Changes in the Hurst exponent of heartbeat intervals during physical activity

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The fractal scaling properties of the heartbeat time series are studied in different controlled ergometric regimes using both the improved Hurst rescaled range (R/S) analysis and the detrended fluctuation analysis (DFA). The long-time "memory effect" quantified by the value of the Hurst exponent $H>0.5$ is found to increase during progressive physical activity in healthy subjects, in contrast to those having stable angina pectoris, where it decreases. The results are also supported by the detrended fluctuation analysis. We argue that this finding may be used as a useful new diagnostic parameter for short heartbeat time series.

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The normal human heart interbeat [(RR) interval] time series, is extremely inhomogeneous and nonstationary. It fluctuates around the mean value in an irregular and complex manner, even under resting conditions [1]. The presence of scaling properties suggests that fractal analysis may provide a method to recognize diseased states of the heart by studying changes in the scaling properties. It has been observed that scaling is degraded in some diseased states [2–4]. Also, the significant scaling differences were found between rest and exercise for healthy individuals [5]. The heart-rate variability (HRV) under controlled physical activity has been partially studied in [6–8]. Some recent studies connected with exercise [5,9] used only healthy subjects in their measurement, and under specific conditions.

In this paper we examine the scaling properties of the heartbeat time series in different controlled ergometric regimes, for healthy individuals as well as for those having stable angina pectoris (SAP). We use ambulatory RR data gathered from stress test, as a possibility to distinguish healthy individuals from individuals with SAP [10]. The long-time Holter data, obtained while the subject is leading a normal everyday life, are not suitable for our analysis since the strength of the physical activity is unknown. The usual diagnosis of SAP through stress tests is based on the shape distortion of the ST wave. We believe that our approach could improve current methods of SAP diagnosis.

Fluctuations in RR intervals during one of our ergometric measurements are shown in Fig. 1. Both time series look very similar and we cannot say which is from a healthy and which is from a SAP individual. Spikes seen in the figure are ectopic heartbeats or *extrasystole* (ES). These spikes are not a sign of SAP, for even a healthy subject can have numerous ES in their RR records.

The time series of RR intervals in our controlled ergometric measurement had a time duration of about 15 min ($\simeq$2000 beats). This type of measurement is used as a routine in the everyday clinical diagnostic procedure because some heart diseases, such as SAP, usually become transparent under physical activities. Each measurement is designed to consists of a resting period (pretrigger Pt), a few stages (P1–P4) of running on an inclined belt, and a period of relaxation (Re). Regimes of physical activity are defined according to the standard Bruce protocol [11] (Table I), with a time duration of 3 min for each program. The pretrigger part has a variable duration and is limited for analysis to the first 30 sec in each measurement. The relaxation period is restricted to 6 min.

In the present paper only regimes P1 and P2 are analyzed. Except in a few singular cases (Fig. 1; bottom), our SAP patients were not able to complete regimes higher than P2, and the measurements for them were stopped for medical reasons.

The ECG ergometric data were digitized at a sampling time of 1 ms by the WaveBook 512 (Iotech. Cal. USA), and transferred to a computer for further analysis. All questionable portions of RR intervals were excluded manually, and only segments with >90% sinus beats were included in the final analyses.

RR records with numerous ES (more than 11% of the whole beat number) were totally excluded from consideration. Some ES, if existing in regimes with longer duration (exercise programs and relaxation periods), were not excluded. However, in short-time rest-condition measurements (Pt periods), because of small beat number and their high influence on the result, all ES were excluded. We believe that ES should be kept, in general, during the analysis since they also contain information about the dynamical state of the heart [12].

In order to estimate the Hurst exponent in series of RR intervals during controlled physical activity, we apply the rescaled range (R/S) method [13,14]. We are interested in the capability of the R/S method to distinguish the patients with SAP from the healthy subjects.

The nonstationarity in the RR time series, caused by physical activity, is removed by a third-order polynomial regression, separately for each regime of measurement. We have found that this simple polynomial regression successfully removes the influence of exercise on the RR data profile, and even removes some of the unusual drifts sometimes found in RR recording at rest. The improved R/S analysis is then performed on the data representing deviations of RR intervals from a trendline [Figs. 2(b) and 2(c)].

Denoting by {u(n)} the deviation of RR data from the trendline, we calculate the running means $\overline{u}(n)$ for a given n and the accumulated deviations from the mean $X(l,n), l=1,\ldots,n$ using

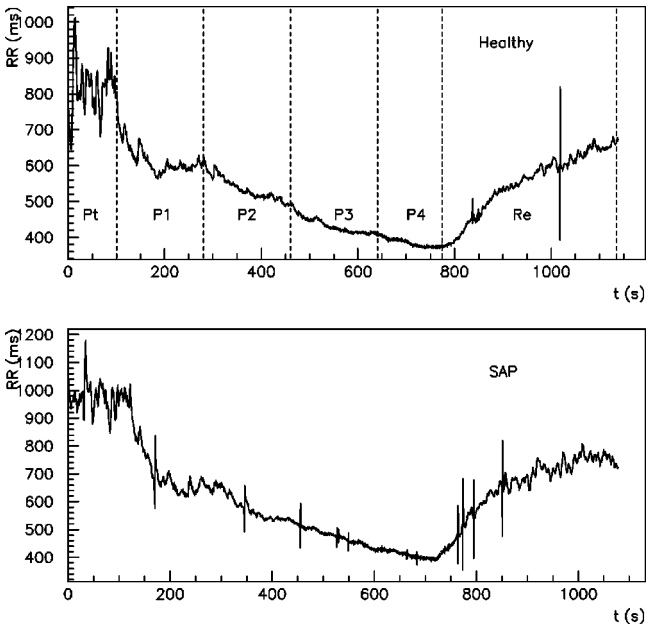

FIG. 1. RR intervals of a healthy (top) and a SAP (bottom) subject in one of our ergometric measurements. The global nonstationarity as result of physical activity is clearly seen. Pt corresponds to the rest-condition measurement, P1–P4 to running stages on a belt with increasing intensity, and Re to a relaxation period after stopping the moving belt.

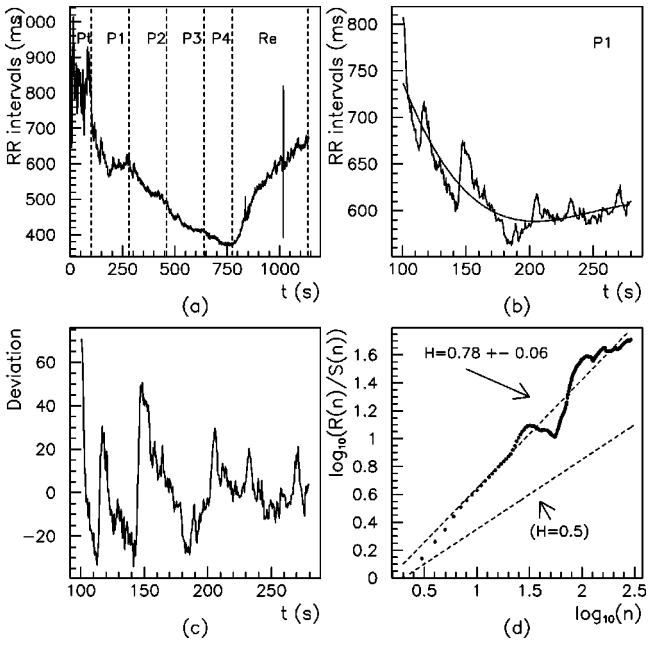

FIG. 2. An example of R/S analysis; (a) Typical shape of RR interval sequence, (b) RR intervals in P1 with a third-order polynomial regression trendline, (c) deviations from the polynomial trendline, and (d) R/S analysis of data in (c) in comparison with the random data ($H=0.5$).

$$\overline{u}(n) = \frac{1}{n}\sum_{k=1}^{n} u(k),$$

$$X(l,n) = \sum_{k=1}^{l} [u(k) - \overline{u}(n)].$$

The range $R(n)$ is defined as a distance between the minimum and the maximum value of X:

$$R(n) = \max_l X(l,n) - \min_l X(l,n).$$

The rescaled range (R/S) is obtained by dividing $R(n)$ with the standard deviation $S(n)$:

$$S(n) = \sqrt{\frac{1}{n}\sum_{k=1}^{n} [u(k) - \overline{u}(n)]^2}.$$

R/S is expected to show a power-law dependence on the box size n:

$$R(n)/S(n) \sim n^H,$$

where H is the Hurst exponent. The relationship between the fractal dimension D and H is [14]

$$D = 2 - H.$$

The time series can be divided into three distinct categories: $H<0.5$, $H=0.5$, and $H>0.5$. The case $H=0.5$ corresponds to random or uncorrelated data. If $H>0.5$, the data are persistent and characterized by long-time correlations or "memory" effects on all time scales. The strength of the persistence increases as H approaches 1.0. The time series with $H<0.5$ is antipersistent, which means that the time series data are negatively correlated.

Our modification relative to the standard Hurst R/S analysis [14,15] consists of using only one box whose width increases from $n=2$ to $n=N$ (the whole series), so that in this way the ordering in the initial series is preserved. In our procedure the boxes of larger width become more relevant for determining the Hurst exponent. The pronounce differences between healthy and SAP subjects are observed in this case.

The R/S analysis was performed on four separate regimes: resting Pt, running programs P1 and P2, and relaxation Re. However, the programs P1 and P2 are only relevant for our analysis and results. The Pt period was too short

TABLE I. Defined regimes in ergometric measurement: Bruce protocol [11].

Program	Belt angle (°)	Belt velocity (km/h)
P1	10	2.7
P2	12	4
P3	14	5.5
P4	16	6.9
P5	18	8

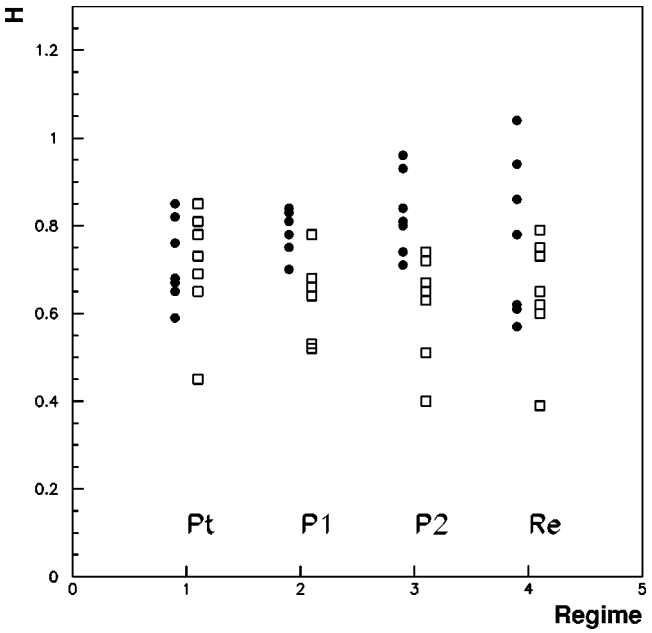

FIG. 3. The H from R/S analysis for each subject in the group (scatter plot), in different regimes of ergometric measurement. Dots denotes healthy subjects and squares SAP subjects.

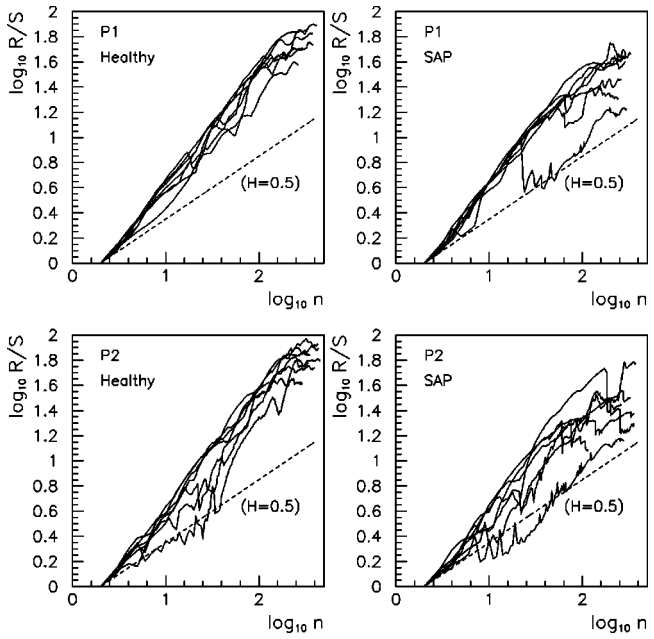

FIG. 4. Individual R/S points as a function of the box-size n in P1 and P2 regimes. Points are connected for clarity by lines for each individual.

(30–50 heartbeats) and the Re period started when the moving belt was stopped, usually somewhere inside of the P3 or P4 programs, for medical reasons.

Our patients were divided into two groups: the group with evidence of ishemic ST-segment depression of more than 1 mV (SAP subjects) and the control group of healthy subjects. The selection of subjects was performed by a cardiologist according to the generally accepted medical knowledge.

As pointed out earlier, our R/S calculation was based on the deviation of the original RR intervals from the trendline. The main steps are shown in Fig. 2 for P1 regime. The procedure adopted here is to calculate R/S for a box of n elements, starting with the first two elements. In each next step one more element is added, and R/S is calculated for the wider box. The process is continued until the box of length N (the whole data set) is reached. H is evaluated as a slope of the least-squares fit line in the log(R/S) versus log(n) plot, using the whole span of the data. In this way, we preserve the ordering of RR intervals during the calculation.

Figure 3 shows the values of H from the R/S analysis for each individual during ergometric measurements. It involves 14 independent measurements on seven healthy plus seven SAP subjects.

Depending on the regime type, H generally exceeds 0.5. In the Pt regime, both healthy and SAP subjects have H about 0.7 and we cannot distinguish these two groups. The situation changes in regimes under physical activity. The difference between healthy and SAP subjects is clearly seen in the P1 and P2 programs. The H for healthy subjects increases with increasing running intensity, while H for SAP subjects decreases in the same (P1 and P2) regimes. Significance of the separation is estimated by the t-test. We find the value 6.18 (4.65) for P1 (P2) regime, which corresponds to the confidence level $P < 0.001$.

For better understanding of our results, shown in Fig. 3, the data points of log(R/S) as a function of log(n), for each subject are plotted in Fig. 4, for P1 and P2 regimes, respectively. A power law fit to the entire data, including the region where one observes a saturation of R/S, is made (Figs. 3 and 4). Since there will be many points in that region, the fit is going to be strongly constrained by that saturation regime, leading to an underestimation of the exponent. Apparently, linear trends observed in SAP patients tend to be globally closer to random data behavior ($H=0.5$). We observed that individual R/S points for larger n became more spread for SAP subjects, in comparison to healthy subjects. Also, the oscillations around linear trendlines are larger for SAP patients than for healthy subjects. In general, the slope of R/S analysis is n dependent and oscillates around the linear trend for large n. Similar behavior has been observed in the R/S analysis of DNA sequence [16]. An oscillating behavior reflects local nonhomogenities, remaining drifts, and presence of ES in RR data, which were not canceled in our method of R/S calculation. Such oscillations around $H=0.5$ are also observed when RR data points are randomly shuffled. Therefore, a very good linearity in power law is generally not observed, and scaling exponent (H) represents the global behavior of linear trends with oscillations around it included. This type of behavior suggests the presence of multiple timescale processes related to multifractality of the RR data under study [3].

We have also compared our results to the usual method of detrended fluctuation analysis (DFA) [2,17]. Our results with the R/S method are supported by the DFA, which shows similar behavior of distinguishing healthy from SAP subjects in the P2 regime. We have used the first-order DFA, on the data set with a third-order polynomial trend removed. For consistency, in the detrending procedure we have included

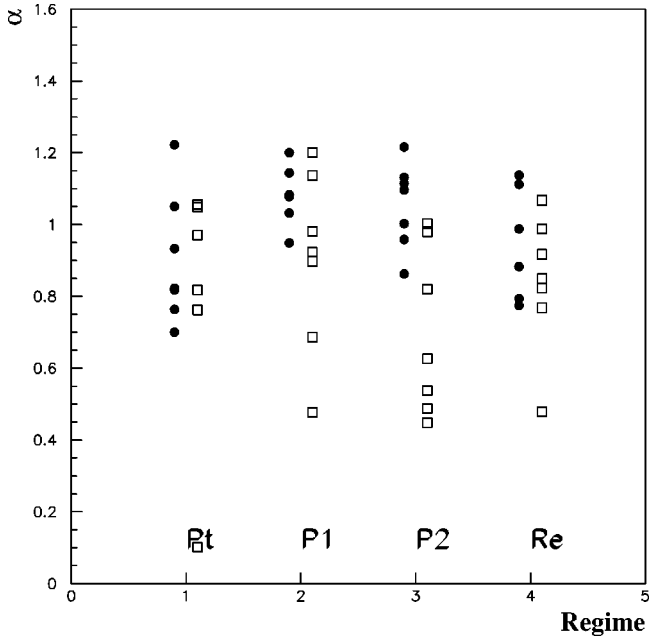

FIG. 5. The scatter plot of the DFA scaling exponents α.

the maximal box sizes of width $n=N$ (whole data set) and $n=[N/2],[N/3]$. These box sizes have also been included in the R/S analysis, where n means that there are exactly n points in each box. The importance of long boxes for the determination of α is less than in the corresponding R/S method. We have found that the DFA is less efficient than the R/S method in separating healthy subjects from SAP patients in P1 and P2 programs (Fig. 5). As estimated by *t*-test, the significance of the separation is 2.88 (4.67) for the P1 (P2) regime, which corresponds to the level of probability $0.01<P<0.02$ (P1) and $P<0.001$ (P2). That is, separation in P1 is less pronounced with the DFA method than it is in comparison to the corresponding R/S result. In the P2 regime, the confidence level of DFA is the same as it was in the R/S analysis.

Our results with the R/S analysis show a clear separation between SAP and healthy subjects in the P2 regime of physical activity. Further studies in larger populations are needed to confirm this result. If the observed trend would continue in larger statistics, the R/S analysis could become a useful method in separating SAP subjects from healthy ones, especially in borderline cases where a clinical diagnosis cannot be set from electrocardiogram (ECG) measurements only.

In conclusion, we are reporting on an analysis of heart-rate data during exercise that appears to provide a window into the diagnosis of angina pectoris. We have shown that fluctuations in heartbeat time series in controlled ergometric regimes exhibit fractal properties when analyzed using the improved rescaled range (R/S) method (Fig. 4), as well as when using the DFA (Fig. 5). The R/S analysis for ergometric measurements is described by the Hurst empirical law $R/S\sim n^H$, for $2\leqslant n<400$. Figure 2(d) shows data for the range 3–260 and, in this case, a good power-law behavior is observed for only 3–20. Oscillatory behavior of the data points around the linear trendline is seen in each case, as is shown in Fig. 4. These oscillations arise from the multiscale nature of RR data that were not canceled out by our way of R/S analysis. They are a signature of multifractality in RR data. The Hurst exponent H during progressive physical activity is generally $H>0.5$ and increases for healthy subjects, in contrast to SAP subjects where it is found to decrease.

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