LONG-TIME AUTOCORRELATION FUNCTION OF ECG SIGNAL FOR HEALTHY VERSUS DISEASED HUMAN HEART

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Long-time ECG time series for healthy subjects and diseased patients are analysed. In the first case, the power spectrum has the $1/f$ shape in a broad frequency range. However, its behaviour for very low and very high frequency is different and the entire spectrum is integrable. For patients with post-ictal heart rate oscillation in partial epilepsy the $1/f$ noise is not present. We determine the power spectrum by evaluating the Fourier transform of the signal in both cases and calculate the signal autocorrelation function. It falls with time faster for diseased patients then for healthy people. The presented method can serve as a diagnostic tool of some heart diseases.

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1. Introduction

A long-time tracing of ECG signal from a human heart is able to reveal [1] some pathological forms of arrhythmia but also the spectral structure of the heart rate variability. The shape of the spectral function and long-time autocorrelations of the signal carry a new information, comparing with a standard, short-time, ECG examination and offers, potentially, a new diagnostic tool.

It has been established [2] that the power spectrum of heart rate exhibits the $1/f$ behaviour over a broad frequency domain. This kind of noise, called also a "flicker noise", is widespread in nature. Its presence has been demonstrated in vacuum tubes [3], carbon resistors, but also for see level

fluctuations [4] and in astronomy [5]. The human heart is not the only biological system among living organisms for which these spectral properties have been shown. Recently, it has been reported for the MEG signal from the human brain [6].

The source of the $1/f$ noise for the human heart is not known. A possible mechanism of its generation stems from the obvious observation that this noise comprises many time scales. A typical power spectrum, connected with the exponential decay of the autocorrelation function, has a Lorentzian form: $P(f) = \tau/(1 + \tau^2 f^2)$, where τ is the correlation time which defines the time scale. We can get formally the $1/f$ noise assuming that the time τ, instead of being a constant value, is given by some probability distribution, *e.g.* the lognormal distribution. Physically it means that the phenomenon we are dealing with results from many underlying processes characterised by various time scales, effectively producing a scale-invariant process.

One can easily imagine candidates for such processes in human heart. The His-Purkinje system takes a single nerve pulse and branches it out. Each pathway of this pulse has its own time scale [7]. Moreover, physiological control systems operate on different time scales; the blood pressure is regulated by at least nine different systems that operate on time scales from a few seconds to a few hours [8]. On the other hand, some pathological conditions, affecting metabolic and biochemical parameters, can destabilise His-Purkinje cell membranes. The power spectrum possesses in this case additional peaks because some sort of regular rhythm of heart rate is generated [9].

The self-similar (more precisely: self-affine) nature of the heart rate can be demonstrated by plotting the heart rate data for different resolutions, on different time scales. This means that if we take a sufficiently long time interval and magnify a portion of it, we get a pattern qualitatively similar to the original interval [10]. Therefore, fractal properties are expected. The self-similarity implies power-law dependences of scaled quantities which manifest themselves as a straight line on log-log plots. It is the case, for example, for the detrended fluctuation analysis method [11] which reveals long-range anticorrelations in the heartbeat fluctuations [12]. Moreover, the Hurst analysis is able to demonstrate the self-similar correlations in the heart rate data [13]. However, the heartbeat time series possesses a rather complicated self-similar structure and is not homogeneous enough to be described uniquely by a single singularity exponent. In order to take into account all scaling properties of the signal, one should determine the entire multifractal spectrum [14]. Analysis of fractal characteristics for various cardiac pathologies indicates significant alterations in short and long-range heartbeat correlation properties, suggesting possible clinical applications [15].

In this paper we perform an analysis of ECG signals for a healthy subject and compare the results with those obtained for a patient with a heart disease. In Sec. 2 we present the ECG time series and details of the measurement. The method of extraction of QRS complexes from those time series is described in Sec. 3. The power spectrum for 24 hours heart rate data, for both healthy and diseased cases, are presented in Sec. 4. Sec. 5 is devoted to the heart rate autocorrelation function, its time dependence is calculated for both cases. The most important results are summarised and discussed in Sec. 6.

2. Description of data

The first group of data consists of five electrocardiographic recordings of subjects without clinical evidence of cardiac disease. The healthy group between 25 and 45 years of age underwent a complete physical examination and their medical history revealed no cardiovascular disease. The ECG recordings were monitored: 24 hours for one subject and 8–10 hours for four other subjects. We used three channel semiconductor holters of the "Medilog Oxford" type. Measurements have been performed in the Cardiological Department of the Military Hospital in Cracow.

The second group comprises of three 8 hours ECG recordings of subjects with post-ictal heart rate oscillation in partial epilepsy. The patients ranged in age from 31 to 48 years had partial seizures and post-ictal cardiac oscillations associated with abnormal heart rhythms and Mayer waves [16]. Mayer waves are spontaneous oscillations at frequencies 0.05–0.1 Hz in cerebral blood flow velocity and represent baroreflex activation. These oscillations are caused by action of the sympathetic nervous system and result from time delays in the baroreflex feedback loop for the control of sympathetic nerve activity [17]. The mechanism of post-ictal oscillation in heart rate during partial epilepsy is described in Refs. [18]. The ECG recordings of the group of diseased people has been taken from MIT-BIH database [19].

3. Detection of the QRS complexes

Analysis of the data requires, as a first step, an identification of QRS complexes from ECG recording. The 3-lead ECG recordings were performed. For the purpose of our analysis, it was sufficient to take only one channel; we chose the second one for which the signal amplitude is the largest. Fig. 1 shows how the original ECG signal has been modified to obtain the heart rate time series useful for our studies. From the ECG signal we subtract the local average of that signal, taken over subsequent five points. Crossing of the threshold value with the averaged ECG signal is used to find local

minima corresponding to the sequence of heart beats. We determine the position of each QRS, as corresponding to its minimum. We accept that minimum if the amplitude value is positioned below an assumed threshold. Applying the above procedure, we preserve the original interbeat intervals and avoid unimportant fluctuations caused by the changes of the ECG signal base line level. The accurate detection of QRS peak locations is crucial to study long term heart rate variability. The occurrence time of the n^{th} QRS we denote by t_n. The signal we will analyse in this paper, $X(t)$, is defined as $X(t) = t_n - t_{n-1}$. More details about the detection algorithm of the QRS complexes can be found in Ref. [20].

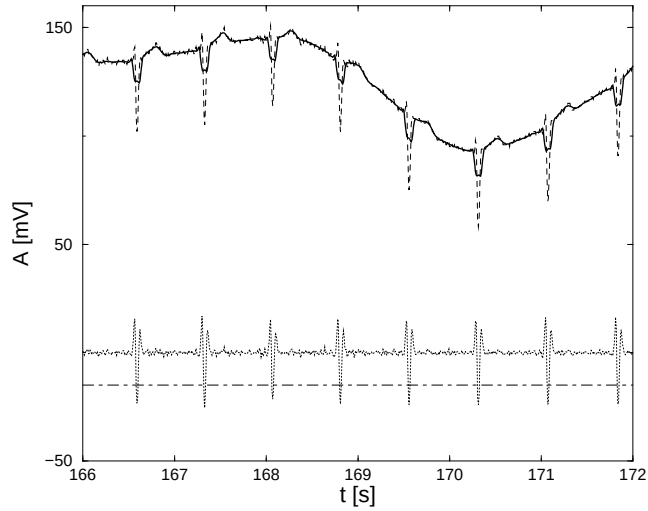

Fig. 1. The principle of the QRS detection method. In the upper part of the figure the measured ECG signal (dashed line) together with the ECG signal calculated as a local averaged over 5 points are presented (solid line). In the lower part of the figure the difference of those two signals is plotted (dotted line). A QRS was accepted if amplitude value outstripped the threshold value −15 mV (dot-dashed line).

4. The spectral analysis

The power spectrum $P(f)$ is defined by the Fourier transform of the time signal $X(t)$ in the following way:

$$P(f) \sim \left| \int_0^\infty X(t) \cos(2\pi f t) dt \right|^2 . \tag{1}$$

To perform the Fourier integral, we have used the FFT algorithm [21].

4.1. The healthy heart

First we consider the heart rate $X(t)$ for the healthy subject and calculate its power spectrum according to Eq. (1). The result is presented in Fig. 2. The curve has the shape $1/f$ within the frequency interval (f_1, f_2), where $f_1 = 0.003$ Hz and $f_2 = 0.1$ Hz. At higher frequency a peak originating from the breathing process emerges: it is centred around 0.2 Hz and reaches up to 0.3 Hz. In the highest frequency limit, the shape $1/f^2$ is clearly visible. This kind of noise, called the brown noise, is defined as an integral from the white noise and it characterises the Wiener process (diffusion).

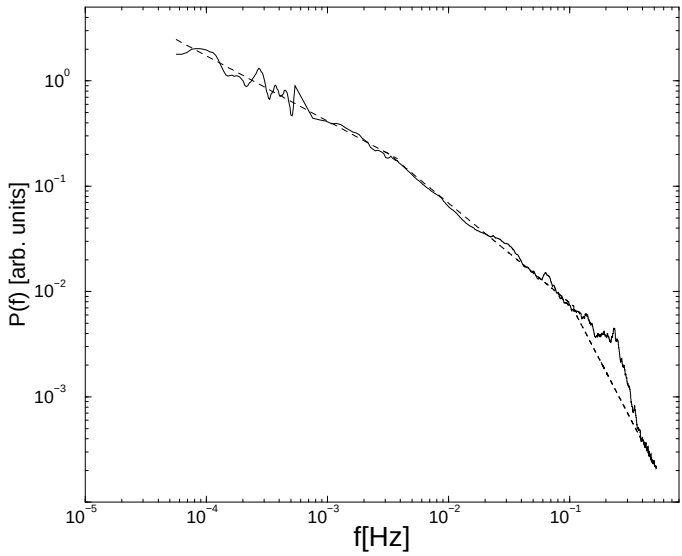

Fig. 2. The power spectrum of the heart rate for a healthy subject (solid line) and its power-law analytical representation, according to Eq. (2) (dashed line).

The $1/f$ noise is difficult to handle mathematically because the integral $\int_0^\infty P(f)df$ diverges at both ends in this case. For stationary processes the integral must assume a finite value [22]. Consequently, in any theory of such processes the $1/f$ law can only appear for intermediate frequencies, *i.e.* the spectrum must possess cut-off at some high, as well as some low, frequency value. Such requirement is obvious if characteristic times are limited from above and from below. For example, the McWhorter theory [23] of noise in semiconductors predicts the spectrum consisting of three parts: the flat spectrum at small frequencies (white noise), $1/f$ segment and, finally, the brown noise ($\sim 1/f^2$).

For the human heart we are not able to point out what a mechanism is responsible for the appearance of $1/f$ behaviour and we cannot prove stationarity itself. However, the data themselves indicate the existence of both frequency cut-offs. Since the time series has a limited length, the spectrum is determined with a finite accuracy. The error manifests itself as oscillations of the power spectrum curve and it is especially large at low frequencies. That frequency domain is the most important because it corresponds to the long-time behaviour of the system. Nevertheless, the shape of the curve in this region (Fig. 2) apparently obey the power law $f^{-\alpha}$ with the numerically estimated exponent $\alpha = 0.65$. Therefore, the power spectrum for the case under consideration is integrable at both sides, suggesting that we are dealing with some sort of stationary process.

The shape of power spectrum at small frequencies is determined by long-time limit of the heart rate signal $X(t)$. Therefore, the length of the data set is crucial for the above analysis. One can ask to what extend our conclusions are sensitive on the finiteness of the time series. In order to check that, we reduced length of the data by 20%. The resulting power spectrum exhibits enhanced oscillations at small frequencies. However, the power law in this region is still recognisable and the fitted exponent differs from the value for the full data by less then 2%. The rest of the spectrum is only slightly distorted by shortening of the time series.

We can then conclude that the power spectrum of the healthy heart rate can be represented by a juxtaposition of three power law dependences:

$$P(f) = \begin{cases} \frac{A}{f^{0.65}} & \text{for } f < f_1 \,, \\ \frac{B}{f} & \text{for } f_1 \leq f \leq f_2 \,, \\ \frac{C}{f^2} & \text{for } f > f_2 \,, \end{cases} \tag{2}$$

where the constants A, B and C are evaluated from the continuity requirement; the frequencies dividing subsequent intervals are: $f_1 = 0.003$ Hz and $f_2 = 0.1$ Hz. Assuming the above Ansatz for the further analysis, allows us to get rid of statistical fluctuations, as well as of some details which are rather trivial, *e.g.* the peak from respiration.

We performed the same analysis for the other five cases of healthy subjects. It confirmed the presence of $1/f$ noise. However, it was impossible to study the low frequency limit because time series were considerably shorter.

4.2. A pathological case

We have performed a similar analysis for the data originated from a patient with post-ictal cardiac oscillations. The power spectrum is presented in Fig. 3. The shape of the curve differs substantially from that obtained for

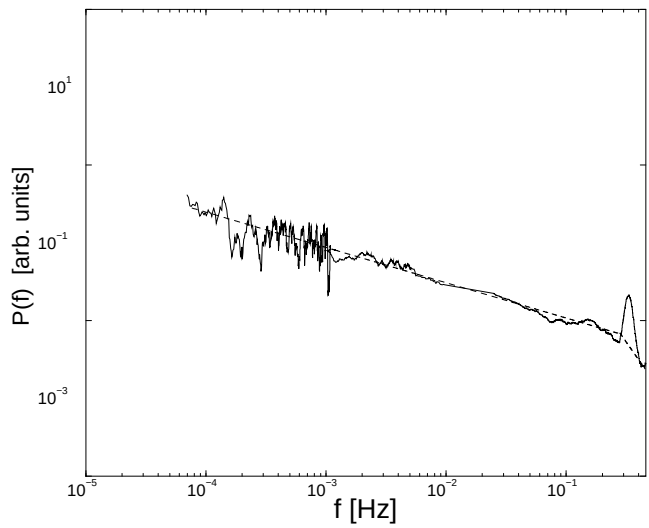

Fig. 3. The power spectrum of the heart rate for a subject with post-ictal heart rate oscillation in partial epilepsy (solid line) and its power-law analytical representation, according to Eq. (4) (dashed line).

the healthy heart. It obeys the power law dependence in a broad frequency range but the exponent is unique and much smaller, it equals 0.5. Therefore, the noise $1/f$ is lacking. For pathological case a sharp peak develops at high frequency edge of the spectrum, at about $f = 0.3$ Hz. It originates from the respiratory sinus arrhythmia. Oscillations on the left hand side of that respiratory peak can be interpreted as Mayer waves [24]. Since they are very small, we neglect them in further analysis. The respiratory sinus arrhythmia peak is much more distinguished than the respiratory peak for the case of healthy heart. It can be parameterised by a Gaussian. If we take this peak into account, the power spectrum for the pathological case can then be cast in the following form:

$$P(f) = \begin{cases} \frac{A}{f^{0.5}} & \text{for } f < f_1 \,, \\ B \exp\left(-\frac{(f-f_0)^2}{2\sigma^2}\right) & \text{for } f_1 \leq f \leq f_2 \,, \\ \frac{C}{f^2} & \text{for } f > f_2 \,, \end{cases} \tag{3}$$

where $f_0 = 0.33$ Hz, $\sigma = 0.03$ Hz, $f_1 = 0.27$ Hz and $f_2 = 0.38$ Hz.

Alternatively, we can consider the power spectrum with the respiratory peak removed, similarly as for the case of healthy heart, and obtain the following simple parameterisation:

$$P(f) = \begin{cases} \frac{A}{f^{0.5}} & \text{for } f < f_1 \,, \\ \frac{C}{f^2} & \text{for } f > f_1 \,, \end{cases} \tag{4}$$

where $f_1 = 0.278$ Hz.

5. The determination of the heart rate autocorrelation function

For any stochastic process, a quantity of interest is its autocorrelation function which is a measure of the influence of the process value $X(\tau)$ at some initial time τ on its value at time $\tau + t$. It is defined as the so-called lagged product sum (time average):

$$C(t) = \lim_{T\to\infty} \frac{1}{T} \int_0^T X(\tau) X(\tau + t) d\tau. \tag{5}$$

In the case of heart rate dynamics, this quantity allows us to quantify a memory of the heart, to determine to what extend the information about heart activity is preserved with time.

It is possible to evaluate $C(t)$ directly from the Eq. (5). However, that procedure is not convenient because it leads to large rounding errors [25]: we are interested in system's behaviour at large times for which $C(t) \to 0$. To avoid that difficulty, we derive the autocorrelation function as the Fourier transform from the power spectrum $P(f)$, using the Wiener–Khinchin theorem [26]:

$$C(t) = 4\pi \int_0^\infty P(f) \cos(2\pi t f) df \,, \tag{6}$$

where we have taken into account that $P(f)$ is an even function. The advantage of this method stems from the fact that asymptotic behaviour of $C(t)$ is determined by small frequencies and $P(f)$ has its maximum at $f = 0$. Therefore, in the most important region the rounding errors are small.

Let us consider first the case of the healthy heart. Inserting the power spectrum (2) into Eq. (6), we get the following expression:

$$C(t) = A \int_0^{2\pi f_1} (2\pi f)^{-0.65} \cos(2\pi f t) df + B[\mathrm{ci}(2\pi f_1 t) - \mathrm{ci}(2\pi f_2 t)] + \frac{C}{2\pi}\left[\frac{\cos(2\pi f_2 t)}{2\pi f_2} + t\,\mathrm{si}(2\pi f_2 t) - \frac{\pi}{2}\right], \quad (7)$$

where $\mathrm{si}(x)$ and $\mathrm{ci}(x)$ denote the integral sine and integral cosine, respectively. This result is shown in Fig. 4. The autocorrelation function falls very slowly with time — after a few hours a considerable amount of information about the initial state of the system still remains. The asymptotic time dependence of $C(t)$ is easy to determine. Since in this limit only small values of frequency contribute to the Fourier integral, we can take into account only the first branch in (2) and extend the upper limit to the infinity:

$$\int_0^{2\pi f_1} (2\pi f)^{-0.65} \cos(2\pi f t) df \approx \int_0^{\infty} (2\pi f)^{-0.65} \cos(2\pi f t) df \sim t^{-0.65} \quad (t \to \infty)\,.$$

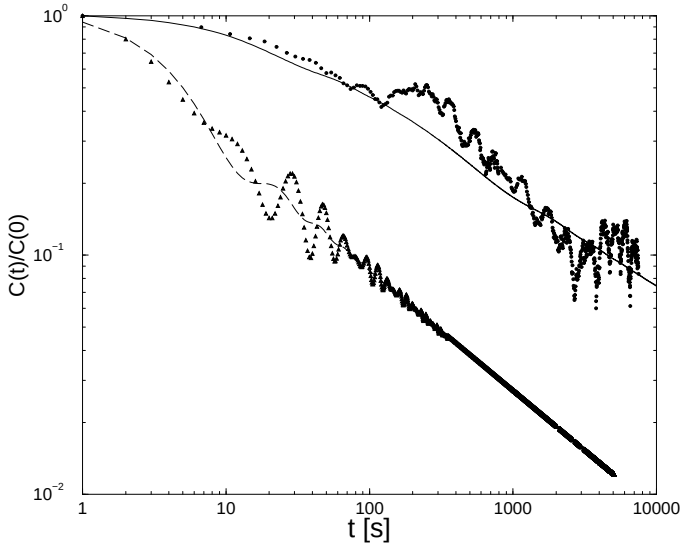

Fig. 4. The heart rate autocorrelation function for healthy subjects calculated from the numerically estimated power spectrum via Eq. (6) (dots) and from its analytical representation according to Eq. (2) (solid line). The autocorrelation function for a diseased patient has been calculated from both analytical representations: with the respiratory sinus arrhythmia peak, using Eq. (3) (triangles), and without it, using Eq. (4) (dashed line).

For comparison, we have calculated the same quantity by inserting to Eq. (6) the original power spectrum in the numerical form, instead of its analytical representation (2). The curve, also presented in Fig. 4, is very rough but the general tendency of its fall-off agrees with the analytical result.

We have performed the same calculations for the pathological case. The autocorrelation function obtained by taking the Fourier transform from the expression (3) is shown in Fig. 4. A characteristic feature of this curve is the presence of oscillations which die down at about $t = 100$ s. They correspond to respiratory sinus arrhythmia peak in the power spectrum (Fig. 3). It becomes clearly visible if we evaluate $C(t)$ from the power spectrum analytical representation without that peak (Eq. (4)). The autocorrelation function declines faster in the pathological case, comparing with the result for the healthy heart. Asymptotically, it approaches the power law dependence: $t^{-0.5}$.

6. Summary and conclusions

We have presented power spectra obtained from analysis of heart rate data for both healthy and diseased cases. The healthy heart exhibits the spectrum typical for the $1/f$ noise which can be interpreted as a result of contribution of many processes possessing different time scales. The $1/f$ shape of the spectrum is restricted to some interval of intermediate frequencies. At high frequency limit a typical brown noise is observed. That fact can be an indication that only one characteristic time prevails there. At low frequency the spectrum obeys the power law with such an exponent that the entire spectrum is integrable. Therefore, there exists a finite variance and the heart rate seems to behave like a stationary stochastic process. On the other hand, there is no trace of $1/f$ pattern for patients with post-ictal heart rate oscillation in partial epilepsy. The power spectrum still obeys the power law but the exponent is smaller.

Our aim was to extract from the numerical power spectrum its most essential features, eliminating unimportant details, statistical fluctuations and measurement errors. We have shown that algebraic dependence of $P(f)$ on frequency, with different indices for low, intermediate and high frequency, can properly represent the measured power spectrum. This parameterisation allowed us to obtain a simple and clear time dependence of the heart rate autocorrelation function $C(t)$ and to extrapolate it to large times, regardless the finite length of the time series. $C(t)$ declines very slowly with time, asymptotically according to the power law.

The heart rate autocorrelation function dies down more rapidly for the pathological case than for the healthy heart. Therefore, the diseased heart looses its memory faster, it behaves more "random". The difference is espe-

cially prominent at relatively short times. These observations are, in fact, consistent with a more elaborate multifractal analysis [27] which indicates a significantly higher degree of dynamical complexity of healthy human heartbeat, compared to the pathologic conditions. In many cases, however, the characteristics which are relevant from the practical point of view seem to be quantifiable already using the method discussed here.

Some methods based of statistical analysis of ECG signals *e.g.* mean fluctuation function, can also be useful in distinguishing healthy subjects from patients with cardiac pathology [28]. Therefore, they can serve as a tool for diagnostic aims. Similarly, the spectral analysis described in this paper, which takes into account long-time behaviour of the heart rate, has a diagnostic advantage over traditional cardiological methods. A short-time ECG examination is in many cases not sufficient to recognise the pathology what results in a sudden epileptic death [29]. The applicability of presented methods for diagnostic aims in the case of post-ictal heart rate oscillation in epilepsy suggests that other cardiac diseases can be dealt with in a similar way. However, that problem must be addressed individually for each particular illness.

The statistical properties of measured heart-rate signals can be regarded either as some sort of intrinsic noise or as a result of an underlying nonlinear deterministic dynamics [30]. One can also consider a possibility of a model combining both approaches in the form of a nonlinear Langevin equation. Since the noise exhibits long-time autocorrelations, the apparent deterministic chaos would be suppressed in this case and characterised by Lapunov exponents smaller, compared to the purely deterministic system [31]. They can even fall to zero for large noise amplitude. This problem of regularisation by the noise needs to be checked for specific models of heart dynamics.

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