

Fractal behavior in heartbeat dynamics

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PHYS498BIN: Statistical Physics Applied to
Biological Information and Complexity

December 11, 2001

*1. Mathematics is the language of nature.
2. Everything around us can be represented
and understood through numbers.
3. If you graph these numbers, patterns emerge.
Therefore: There are patterns everywhere in nature.
-Max Cohen in Π*

1 Introduction

Many simple systems in nature have correlation functions that decay with time in an exponential way. For systems comprised of many interacting subsystems, physicists discovered that such exponential decays typically do not occur. Rather, correlation functions were found to decay with a power-law form. The implications of this discovery is that in complex systems, there is no single characteristic time. Since at large time scales a power-law is larger than an exponential function, correlations described by power-laws are termed “long-range” correlations [1].

The study of the statistical properties of heartbeat interval sequences has been the interest of many researchers in recent years. The healthy heartbeat is generally thought to be regulated according to the classical principle of homeostasis whereby physiologic systems operate to reduce variability and achieve an equilibrium like-state. However, a group of researchers composed by physicists from Boston University, Harvard Medical School and elsewhere [2, 3, 4, 5], have found that under normal conditions, beat-to-beat fluctuations in heart rate display the kind of long-range correlations typically ex-

hibited by physical dynamical systems far from equilibrium [5].

The group has studied scale-invariant properties of the human heartbeat time series. In their studies, they sample beat-to-beat heart rate fluctuations over very long time intervals for different cases. They study healthy subjects awake and asleep, subjects with heart failure, and even subjects in the space station MIR (Figure 1). The group has performed different studies in heart-beat diagnosis over the last years. In this paper, I will review their findings on the multifractality in human heartbeat dynamics.

2 Fractality of the human heartbeat

In their initial study of the human heart, records of up to 24 hour intervals of heartbeats were obtained from several subjects and represented by time series from the sequential intervals between beat n and beat $n + 1$, denoted by $B(n)$ (Figure 2). Then they introduce a mean fluctuation function $F(n)$, defined as

$$F(n) \equiv \overline{|B(n'+n) - B(n')|}, \quad (1)$$

where the bar denotes an average over all values of n . $F(n)$ quantifies the magnitude of the fluctuations over different time scales n . Figure ??c shows a log-log plot of $F(n)$ vs. n . The plot is linear over a broad physiologically relevant time scale and shows that

$$F(n) \sim n^\alpha \quad (2)$$

and that the exponent α is very different for the healthy and diseased patients. For the healthy patients, α is close to zero, and for the diseased cases, $\alpha \sim 0.5$, corresponding to a random walk!

They further introduce a new stochastic variable, the timebeat intervals $I(n) \equiv B(n+1) - B(n)$. With it, they could perform power spectra analysis (because $I(n)$ is more stationary [6]), and this analysis yielded:

$$S_I(f) \sim \frac{1}{f^\beta} \quad (3)$$

where $\beta = 2\alpha - 1$, and can serve as an indicator: (i) $\beta = 0$, white noise, (ii) $0 < \beta < 1$, $I(n)$ is correlated so that positive values of I are likely to be close in time to each other, (iii) $-1 < \beta < 0$, $I(n)$ is negatively correlated.

For the subjects with heart failure, they found $\beta \sim 0$ in the low frequency region confirming that the $I(n)$ are not correlated for long time scales, as opposed to the healthy subject, that had $\beta \sim -1$, indicating non-trivial long-range correlations in $B(n)$, and a negative feed-back system that prevents the heart to hit the extremes.

Later, using an alternative analysis, the *detrended fluctuation analysis* (DFA), they proved that the fractal coefficient for sleeping subjects is lower than the one for awake subjects, for both healthy and heart failure patients. The fact that the heart failure patients are physically restrained because of their disease, rules out changes on the behavior due to activity stimuli. This was further confirmed by analysing the data of cosmonauts in the MIR space station, that were subject to high stress activity and zero gravity, and obtaining similar results as the healthy patients.

So, the sleep-wake scaling differences are due to intrinsic changes in the cardiac control mechanisms, and fluctuation cardiac dynamics exhibit scale-free behavior for both.

3 Multifractality of the human heartbeat

In view of the heterogeneous nature of the heartbeat interval time series, they further suggested that a single exponent is not enough to characterize the complexity of the cardiac dynamics, and that a multifractal approach is necessary (See figure 4). To test the hypothesis that an infinite number of exponents is required to characterize healthy dynamics, a multifractal analysis of heartbeat interval time series has been performed and the fractal dimension, $D(h)$ has been calculated using wavelet methods.

The properties of the wavelet transform make wavelet methods attractive for the analysis of complex nonstationary time series such as one encounters in physiological signals. The group used n -order derivatives of the Gaussian function, allowing them to estimate the singular behavior and the corresponding exponent h at a given location in the time series. The higher the order n of the derivative, the higher the order of the polynomial trends removed and the better the detection of the temporal structure of the local scaling exponents in the signal. After obtaining the value h at each point of the time series, they obtained the partition function $Z_q(a)$, the sum of the q th powers of the local maxima of the modulus of the wavelet transform coefficients at scale a .

For small scales,

$$Z_q(a) \sim a^{\tau(q)}. \quad (4)$$

The scaling exponents $\tau(q)$ can reveal different aspects of cardiac dynamics. For example, for positive q , $Z_q(a)$ reflects the scaling of the large fluctuations, for negative q , small fluctuations. The fractal dimensions $D(h)$ are defined through a Legendre function of $\tau(q)$

$$D(h) = qh(q) - \tau(q), h(q) \equiv \frac{d\tau(q)}{dq}. \quad (5)$$

Monofractals display a linear $\tau(q)$ spectrum, $\tau(q) = qH - 1$, where H is the global Hurst exponent. For multifractal signals, $\tau(q)$ is a nonlinear function $\tau(q) = qh(q) - 1$ where h is not a constant (Figure 5). Analysing different subjects they found that for all subjects $Z_q(a)$ scales as a power law. For all healthy subjects $\tau(q)$ is a nonlinear function indicating that the heart rate of healthy humans is a multifractal signal. Fig 5b shows that for healthy subjects, $D(h)$ has nonzero values for a broad range of local Hurst exponents h . Furthermore, the multifractality can not be explained by activity, because the sleeping subjects display the same dynamics. In contrast, subjects with a pathological condition show a loss of multifractality, $D(h)$ is composed only by a narrow range of exponents h . Moreover, even when the same exponent h appears in both healthy and heart failure subjects, the fractal dimension associated with it is smaller for the heart failure subjects.

4 Conclusions

The analysis performed by this group show a very important insight into the dynamics of heartbeats. They conclude that the different scaling behavior in health and disease must relate to the underlying mechanism. Applications of this analysis may lead to new diagnostics for patients at high risk of cardiac disease and sudden death [6] with the advantage of a non-invasive method. Like these, analysis using physical approaches to health issues can improve medicine. This is very exciting for physicists that like to build models, for they can actively contribute to exciting areas like this one without necessarily having to get involved in applied physics for the development of new technologies. .

References

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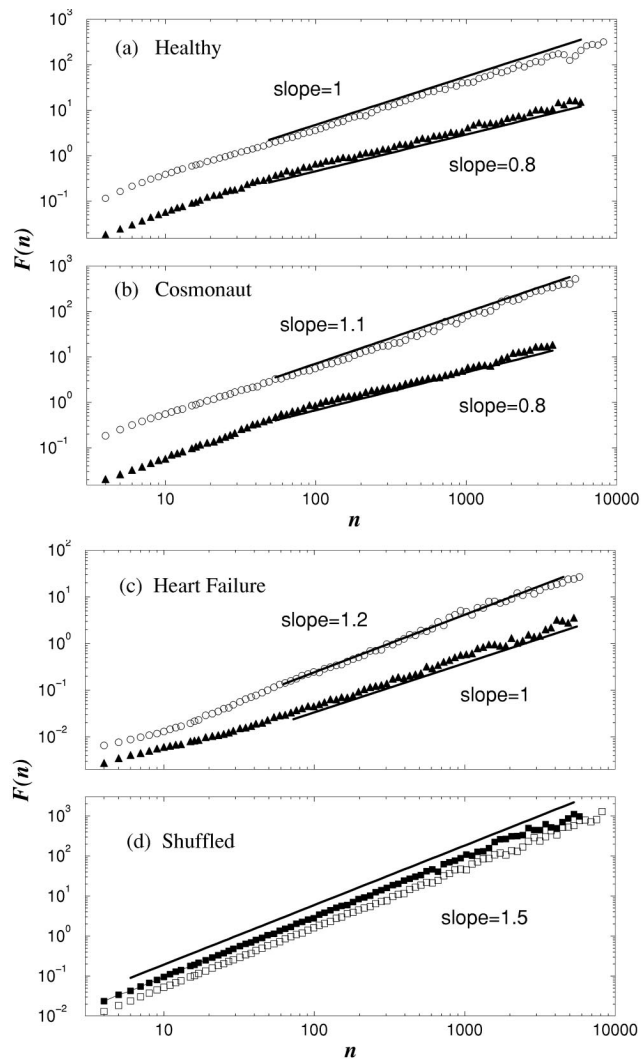


Figure 1: Plots of $\log F(n)$ vs $\log n$ for 6 h wake (open circles) and sleep records (filled triangles) of (a) one typical healthy subject, (b) one cosmonaut (during orbital flight); and (c) one patient with congestive heart failure. Note the systematic lower exponent for the sleep phase (filled triangles), indicating stronger anticorrelations. (d) As a control, we reshuffle and integrate the interbeat increments from the wake (open squares) of the healthy subject presented in (a). We find a Brownian noise scaling over all time scales for both wake and sleep phases with an exponent $\alpha=1.5$, as one expects for random walk-like fluctuations. Reproduced from [2]

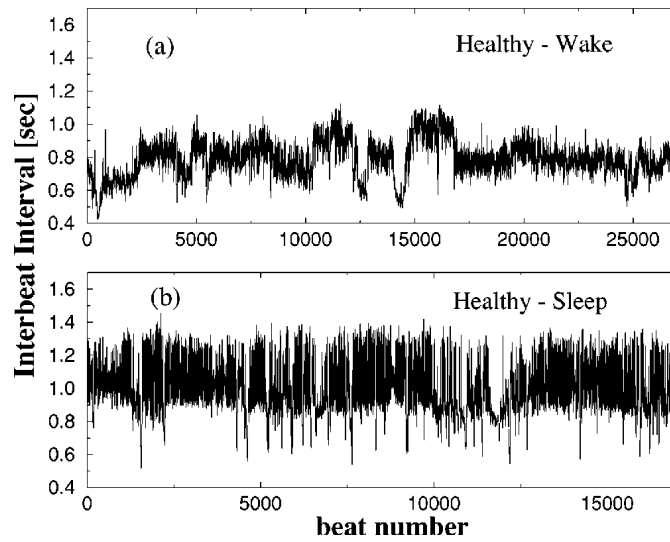


Figure 2: Consecutive heartbeat intervals are plotted versus beat number for 6h recorded from the same healthy subject during: (a) wake period and (b) sleep period. (Note that there are fewer interbeat intervals during sleep due to the larger average of the interbeat intervals, i.e., slower heart rate. Reproduced from [2])

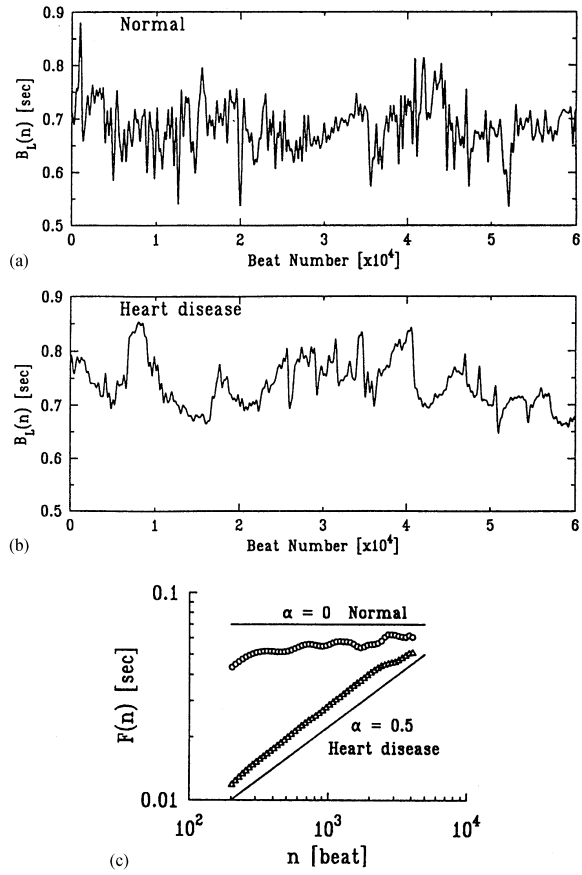


Figure 3: The interbeat interval $B_L(n)$ after low pass filtering for (a) healthy subject and (b) a patient with severe cardiac disease. The healthy heartbeat time series shows more complex fluctuations compared with the diseased heart rate fluctuation pattern that is close to a random walk. (c) Log-log plot of $F(n)$ vs n .

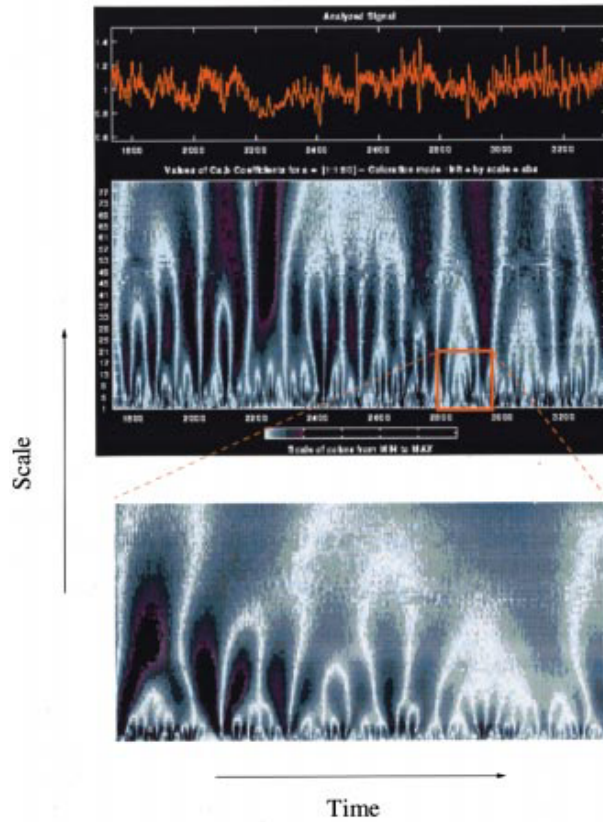


Figure 4: Color-coded wavelet analysis of a heartbeat interval signal. The x-axis represents time (1700 beats) and the y-axis indicates the scale of the wavelet used ($a=1,2,\dots,80$; i.e., from 5 to 5 min) with large scales at the top. This wavelet decomposition reveals a self-similar fractal structure in the healthy cardiac dynamics - a magnification of the central portion of the top panel with 200 beats on the x-axis and wavelet scale $a=1,2,\dots,20$ on the y-axis shows similar branching patterns (lower panel). Reproduced from [2].

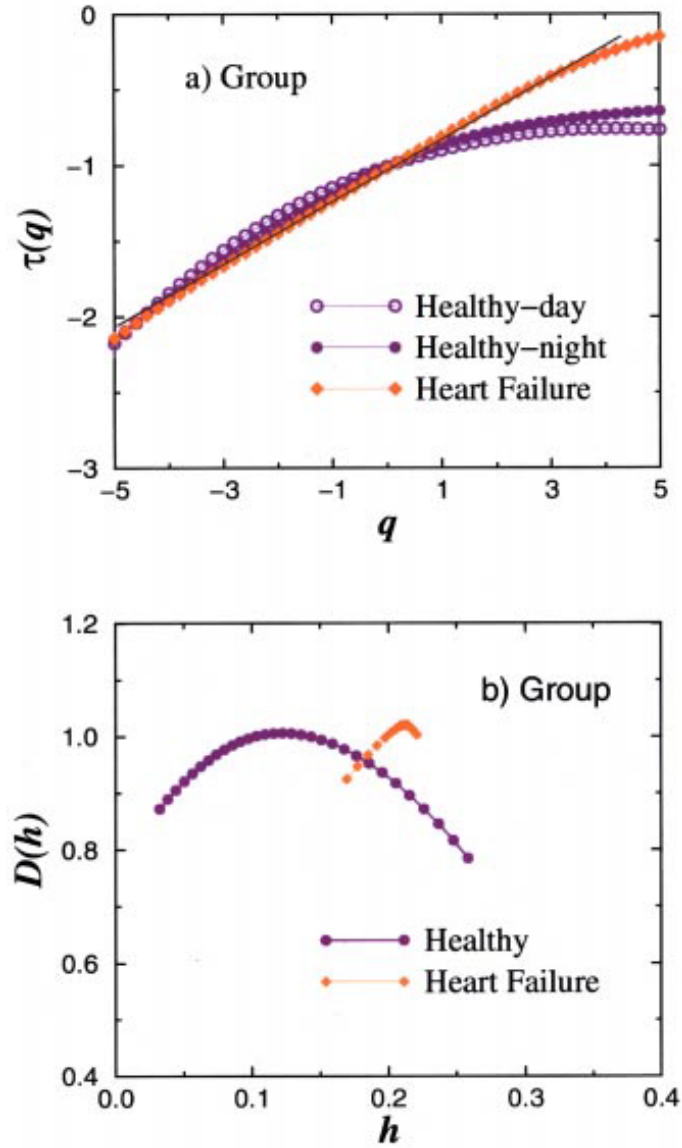


Figure 5: (a) Multifractal spectrum $\tau(q)$ of the group averages for daytime and nighttime records for 18 healthy subjects and for 12 patients with congestive heart failure. The results show multifractal behavior for the healthy group and the distinct change in this behavior for the heart failure group. (b) Fractal dimensions $D(h)$ obtained through Legendre transform from the group averaged $\tau(q)$ spectra of (a). The shape $D(h)$ for the individual records and for the group average is broad ($\delta h \sim 0.25$), indicating multifractal behavior. On the other hand, $D(h)$ for the heart failure group is very narrow ($\delta h \sim 0.05$), indicating loss of multifractality.