Fractal component of variability of heart rate and systolic blood pressure in congestive heart failure

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1. There is a substantial non-harmonic or fractal component to the variability of both heart rate and blood pressure in normal subjects. Heart rate is the more complex of these two signals, with respect to the slope, $\beta$, of the $1/f^\beta$ relationship. In congestive heart failure, heart rate spectral power is attenuated, but the fractal and harmonic components of heart rate and systolic blood pressure variability have not been characterized.

2. The groups, each comprising 20 men, were studied during 15 min of supine rest and spontaneous respiration: one with functional class II–IV heart failure (age 52 ± 2 years; mean ± SEM) and a second group of healthy men (age 46 ± 2 years).

3. Total spectral power for heart rate was significantly reduced in heart failure ($P < 0.02$), whereas total spectral power for systolic blood pressure was similar in the two groups. In both heart failure and normal subjects, 65–80% of total spectral power in these two signals displayed fractal characteristics.

4. In heart failure, the slope of the $1/f^\beta$ relationship for heart rate was significantly steeper than in normal subjects ($1.40 ± 0.08$ compared with $1.14 ± 0.05$; $P < 0.05$), indicating reduced complexity of the fractal component of heart rate variability. There was no significant difference in the $1/f^\beta$ slope for systolic blood pressure variability between these two groups, but the blood pressure signals were less complex than heart rate variations in both heart failure ($2.31 ± 0.15$; $P < 0.006$) and normal subjects ($2.47 ± 0.15$; $P < 0.0001$).

5. Parasympathetic nervous system activity, as estimated from heart rate variability was reduced ($P < 0.01$) in patients with heart failure, whereas trends towards increased sympathetic nervous system activity and decreased non-harmonic power were not significant.

6. The non-harmonic components of cardiac frequency are reduced in heart failure. Non-harmonic power is not attenuated, but the complexity of the heart rate signal is less than in subjects with normal ventricular function. A reduction in parasympathetic modulation appears to contribute to this loss of complexity of heart rate. Consequently, the heart rate signal comes to resemble that of blood pressure. In contrast, the variability and complexity of the systolic blood pressure signal is similar in heart failure and normal subjects. This reduced complexity of heart rate variability may have adverse implications for patients with heart failure.

INTRODUCTION

Spectral analysis of heart rate variability (HRV) can provide quantitative information on the relative balance between the parasympathetic and sympathetic neural control of heart rate [1–3]. These harmonic contributions to heart rate variability are superimposed on broad-band non-harmonic noise that is fractal in nature, with its greatest spectral power in the very-low-frequency range [4, 5]. This non-harmonic component can be characterized if the log of spectral power is plotted as a function of the log of frequency. The slope of the $1/f^\beta$ relationship provides an overall estimate of the complexity of these variations [5]. The same analysis can be applied to the blood pressure signal [6].

Approximately 70% of the spectral power of both heart rate and blood pressure displays fractal characteristics [6]. In normal subjects, heart rate is the more complex signal, with a $\beta$ value of approximately 1.0 under resting conditions [1, 7, 9]. Following cardiac transplantation, the slope, $\beta$, for heart rate increases to 2.1 [7]. This approaches the $\beta$ value...
for blood pressure variability (BPV) in normal subjects, which is approximately 2.3 [6]. The difference in the complexity of these two signals in healthy subjects has been attributed to the modulation of heart rate by both parasympathetic (PNS) and sympathetic nervous system (SNS) input [6, 9].

Loss of total heart rate spectral power after myocardial infarction and, in particular, the reduction in spectral power at its very lowest frequencies (where the bulk of non-harmonic power is distributed), is strongly associated with high risk of subsequent mortality and arrhythmic death [10]. The β value for heart rate variability is also an independent predictor of cardiac and arrhythmic death [7]. Indeed, Bigger et al. [7] argue that power-law regression parameters are superior to traditional power spectral bands in predicting these events. Consequently, there is considerable interest in determining the source of harmonic and non-harmonic power, identifying mechanisms by which their loss would predispose to adverse outcome, and in developing and applying pharmacological and non-pharmacological interventions that may augment both components of HRV [11, 12]. In congestive heart failure (CHF), HRV spectral power is attenuated [13–16], but the complexity of the heart rate signal has not been quantified and mechanisms responsible for any loss of complexity have yet to be established. Furthermore, potential non-harmonic contributions to the heart rate and blood pressure signals have not been characterized in this condition.

The present study had three objectives. The first was to compare 1/fβ relationships for HRV and BPV during supine rest and spontaneous respiration in subjects with normal and impaired ventricular function. The second was to determine the effect of CHF on the fractal component of HRV and BPV by comparing these with values acquired from normal subjects studied under similar conditions. The third objective was to investigate potential neural mechanisms responsible for the generation of the 1/f variation by relating these observations to PNS and SNS influences on the harmonic components of heart rate and systolic blood pressure variability.

METHODS

Patients

Twenty men with chronic, stable CHF (New York Heart Association functional class II–IV) and a left ventricular ejection fraction of <40% were studied. Their average age was 52±2 years (mean ±SEM). The mean left ventricular ejection fraction (determined by technetium-99 equilibrium angiography) was 19±2%. Long-term medications included digoxin (n = 14), diuretics (n = 12) and angiotensin converting enzyme inhibitors (n = 18). None were on β-blockers. All were in normal sinus rhythm and had few or no atrial or ventricular extrasystolic beats when screened. Prescribed medications were continued except for diuretic drugs, which were withheld on the morning of the study. Twenty healthy men (age 46±2 years; P>0.05) were studied under identical conditions. All subjects signed a consent form approved by our institution's Human Subjects Review Committee.

Experimental procedure

Data were acquired with subjects in the supine position, breathing spontaneously in a quiet, motionless and awake state, with eyes open. Lead II of the ECG was recorded continuously and inscribed onto paper by an ink recorder (Gould model 2800S, Cleveland OH, U.S.A.). In a subgroup of 16 of these subjects with heart failure (mean age 51 years) and in 16 healthy subjects (mean age 48 years), a volume-clamp cuff was placed on the left middle finger for continuous non-invasive beat-by-beat recording of blood pressure [17] (Ohmeda 2300 Finapres). The analogue output of the ECG amplifier was discriminated to yield a train of rectangular impulses corresponding to the QRS complexes. The impulse train was processed on a real-time basis with a microcomputer via a 12-bit analogue-to-digital converter (DAS-16, Metrabyte) at a sampling frequency of 1000 Hz and stored sequentially for data analysis. Immediately after detecting an R wave, the computer algorithm tracked the pressure channel and identified the systolic and diastolic pressures as the highest and lowest values occurring before the next R wave.

Spectral analyses

Heart rate and blood pressure signals were submitted to coarse graining spectral analysis (CGSA). The details of this technique have been reported previously [18, 19]. The rationale for using CGSA, as opposed to conventional spectral analysis techniques, was to specifically identify and extract the fractal component of the HRV and BPV signals [1, 6, 20]. The residual estimates of low- and high-frequency power in the harmonic components are then no longer biased by the broadband, 'non-white' signal. This method therefore provides a more precise estimate of the harmonic contributions to low- and high-frequency power, an advantage that is particularly important in heart failure, a condition in which harmonic power is reduced and the probability of distortion across these frequencies is increased [11].

Seven minutes of data were acquired and searched for extra or missing beats that could corrupt the HRV analysis. These were edited and replaced by substitute RR intervals calculated by linear interpolation from adjacent cycles [18, 19]. Spectra were calculated as ensemble averages of 256-beat sequences taken from a time series containing approximately 400–500 beats.
Total spectral power ($P_T$) was divided into its fractal ($P_f$), low-frequency harmonic (0.0–0.15 Hz, $P_L$) and high-frequency harmonic (0.15–0.50 Hz, $P_H$) components, with total harmonic power ($P_{TH}$) comprising the sum of $P_L$ and $P_H$. Because the PNS is responsible for generating high-frequency power [21], the ratio ($P_H/P_T$) was used to estimate the vagal contribution to total spectral power and termed the parasympathetic indicator. The ratio, $P_L/P_H$, generally considered to be an index of SNS activity [1, 21] or 'sympathovagal balance' [3], was termed the SNS indicator in the present study.

The fractal component was plotted in a log power versus log frequency plane with $\beta$ estimated as the slope of the linear regression of this $1/f^\beta$ plot. The linear regression was calculated for Fourier components from 2.5% of Nyquist frequency to the point corresponding to 0.3 Hz. Subsequently, the regression upper limit was expanded to the end of the Fourier components (depending on linearity of decay beyond 0.3 Hz), and adopted the regression slope when the root mean square of the error showed minimal value [1, 6, 22].

Beat-to-beat values of systolic blood pressure were aligned sequentially to construct a 'systogram', and the mean RR-interval was used to obtain an equally spaced time series for BPV. CGSA was performed on these values for systolic blood pressure, using the same algorithms as for HRV. $P_f$, $P_L$ and $P_H$ components of BPV were derived from this analysis.

**Statistical analysis**

Mean values $\pm$ SEM are reported throughout. For comparisons of heart rate and blood pressure variability within subject groups, normally distributed data were submitted to paired $t$-tests, whereas non-normally distributed data were submitted to the non-parametric Mann–Whitney rank sum test (SigmaStat, Jandel Corp. 1994, San Rafael, CA, U.S.A.). Statistical significance was accepted if $P < 0.05$.

**RESULTS**

**HRV**

Subjects with heart failure had a significantly shorter RR-interval (higher heart rate) than healthy subjects (824 $\pm$ 37 ms as compared with 960 $\pm$ 37 ms in normal subjects; $P < 0.005$) and a marked reduction in all harmonic aspects of their HRV (Table 1; Figure 1). Mean values for $P_T$ were 922 ms$^2$ in the heart failure patients and 1736 ms$^2$ in normal subjects ($P < 0.03$). Similar reductions in spectral power were observed for $P_L$ ($P < 0.02$) and $P_H$ ($P < 0.004$). Compared with healthy subjects, the PNS indicator ($P_H/P_T$) was reduced ($P < 0.006$). There was a trend towards an increased SNS indicator ($P_L/P_H$) in the heart failure group, but this did not achieve significance ($P = 0.15$), owing to the considerable variation in this ratio in individual subjects within these two groups.

The fractal component of HRV in CHF patients was less than half the value of normal subjects when represented in absolute units ($P_f$) ($P = 0.06$), but there was no difference between the two groups in the percentage of $P_T$ with fractal characteristics (approximately 65–70%). The slope $\beta$ of the $1/f^\beta$ relationship was significantly steeper in heart failure (1.40 $\pm$ 0.08) (Table 1) than in normal subjects (1.14 $\pm$ 0.05; $P < 0.05$).

**BPV**

There was no significant difference between heart failure and healthy subjects in mean values for rest-

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**Table 1. Comparison of spectral indices for heart rate and blood pressure variability between CHF patients and normal subjects.** Values shown represent means $\pm$ SEM; NS, not significant.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Heart rate variability</th>
<th>Blood pressure variability</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>CHF ($n = 20$)</td>
<td>Normal ($n = 20$)</td>
</tr>
<tr>
<td>$P_T$ (ms$^2$)</td>
<td>922 $\pm$ 237</td>
<td>1736 $\pm$ 392</td>
</tr>
<tr>
<td>$P_L$ (ms$^2$)</td>
<td>232 $\pm$ 76</td>
<td>428 $\pm$ 106</td>
</tr>
<tr>
<td>$P_H$ (ms$^2$)</td>
<td>49 $\pm$ 14</td>
<td>236 $\pm$ 80</td>
</tr>
<tr>
<td>$P_{TH}$ (ms$^2$)</td>
<td>281 $\pm$ 79</td>
<td>664 $\pm$ 181</td>
</tr>
<tr>
<td>$P_f$ (ms$^2$)</td>
<td>640 $\pm$ 164</td>
<td>1071 $\pm$ 253</td>
</tr>
<tr>
<td>$P_L/P_T$ (%)</td>
<td>70 $\pm$ 2.7</td>
<td>66 $\pm$ 3.6</td>
</tr>
<tr>
<td>$P_L/P_H$</td>
<td>0.06 $\pm$ 0.01</td>
<td>0.13 $\pm$ 0.02</td>
</tr>
<tr>
<td>$P_H/P_T$</td>
<td>32.57 $\pm$ 18.7</td>
<td>5.33 $\pm$ 2.63</td>
</tr>
<tr>
<td>$P_L/P_{TH}$</td>
<td>0.04 $\pm$ 0.06</td>
<td>0.66 $\pm$ 0.04</td>
</tr>
<tr>
<td>$P_H/P_T$</td>
<td>0.19 $\pm$ 0.03</td>
<td>0.24 $\pm$ 0.03</td>
</tr>
<tr>
<td>Slope ($\beta$)</td>
<td>1.40 $\pm$ 0.08</td>
<td>1.14 $\pm$ 0.05</td>
</tr>
</tbody>
</table>
ing systolic blood pressure (128 ± 4 mmHg compared with 123 ± 3 mmHg) or any of the harmonic spectral power indices for systolic BPV (Figure 2). Absolute and relative fractal power (77% and 69%) and the slope β of the fractal component of BPV were also similar in the two groups (Table 1).

Within-group comparisons of HRV and BPV

In healthy subjects, the PNS indicator (P_{II}/P_{III}) for HRV was higher than the corresponding value for BPV (P < 0.0006), yet the contribution of P_{II} to P_{III} was significantly less for heart rate than for blood pressure (P < 0.0004) (Figure 3). The slope β of the 1/f^β relationship for BPV was significantly steeper than the slope β for HRV (P < 0.0001), indicating that, in healthy subjects, the blood pressure signal is much less complex than the heart rate signal (Table 2; Figure 4).

In contrast, there was no significant difference in the harmonic components of BPV and HRV in heart failure. In particular, the ratios P_{II}/P_{III}, P_{II}/P_{III} and P_{II}/P_{III} for BPV and HRV were similar in these patients (Figure 3). As was the case in healthy subjects, the slope β of the 1/f^β for BPV was significantly steeper that the slope β of the 1/f^β relationship for HRV (P < 0.006), indicating that the HRV signal remains more complex than the BPV signal, despite the significant reduction in harmonic spectral power in this condition (Table 2; Figure 4).

DISCUSSION

Spectral analysis of HRV provides a quantitative estimate of the modulation of heart rate by the
Fractal spectra in heart failure

autonomic nervous system [3, 23–25]. Respiration is the primary rhythmic stimulus to harmonic power at frequencies between 0.15 and 0.50 Hz and is effected by efferent vagal discharge. Indeed, most of the variability in the low (0.0 Hz–0.15 Hz) and high (0.15 Hz–0.50 Hz) frequency ranges of the power spectrum in healthy humans appears to be a function of vagal input, as it is attenuated or abolished by atropine [2], but there remains a significant residual component of HRV, particularly in the low frequency range, that is mediated by oscillations in efferent SNS activity. These observations relate primarily to the generation of harmonic power, which comprises the minority of total spectral power in normal subjects. The sources of fractal power, and hence the overall complexity of the heart rate signal, have not been as clearly defined.

In the present study, the harmonic, non-harmonic and $1/f^\beta$ characteristics of heart rate and blood pressure variability were examined in men with normal or impaired left ventricular systolic function during supine rest and spontaneous respiration. Between 65 and 80% of the power in the heart rate and blood pressure spectra in both groups displayed non-harmonic characteristics. The fractal component of HRV exhibited power-law scaling, with slopes ($1/f^\beta$) of 1.14 in normal subjects and 1.40 in those with systolic dysfunction ($P < 0.05$), indicating reduced complexity of the heart rate signal in these patients. In contrast, the slope, $\beta$, of the fractal component of systolic BPV was similar in healthy and heart failure subjects. In both groups, the non-harmonic component of BPV was less complex than the non-harmonic component of HRV, in that the slope $\beta$ for BPV was significantly greater than the corresponding value for HRV. Observations in normal subjects confirm previous reports, in younger subjects, of fractal components having power-law scaling, with spectral exponents ($\beta$) of approximately 1.0 for HRV and 2.3 for BPV [1, 6, 9]. The present findings for HRV and BPV in patients with impaired left ventricular function are novel and, when considered in the context of the entire power spectrum, indicate that the autonomic nervous system contributes to the generation of complexity in the $1/f$ component of these signals in both healthy and heart failure subjects.

CHF is a condition of PNS withdrawal and SNS activation [12, 26–30]. Both time- and frequency-domain indices of HRV are reduced, as compared with normal subjects, and there is an inverse relationship between such variability and prognosis [10, 28, 31]. The attenuation of spectral power in heart failure has been attributed to both neural and mechanical mechanisms. Neural factors include a reduction in the baroreflex regulation of heart rate, as a result of one or more of baroreceptor afferent nerve desensitization, altered central integration or reduced sino-atrial responsiveness to the neurotransmitters acetylcholine and noradrenaline [29, 30, 33, 34]. Stretch of the sino-atrial node, which may occur in patients with high right atrial pressures, also reduces the high-frequency component of heart rate

![Table 2. Within-group comparisons of harmonic and fractal components of heart rate and blood pressure variability. Values shown represent means ± SE; NS, not significant.](image)

<table>
<thead>
<tr>
<th>Variable</th>
<th>CHF (n = 16)</th>
<th>Normal subjects (n = 16)</th>
</tr>
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<tbody>
<tr>
<td></td>
<td>HRV</td>
<td>BPV</td>
</tr>
<tr>
<td>Pr/Pt (%)</td>
<td>71 ± 3.1</td>
<td>76 ± 3.9</td>
</tr>
<tr>
<td>Pr/Pf</td>
<td>0.03 ± 0.01</td>
<td>0.04 ± 0.02</td>
</tr>
<tr>
<td>Pr/Pf</td>
<td>0.76 ± 0.05</td>
<td>0.78 ± 0.06</td>
</tr>
<tr>
<td>Pr/Pf</td>
<td>0.20 ± 0.03</td>
<td>0.18 ± 0.03</td>
</tr>
<tr>
<td>Slope ($\beta$)</td>
<td>1.31 ± 0.09</td>
<td>2.31 ± 0.15</td>
</tr>
</tbody>
</table>
variability, even after vagotomy, suggesting that mechanical factors also contribute to the loss of spectral power observed in CHF [35]. Ponikowski et al. [36] have identified and characterized a discrete very low frequency rhythm (<0.04 Hz) in the majority of their patients with chronic heart failure, and have hypothesized that these oscillations arise from enhanced peripheral chemoreceptor sensitivity.

Because spectral power is attenuated across virtually all frequencies [14–16], we hypothesized that any impairment in the modulation of heart rate by the autonomic nervous system would affect not only the harmonic oscillations generated by PNS and SNS input, but also non-harmonic spectral power and the more complex 1/f components of the heart rate signal [11]. Our findings in the present study lead us to conclude, firstly, that the complexity of the heart rate signal is reduced in heart failure, second, that complexity can be dissociated from spectral (fractal) power in this condition and, third, that parasympathetic withdrawal contributes to this loss of complexity in the heart rate signal.

The latter proposal is based on several lines of evidence. Firstly, the frequency-domain characteristics of the subjects with impaired ventricular systolic function parallel responses to the orthostatic stress of lower-body negative pressure in healthy subjects: an acute loss of complexity in the HRV signal (i.e. a steeper slope for $\beta$) and a corresponding decrease in the ratio $P_{HR}/P_{r}$, which is an index of the contribution of the PNS to harmonic power [1, 6]. These effects are mediated by parasympathetic withdrawal and cardiac sympathetic activation as reflex responses to arterial and cardiopulmonary baroreceptor unloading during this manoeuvre [37, 38]. Mechanical factors, as proposed by Horner et al. [35], cannot be invoked, as atrial stretch is reduced by lower-body negative pressure. Second, the PNS accounts for the high degree of complexity in the non-harmonic component of HRV in subjects with normal ventricular function. Both vagal blockade with atropine [39] and vagal withdrawal during exercise [40] increase $\beta$ significantly, whereas $\beta$-adrenergic receptor blockade with propranolol does not affect the slope of the $1/f^\beta$ relationship [9]. These observations therefore provide evidence that altered vagal modulation of heart rate can influence the broad-band fractal component of the HRV spectrum, in addition to its harmonic components.

Third, with age, there is a decline in the parasympathetic control of heart rate via the arterial baroreceptor reflex [41], and a decrease in the complexity of the heart rate signal: $\beta$ rises steadily between the third and tenth decade of life [5, 42].

This high degree of complexity of heart rate variability in normal subjects implies that neural mechanisms modulating heart rate are well regulated and tightly controlled under resting conditions [1, 6, 43]. During graded lower-body negative pressure, oscillatory patterns in the time-domain representations of pulse interval are damped, then disappear, resulting in a ‘flat-line’ appearance; $\beta$ steepens to a slope of 3.0; i.e., these HRV spectra come to resemble those of subjects with severe heart failure. At this point, some subjects experience presyncope or syncope. A similar reduction in the complexity of heart rate variability in CHF may well compromise the ability of such patients to respond rapidly, flexibly and appropriately when faced with emotional or physical threats to cardiovascular homeostasis [44]. Neural responses to tilt, for example, are attenuated, relative to the severity of heart failure [25].

Experiments in dogs [20, 44], and in humans [6, 44a] indicate that the variability of blood pressure also has fractal characteristics, in that this signal displays self-similarity [19] and can be viewed over a range of time scales, with information encoded over both the short and the long term. As with heart rate, the slope of the $1/f^\beta$ relationship can be derived as an index of the complexity of blood pressure variation. The complexity of the blood pressure signal is reduced markedly (but not abolished completely) if the tonic inhibition of sympathetic vasoconstrictor outflow is removed by total sino-aortic and cardiopulmonary baroreceptor denervation [44].

In a previous study in healthy normal males [6], the slope for the $1/f^\beta$ relationship for BPV was 2.31 during supine rest. Unlike the slope $\beta$ for heart rate, this value for BPV did not increase during the acute stimulus of lower-body negative pressure [6]. These observations indicate that the BPV signal is less complex than the HRV signal, owing to the absence of direct modulation by the PNS. Within-group comparisons of the vagal contributions to the harmonic components of HRV and BPV spectra in normal subjects provide support for this concept: BPV ratios of $P_{L}/P_{HP}$ and $P_{L}/P_{r}$ were significantly higher, whereas the PNS indicator ($P_{HR}/P_{r}$) was significantly lower, as compared with the corresponding HRV ratios.

If the lack of direct PNS modulation renders the blood pressure signal less complex than the heart rate signal in normal subjects, then any impairment
of vagal tone in CHF patients should have little, if any, impact on their blood pressure variability, and the blood pressure signal of subjects with normal and impaired ventricular function should display similar harmonic and non-harmonic characteristics. Indeed, there was no difference in either the (P_L/P_H, P_T/P_Y) or the non-harmonic (P_F or P_T/P_Y) components of BPV between normal and heart failure subjects.

That congestive failure reduces the variability and complexity of the heart rate signal, without affecting the variability or the complexity of the blood pressure signal, indicates that the neural regulation of these two variables is independent, and that impairment of such regulation in CHF is relatively selective for heart rate. Prior observations in normal subjects are consistent with the first of these conclusions, in that the contrasting effects of graded lower-body negative pressure on values of $\beta$ for HRV and BPV indicate that these two systems must be regulated independently in response to this brief stimulus [6]. The complexity of heart rate and blood pressure signals provides information as to the relative efficiency with which heart rate and blood pressure maintain cardiovascular homeostasis in normal subjects. Whereas heart rate is regulated quickly, on a beat-to-beat basis, primarily by the PNS, BPV arises from the interaction between several factors, including changes in stroke volume, the rhythmicity of baroreceptor-mediated oscillations in sympathetic nerve traffic to blood vessels [44], the efficiency of neural effector transduction, humoral factors and/or intrinsic vascular smooth-muscle properties. Many of these mechanisms behave as 'low-pass' filters. The similar complexity of BPV in subjects with normal and impaired ventricular function is consistent with the concept that mechanisms responsible for regulating this variable (e.g. the arterial baroreflex control of sympathetic nerve traffic [44, 45, 46]) are not impaired significantly in heart failure.

The principal limitation to this study is that our methods for computing the variability and complexity of heart rate precludes the study of many patients with end-stage, severe congestive failure, whose heart rate is invariable. It should also be noted that subjects with heart failure were exposed to digoxin and/or angiotensin-converting inhibitor therapy. However, because the effect of these medications would be to enhance HRV [33, 47], they cannot account for the significant decrease in harmonic and non-harmonic spectral power in CHF, but may explain the high residual fractal power in some of these subjects (Table 1). Relative preservation of fractal power may also contribute to the discrete very-low-frequency rhythm referred to earlier [36].

In CHF, heart rate is less variable and the heart rate signal less complex than in subjects with normal ventricular function. The loss of complexity most likely reflects a reduction in parasympathetic modulation of heart rate. As a result, the heart rate signal comes to resemble the blood pressure signal, which displays similar variability and complexity in heart failure and normal subjects. Bigger et al. [7] have recently provided compelling evidence that the slope of the spectral exponent derived from ambulatory monitoring of the ECG has associations with mortality end-points after acute myocardial infarction that are comparable with or superior to the predictive value of power loss in conventional spectral bands. In patients with a slope steeper than 1.372, the 3 year mortality rate was 41.5%, in contrast to 14.2% in patients whose slope was equal to or less than that value. The prevalence of systolic dysfunction in these subjects was not reported. The average slope of this relationship in our heart failure group was 1.40. This reduction in heart rate complexity may well have adverse prognostic implications for these patients with heart failure.

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