

A Healthy Human Heart Operates in a Critical State

Term Paper for Physics 563*

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December 9, 2005

Abstract: Data is presented to show that successive increments in the beat-to-beat intervals of healthy human hearts (HHRI) display scale-invariant, long range autocorrelations that vanish in subjects with heart diseases. Evidence of data collapse and non Gaussianity observed in the probability distribution functions (PDF) of the HHRI is discussed. Finally, experimental data is presented to prove that these hallmarks of criticality are observed only during normal daily activity and a phase transition occurs during prolonged sleep and strenuous exercise. This was the first reported discovery of a dynamic phase transition in a biological control system. The results presented strongly support the hypothesis[2] that a healthy human heart operates far from equilibrium and is controlled to converge continually to a critical state. Open questions and possibilities of future research helping clinical diagnosis of heart diseases are briefly discussed.

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1.Introduction

It is a commonly held notion that disease and aging arise from stress that acts on an orderly and ‘programmed’ system, and decreases the ‘order’ by provoking erratic responses or by upsetting the body’s normal periodic rhythms. However, various physiological systems behave erratically in young and healthy humans. Counter intuitive as it may seem, as we age/become ill, some of our systems lose the ability to behave erratically and more regular and periodic behaviour sets in. In this term paper, I will briefly discuss one such fascinating system: the human heart.

Most of us have measured our pulse rate to be ~ 72 /min when we are normal and higher/lower when we are sick. Almost all of us would say that our heart beats at a nearly constant rate. Clinicians conventionally describe the normal electrical activity of the heart as “regular sinus rhythm”. However, the interval between beats fluctuates in a complex and apparently erratic manner in the heart of a healthy subject, even at rest (see Fig. 1).

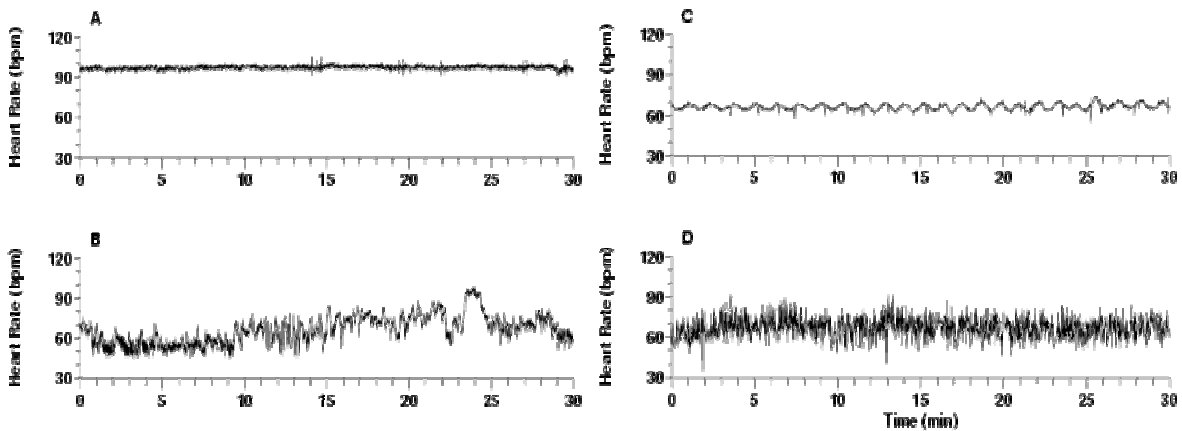


Fig. 1: Heart beat rate in beats per min as a function of time(min). A and C depict a diseased (congestive) heart. B shows a healthy human heart and D shows the heart beat rate trace of a subject with cardiac arrhythmia/ atrial fibrillation. Clearly the healthy human heart shows erratic variation in beat rate while the diseased heart rate seems to be periodic.

Understanding heart beat dynamics has been a long standing challenge now. Fourier analysis of heart rate data taken over a long period of time in healthy subjects typically shows a $1/f$ –type spectrum[14]. Very long range correlation properties of heart beat rates reveal interesting information about healthy vs. diseased subjects. A healthy human heart rate (HHR) belongs to a special class of non linear signals and the probability distribution function of the increment in beat intervals of HHR display non gaussianity and scaling properties.

Primarily, two models were proposed to understand these observations, (i) a random multiplicative cascade process[4]and (ii) critical state like dynamics[7]. The models were suggested based on the resemblance of the behaviour of the structure function of HHRI to that of spatial velocity differences in hydrodynamic turbulence[4] and the scale invariant properties of HHRI to those of systems in the critical region[7] respectively.

Recently [2,3,5] several conventional techniques (fourier transforms, wavelet transforms, method of surrogate data analysis) together with new methods have been used to study HHR data. These studies indicate that the heart is operating in a critical region quite convincingly. Phase transitions between different behavioural states have been detected in these data, and this is said to be the first discovery of any such dynamics in a biological control system. This work has led to what might be a potential indicator and perhaps quantifier of a healthy heart or a diseased heart. It is quite interesting that a biological control system as intricate and complicated as our heart shows signatures of criticality just like those observed in simple physical systems and that perhaps we can get away with using crude models and our knowledge of critical systems to obtain clinically relevant results!

This paper will mostly present experimental observations listed above, and discuss the main idea behind the data analysis methods used wherever necessary. We will follow the experiments in a logical order until we are hopefully convinced that the heart does operate in a critical state! We do not yet have a robust model or even know all the parameters involved in heart beat dynamics. Any relevant information on the physiology that is known to have provided useful insights into this problem, I shall provide references to. In the concluding section, we shall discuss open problems these results point to, and possible future directions.

2. Experimental Results, Physical Models and Discussion

We first briefly discuss initial results that motivated heart rate dynamics modeling. Next, we shall discuss physical models and the recent experiments done to test their validity.

2.1 Initial Results

Peng et al [1] analysed data from the digitized electrocardiograms of beat-to-beat variations over very long time intervals (upto 24 h $\sim 10^5$ beats) recorded with an ambulatory monitor in healthy subjects as well as subjects with heart disease. The data consisted of a time series obtained by plotting the sequential intervals between beat n and $n+1$ denoted by $b(n)$. After filtering out very high frequencies (to observe large time scale behaviour), they plotted the mean fluctuation function defined as $F(n) = \langle |b(i+n) - b(i)| \rangle$ where $\langle . \rangle$ denotes statistical averaging. It is seen that $F(n)$ which is the magnitude of fluctuations, scales like a power of n as: $F(n) = n^\alpha$. α was found to be close to 0 for the healthy heartbeat data and 0.5 for the diseased case. It is interesting to note that $\alpha = 0.5$

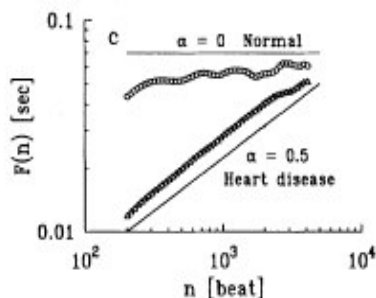


Fig 2a (from Ref[1]): $F(n) = n^\alpha$

case corresponds to the random walk case, so that the long time scale behaviour of a diseased heart can be interpreted as a random stochastic process. Fig. 2a depicts these results.

Fig. 2b depicts the plot of power spectral density function (squared modulus of the fourier transform of time series $b(i)$) vs. frequency. We can ignore the

legend on the figure. For our purposes, all we need to know is that various time series records (shown in different colored circles) have been used to compute the power spectral density function. We can see the $1/f$ scaling form of the spectral density function in the figure.

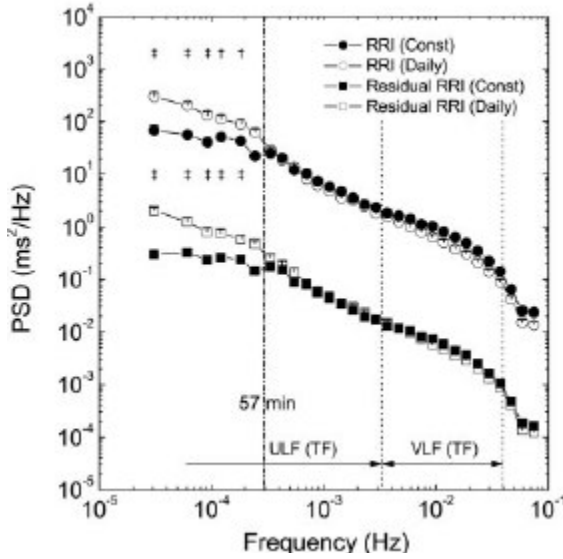


Fig2b: Power spectral density plot, from Ref[14]

Are these differences in scaling properties of the healthy and diseased heart observed because of their underlying statistical properties being different? Peng et al defined an increment function $I(n)=b(n+1)-b(n)$ and plotted its histogram(not shown here) for both the normal and diseased cases, and the histograms overlapped exactly.

If the increments have the same underlying distribution function, the correlations between successive increments should affect the scaling properties and hence provide useful insight into the healthy vs. diseased case. Shown below in log-log form is the power spectra (plot of the square of the fourier transform of $I(n)$ vs frequency) from the same data sets. $S(f) \sim f^\beta$, where $\beta = 1 - 2\alpha$ [12]. If β is negative, $I(n)$ is correlated such that positive(or negative) values of $I(n)$ lie close together, and if β is positive, $I(n)$ is organized such that positive and negative values alternate more often(anticorrelated). $\beta=0$ corresponds to white noise(uncorrelated case).

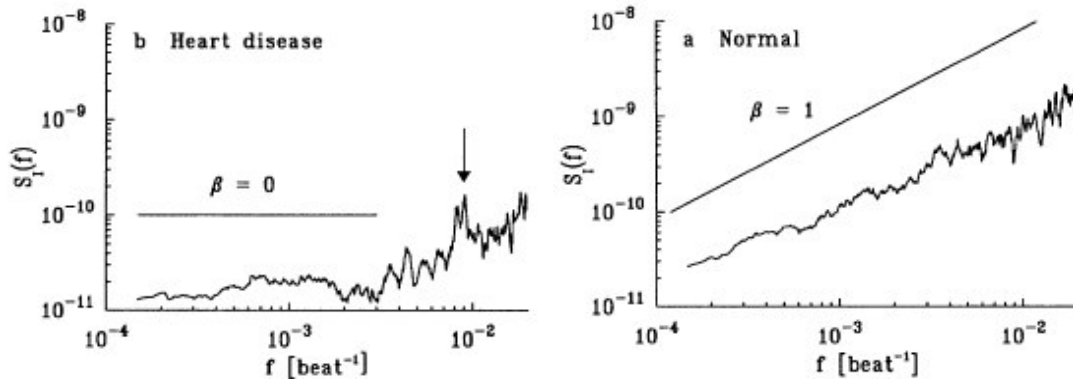


Fig 3(from Ref[1]): power spectra

From fig 3, we observe that the beat increments are uncorrelated over long time scales in the diseased case, and they are strongly anticorrelated in the healthy case! This means that there must exist a nonlinear feedback system that kicks the heart rate away from extremes.

We see that the anticorrelations exist over a long range of time scales. Lack of a characteristic time scale helps prevent excessive mode locking behaviour in which the heart cannot adapt to change quickly. In severe cases of heart failure, breakdown of long range correlations is observed. Analogous transitions to highly periodic regimes have been reported in a wide range of other heart diseases (associated with breathing disorders and fibrillation of the arteries).

2.2 Non Physiological Models of Healthy Heart Rate Variability

2.2.1 Random Multiplicative Cascade Model : A Turbulence Analogy

Lin and Hughson[4] used a turbulence analogy and proposed that the heart beat is a random multiplicative cascade process. Here, we shall just list some basic ideas that were relevant to the model, but not delve very deeply into turbulence. A good review of turbulence in fluids can be found in Ref[6] .

Fully developed turbulence(FDT) and HHR, both seemed to demonstrate a 1/f like spectrum and a non gaussian pdf. To model these similarities, the idea of structure function and cascades was applied. Let $b(i)$ (beat interval) and $I(n)$ (beat increment) be defined as before. The structure function is defined by $S_q(n) = \langle |I(n)|^q \rangle$ where $\langle \cdot \rangle$ denotes a statistical average and $q > 0$ is real. Lin and Hughson then formulated a scaling form for the structure function similar to the Kolmogorov theory of FDT[6].

$$S_q(n) \sim n^{e(q)} \tag{1}$$

The Kolmogorov theory of FDT [6,14] gives a similar scaling form for the structure function, where l (length scale of observation) replaces n . This form for FDT is valid over all the length scales l in the inertial range where a balance exists between the energy dissipated in the flow and the energy injected at a large scale(see Ref[6]).Lin and Hughson analysed heart beat data and the structure function showed power law behaviour. Further careful numerical investigation suggested that

$$\frac{e(q)}{e(p)_{FDT}} \sim \frac{e(q)}{e(p)_{HHR}} \tag{2}$$

for some fixed integer p .

It was found that for $p=3$, the FDT structure function was the same as the HHR one. From these qualitative similarities, they concluded that perhaps the statistical properties of the HHR signal and velocity increments in FDT are the same. In FDT, a cascade method (random multiplicative cascades)was proposed[13] to account for the statistical properties.

The main idea is to first pick a random flat field $r_0(t)=c_0$, and then repeatedly cascade as: (a) divide the time domain into random subintervals and (b) multiply a random factor to the field at the subinterval. The field after J cascades is given by:

$$r_j(t) = c_0 \prod_{j=1}^J w_j(t) \quad \text{where } w_j(t) \text{ is the time scale component such that:}$$

$$\langle w_j \rangle = 1 \tag{3}$$

The field r updates its values at random time variables which in turn form the random subintervals. The number of such time intervals $N(j)$ at the j th cascade level describes the time scale of $w_j(t)$. For a self-similar process, it is necessary that $R \langle N(j) \rangle = \langle N(j+1) \rangle$ where R is a constant > 1 . Hence we see that random multiplicative cascades redistribute a finite quantity from coarser to finer scales.

Lin and Hughson[4] used the same cascade method to model HHR data and managed to simulate the power law spectrum and a few other statistical properties of the actual data. I will not elaborate on this work as it will later be shown that HHR do not follow random multiplicative cascades.

2.2.2 Critical State Model

Based on the PDF of the heart rate increments showing non Gaussian scale invariant behaviour, Peng et al.[7] put forth a critical state like model for the heart. They relate the heart rate mechanism is to competing neuroautonomic input: Parasympathetic stimulation decreases the firing rate of pacemaker cells in the heart's sinus node. Sympathetic stimulation has the opposite effect. The nonlinear interaction between these two branches of the nervous system was postulated to drive the heart into a critical state.

2.3 Recent Results

We will follow the findings and methods of very recent papers by Kiyono and coworkers[2,3] to illustrate the hypothesis that a healthy heart operates in a critical state. As before, the time series of interbeat intervals ($b(i)$ where i is the beat number) was analysed. The time series was normalized to have zero mean and unit variance. To get a time trace of the heart beat data, we integrate the time series $b(i)$ to obtain $B(i)$

$$B(i) = \sum_{j=1}^i b(j) \tag{4}$$

Now, as we can see from fig. 1, the healthy human heart rate trace is quite “patchy” or nonstationary, i.e. its mean value keeps changing in some particular trend continuously. We, however would like to study the correlation of the fluctuations about the mean, and the ‘trend’ in the mean is like a background that we wish to ignore. To do this, we divide the time series into segments of length $2s$. And within each time segment, we fit the $B(i)$ ’s to a polynomial function of order d ($d=3$ was used in the papers[2,3] as greater values didn’t seem to make a difference to the detrending procedure) as:

$$B(i) = \sum_{j=-s}^s C(j)F(B(j)) \tag{5}$$

where $C(j)$ s are some constants and $F(B(j))$ is a polynomial function of order d . We have now obtained the d th order polynomial trend in the data. Now we find the deviation of the actual data $B(i)$ from the fitted $B^*(i)$, and this gives us the fluctuation $B^*(i)$ about the trend. We now define the heart rate increments at a scale ‘ s ’ as

$$\Delta_s B(i) = B^*(i + s) - B^*(i) \quad (6)$$

Kiyono and coworkers analysed two experimental and two computer data sets. The first data set consisted of daytime(1200 to 1800 hrs) heart rate data of about 10^4 heartbeats from 50 healthy subjects(10 females and 40 males ages 21-76).The recruitment of these subjects was done after a thorough medical screening! The second experimental data set consists of data of seven 26 h long periods collected when the subjects (7 healthy males) underwent “constant routine”(CR) protocol, where known behavioural factors affecting heart rate(eg. Exercise, sleep, diet) were eliminated. The third data set consisted of surrogate data and its purpose and construction is discussed below.

The initial results seem to motivate the fact that there exists a nonlinear feedback system at play which is leading to long range correlations and ‘patchy’ data as shown in fig. 1. How do we look at the data and confirm that there are indeed non linear signals, instead of a Gaussian stochastic process (Gaussian noise term added to the data)? The method of surrogate data analysis[9] was used to test this ‘null hypothesis’. The idea is that if the data is nonlinear, there is some phase relationship between different fourier components, where as the fourier components of linear Gaussian noise would have random phases. Hence, surrogate data is generated by fourier transforming the original data set $\{b(i)\}$, retaining the amplitudes but randomizing the phases and inverting the transform to obtain $\{b(i)\}_{\text{surr}}$. Thus, both the original and surrogate data have the same fourier spectrum and only differ in their phase correlation properties, if any. By analyzing the PDF of $\Delta_s B(i)$ for both these data sets, if we find no difference, it means that we cannot ascertain the presence of nonlinear effects in our data.

The last set of heart beat increments were generated following the turbulence scenario from the random cascade model with the same parameters J , R (defined in sec 2.2.1)and the same variance for the distribution of $w_j(t)$ as used by Lin and Hughson[4].

PDF’s of $\Delta_s B(i)$ for healthy humans, which were standardized by dividing the heart rate increments in each record by the standard deviation, were found to be non-Gaussian for a wide range of scales $8 < s < 4096$ irrespective of whether the subjects were in normal daily activity where the mean rate of heart beat was continuously changing or in CR. On the other hand, the PDF of the surrogate data are near Gaussian, although non gaussianity is found in fine scales. Hence, we have confirmed non linear features in the actual data which do not show up in the surrogate data set. The PDFs of the cascade model show continuous deformation from Gaussian at large scales to non Gaussian with stretched exponentials at fine scales. Intuitively we can deduce this as follows. Cascading suppresses the tail values and amplifies the values around the peak of a field at small time scales, and so we find exponential tails and sharp peaks. At larger time scales, the field

should be uniformly distributed by the cascading process and we observe near Gaussian PDFs. However, the PDFs of daily routine and CR show non gaussianity consistently irrespective of the scale. See Fig. 4 for details.

For a quantitative comparison, the data was fitted to the Castaing's equation(see Ref[10]) which is basically a non gaussian PDF described by:

$$P_s(x) = \int P_L\left(\frac{x}{\sigma}\right) \frac{1}{\sigma} G_{s,L}(\ln \sigma) d(\ln \sigma) \quad (7)$$

where P_L is the increment PDF at a large scale $L > s$ and the self similarity kernel $G_{s,L}$ models the nature of the random multiplicative process. The assumption is that both P_L and $G_{s,L}$ are Gaussian,

$$G_{s,L}(\ln \sigma) = \frac{1}{\sqrt{2\pi\lambda}} \exp\left(\frac{-\ln^2 \sigma}{2\lambda^2}\right) \quad (8)$$

Note that as λ tends to 0, the probability distribution function converges to Gaussian and for large values of λ , there is a stronger peak about the mean value and a flatter tail. It is seen that this fitting is robust and models the data nearly accurately. The behaviour of λ^2 of the fit with the scale s shows interesting behaviour as it contains information about deviation from gaussianity. Within the turbulent cascade picture, the parameter λ^2 can be interpreted as being proportional to the number of cascade steps and is known[15] to decrease linearly with $\log s$. The cascade heart model data set shows this trend, where as the normal daily routine and CR do not show any significant difference in λ^2 with scale. Fig. 6 shows relevant details. This shows the absence of any cascade process in heart beat rate. Moreover, the PDFs of daily routine and CR at different scales when superimposed, collapse onto the same curve(see fig.5) which is typical of systems near the critical point(data collapse). The range of scales where such scaling is observed (10 beats to 10000 beats) is compatible with the $1/f$ scaling and multifractality data[14,5]. Such scaling is absent in the cascade model. Hence, we can conclude that the cascade model doesn't accurately depict the statistical properties of HHR. The scale invariance seems to be robust and independent of behavioural modifiers altering the mean heart level, and this strongly supports the hypothesis that a healthy heart operates in a critical state.

This work assumed that averaging the PDFs of different healthy subjects in one data set to obtain the PDF of increments and then scaling the PDF by the variance in the data set is a valid description. Previously, using wavelet transform for subtracting the background trend, Ivanov et al.[5] showed that when scaled appropriately(by the variance in our case), the PDFs of individual healthy subjects do collapse onto on curve, and hence the PDF of healthy heart rate increments can be described by one curve for all individuals in a data set. This was not observed in the case of diseased patients. Note that instead of using $\Delta_s B(i)$ as we defined it or wavelet transforms[5],if we had used the PDF of the increments directly, we could not have differentiated between healthy and diseased cases(see sec 2.1). This shows that correcting for non stationarity of data is important.

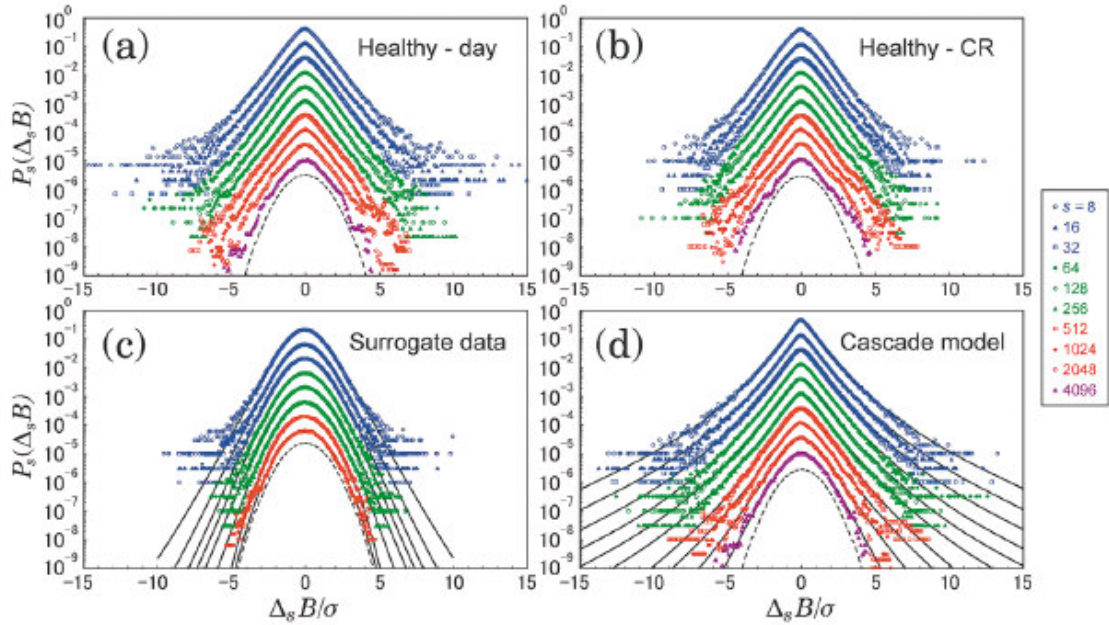


Fig. 4*: Deformation of increment PDF's across scales. Standardized PDF's (in logarithmic scale) of $\Delta_s B(i)$ for different time scales are shown for (from top to bottom) $s = 8, 16, 32, 64, 128, 256, 512,$ and 1024 beats. These PDF's are estimated from all samples in each group. The dashed line is a Gaussian PDF for comparison. (a) The PDF's from daytime (12:00–18:00 h) heart rate time series from healthy subjects. (b) From healthy subjects during constant routine protocol. (c) From surrogate time series for (b). (d) From a cascade model. Note in the last case (d) the continuous deformation and the appearance of fat tails when going from large to fine scales. In solid lines, we superimpose the deformation of the PDF using Castaing's equation with the log-normal selfsimilarity kernel, providing an excellent fit to the data.

*Figure and caption from From Ref [2]

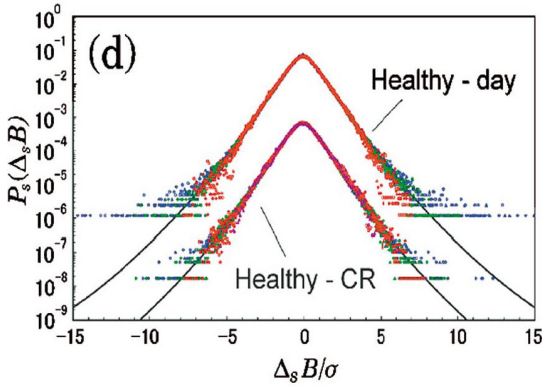


Fig. 5*: Superposition of standardized PDF's at different scales shown in Figs. 4(a) and 4(b), where we use the scale range $8 < s < 2048$ and $8 < s < 4096$, respectively. In solid lines, we superimpose the PDF using the Castaing's equation ($\lambda^2 = .16$) for both groups

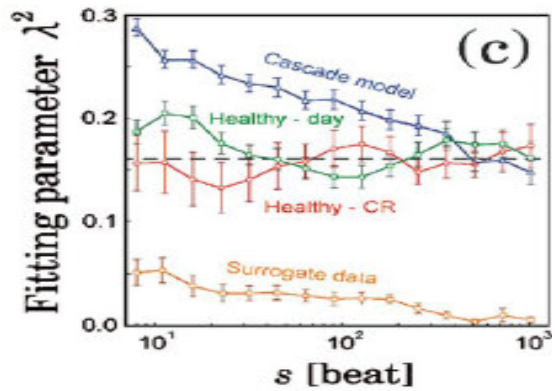


Fig. 6* The dependence of the fitting parameter λ^2 on scale s . Error bars indicate standard error of the group average values of λ^2 .

*figures and captions from Ref[2]

Finally, we ask: If the healthy heart is close to the critical point, then can we ever observe a breakdown of critical characteristics, i.e. a continuous phase transition? So far, in healthy human subjects, we always observed long range correlations and our PDFs showed similar behaviour irrespective of behavioural modifiers affecting the mean heart rate. So, then in what phase of a healthy human's life would we see a breakdown of criticality, if any?

The answer (or atleast part of it) turns out to be : during prolonged sleep and exercise[3]. As in figure 2, we calculate the mean square displacement $S_2(s) = \langle |\mathbf{B}(\mathbf{i}+\mathbf{s})-\mathbf{B}(\mathbf{i})|^2 \rangle$. It is related to the so called Hurst exponent \mathbf{H} as $s^{2\mathbf{H}}$. For a random process $\mathbf{H}=1/2$ and $\mathbf{H} = (2-\alpha)/2 > 1/2$ (α is as defined before)where represents long range correlations. Figure 7 depicts the scaling behaviour of S with s in various states(see caption below figure). It is seen that the sleep and exercise states show a crossover in the scaling behaviour, the crossover point being about 3 minutes or so for both states.

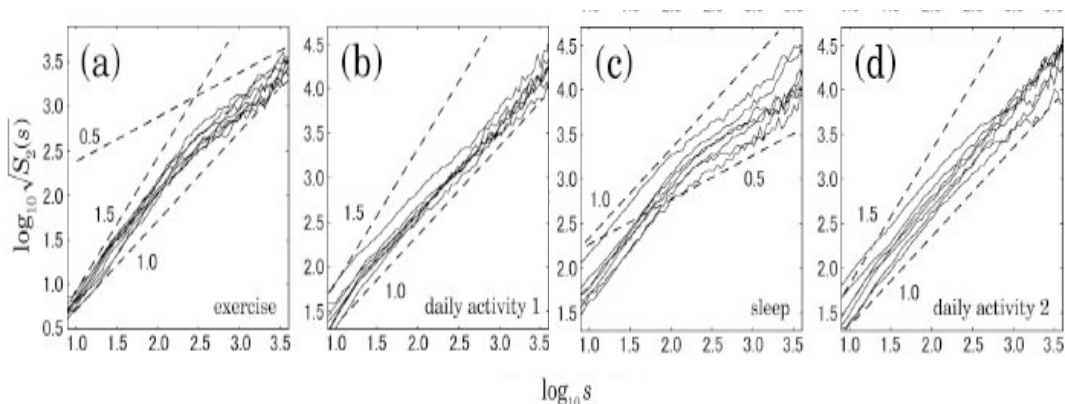


Fig. 7* : Scale dependence of the square root of the mean square displacement function of seven records from healthy records from healthy subjects during (a)constant exercise, (b)usual daily activity after the exercise, (c) sleep and (d) usual daily activity the next day morning.

* Figure and Caption from Ref[3]

This indicates existence of a phase transition. Now let us see what information was obtained from the PDF analysis as explained before. First, let us write down

$$\Delta_s B(i) = \varepsilon_s(i) e^{w_s(i)} \quad (9)$$

We note that $\varepsilon(i)$ determines the sign of $B(i)$ while $w(i)$ modulates the amplitude. We now fit the Castaing's equation to the data as before and use the above equation to estimate the variance λ^2_s of w_s . The non Gaussian PDFs of the two records of daily activity show striking resemblance. The scale invariance of the non Gaussian pdf in a range of 20-1000 beats however disappears in sleep states, in which the non Gaussian fluctuations at a scale of ~ 100 beats are dominant. The λ^2 for constant exercise is much smaller than other states, implying that it is nearly Gaussian. See Fig.8 for details.

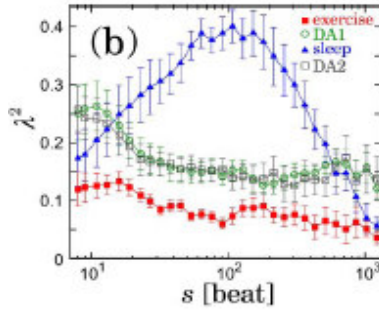


Fig. 8 : From Ref [3].

Now, it is important to note that the Gaussian noise with uncorrelated w_s in eqn (9) above and the long range correlated w_s are indistinguishable so far, as we have only looked at one point statistics. In order to observe possible non Gaussian fluctuations in w and their origin, we have to establish the correlation properties of w_s for different scales. Kiyono et al.[3] introduced an alternative method to study these fluctuations (see Ref [11]). At a scale s , they defined the local energy fluctuations or the mean square fluctuation as

$$\sigma_s^2(i) = \frac{1}{s} \sum_{j=1+s(i-1)}^{si} \Delta_s B(j)^2 \quad \text{and} \quad (10)$$

$$w_{s,avg}(i) = \frac{1}{2} \log \sigma_s^2(i)$$

The average $w_{s,avg}$ defined above was used to evaluate the correlation properties of w_s in eqn(9) by using the magnitude correlation function(Read $w_{s,=w_{s,avg}}$):

$$C(\tau; s_1, s_2) = \langle [w_{s_1}(i) - \langle w_{s_1} \rangle] [w_{s_2}(i + \tau) - \langle w_{s_2} \rangle] \rangle \quad (11)$$

This quantifies the clustering of local magnitude fluctuations. The magnitude fluctuations for the daily activity state are strongly correlated not only at a fixed scale but a wide range of scales, which means that the fluctuations at a short time scale strongly affect those generated at other ranges of time scales. On the other hand, the magnitude correlations for the sleep state and exercise are weak between different time scales. Fig. 9 below has relevant details.

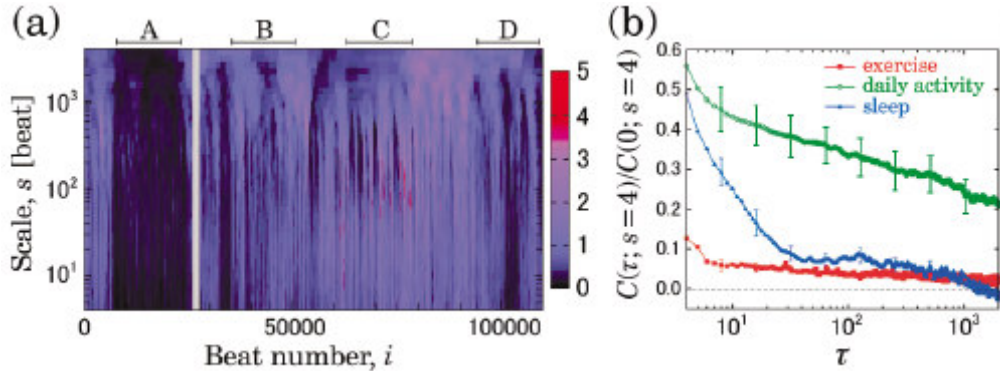


Fig 9 *: (a) Local energy fluctuations $\frac{\sigma_s^2(i)}{\langle \sigma_s^2(i) \rangle}$ of the heart rate increments $\Delta_s B(i)$ obtained at each scale (resolution) s ; (b) one-scale magnitude correlation functions, $C(\tau; 4; 4)/C(0; 4; 4)$

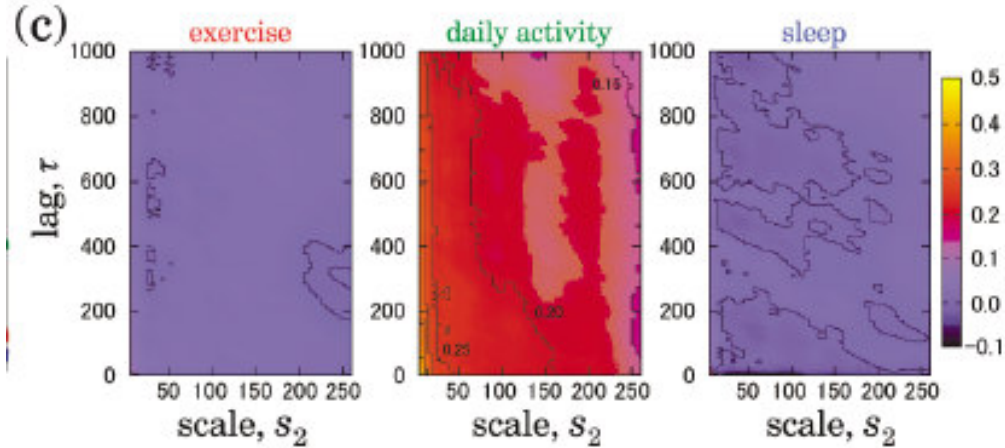


Fig 9 (c) two-scale magnitude correlation functions for each state. The color scales and contour lines (at 0.05 resolution increment levels) represent values of $C(\tau; 4, s_2)/\sigma_4\sigma_{s_2}$. For exercise and sleep states, contour lines of only $C(\tau; 4, s_2)=0$ can be seen.. The one-scale and two-scale magnitude correlation functions are estimated from all the records for each state.

*figure and caption from Ref [3]

We have now demonstrated a striking difference between waking, exercise and sleep states using two scale correlation functions.

3.Conclusions

In conclusion, the authors have demonstrated that a healthy human heart operates in a critical state, and exhibits phase transition like dynamics between different behavioural states, with a dramatic departure from criticality. We have elucidated strong evidence of there being non linear feedback loops at play in this control system and shown that the signals cannot be explained as some random cascade process. We shall now try to interpret the larger picture these results provide.

First, we do not yet know what the exact mechanism for critical heart rate dynamics may be. We have at least ruled out that is a cascade process, and it does seem unlikely that cascading heart beats might provide any biological advantage. Physiological models have pointed out that there are competing nervous systems that are play in the circulatory system, and the non linear interaction between them causes seemingly erratic but biologically balanced heart beat rate. There exists a model (see Ref[8]) which is based on synchronization between heart beat rate and respiration consisting of antagonistic and delayed feedback loops. Perhaps, we could analyse this model more carefully to see that if the time scales of the feedback loops match the scales over which we observe critical scale invariance. This might provide us better insight into the mechanism of heart beat.

What could be the reason that the heart operates in a critical state? Naively, we can say that this might be increasing its functional abilities in some way. Based on studies of traffic flow models which have revealed that the efficiency of transportation is maximum at the critical point (between the “uncrowded” to “congested” states), some people[2] have suggested that the heart might operate in a critical state to cope with the continuous

load on it. They also suggest that complete breakdown of these criticality characteristics leads to heart disease. Is it feasible to use the physiological model (see Ref[8]) mentioned above and study its efficiency of operation in its parameter space? Perhaps, we will then find that it is maximum in the critical region. If we could do this, then we could obtain some kind of a “phase diagram” for the healthy heart. With a reasonable understanding of the heart beat system physiologically and some modeling at a crude level(given that the healthy heart is in a critical state), we can perhaps observe trends in the crossover region from healthy to diseased state and this might help early diagnosis of cardiac disorders.

Now, we address the most recent results on phase transition like dynamics between waking and sleep states, exercise and daily activity states. The exercise state PDFs looked nearly Gaussian, perhaps because the non linear feedback system doesn't work very well during exercise. Is it advantageous for long range correlations to break down during strenuous exercise or is it simply inability of the control system to cope with the load? I propose that a possible way to find out would be to do similar analysis on (a) normal healthy subjects and (b) trained athletes during strenuous exercise. Maybe we could see then if the criticality characteristics breakdown at the same time scale for both cases or whether they breakdown at longer time scales in trained athletes. In the sleep state, there seems to be a different sort of nonlinear mechanism at play, which doesn't operate over very long time scales, but typically about 100 beats. Further physiological data might be needed to understand this rather interesting finding.

I do not know whether we would ever be able to obtain a “phase diagram” of this complicated control system, by defining relevant parameters and measuring them. However, the work done so far definitely inspires further research on this problem. This is a rather new field and open to exciting ideas and experiments.

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