NEW COMPUTATIONAL APPROACHES TO ANALYSIS OF INTERBEAT INTERVALS IN HUMAN SUBJECTS

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We investigate the Markov nature, Cascade of information from large time scale to small scale and extended self-similarity properties of the beat to beat fluctuations of healthy subjects as well as those with congestive heart failure. To check the Markov nature, we use a novel inverse method that utilizes a set of data to construct a simple equation that governs the stochastic process for which the data have been measured, hence enabling us to reconstruct the stochastic process. The inverse method provides a novel technique for distinguishing the two classes of subjects in terms of a drift and a diffusion coefficients which behave completely differently for the two classes of subjects. To investigate the cascade of information from large to small time scales we also analyze the statistical properties of interbeat intervals cascade by considering the joint probability distribution \( P(\Delta x_2, \tau_2; \Delta x_1, \tau_1) \) for two interbeat increments \( \Delta x_1 \) and \( \Delta x_2 \) of different time scales \( \tau_1 \) and \( \tau_2 \). We present evidence that the conditional probability distribution \( P(\Delta x_2, \tau_2|\Delta x_1, \tau_1) \) may obey a Chapman-Kolmogorov equation. The corresponding Kramers-Moyal (KM) coefficients are evaluated. It is shown that while the first and second KM coefficients, i.e., the drift and diffusion coefficients, take on well-defined and significant values, the higher-order coefficients in the KM expansion are very small. As a result, the joint probability distributions of the increments in the interbeat intervals obey a Fokker-Planck equation. Finally we analyze the extended self-similarity (ESS) in the beat-to-beat fluctuations in the heart rates of healthy and congestive heart failure subjects. The data were measured both during the day and at night, and for both male and female subjects with a wide range of ages. The proposed methods provide the novel techniques for distinguishing the two classes of subjects in terms of the drift and diffusion coefficients, intermittency exponents which behave differently for two classes of the subjects, namely, healthy subjects and those with congestive heart failure.

1. Introduction

Physiological data and time series are generated by complex self-regulating systems that process inputs with a broad range of characteristics. 1−3 Many physiological time series seem to be highly chaotic, represent nonstationary data, and fluctuate in an irregular and complex manner. A hypothesis advocated by some is that the seemingly chaotic structure of physiological time series arise from external and intrinsic perturbations that push the system away from a homeostatic set point. An alternative hypothesis is that the fluctuations are, at least in part, due to the system’s underlying dynamics.

It has also been suggested that physiological time series may possess fractal and self-similar properties. However, until recently, the analysis of the fractal properties of such fluctuations was restricted to computing their characteristics based on the second moment of the data, such as the power spectrum and the two-point autocorrelation function. These analyses indicate that the fractal behavior of healthy, free-running physiological systems is often characterized by \( 1/f \)-like scaling of the power spectra over a wide range of time scales. 4−8 A time series that exhibits such power-law long-range correlations and is homogeneous (i.e., different parts of the series have identical statistical properties) is called a monofractal series. Many physiological time series, however, are heterogeneous in the sense that different parts of the series are characterized by distinct statistical properties. In addition, there is also some evidence that physiological dynamics may exhibit nonlinear properties. 9−15 Such features are often associated with multifractal behavior, i.e., the presence of long-range power-law correlations in the higher moments of the time series, which are nonlinear functions of the scaling exponents of the second
moment. However, up until recently, robust demonstration of multifractality of nonstationary time series had been hampered by problems related to significant bias in the estimate of the singularity spectrum, due to diverging negative moments of the time series. Moreover, the classical approaches, based on the box-counting technique and structure function formalism, fail when a fractal function is composed of a multifractal singular part embedded in regular polynomial behavior. Such problems were recently addressed by using a wavelet-based multifractal formalism, which demonstrated that healthy human heartbeat dynamics exhibits even higher complexity than previously expected from the monofractal, 1/f scaling, and is characterized by a broad multifractal spectrum.

Among physiological time series, the study of the statistical properties of heartbeat interval sequences has attracted much recent attention. Extensive analysis of interbeat interval variability has been carried out, as it represents an important quantity for elucidating the possibly nonhomeostatic physiological variability. Moreover, (i) the heart rate is under direct neuroautonomic control; (ii) interbeat interval variability is readily measured by noninvasive means, and (iii) analysis of the heart rate dynamics may provide important practical diagnostic and prognostic information. Figure 1 shows cardiac interbeat time series (the output of a spatially- and temporally-integrated neuroautonomic control system) for healthy subjects and those with congestive heart failure (CHF), exhibiting erratic fluctuations and patchiness. In the conventional approaches to analyzing such data, it is assumed that there is no meaningful structure in the apparent noise and, therefore, one should not expect to gain any understanding of the underlying system through the study of such fluctuations. Therefore, the fluctuations are usually ignored in conventional studies that focus on averaged quantities. In fact, such fluctuations are often labeled as noise to distinguish them from the true time series of interest.

However, by adapting and extending methods developed in modern statistical physics and nonlinear dynamics, physiological fluctuations shown in Figure 1 can be shown to exhibit an unexpected hidden scaling structure. Moreover, the dynamics of the fluctuations and the associated scaling features may be shown to change with pathological perturbations. These discoveries have raised the possibility of identifying the origin of such temporal structures and their alterations with a disease, (i) may elucidate certain basic aspects of heart rate control mechanisms, and (ii) may have potential for clinical monitoring.

In this review we describe new computational approaches, based on new theoretical concepts, for analyzing physiological time series, and in particular the beat-to-beat fluctuations in the heart rates of human subjects. The theoretical concepts are based on the possible Markov properties of the time series, a cascade of information from large time scales to small ones that are built based on the increments in the time series, and the extended self-similar properties of the beat-to-beat fluctuations of healthy subjects as well as those with CHF. We describe a recently-developed novel method that utilizes a set of data (e.g., time series) for a given phenomenon that contains a degree of stochasticity, and numerically constructs a relatively-simple equation that governs the phenomenon. In addition to being highly accurate, the method is quite general. It, (i) is capable of providing a rational explanation for complex features of the phenomenon under study, and (ii) requires no scaling feature or assumption. As an example, the method is applied to analyze cardiac interbeat intervals.

In addition, we describe new methods for computing the Kramers-Moyal (KM) coefficients for the increments of interbeat intervals fluctuations, $\Delta x(\tau) = x(t+\tau) - x(t)$. Here, $\Delta x$ is the interbeat increments defined as, $\Delta x \equiv \Delta x/\sigma_x$, where $\sigma_x$ is the standard deviations of the increments in the interbeats data. The significance of computing the KM coefficients is that, whereas the first and second KM coefficients that represent, respectively, the drift and diffusion coefficients in a Fokker-Planck (FP) equation, have well-defined values, the third- and fourth-order KM coefficients may be small. If so, a FP evolution equation can be numerically constructed for the probability density function (PDF) $P(\Delta x, \tau)$ which, in turn, is used to gain information on the evolution of the PDF as a function of the time scale $\tau$ (see also Jafari et al. for another interesting and carefully-analyzed example of the application of the method to stochastic phenomena). Finally, we address the question of whether there are characteristic differences between the extended self-similar properties of healthy subjects and those with CHF.

We show that the application of the methods that are described in this review may have the potential for leading to a novel diagnostic tool for distinguishing healthy subjects from those with CHF.

### A Regeneration Method

We begin by describing the steps that lead to the numerical construction of a stochastic equation that describes the phenomenon, based on the (stochastic) data set, which is then utilized to reconstruct the original time series. Two basic steps are involved in the numerical analysis of the data and their reconstruction:

1. The data are first examined to see whether they follow a Markov chain and, if so, estimate the Markov time scale $t_M$. As is well-known, a given process with a degree of stochasticity may have a finite or an infinite Markov time scale, which is the minimum time interval over which the data can be considered as a Markov process.

2. To determine the Markov scale $t_M$, we note that a complete
characterization of the statistical properties of stochastic fluctuations of a quantity \( x(t) \) requires the numerical evaluation of the joint PDF \( P_n(x_1, t_1; \ldots; x_n, t_n) \) for an arbitrary \( n \), the number of the data points in the time series \( x(t) \). If the time series \( x(t) \) is a Markov process, an important simplification can be made as \( P_n \), the \( n \)-point joint PDF, is generated by the product of the conditional probabilities \( P(x_{i+1}, t_{i+1} | x_i, t_i) \), for \( i = 1, \ldots, n - 1 \). A necessary condition for \( x(t) \) to be a Markov process is that the Chapman-Kolmogorov (CK) equation, \(^36\)

\[
P(x_2, x_3 | x_1, t_1) = \int d(x_3) P(x_2, x_3 | x_3, t_3) P(x_3, x_1 | x_1, t_1) ,
\]

(1)

should hold for any value of \( t_3 \) in the interval \( t_2 < t_3 < t_1 \). One should check the validity of the CK equation for various \( x_1 \) by comparing the directly-computed conditional probability distributions \( P(x_2, x_3 | x_1, t_1) \) with the ones computed according to right side of Eq. (1). The simplest way to determine \( t_M \) for stationary or homogeneous data is the numerical computation of the quantity, \( S = [P(x_2, x_3 | x_1, t_1) - \int dx_3 P(x_2, x_3 | x_3, t_3) P(x_3, x_1 | x_1, t_1)] \), for given \( x_1 \) and \( x_2 \), in terms of, for example, \( t_3 - t_1 \) (taking into account the possible numerical errors in estimating \( S \)). Then, \( t_M = t_3 - t_1 \) for that value of \( t_3 - t_1 \) for which \( S \) vanishes or is nearly zero (achieves a minimum).

(2) Numerical construction of an effective stochastic equation that describes the fluctuations of the quantity \( x(t) \), representing the time series, constitutes the second step. The CK equation yields an evolution equation for the PDF \( P(x, t) \) across the scales \( t \). The CK equation, when formulated in differential form, yields a master equation which takes the form of a FP equation:

\[
\frac{d}{dt} P(x, t) = \left[ -\frac{\partial}{\partial x} D^{(1)}(x, t) + \frac{\partial^2}{\partial x^2} D^{(2)}(x, t) \right] P(x, t) .
\]

(2)

The drift and diffusion coefficients, \( D^{(1)}(x, t) \) and \( D^{(2)}(x, t) \), are computed directly from the data and the moments \( M^{(k)} \) of the conditional probability distributions:

\[
D^{(k)}(x, t) = \frac{1}{k!} \lim_{\Delta t \to 0} M^{(k)} ,
\]

(3)

\[
M^{(k)} = \frac{1}{\Delta t} \int dx' (x' - x)^k P(x', t + \Delta t | x, t) .
\]

(4)

Note that the above FP formulation is equivalent to the following Langevin equation: \(^28\)

\[
\frac{d}{dt} x(t) = D^{(1)}(x) + \sqrt{D^{(2)}(x)} f(t) ,
\]

(5)

where \( f(t) \) is a random force with zero mean and Gaussian statistics, \( \delta \)-correlated in \( t \), i.e., \( \langle f(t)f(t') \rangle = 2\delta(t - t') \). We note that the numerical reconstruction of a stochastic process does not imply that the data do not contain any correlations, or that the above formulation ignores the correlations.

Equation (5) enables us to reconstruct a stochastic time series \( x(t) \), which is similar to the original one in the statistical sense. The stochastic process is regenerated by iterating Eq. (5) which yields a series of data without memory. To compare the regenerated data with the original ones, we must take the temporal interval in the numerical discretization of Eq. (5) to be unity (or renormalize it to unity). However, the Markov time is typically greater than unity. Therefore, we correlate the data over the Markov time scale \( t_M \), for which there are a number of methods. \(^27,28,37-39\) A new technique that we have used in our own studies, which we refer to as the kernel method, is one according to which one considers a kernel function \( K(u) \) that satisfies the condition that,

\[
\int_{-\infty}^{\infty} K(u)du = 1 ,
\]

(6)

such that the data are determined, or reconstructed, by

\[
x(t) = \frac{1}{nh} \sum_{i=1}^{n} x(t_i) K \left( \frac{t - t_i}{h} \right) ,
\]

(7)

where \( h \) is the window width. For example, one of the most useful kernels is the standard normal density function, \( K(u) = (2\pi)^{-1/2} \exp(-\frac{1}{2}u^2) \). In essence, the kernel method represents the data as a sum of ‘bumps’ placed at the observation points, with its function determining the shape of the bumps, and its window width \( h \) fixing their width. It is evident that, over the scale \( h \), the kernel method correlates the data.

FIG. 1. Interbeats fluctuations of healthy subjects (top), and those with congestive heart failure (bottom).
3. Analysis of Fluctuations in Human Heartbeats

To show how the above reconstruction method is used in practice, and to demonstrate its utility, the method has been applied to reconstruction of the fluctuations in the human heartbeats of both healthy and ill subjects by taking \( h \approx t_M \). Recent studies\(^6\);\(^26\);\(^40\)–\(^43\) reveal that under normal conditions, beat-to-beat fluctuations in the heart rates might display extended correlations of the type typically exhibited by dynamical systems far from equilibrium. It has been argued,\(^40\);\(^41\) for example, that the various stages of sleep might be characterized by extended correlations of heart rates separated by a large number of beats. While the existence of extended correlations is an interesting and important result, we show that the Markov time scale \( t_M \), and the drift and diffusion coefficients of the interbeat fluctuations of healthy subjects and those with CHF help one to better distinguish the two classes of subjects, particularly at the early stages of the disease, as these quantities have completely different behaviour.

Both daytime (12:00 pm to 18:00 pm) and nighttime (12:00 am to 6:00 am) heartbeat time series of healthy subjects, and the daytime records of patients with CHF have been analyzed by this method.\(^45\);\(^46\) The data base includes 10 healthy subjects (7 females and 3 males with ages between 20 and 50, and average age of 34.3 years), and 12 subjects with CHF (3 females and 9 males with ages between 22 and 71, and average age of 60.8 years). Figure 1 presents the typical data.

As the first step, the Markov time scale \( t_M \) of the data is computed. From the daytime data for healthy subjects the values of \( t_M \) are computed to be (all the values are measured in units of the average time scale for the beat-to-beat times of each subject), \( t_M = 3, 3, 3, 1, 2, 3, 3, 2, 3 \).
and 2. The corresponding results for the nighttime records are, $t_M$ are 3, 3, 1, 3, 3, 2, 3, 3, 2 and 3, respectively, comparable to those for the daytime. On the other hand, for the daytime records of the patients with CHF, the computed Markov time scales are, $t_M = 151, 258, 760, 542, 231, 257, 864, 8, 366, 393, 385,$ and 276. Therefore, the healthy subjects are characterized by $t_M$ values that are much smaller than those of the patients with CHF. Thus, one has an unambiguous quantity for distinguishing the two classes of patients.

Next, the validity of the CK equation for describing the phenomenon is checked for several $x_1$ triplets by comparing the directly-computed conditional probability distributions $P(x_2, t_2|x_1, t_1)$ with the ones computed according to right side of Eq. (1). Here, $x$ is the interbeat and for all the samples we define, $x \equiv (x - \bar{x})/\sigma$, where $\bar{x}$ and $\sigma$ are the mean and standard deviations of the interbeats data. In Figure 2, the two PDFs, computed by the two methods, are compared. Assuming the statistical errors to be the square root of the number of events in each bin, the two PDFs are statistically identical.

The corresponding drift and diffusion coefficients, $D^{(1)}(x)$ and $D^{(2)}(x)$, are displayed in Figure 3, demonstrating that, in addition to the Markov time scale $t_M$, the two coefficients provide another important indicator for distinguishing the ill from the healthy subjects: For the healthy subjects the drift $D^{(1)}$ and the diffusion coefficients $D^{(2)}(x)$ follow (approximately) respectively, linear and quadratic functions of $x$, whereas the corresponding coefficients for patients with CHF follow (approximately) third- and fourth-order equations in $x$. Thus, for the healthy subjects,

$$D^{(1)}(x) = -0.12x,$$

$$D^{(2)}(x) = 0.05 - 0.042x + 0.07x^2,$$

whereas for the patients with CHF,

$$D^{(1)}(x) = -0.0026x - 0.0018x^2 - 0.0007x^3,$$

$$D^{(2)}(x) = 0.0006 - 0.0007x + 0.0005x^2 + 0.0003x^3 + 0.0002x^4.$$

Note that the final results for the Langevin equation are the same as those obtained by Knuusela\textsuperscript{44} by a different method. For other data bases measured for other patients, the functional dependence of $D^{(1)}$ and $D^{(2)}(x)$ would be the same, but with different numerical coefficients. The order of magnitude of the coefficients would be the same for all the healthy subjects, and likewise for those with CHF (see also Wolf \textit{et al.}\textsuperscript{20}). Moreover, if one analyzes different parts of the time series separately, one finds, (1) practically the same Markov time scale for different parts of the time series, but with some differences in the numerical values of the drift and diffusion coefficients, and (2) that the drift and diffusion coefficients for different parts of the time series have the same functional forms, but with different coefficients in equations such as (8)-(11). Hence, one can distinguish the data for sleeping times from those patients when they are awaken.

There is yet another important difference between the heartbeat dynamics of the two classes of subjects: Compared with the healthy subjects, the drift and diffusion coefficients for the patients with CHF are very small, reflecting, in some sense, their large Markov time scale $t_M$. Large Markov times $t_M$ imply longer correlation lengths for the data, and it is well-known that the diffusivity in correlated system is smaller than those in random ones. Hence, one may use the Markov time scales, and the dependence of the drift and diffusion coefficients on $x$, as well as their comparative magnitudes, for characterizing the dynamics of human heartbeats and their fluctuations, and to distinguish healthy subjects from those with CHF.

How accurate is the reconstruction method? Shown in Figure 4 is a comparison between the original time series $x(n)$ and those reconstructed by the Langevin equation [by, for example, using Eqs. (4), (8) and (9)] and the kernel method. While both methods generate series that look similar to the original data, the kernel method appears to better mimic the behavior of the original data. To demonstrate the accuracy of Eq. (7), Figure 5 compares the second moment of the stochastic function, $C_2(m) = \langle |x(0) - x(m)|^2 \rangle$, for both the measured and reconstructed data using the kernel method. The agreement between the two is excellent. However, it is well-known that such agreements is not sufficient for proving the accuracy of a reconstruction method. Hence, the accuracy of the reconstructed higher-order structure function, $S_n = \langle |x(t_1) - x(t_2)|^n \rangle$, which was also checked. It was found that the agreement between $S_n$ for the original and reconstructed time series for $n \leq 5$ is excellent, while the difference between higher-order moments of the two times series, which are related to the tails of the PDF of the $x$-increments, increases.

**The Cascade of Information from Large to Small Time Scales**

One may be argue that if long-range, nondecaying correlations do exist in the time series, then one cannot use the above reconstruction method for analyzing them because, as is well-known, if a time series represents a Markov process, the correlations in it decay exponentially, and long-range, nondecaying correlations cannot be represented by exponentially decaying correlations. Aside from the fact that even in such cases the method described above provides an unambiguous way of distinguishing healthy subjects from those with CHF, which we believe is more effective than simply analyzing the data to see what type of correlations may exist in the data,
we argue that the nondecaying correlations do not, in fact, pose any limitations to the fundamental ideas and concepts of the reconstruction method described above.

The reason is that, even if the above reconstruction method fails to describe long-range, nondecaying correlations in the data, one can still analyze the data based on an important result recently pointed out by Friedrich, Peinke, and co-workers,\textsuperscript{29,33,37,39} as well as by Jafari et al.\textsuperscript{28} They studied the evolution of the PDF of several stochastic properties of turbulent free jets, and rough surfaces. They pointed out that the conditional PDF of the increments of a stochastic field, such as the increments in the velocity field in the turbulent flow or heights fluctuations of the rough surface that were studied, satisfies the CK equation, even if the velocity field (or the height function) itself contains long-range, nondecaying correlations. This enabled them to derive a FP equation for describing the systems under study. Hence, one has a way of analyzing correlated stochastic time series or data in terms of the corresponding FP and CK equations. We now describe the conditions under which such a formulation can be utilized.

One computes the Kramers-Moyal (KM) coefficients for the increments of interbeat intervals fluctuations, \( \Delta x(\tau) = x(t + \tau) - x(t) \), rather than the time series \( x(t) \) itself. One then checks whether the first and second KM coefficients that represent, respectively, the drift and diffusion coefficients in the FP equation, have well-defined and finite values, while the third- and fourth-order KM coefficients are small. According to the Pawula’s theorem,\textsuperscript{37} the KM expansion,

\[
\frac{\partial}{\partial t} P(x, t | x_0, t_0) = \sum_{k=1}^{\infty} \left( -\frac{\partial}{\partial x} \right)^k \left[ D^{(k)}(x, t) P(x, t | x_0, t_0) \right],
\]

(12)
Fig. 8. The drift and diffusion coefficients $D^{(1)}(\Delta x)$ and $D^{(2)}(\Delta x)$ are estimated from the Eq. (3) for typical patients with heart failure, and follow linear and quadratic behavior, respectively.

Fig. 9. Generalized scaling analysis of a typical healthy subject. Structure functions $S_q$ are displayed versus $S_3$ in log-log scale.

can be truncated after the second (diffusive) term, provided that the third- and fourth-order coefficient $D^{(4)}$ vanish, or are very small compared with the first two coefficients. If so, which is often the case, then the KM expansion, Eq. (12), reduces to a FP evolution equation. In that case, the FP equation is numerically constructed (by computing the drift and diffusion coefficients) for the PDF $P(\Delta x, \tau)$ which, in turn, is used to gain information on evolution of the shape of the PDF as a function of the time scale $\tau$. In essence, if the first two KM coefficients are found to have numerically-meaningful values (i.e., not very small), while the third and higher coefficients are small (compared with the first two coefficients), the above reconstruction method - Eqs. (2)-(4) - are used for the increments of the time series, rather than the time series itself.

Therefore, carrying out the same type of computations described above, but now for the increments $\Delta x(t)$, the following results are computed for the healthy subjects,

$$D^{(1)}(\Delta x, \tau) = -0.03\Delta x - 0.0046,$$

$$D^{(2)}(\Delta x, \tau) = \left(0.01 + \frac{0.11}{\tau}\right)(\Delta x)^2 + \left(0.057 + \frac{0.287}{\tau}\right),$$

whereas for the patients with CHF we obtain,

$$D^{(1)}(\Delta x, \tau) = -0.013\Delta x - 0.0018,$$

$$D^{(2)}(\Delta x, \tau) = \left(0.005 + \frac{0.005}{\tau}\right)(\Delta x)^2 + \left(0.013 + \frac{0.066}{\tau}\right).$$

Estimates of the above coefficients are less accurate for large values of $\Delta x$. Also computed are the average of the coefficients $D^{(1)}$ and $D^{(2)}$ for the entire set of the healthy subjects, as well as those with CHF. Moreover, $D^{(4)}$ is about $\frac{1}{10}D^{(2)}$ for the healthy subjects, and about $\frac{1}{20}D^{(2)}$ for those with CHF. Therefore, the KM expansion can indeed be truncated beyond the second term, and the FP formulation is numerically justified.

Equations (13)-(16) state that the drift coefficients for the healthy subjects and those with CHF have the same order of magnitude, whereas the diffusion coefficients for the given $\tau$ and $\Delta x$ differ by about one order of magnitude. This points to a relatively simple way of distinguishing the two classes of the subjects. Moreover, the $\tau$-dependence of the diffusion coefficient for the healthy subjects is stronger than that of those with CHF (in the sense that the numerical coefficients of the $\tau^{-1}$ are larger for the healthy subjects). These are shown in Figures 7 and 8. Note also that these results are consistent with those presented earlier for the time series $x(t)$ itself, in terms of distinguishing the two classes of patients through their different drift and diffusion coefficients.
The strong \( \tau \)-dependence of the diffusion coefficient \( D^{(3)} \) for the healthy subjects indicates that the nature of the PDF of their increments \( \Delta x \) for given \( \tau \), i.e., \( P(\Delta x, \tau) \), is intermittent, and that its shape should change strongly with \( \tau \). However, for the patients with CHF the PDF is not so sensitive to the change of the time scale \( \tau \), hence indicating that the increments’ fluctuations for these patients is not intermittent. These results are completely compatible with the recent discoveries that the interbeat fluctuations for healthy subjects and those with CHF have fractal and multifractal properties, respectively.18

The Extended Self-Similarity of Interbeat Intervals in Human Subjects

In this section, we describe another computational method for distinguishing healthy subjects from patients with CHF. The method is based on the concept of extended self-similarity (ESS) of a time series. This concept is particularly useful if the time series for interbeat fluctuations (or other types of time series) do not exhibit scaling over a broad interval, which is often the case. In such cases, the time interval in which the structure functions of the time series, i.e.,

\[
S_q(\tau) = \langle |x(t+\tau) - x(t)|^q \rangle ,
\]

behaves as

\[
S_q(\tau) \sim \tau^{\zeta_q} ,
\]

is small, in which case the existence of scale invariance in such data can be questioned. The possibility of the existence of scale invariance in such data can be checked via the concept of ESS.

The ESS is a powerful tool for checking non-Gaussian properties of data,48,49 and has been used extensively in research on turbulent flows. Indeed, when analyzing the interbeat time series for human subjects, one can, in addition to the \( \tau \)-dependence of the structure function, compute a generalized form of scaling using the ESS concept. In many cases, when the structure functions \( S_q(\tau) \) are plotted against a structure function of a specific order, say \( S_3(\tau) \), an extended scaling regime is found according to,48,49

\[
S_q(\tau) \sim S_3(\tau)^{\zeta_q} ,
\]

Clearly, meaningful results are restricted to the regime where \( S_3 \) is monotonic. For any Gaussian process the exponents \( \zeta_q \) follow a simple equation,

\[
\zeta_q = \frac{1}{3} q .
\]

Therefore, systematic deviation from the simple scaling relation, Eq. (20), can be interpreted as deviation from Gaussianity. An additional remarkable property of the ESS is that it holds rather well even in situations when the ordinary scaling does not exit, or cannot be detected due to small scaling range (which is the case for the data analyzed here).

Using the ESS concept, we analyzed50 the fluctuations in human heartbeat rates of healthy subjects and those with CHF, analyzed earlier with the reconstruction method. The results are shown in Figure 9. For a typical healthy subject an improved scaling behavior of the time series is indicated by the ESS. In Figure 10 the computed scaling exponents \( \zeta_q \) of the structure functions are plotted against the order \( q \). A mono-fractal time series corresponds to linear dependence of \( \zeta_q \) on \( q \), whereas for a multifractal time series \( \zeta_q \) depends nonlinearly on \( q \). The constantly changing curvature of the computed \( \zeta_q \) for the healthy subjects suggests multifractality of their corresponding time series. In contrast, \( \zeta_q \) is essentially linear for the patients with CHF, indicating mono- or simple fractal behavior (see also Ivanov et al.18).

It is well-known that the moments with \( q < 1 \) and \( q > 1 \) are related, respectively, to the frequent and rare events in the time series.48,49 Thus, for the data considered here one may also be interested in the frequent events in the interbeats. In Figure 11 we show the results for the moment \( q = 0.1 \) against a third-order structure function for healthy subjects and those with CHF. There are two interesting features in Figure 11. First, the starting point of \( S_{0.1}(\tau) \) versus \( S_3(\tau) \) is different for the data for healthy subjects and patients with CHF. To determine the distance form the origin, we define,51

\[
T(\tau = 1) = [S_{0.1}^2(\tau = 1) + S_3^2(\tau = 1)]^{1/2} .
\]

The second important feature of Figure 11 is that there is a well-defined \( \tau^* \) beyond which the plot of \( S_{0.1}(\tau) \) versus \( S_3(\tau) \) is multi-valued. One can estimate \( \tau^* \) by checking when \( S_3(\tau) > S_3(\tau + 1) \). Moreover, if we define \( T(\tau^*) \) by,

\[
T(\tau^*) = [S_{0.1}^2(\tau^*) + S_3^2(\tau^*)]^{1/2} ,
\]

then, there is a time scale \( \tau^* \) such that the values of the third moment before and after \( \tau^* \) are almost the same. Thus, the quantity \( \tau^* \) plays a role of a local mirror on the time axis. In other words, locally, \( S_3(\tau) \) for \( \tau < \tau^* \) and \( \tau > \tau^* \) has almost the same value. In Figure 11, we show the time scale \( \tau^* \) and, therefore, indicate that values of \( T(\tau = 1) \) and \( T(\tau^*) \) of the interbeat fluctuations of the healthy subjects and patients with CHF are different.

Table 1 presents the computed values of \( T_1 = T(\tau = 1) \) for both healthy subjects and those with CHF. To compute these results, we first rescaled the data sets by their
standard deviation and, therefore, the $T_1$ values are dimensionless. The average value of $T_1$ for the healthy subjects is, $\bar{T}(\tau = 1) \simeq 0.5848$, with its standard deviation being, $\sigma \simeq 0.065$. The corresponding values for the daytime records of the patients with CHF are, $\bar{T}(\tau = 1) \simeq 0.5077$ and $\sigma \simeq 0.03$, respectively. Therefore, on average, healthy subjects possess $T_1$ values that are greater than those of the patients with CHF. However, note that $T_1$ for the various data sets do not have large enough differences to be able to distinguish unambiguously the data sets. Indeed, as Table 1 indicates, three of the data sets (belonging to the day- and nighttime records of one healthy subject, with the third one belonging to the daytime of another healthy subject) overlap.

To develop a more definitive criterion for distinguishing the data for various subjects, we compute values of $T(\tau^*)$. The results are listed in Table 2. It is evident that, in this case, there is no overlap in the data sets. Indeed, the $T(\tau^*)$ values for the healthy subjects are larger, by a factor of about 3, than those for the patients with CHF, hence providing an unambiguous way of distinguishing the data sets for healthy subjects and those with CHF.

### Comparison with other Methods

Stanley and co-workers$^{6,18,26,41-43,52,57,59}$ and others$^{53-56,58}$ analyze the type of data that we consider in this paper by different methods. Their analyses indicate that there may be long-range correlations in the data, characterized by self-affine fractal distributions, such as the fractional Brownian motion, the power spectrum of which is given by, $S(f) \sim f^{-(2H+1)}$. They distinguish healthy subjects from those with CHF in terms of the type of correlations that might exist in the data: negative or antipersistent correlations for $H < 1/2$, as opposed to positive or persistent correlations for $H > 1/2$. The reconstruction method described in the present review analyzes the data in terms of the properties of Markov processes. As a result, it distinguishes the data for healthy subjects from those with CHF in terms of the differences between the drift and diffusion coefficients of a Fokker-Plank equation which, in our view, provide a clearer and more physical way of understanding the differences between the two groups of the subjects. In addition, the computational approach described in this review provides an unambiguous way of reconstructing the data, hence providing a means of predicting the behavior of the data over periods of time that are on the order of their Markov time scales.

On the other hand, Lin$^{60}$ argued that the daytime

TABLE I. Values of $T_1$ for the healthy subjects (day time and night time) and those with CHF.

|       | Healthy | CHF  |
|-------|---------|------|
| $T_1$ | 0.658   | 0.557|
|       | 0.672   | 0.565|
|       | 0.614   | 0.539|
|       | 0.505   | 0.526|
|       | 0.583   | 0.512|
|       | 0.581   | 0.493|
|       | 0.576   | 0.492|
|       | 0.558   | 0.481|
|       | 0.494   | 0.469|
|       | 0.480   | 0.443|
heart rate variability of healthy subjects may exhibit discrete scale-invariance (DSI). A stochastic process \( x(t) \) possesses continuous scale-invariant symmetry if its distribution is preserved under a change of variables, \( t \rightarrow \lambda t \) and \( x \rightarrow x/\mu \), where \( \lambda \) and \( \mu \) are real numbers, so that,

\[
x(t) = \frac{1}{\mu} x(\lambda t) .
\]

If Eq. (23) holds only for a countable (discrete) set of values of \( \lambda \), \( x(t) \) is said to possess DSI, which implies a power-law behavior for \( x(t) \) that has a log-periodic correction of frequency \( 1/\log \lambda \), so that,

\[
x(t) = \zeta F(\log t / \log \lambda) ,
\]

with \( \gamma = \log \mu / \log \lambda \), and \( F(x) = F(x + 1) \) being a period scaling function. Generally speaking, one may write, \( x(t) = c(t)t^\zeta \), with, \( \zeta = \gamma + 2n\pi i / \log \lambda \), with \( n = 1, 2, \ldots \). The existence of log-periodicity was first suggested by Novikov\(^6\) in small-scale energy cascade of turbulent flows. It has been argued\(^6\) that log-periodicity may exist in the dynamics of stock market crashes,\(^6\) turbulence,\(^6\) earthquakes,\(^6\) diffusion in disordered materials,\(^6,6,6\) and in fracture of materials near the macroscopic fracture point.\(^6\) The log-periodicity, if it exists in the heart rate variability (HRV), implies the existence of a cascade for the multifractal spectrum of HRV, previously reported by others. However, Lin’s method, neither provides a technique for distinguishing the HRV of healthy people from those with CHF, nor can it predict the future behavior of HRV based on some data at earlier times.

Although it remains to be tested, we believe that, together, all the computational methods that have been described in this review are more sensitive to small differences between the data for the two groups of the subjects and, therefore, might eventually provide a diagnostic tool for early detection of CHF in patients.

Finally, the computational approaches described in this review are quite general, and may be used for analyzing times series that represent the dynamics of completely unrelated phenomena. For example, we have used the concepts of Markov processes and extended self-similarity to develop\(^5\) a method for providing short-term alerts for earthquakes, as well as making predictions for the price of oil.\(^5,6\)

**TABLE II. Values of the \( T(\tau^*) \) for the healthy subjects (day time and night time) and for those with CHF.**

| \( \text{Healthy} \) | \( \text{CHF} \) |
|---------------------|----------------|
| 3.08                | 0.741          |
| 2.68                | 0.714          |
| 2.34                | 0.685          |
| 1.92                | 0.681          |
| 1.86                | 0.675          |
| 1.42                | 0.632          |
| 1.40                | 0.573          |
| 1.22                | 0.728          |
| 1.20                | 0.552          |
| 1.15                | 0.465          |

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