The Effects of Emotions on Short-Term Power Spectrum Analysis of Heart Rate Variability
Rollin McCraty, Mike Atkinson, William Tiller, Glen Rein, and Alan D. Watkins

This study utilizes heart rate variability analysis to examine a new method of intentionaly shifting emotional states, and demonstrates that positive emotions lead to alterations in sympathovagal balance that may be beneficial in the treatment of hypertension. Anger, on the other hand, was shown to significantly increase sympathetic activation.
The Effects of Emotions on Short-Term Power Spectrum Analysis of Heart Rate Variability

Rollin McCratty, MA, Mike Atkinson, William A. Tiller, PhD, Glen Rein, PhD, and Alan D. Watkins, MBBS

The mathematic transformation of heart rate variability (HRV) into power spectral density (PSD) is often used as a noninvasive test of integrated neurocardiac function, because it can help distinguish sympathetic from parasympathetic regulation of the sinoatrial node. HRV and PSD analyses have been used to monitor a variety of pathologic states, and to predict mortality after myocardial infarction and congestive heart failure, and during coronary angiography; they have also been used to determine risk of rejection after cardiac transplantation. Recent work has suggested that HRV and PSD analyses also can be used to characterize a number of psychological illnesses, including major depression and panic disorders, providing a potential link between emotional states and HRV. In addition, a number of studies assessing HRV after mental stress and a recent study looking at hostility have all reported increased sympathetic and decreased parasympathetic activity. Decreased parasympathetic tone has been reported after acute myocardial infarction, hypertension, and heart failure. These findings may explain why emotional and personal stress has been identified as an independent risk factor in cardiac death after acute myocardial infarction and may predict the risk of developing hypertension. This study assesses sympathovagal balance using PSD analysis of HRV during the emotional states of appreciation and anger. We have developed a simple method termed the “freeze-frame” method for consistently producing desired shifts in parasympathetic tone and sympathovagal balance, and have tested it in both normal persons and subjects with a number of pathologic states. The technique has been successfully used in a number of applications to reduce stress and emotional reactivity.

... from alcohol, caffeine, and nicotine for 4 hours before testing. They had no knowledge of the parameters being measured, and had received training in the freeze-frame technique to help manage their mental and emotional responses.

Facilitation of positive emotional states was accomplished utilizing the freeze-frame method. Briefly stated, this technique instructs subjects to consciously disengage from unpleasant mental and emotional reactions by shifting attention to the heart, which most people associate with positive emotions, and focus on sincerely feeling appreciation or a similar positive emotion toward someone in contrast to mentally recalling or visualizing a past positive experience. Previous experience with this technique has shown that it is an effective method of shifting focus of attention away from current stressors. The conscious shifting of awareness to a positive emotional feeling state appears to be a key to the successful application of this maneuver.

The emotion of anger was self-induced by asking subjects to recall situations in their lives that still arouse feelings of anger and/or frustration. This self-recall method has been shown to effectively induce the emotional states of anger and frustration. Subjects were asked to maintain the desired feelings throughout the 5-minute test period. Five-minute recording periods were selected because this was the minimal time required to resolve frequencies down to 0.01 Hz in the HRV power spectrum and the maximal time that most subjects could sustain the emotional focus.

Subjects were seated in straight, high-back chairs to minimize postural changes, and fitted with silver/silver chloride disposable electrocardiographic electrodes. The positive electrode was located on the left side of the chest over the sixth rib and the reference was placed in the right supraventricular fossa. Electrocardiographic measurements were recorded throughout the entire 15-minute rest period, of which the last 5 minutes were used as the baseline. Following this, the subjects were asked to consciously experience appreciation or anger for the next 5 minutes. Heart rate, short-term HRV, and PSD measures were calculated from these two 5-minute periods. Four subjects were studied per session. A total of 6 sessions were conducted over a 2-week period, all at the same time of day (11 A.M.). Before each session, subjects were asked to refrain from talking, falling asleep, exaggerated body movements, and intentionally altering their respiration. Subjects were carefully monitored to ensure there were no significant respiratory or postural changes during the session.

The short-term HRV signal was in the form of an RR interval tachogram. PSD was obtained from the analysis of successive discrete RR interval series taken from the electrocardiographic signal, sampled at 256 Hz. Analyses of HRV, fast-Fourier transforms, PSD (calcu-
TABLE I Effects of Anger on Cardiovascular Variables

<table>
<thead>
<tr>
<th>Variable</th>
<th>Baseline</th>
<th>During Anger</th>
<th>Wilcoxon W</th>
<th>Wilcoxon T</th>
<th>p Value</th>
<th>Change Scores</th>
<th>t</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>LF</td>
<td>0.025 ± 0.008</td>
<td>0.072 ± 0.029</td>
<td>78</td>
<td>0</td>
<td>&lt;0.01</td>
<td>0.047 ± 0.029</td>
<td>5.651</td>
<td>0.0001</td>
</tr>
<tr>
<td>MF</td>
<td>0.01 ± 0.004</td>
<td>0.019 ± 0.006</td>
<td>76</td>
<td>1</td>
<td>0.009</td>
<td>0.009 ± 0.007</td>
<td>4.643</td>
<td>0.0007</td>
</tr>
<tr>
<td>HF</td>
<td>0.023 ± 0.011</td>
<td>0.025 ± 0.012</td>
<td>28</td>
<td>25</td>
<td>NS</td>
<td>0.002 ± 0.013</td>
<td>0.625</td>
<td>0.5446</td>
</tr>
<tr>
<td>LF + MF + HF</td>
<td>0.058 ± 0.018</td>
<td>0.116 ± 0.04</td>
<td>78</td>
<td>0</td>
<td>&lt;0.01</td>
<td>0.058 ± 0.036</td>
<td>5.593</td>
<td>0.0002</td>
</tr>
<tr>
<td>LF/HF</td>
<td>1.312 ± 0.573</td>
<td>3.277 ± 1.721</td>
<td>78</td>
<td>0</td>
<td>&lt;0.01</td>
<td>1.956 ± 1.5</td>
<td>4.537</td>
<td>0.0008</td>
</tr>
<tr>
<td>MF/(LF + HF)</td>
<td>0.214 ± 0.064</td>
<td>0.212 ± 0.09</td>
<td>35</td>
<td>35</td>
<td>NS</td>
<td>-0.002 ± 0.11</td>
<td>-0.054</td>
<td>0.9582</td>
</tr>
<tr>
<td>HR</td>
<td>68.51 ± 9.67</td>
<td>72.42 ± 8.68</td>
<td>78</td>
<td>0</td>
<td>&lt;0.01</td>
<td>3.917 ± 9.829</td>
<td>1.38</td>
<td>0.1949</td>
</tr>
<tr>
<td>HR SD</td>
<td>3.37 ± 0.63</td>
<td>5.08 ± 1.12</td>
<td>78</td>
<td>0</td>
<td>&lt;0.01</td>
<td>1.710 ± 1.187</td>
<td>4.992</td>
<td>0.0004</td>
</tr>
</tbody>
</table>

Values are expressed as mean ± SD unless otherwise noted.
HF = high-frequency power; HR = heart rate; LF = low-frequency power; MF = medium-frequency power.

TABLE II Effects of Appreciation on Cardiovascular Variables

<table>
<thead>
<tr>
<th>Variable</th>
<th>Baseline</th>
<th>During Appreciation</th>
<th>Wilcoxon W</th>
<th>Wilcoxon T</th>
<th>p Value</th>
<th>Change Scores</th>
<th>t</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>LF</td>
<td>0.025 ± 0.014</td>
<td>0.052 ± 0.044</td>
<td>66</td>
<td>6</td>
<td>&lt;0.01</td>
<td>0.027 ± 0.03</td>
<td>3.098</td>
<td>0.0101</td>
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<tr>
<td>MF</td>
<td>0.023 ± 0.016</td>
<td>0.173 ± 0.169</td>
<td>78</td>
<td>0</td>
<td>&lt;0.01</td>
<td>0.150 ± 0.166</td>
<td>3.141</td>
<td>0.0094</td>
</tr>
<tr>
<td>HF</td>
<td>0.019 ± 0.013</td>
<td>0.031 ± 0.025</td>
<td>56</td>
<td>0</td>
<td>&lt;0.01</td>
<td>0.012 ± 0.02</td>
<td>2.011</td>
<td>0.0595</td>
</tr>
<tr>
<td>LF + MF + HF</td>
<td>0.020 ± 0.027</td>
<td>0.256 ± 0.203</td>
<td>78</td>
<td>0</td>
<td>&lt;0.01</td>
<td>0.189 ± 0.19</td>
<td>3.434</td>
<td>0.0056</td>
</tr>
<tr>
<td>LF/HF</td>
<td>1.588 ± 0.453</td>
<td>2.024 ± 1.075</td>
<td>26</td>
<td>26</td>
<td>NS</td>
<td>0.436 ± 1.24</td>
<td>1.219</td>
<td>0.2482</td>
</tr>
<tr>
<td>MF/(LF + HF)</td>
<td>0.538 ± 0.251</td>
<td>2.636 ± 3.244</td>
<td>78</td>
<td>0</td>
<td>&lt;0.01</td>
<td>2.097 ± 3.179</td>
<td>2.285</td>
<td>0.0431</td>
</tr>
<tr>
<td>HR</td>
<td>73.599 ± 9.675</td>
<td>73.867 ± 9.283</td>
<td>0</td>
<td>39</td>
<td>NS</td>
<td>0.268 ± 10.414</td>
<td>0.089</td>
<td>0.9305</td>
</tr>
<tr>
<td>HR SD</td>
<td>3.391 ± 0.836</td>
<td>5.968 ± 2.189</td>
<td>78</td>
<td>0</td>
<td>&lt;0.01</td>
<td>2.577 ± 2.18</td>
<td>4.095</td>
<td>0.0018</td>
</tr>
</tbody>
</table>

Values are expressed as mean ± SD unless otherwise noted.
Abbreviations as in Table I.

lated as [beats/min]²/Hz), and time-domain measurements were performed using DADISP/32 digital signal processing software (DSP Development Corporation, Cambridge, Massachusetts). As described by other investigators, we divided the power spectrum into 3 major frequency ranges (low frequency [LF], medium frequency [MF], and high frequency [HF]). The integral of the power spectrum within each region was calculated. The LF region (0.01 to 0.08 Hz) is primarily considered a measure of sympathetic activity with a minor parasympathetic component. In contrast, the HF region (0.15 to 0.5 Hz) is associated with respiratory sinus arrhythmia and is almost exclusively due to parasympathetic activity. The LF/HF ratio has been used as a measure of sympathovagal balance. The MF region (0.08 to 0.15 Hz) has been used as an indirect indicator of activity in the baroreceptor feedback loop controlling blood pressure. Power in the MF region is thought to be mixed sympathetic and parasympathetic activity, but predominantly the latter. In addition, total (sympathetic and parasympathetic) power (LF + MF + HF) and the MF/LF + HF ratio were calculated. The latter ratio was calculated to provide a measure of the MF power relative to the LF and HF regions because our experience has shown this area to be highly responsive to changing emotional states.

The Mann-Whitney rank-sum test was used to compare baseline values between the 2 groups for all variables and for comparison between anger and appreciation. Comparison of unadjusted baseline data with those during emotional expression within the 2 groups was also performed using the Wilcoxon signed rank-sum test utilizing the sum of the ranks for positive and negative differences for each group. Wilcoxon p values were taken from the table of critical values for the Wilcoxon signed rank test. Because there was a significant intergroup difference in baseline variables (MF and MF/(LF + HF) ratio), data were also analyzed after adjustment for baseline differences. Regressed change scores were calculated and analyzed for significance using 2-tailed t tests for unequal variance against a value of zero.

Comparison of the time-domain traces revealed that most subjects developed a rhythmic sine wave-like pattern during appreciation compared with anger or baseline. Raw data values for change in heart rate and heart rate SD for each 5-minute period are presented in Table I (n = 12). The results from both t tests and the Wilcoxon tests were the same except where noted. Heart rate SD significantly increased during both anger (p < 0.01) and appreciation (p < 0.01). Heart rate, on the other hand, was unchanged during appreciation and increased during anger (p < 0.01). However, the increase in heart rate in the anger group was not significant using baseline-adjusted scores. Means and SDs of baseline-adjusted scores for each variable are presented in Tables I and II. Figures 1 and 2 show the graphic representations. Results show that both emotions caused an overall autonomic activation as demonstrated by an increase in total autonomic activity (LF + MF + HF) and in mean heart rate SD. Thus, total autonomic activity increased significantly during anger (p < 0.01) and during appreciation (p < 0.01). However, the 2 emotional states produced different effects on sympathovagal balance. Anger resulted in a significant increase in LF power (p < 0.01) with no change in HF power. In contrast, appreciation pro-
duced an increase in LF (p < 0.01) and HF (p < 0.05) power. The increase in HF power was not significant when baseline-adjusted change scores were used. Because of the increase in LF power, the LF/HF ratio was significantly increased during anger (p < 0.01) and remained unchanged during appreciation. Activity in the MF region was significantly increased during anger (p < 0.01) as well as during appreciation (p < 0.01). In the MF/(LF + HF) ratio, appreciation produced a significant increase (p < 0.01), whereas anger showed no change. The mean power spectra for all 12 subjects before and during the 2 emotional states are presented in Figure 2. There is a clear shift in spectral power to the MF (0.1 Hz) region during appreciation and to the LF or sympathetic region.

FIGURE 2. Mean power spectral density (PSD) before and during emotional states for both groups.
during anger. When comparing anger with appreciation, the LF power was significantly greater during anger (p = 0.032), whereas the MF power was greater during appreciation (p < 0.0001). The LF/HF ratio was significantly greater during anger (p = 0.037), whereas the MF/(LF + HF) ratio was significantly greater during appreciation (p < 0.0001).

There is now a substantial body of evidence indicating the impact of sympathovagal balance on morbidity/mortality for cardiovascular disease. The detrimental effects of mental and emotional stress on cardiovascular function in humans have also been well documented. However, with the exception of 2 studies investigating the effects of joy and happiness, we are unaware of any studies examining the effects of positive emotions on cardiovascular function. Similarly, there is a paucity of data on the effects of positive and negative emotions on time-domain HRV or PSD analysis.

In this study, both anger and appreciation caused an overall increase in autonomic activation measured as the power in all frequencies of the HRV power spectrum and short-term HRV measured by heart rate SD. The 2 emotional states, however, could be distinguished by power spectral analysis. Although both emotional states produced an increase in sympathetic activity, in the case of anger, sympathetic activation was responsible for the significant increase in the LF/HF ratio. These results extend previous studies by demonstrating that, similar to mental stress, feelings of anger produce a sympathetically dominated power spectrum. The use of the freeze-frame technique to enhance feelings of appreciation, on the other hand, enabled subjects to shift their sympathovagal balance in the opposite direction toward increased MF and HF predominance. Previous experience with subjects untrained in the freeze-frame technique has shown that simply experiencing feelings of anger produces increased sympathetic activity, whereas experiencing appreciation normally only increases total autonomic power. The freeze-frame technique is required to produce the changes in HRV and PSD analysis seen in this study.

A recent study by Sloan et al suggests that mental stress increases LF activity and decreases MF and HF activity. Similarly, anger expressed by type A personalities produces an increase in sympathetic activity and elevated neuroendocrine responses. In keeping with these data, we also found that self-recalled anger caused sympathetic activation, as indicated by increased LF power and an increased LF/HF ratio. In this study, we observed an increase in MF power during emotional states. In contrast to anger, this increase was seen during appreciation whether expressed as a percentage of total power or as the MF/(LF + HF) ratio. Although our data do not examine mechanisms, there is evidence that this increase in MF power during appreciation reflects increased baroreceptor-afferent activity. Baroreceptor activity is known to inhibit sympathetic efferent outflow to peripheral vascular beds, whereas stress increases sympathetic outflow, inhibiting baroreflex activity. All 12 subjects in the appreciation group had an increase in the MF region of their power spectra, which is clearly displayed in the average power spectra illustrated in Figure 2. It is possible that this increase in MF power may have important implications in the control of hypertension, since baroreflex sensitivity is reduced in many hypertensive persons. This view is supported by anecdotal evidence from our laboratory, where we have observed dramatic reductions in blood pressure in hypertensive patients after they have learned to shift their attention to the heart and engage positive emotional states. The reduction in blood pressure observed is usually accompanied by an increase in MF power. A large scale clinical study is now being designed to test this hypothesis.

Various psychological interventions have been shown to reduce sympathetic drive and/or increase vagal activity. For example, subjects can be trained to consciously control their heart rate using biofeedback techniques. However, this is probably mediated by controlling respiratory rate, thereby increasing vagal activity. More recently, it was demonstrated that neutral hypnosis or operant conditioning of the heart rate can produce a decrease in the LF/HF ratio that is independent of controlled breathing techniques.

Our results suggest that emotional experiences play a role in determining sympathovagal balance independent of heart rate and respiration. The shifts in sympathovagal balance toward increased MF and HF power were physiologic manifestations of experiencing the emotional state of appreciation. This study, we believe, is the first to demonstrate that positive emotions can significantly influence HRV and PSD. Although we only examined a few patients over a short period of time, results support previous work and suggest that psychological interventions that minimize negative reactions, such as anger, hostility, and anxiety, and enhance positive emotional states, such as appreciation, could significantly impact cardiovascular function. Larger studies assessing the effects of this type of behavioral intervention and examining serial assessments of HRV and outcomes are necessary to determine the clinical utility of this type of intervention.

In summary, this work extends previous findings by demonstrating that anger produces a sympathetically dominated power spectrum, whereas appreciation produces a power spectral shift toward MF and HF activity. Results suggest that positive emotions lead to alterations in HRV, which may be beneficial in the treatment of hypertension and in reducing the likelihood of sudden death in patients with congestive heart failure and coronary artery disease.


