3.09[ns]	9.46**	6.20*
Treatment	40.76	2.67	52.73	4.29			
Control	34.30	2.03	40.44	2.52			

* $p < 0.05$. ** $p < 0.01$

example, change in HRV (phasic HRV) in response to internal or environmental stressors may represent a more important outcome associated with biofeedback interventions than any potential change in tonic level of HRV. Therefore, improved phasic HRV in the context of stress may be more relevant and compelling than changes in tonic HRV.

Evidence was present, but limited, in the current study for changes in phasic HRV following *emWave* treatment. Neither the control nor treatment group showed evidence of change in LF HRV, HF HRV, SDNN, or RMSSD during stress presentations from the pre- to the post-treatment assessment of reactivity. However, the treatment group exhibited higher pNN50 during stress relative to the control group at the post-treatment session. As pNN50 is largely indicative of parasympathetic function (Task Force 1996), this effect suggests that *emWave* treatment resulted in increased parasympathetic augmentation, or less parasympathetic withdrawal, in response to stress.

The current study is one study, among very few studies in the current literature that have assessed phasic changes during stress in HRV following biofeedback treatment. Another study utilized the *Freeze-Frame* (prior version of *emWave*) with children in middle school and found no changes in reactivity to stress (McCraty et al. 1999), but it did not measure pNN50. The only other study offering results for comparison found that participants receiving traditional HRV biofeedback showed increased pNN50 (hand grip and cold pressor tasks) and LF HRV (hand grip task only) response to stress between pre- and post-treatment periods, while the control group did not show increases across the same time points (Hallman et al. 2011). Although evidence is limited, the current study and previous findings from Hallman et al. (2011) suggest that HRV biofeedback may function by increasing parasympathetic activation in the context of stress. It is possible, then, that HRV biofeedback might effectuate physiological mitigation of the stress response by supporting parasympathetic activity to buffer sympathetic reactions to stress.

Although Hallman et al. (2011) also found increased LF HRV during a hand grip stressor, this finding was not replicated with the cold pressor task in their study, and results of the current study did not reveal any change in LF HRV during phasic responses to stress. Of note, neither the current results nor those of Hallman et al. (2011) showed significant changes in HF HRV or SDNN in reaction to stress. Therefore, changes in parasympathetic activity during stress appears to be susceptible to change following HRV biofeedback treatment, but changes in other HRV parameters in addition to pNN50 appear less likely to be observed.

It should be noted that exploratory analyses of treatment responders in the current study indicated that effects of

emWave biofeedback on HRV reactivity may extend beyond the single measure of pNN50. When the two participants who appeared to respond to treatment differently than other treatment participants were excluded and analyses were repeated, significant Group by Time interactions emerged for LF HRV, SDNN, and RMSSD during stress. This pattern of results suggests a more general increase in HRV during stress following *emWave* intervention, as opposed to an isolated effect on parasympathetic functioning (i.e., pNN50). No established parameters exist to differentiate treatment responders from non-responders per se for treatment using HRV biofeedback, so excluding these two participants introduces selection bias into the study analyses. The two participants may represent a subset of individuals whose phasic physiology does not respond to *emWave* treatment, whereas the majority of participants who received *emWave* training represented a group that did experience changes in phasic physiology following treatment. This will be an avenue for future studies to pursue.

Effect of emWave Treatment on HRV Recovery From Stress

Whereas results of the current study support a treatment effect of the *emWave* on pNN50 during stress, there were no effects of treatment on recovery from stress for pNN50, or any other HRV parameter. Therefore, results of the present study do not support the efficacy of *emWave* treatment in improving physiological restoration of the autonomic nervous system's tonic levels of activity following exposure to stress. In their study of implementing HRV biofeedback and other self-regulation treatments in middle schoolers, McCraty and associates (1999) found improvements in LF HRV during recovery from stress. As noted above, however, interpretation of the results of this study is difficult due to the lack of a comparison group. Ultimately, insufficient data exists to date indicating efficacy of *emWave* treatment in improving HRV recovery from stress.

Two traditional HRV biofeedback studies assessed changes in HRV during recovery from stress. Although significant increases in HF HRV between periods of stress and recovery during a physical stressor task have been observed (Nolan et al. 2005), resting levels were not controlled in this study. Although Hallman et al. (2011) showed increases from pre- to post-treatment in SDNN during recovery from a hand grip stressor, no change in SDNN during recovery from the cold pressor task was observed, and no changes were observed among all other HRV outcomes (i.e., HF HRV, LF HRV, and pNN50). Overall, evidence for improvement in HRV during recovery from stress following HRV biofeedback is weak.

In sum, the physiological benefits associated with *emWave* biofeedback training appear to be less related to

change in parasympathetic tone and more likely linked to the increased HRV that occurs in response to stress. Although change in HRV response to stress is a plausible mechanism through which HRV biofeedback may confer clinical improvements in stress-related illnesses, it is also possible that HRV biofeedback treatment exhibits clinical efficacy via another mechanism. For example, biofeedback practice may produce physiological effects other than changes in HRV, such as changes in inflammatory processes, which could facilitate improvement in clinical outcomes (Lehrer et al. 2004). Further, some literature supports changes in baroreflex function as a consequence of HRV biofeedback treatment (Lehrer et al. 2003; 2004) or baroreflex biofeedback (Reyes del Paso and Gonzalez 2004). Baroreflex function was not measured directly in the current study, so it is unknown currently whether *emWave* treatment exercises the baroreflexes and/or strengthens the baroreflex response to stress. However, given that no change in any measure of HRV tone was detected following *emWave* treatment sessions, and yet, participants exhibited increased pNN50 during post-treatment stress exposures, it is possible that baroreflexes were being stimulated and exercised during treatment sessions. Consequently, participants who exercised their baroreflexes during training displayed their improved baroreceptor "fitness" following treatments, but only when such reflex action was warranted (i.e., during stress).

Effect of *emWave* Treatment on Self-report Measures

State affect in the context of stress was assessed to supplement measures of physiological indices of reactivity and recovery. No significant interactions emerged for any of the MAACL-R subscales. Therefore, there were no apparent treatment effects of the *emWave* training protocol on affective responses to stress. However, participants were instructed not to use the quick coherence technique during the post-treatment assessment, so changes in affective responding may not have been anticipated. No previous HRV biofeedback study that assessed phasic stress responses assessed affective responses as part of the stressor protocols, so comparisons to previous investigations were not possible.

As most studies examining the effects of *emWave* treatment focused on non-physiological outcomes, self-report measures of general stress and distress secondary to psychological symptoms were included for purposes of supplementing physiological outcome indices. No interactions emerged for scores on the BSI or any subscales of the PSS to demonstrate that *emWave* treatment affected psychological symptom distress or overall self-reported stress levels. Although improvement on these types of variables following HRV biofeedback training has been reported

(Zucker et al. 2009), most prior studies that have employed control groups have failed to find significant improvement among those receiving biofeedback when compared with control participants (Henriques et al. 2011; Nolan et al. 2005; Swanson et al. 2009).

This study is not without limitations. The *emWave* program was not designed for use in clinical trials and does not have an established protocol. Although those who developed the *emWave* program provided a clinician guide offering some recommended parameters for treatment delivery (Culbert et al. 2007), no evidence-based information regarding implementation of the *emWave* treatment is available. It also is possible that changes in tonic or phasic LF HRV, HF HRV, SDNN, pNN50, and RMSSD do not measure the mechanism through which HRV biofeedback exerts its clinical effect. Improvements in baroreflex functioning may be a viable mechanism through which HRV biofeedback confers clinical improvement across medical and psychological symptoms (see Wheat and Larkin 2010), but baroreflex functioning was not measured in the current study. Additionally, respiration was not measured concurrent to either assessment or training sessions and may have accounted for the relative lack of significant findings associated with biofeedback treatment. Courtney et al. (2011) illustrated that some individuals may exhibit dysfunctional breathing patterns that could affect their ability to improve HRV and obtain coherence during biofeedback. As breathing patterns were not measured in this study, discerning whether breathing dysfunctions existed among study participants was not possible, which may have affected results. The only adherence/practice data obtained for this protocol was self-report, and the findings may have been strengthened by inclusion of more objective adherence measures to illustrate possible dose–response effects. The primary aim of this study was to examine physiological changes in response to *emWave* treatment, so only healthy volunteers were recruited for participation to eliminate potentially confounding disease states and interference by medications less health participants may have been prescribed. In this regard, the magnitude of treatment effects may have been negligible due to a ceiling effect, whereby participants already tended to exhibit adequate physiological responses to stress. A stronger likelihood of altering one's physiological responses to stress may be observed among clinical samples where HRV and associated parameters are known to be compromised. Finally, a few aspects of the current study limit the generalizability of findings. First, our sample did not exhibit an even gender distribution. Our sample also is unique in that the participants were recruited from psychology courses and/or programs of study. Participants may have had more familiarity than individuals not formally educated in psychology with behavioral interventions for psychiatric and/or medical

symptoms or illnesses, and it is possible that they may have been more amenable to engaging readily in *emWave* treatment for that reason.

Due to the paucity of peer-reviewed literature on effects of *emWave* treatment, there are several notable future directions to pursue. Within the context of HRV biofeedback in general, the current study also provides information for areas of improvement in that broader literature as well. It will be informative to test the use of *emWave* in clinical populations to determine whether it may prove to be efficacious in producing physiological and/or clinical changes in those with psychiatric or medical complaints, as well as in those who exhibit affected physiology due to related conditions.

This is the second HRV biofeedback study, and the first utilizing the *emWave*, that found an effect of treatment on pNN50 during reactivity to stress (Hallman et al. 2011). As this effect on parasympathetic responding during stress has been noted across a healthy sample (current study) and a sample of participants with pain (Hallman et al. 2011), as well as across two different modalities of HRV biofeedback, it may represent a reliable treatment effect. Replication of this finding would be beneficial to determine if comparable effects can be noted across additional populations to which HRV biofeedback treatment is delivered.

Ultimately, to rigorously assess the efficacy and effectiveness of *emWave* treatment as a viable way to treat emotional and physical disorders, a standardized treatment approach should be developed for use in empirical assessments of the treatment. For example, investigations addressing treatment development, manual development, specification of session content, determination of treatment length, etc. will assist in maximizing the potential for efficacy of treatment (see Rounsaville et al. 2001) and, importantly, testability of *emWave* treatment as such. Given the ease of use and relatively low cost of the *emWave*, continuing to investigate its effects to establish whether it reliably produces either physiological or clinical change is warranted. The current study documented one way in which *emWave* treatment affected a limited aspect of phasic HRV responses to stress and, in doing so, replicated one other study finding a similar effect. The scientific and clinical communities will benefit from broadening our conceptualization of ways in which HRV biofeedback may confer meaningful clinical changes and, in turn, move us forward to establish which modalities address this mechanism most efficiently and which populations may benefit to the greatest degree.

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