Fluctuations of the human heart beat constitute a complex system that has been studied mostly under resting conditions using conventional time series analysis methods. During physical exercise, the variability of the fluctuations is reduced, and the time series of beat-to-beat RR intervals (RRI) becomes highly non-stationary. Here we develop a dynamical approach to analyze the time evolution of RRI correlations in running across various training and racing events under real-world conditions. In particular, we introduce dynamical detrended fluctuation analysis and dynamical partial autocorrelation functions, which are able to detect real-time changes in the scaling and correlations of the RRI’s. We relate these changes to the exercise intensity quantified by the heart rate. Beyond a certain intensity threshold, RRI’s show strong short-time anticorrelations on different scales, which we relate to physiological hemodynamics. The results demonstrate the feasibility of dynamical statistical analysis of RRI’s to monitor exercise load in real time with wearable devices and without previous knowledge of external parameters such as the maximum heart rate of the individual.

I. INTRODUCTION

The increasing popularity and accuracy of wearable devices and sensors present new opportunities to study human physiology in a continuous, non-invasive manner for a huge number of "subjects" under real-world conditions. These devices enable the measurement of a plethora of physiological and mechanical signals such as the heart rate, beat-to-beat (RR) intervals, overall motion via GPS, motion of specific body locations via accelerations, and skin temperature. These data can be recorded in real time, often at one second intervals, and uploaded to web services. To date, most recorded data are not analyzed in scientific rigour due to a lack of suitable models for the dynamics of physiological signals under various intensities of exercise load, and also due to restricted availability of the data (property of industry and users). This limits the opportunities for a better understanding of complex physiological processes, diagnostics and monitoring for patients in rehabilitation, and the optimal training of athletes. However, it has been long known that a variety of physical conditions and cardiac diseases affect the correlations in RR intervals [1].

In exercise physiology, heart rate variability (HRV) is often used at rest to evaluate recovery, fatigue and over-training. It is known that during exercise the overall variability of the RR intervals is strongly suppressed. Regardless, the RR interval correlations contain valuable information even during exercise 2 3. For example, the determination of certain physiological thresholds such as the ventilatory threshold from the frequency spectrum of HRV has been examined [4]. Often the relative importance of low-frequency (LO: 0.04–0.15 Hz) and high-frequency (HI: 0.15–0.4 Hz) spectral power is studied during exercise. Using this concept as a measure of the relative sympathetic (SNS) and parasympathetic nervous system (PNS) activity during exercise, it has been shown that the PNS activity decreases dramatically during exercise, while the SNS activity remains unchanged up to the ventilatory threshold, whereas it increases abruptly at 110% of the ventilatory threshold [5]. However, it is known that Fourier decomposition of dynamic signals is often hampered by non-stationarity. Karavirta et al. have studied heart rate dynamics and its complexity from short-term recordings during moderate exercise to quantify exercise-induced changes on the cardiovascular system [6]. They found that endurance exercise leads to increased HRV complexity as quantified by the multiscale entropy analysis.

Here, we study real-time correlations of RRI’s during exercise of various intensities. The data were recorded during real-world (outside the laboratory) running exercises and races by using heart-rate monitors linked to a GPS running watch. Two groups of data are analyzed: Everyday runs of varying duration and intensity, and Marathon races. A dynamical version of detrended fluctuation analysis (DDFA) is developed and applied to the RR time series to extract time- and scale-dependent scaling exponents that characterize dynamic multiscale correlations. The scaling exponents are studied both as a function of the exercise time and as a function of the exercise intensity as measured by the binned average heart rate. The findings are supplemented by a direct study of local partial autocorrelation functions for lags of a few beats.

Our main result consists of two observations. First, we
demonstrate that the here developed DDFA method can reliably determine the dynamic, scale-dependent scaling exponent $\alpha(t,s)$. Hence, it provides a powerful method for measuring multiscale correlations of non-stationary physiological signals. Second, we find strong evidence for measuring multiscale correlations of non-stationary intensity. We conclude with some remarks in Sec. V.

It is important to understand the physiological mechanism causing the observed anticorrelations. With increasing exercise intensity, the stroke volume of the heart is relatively stable (after an initial increase at low upright exercise intensity). The increased oxygen demand during exercise is satisfied by a decline in the peripheral vascular resistance due to arteriolar dilation and a resulting increase in the cardiac output. This is the so-called peripheral model of circulatory control [7]. It was found that during treadmill exercise to exhaustion the calculated peripheral resistance dropped by 60% [8]. As the ventricles of the heart do not enlarge during exercise, the increased volume load must be satisfied by an increased heart rate. A possible response to an increased systemic venous return of blood to the heart is the so-called Bainbridge reflex. It is a sympathetic stimulation of the heart rate (tachycardia) due to the increased right atrial pressure, and reflex bradycardia with volume reduction. This reflex has been first discovered in dogs [3] but more recently its presence in human subjects has been confirmed [10]. This observation is consistent with the above mentioned abrupt increase of SNS activity above the ventilatory threshold. We argue that the complex mechanism that maintains the stroke volume and hence the mechanical efficiency of the muscle fibers of the left ventricle in a narrow range during rapid fluctuations in systemic venous return during exercise can cause the observed anticorrelations of the RRI’s.

The paper is organized as follows. In Sec. [11] we describe the heart rate data collected for the study and its preprocessing. In Sec. [14] we describe the methods by starting with the (static) DFA and PACF, followed by the dynamic segmentation and finally by the dynamic versions of these methods, i.e., DDFA and DPACF. Section [V] provides our results for the scaling of the heart-beat fluctuations during exercise and for the relation between scale-dependent RRI correlations and exercise intensity. We conclude with some remarks in Sec. V.

II. MATERIALS

A. Heart Rate Data During Exercise

All heart rate data for this study have been collected during regular running training and racing under real-world conditions, i.e., outside the laboratory. Two groups of data were used for our theoretical analysis. The first group of data, in the following called “group T”, were recorded by human volunteers during their regular running training with freely chosen intensity and volume. The study period was at least 4 weeks, and some subjects provided data over a longer period of time. We obtained institutional approval and informed consent (see acknowledgments for reference). This group involved 12 volunteers (5 female, 7 male, with an age span from 27 to 65 years). Their performances span a wide range from top national level to recreational runners: the personal bests in 10 km range from 29 min 31 sec to 44 min 57 sec, in marathon from 2 hours 43 min 20 sec to 4 hours 26 min 3 sec.

During exercises, heart rate (HR), RR intervals (RRI), running velocity and distance were recorded using a Garmin heart rate monitor HRM4-Run and a GPS watch (Forerunner 935, Garmin Inc., Olathe, KS, USA). A previous study has investigated and validated the accuracy of this HRM [11]. The data were recorded by the GPS watch in the Flexible and Interoperable Data Transfer (FIT) format [12] and subsequently uploaded by the subjects to a web service that we had launched for this study. The total number of exercise files analyzed per subject (samples) varied between 18 and 261, with total covered distances from 150 to 1889 km.

The second group of data, in the following called “group M”, was obtained by selecting randomly the Marathon races of 7 subjects from data uploaded to the Polar Flow web service [13]. Within registration to Polar Flow, the subjects have given their consent for the use of personal data and transferring personal data. The metadata were provided by the users of this web service (all male, with an age span from 28 to 53 years, and Marathon finishing times between 3 hours 30 min and 4 hours 17 min). HR and RRI’s were recorded for this group of subjects with a Polar heart rate monitor H10 and a Pro Strap (Polar Electro Oy, Kempele, Finland). Recently, the RR signal quality of this HRM has been shown to be excellent from low- to high-intensity activities in comparison to a ECG Holter device [14]. In both groups T and M, the subjects provided their maximum heart rate. A summary of all metadata for the two groups is shown in Tab. I.

B. Data preprocessing

Artifacts in the data are removed prior to the analysis by utilizing the following scheme:
TABLE I. Summary of the metadata for the two groups of subjects: Training runs of various durations and intensities (T) and Marathon races (M). For group T shown are the age, gender, resting heart rate $HR_{\text{rest}}$ and maximum heart rate $HR_{\text{max}}$ (as reported by the subject), the personal best (PB) time for 10 km and Marathon (time in hh:mm:ss) within 3 years before this study, the number of exercise samples analyzed, the average heart rate $HR_{\text{avg}}$ of all samples, and the total distance and duration of all samples. For group M all subjects were male, and shown are their age, resting heart rate $HR_{\text{rest}}$ and maximum heart rate $HR_{\text{max}}$ (as reported by the subject), Marathon finishing time, and average heart rate $HR_{\text{avg}}$ during the Marathon.

| Group T | | | | | | |
|---------|---|---|---|---|---|---|
| subject | age [y] | gender | $HR_{\text{rest}}$ | $HR_{\text{max}}$ | PB 10 km | PB Marath. | samples | $HR_{\text{avg}}$ | distance [km] | duration [h] |
| T01     | 48  | m   | 40  | 192 | 00:36:49 | 02:50:49 | 186 | 151 | 1527 | 121.5 |
| T02     | 27  | m   | 40  | 193 | 00:29:31 | –     | 54  | 130 | 576  | 43.5  |
| T03     | 29  | f   | 43  | 200 | 00:36:50 | 02:47:56 | 20  | 155 | 170   | 13.7  |
| T04     | 29  | f   | 50  | 205 | 00:42:30 | –     | 103 | 167 | 1076  | 92.8  |
| T05     | 39  | f   | –   | 200 | 00:44:57 | 03:40:09 | 15  | 176 | 150   | 11.7  |
| T06     | –   | m   | 40  | 192 | 00:35:22 | 02:43:25 | 26  | 153 | 666   | 49.3  |
| T07     | 33  | m   | 42  | 214 | 00:33:56 | 02:43:20 | 261 | 154 | 1889  | 138.7 |
| T08     | 37  | f   | 43  | 200 | –     | 04:26:03 | 20  | 149 | 209   | 22.1  |
| T09     | 27  | m   | 48  | 195 | 00:31:32 | –     | 21  | 143 | 199   | 16.7  |
| T10     | 37  | f   | 45  | 179 | –     | –     | 18  | 154 | 215   | 18.1  |
| T11     | 65  | m   | 47  | 170 | 00:43:10 | 03:13:29 | 26  | 134 | 287   | 28.2  |
| T12     | 47  | m   | 48  | 182 | –     | 03:09:49 | 53  | 129 | 1133  | 95.4  |

| Group M | | | | | | |
|---------|---|---|---|---|---|---|
| subject | age [y] | gender | $HR_{\text{rest}}$ | $HR_{\text{max}}$ | Finishing time [hh:mm:ss] | $HR_{\text{avg}}$ |
| M1      | 53  | m   | 56  | 185 | 03:40:09 | 172 |
| M2      | 50  | m   | 46  | 174 | 03:36:47 | 149 |
| M3      | 28  | m   | 58  | 198 | 04:17:04 | 171 |
| M4      | 34  | m   | 50  | 195 | 04:12:36 | 155 |
| M5      | 43  | m   | 55  | 200 | 03:49:03 | 162 |
| M6      | 39  | m   | 55  | 194 | 04:19:36 | 155 |
| M7      | 50  | m   | 50  | 200 | 03:30:33 | 174 |

TABLE II. Data filtering parameters.

| Group | $RR_{\text{min}}$ (ms) | $RR_{\text{max}}$ (ms) | $l_{\text{med}}$ (beats) | $c_{\text{med}}$ |
|-------|-----------------|-----------------|----------------|----------------|
| M     | 250             | 600             | 15              | 0.026          |
| T     | 250             | 1000            | 11              | 0.03           |

1. RR intervals below or above the threshold values $RR_{\text{min}}$ and $RR_{\text{max}}$ are removed.

2. Compute the local median of the RR intervals $RR_{\text{med}}(t)$ in a moving window of length $l_{\text{med}}$.

3. Remove RR intervals that fall outside the range $(1 \pm c_{\text{med}})RR_{\text{med}}(t)$, where the threshold $c_{\text{med}}$ is a constant.

The values for the filtering parameters are listed in Table II. Acceptable performance of the filter is manually inspected for all the samples in group M and a representative subset of 17 samples from the group T. The filter is particularly adept at removing too long intervals arising from missed beats, which is the most common error in exercise data. As ECG data is not available, we do not attempt to filter the data based on physiological criteria, and merely remove technical artifacts that can be isolated with reasonable certainty.

III. METHODS

A. Detrended Fluctuation Analysis (DFA)

Ever since its introduction in the study of correlations in DNA sequences [15], DFA for time series has been widely employed across multiple disciplines such as physics [17, 18], medicine [11, 19, 20], finance [21], and even music [22, 23]. The DFA method has been extensively studied [24, 25] and it has been expanded to account for effects such as multifractality [26] and cross-correlations [27].

We briefly summarize the conventional DFA algorithm which has been developed to detect correlations in non-stationary time series [16]. First, for a time series $X(j)$ of length $N$ a cumulative summation is performed,

$$Y(k) = \sum_{j=1}^{k} (X(j) - \langle X \rangle),$$

where the mean $\langle X \rangle$ of the time series is subtracted, but that is not strictly necessary for DFA. Conventionally, the integrated time series of Eq. (1) is divided into non-overlapping windows of length $s$. In each window $w$, a local trend is determined as the least-squares fit of a low order polynomial $p_{s,w}(k)$ to the data. (The method
is denoted by DFA-α if the degree of the detrending polynomial is n [24]. The fluctuations are measured as the variance from the local trend \( p_{s,w}(k) \) in each window: \( F_{s,w}^2 = \frac{1}{2} \sum_{k \in w} (Y(k) - p_{s,w}(k))^2 \). These squared fluctuations are averaged over the windows to yield the fluctuation function

\[
F(s) = (F_{s,w}^2)^{1/2}.
\]  

(2)

Allowing the windows to overlap enhances the statistical properties of this estimate [35]. When this procedure is repeated for different window sizes, or scales s, a power-law increase of the fluctuations with the window size may be observed, i.e., \( F(s) \sim s^\alpha \). Here \( \alpha \) is a scaling exponent that can be considered as a generalization of the Hurst exponent \( H \) [36]. See Sec. III D 2 below for more details.

However, experimental time series rarely exhibit exact scaling over several scales. Many previous studies have focused on finding a robust determination of the scaling regimes [37,38], or on extracting a spectra of scaling exponents \( \alpha(s) \) [39,43]. These methods are based on the notion that the spectra may be defined as the local slope of the logarithmic fluctuation function,

\[
\alpha(s) = \frac{d \log F(s)}{d \log s}.
\]  

(3)

In the context of HRV, these methods generalize and expand the conventional division into short (4–16 beats) and long-range (16–64 beats) scaling exponents. In practice, the behavior may also change over time, either due to external influences, or the process itself may comprise several distinct intrinsic modes. This paper develops a methodology that takes these temporal variations into account in a consistent manner.

Depending on the value of the exponent \( \alpha \), different degrees of correlations of the time series or its increments can be identified. The meaning of the different ranges for \( \alpha \) are summarized in Tab. III. The scaling exponent \( \alpha \) is related to other scaling exponents in time series analysis. Scale invariance is also observed in the Fourier domain as a function of the frequency \( f \) with a power spectral density that scales in the low frequency limit as \( P(f) \sim f^{-\beta} \). The exponent \( \beta \) is related to the DFA exponent by the scaling relation \( \beta = 2\alpha - 1 \) [27, 28]. In exercise physiology, the power spectrum of heart rate time series is a frequently employed tool to quantify the cardiological response to exercise. However, analyses in the frequency domain are potentially plagued by non-stationarity. For stationary signals, the autocorrelation function \( C(\tau) = \langle X(\tau_0)X(\tau_0 + \tau) \rangle \) decays for long lags \( \tau \) with a power law \( \sim \tau^{-\beta} \). Then the scaling relation \( \gamma = 2 - 2\alpha \) holds [29]. For more details on the DFA method and its relation to correlation functions, see Appendix A.

### B. Partial Autocorrelation Function (PACF)

It is instructive to supplement DFA analyses by a direct study of correlations at different time scales by computing the autocorrelation function \( C(\tau) \) and the partial autocorrelation function \( C(\tau) \) at lag \( \tau \). The latter has been successfully used to identify the best autoregressive (AR) process to fit a time series, using the fact that \( C(\tau) = 0 \) for all \( \tau > p \) for an AR model of order \( p \) [44]. The autocorrelation function \( C(\tau) \) is dominated by trends in the data, suggesting apparent correlations. On the contrary, \( C(\tau) \) is less affected by trends due to the subtraction of the linear dependence on intermediate lags from the autocorrelation function. If the time series contains oscillations, \( C(\tau) \) shows a periodic pattern with a frequency that modulates the data. On the contrary, \( C(\tau) \) shows anticorrelations. The lags \( \tau \) for which \( C(\tau) \) assumes negative values can be related to the periodicity of the oscillations. More specifically, for a time series \( X(\tau) \) the partial autocorrelation function is given by

\[
C(\tau) = \langle [X(\tau_0) - \hat{X}_{\tau_0\tau}(\tau_0)][X(\tau_0 + \tau) - \hat{X}_{\tau_0\tau}(\tau_0 + \tau)] \rangle
\]  

(4)

where \( \hat{X}_{\tau_0\tau}(\tau') \) is the best linear predictor, determined by

\[
\hat{X}_{\tau_0\tau}(\tau') = c_0 + \sum_{i=1}^{\tau-1} c_i X(\tau_0 + i),
\]  

(5)

where the coefficients \( c_i \) are determined by the conditions

\[
c_0 + \sum_{i=1}^{\tau-1} c_i \langle X(\tau_0 + i) \rangle = \langle X(\tau') \rangle,
\]  

(6)

\[
c_0 \langle X(j) \rangle + \sum_{i=1}^{\tau-1} c_i \langle X(\tau_0 + i)X(j) \rangle = \langle X(\tau')X(j) \rangle
\]  

(7)

for \( j = \tau_0 + 1, \ldots, \tau_0 + \tau - 1 \). The function \( C(\tau) \) can be computed practically from the Yule-Walker equations [44]. The relation between the AR fits and \( C(\tau) \) will be very important in the following analysis. Indeed, it has been shown that a signal with non-trivial periodic behavior can be, for short time scales, successfully fitted by an AR process, and its dominant frequency of oscillation can be extracted from the estimated coefficients. These findings have been also confirmed by DFA [45].

| Scaling exponent | Interpretation | Stationarity   |
|------------------|---------------|---------------|
| \( 0 < \alpha < 1/2 \) | anti-correlated |                |
| \( \alpha = 1/2 \) | white noise   | stationary     |
| \( 1/2 < \alpha < 1 \) | correlated    |                |
| \( \alpha = 1 \) | 1/f (pink) noise | |
| \( 1 < \alpha < 1/2 \) | anti-correlated increments | non-stationary, |
| \( \alpha = 1/2 \) | Brownian noise | stationary     |
| \( 1/2 < \alpha < 2 \) | correlated increments | increments |
C. Dynamic Segmentation

The dynamic behavior of the time series can be studied by performing the analysis in moving temporal windows. However, to guarantee the sufficient statistical accuracy, the length of these segments is dictated by the largest scale \( s \) (DFA) or the lag \( \tau \) (PACF), resulting in diminished temporal resolution for small scales. Therefore, we propose a dynamic segmentation procedure, where the segment length is varied as a function of the scale or the lag:

1. Choose a function for determining the segment lengths \( \ell(s) \) as a function of the scale \( s \). Here we adopt a simple linear relationship \( \ell(s) = as \) where \( a \) is a constant. Smaller values increase the temporal resolution but also the statistical noise. The dynamic length factor \( a \) itself may also be varied for different scales, but it is important that it remains constant within each scale.

2. For each scale divide the time series into segments of length \( \ell(s) \). The segments themselves may be overlapping if desired for smoother results. Identify the segments \( S_{s,t} \) by their temporal indices \( t \), which may be, e.g., the mean time within the segment or any other suitable quantity.

D. Dynamic Detrended Fluctuation Analysis (DDFA)

1. Procedure

The dynamic segmentation together with maximally overlapping windows in the DFA scheme enables the following procedure for dynamic DFA (DDFA):

1. Perform the dynamic segmentation for each scale \( s \). The value of \( a = 5 \) was found to be an acceptable value for the dynamic length factor.

2. Utilizing overlapping windows, compute the fluctuation function in each segment \( S_{s,t} \) at scales \( \{s - 1, s, s + 1\} \). Denote the logarithmic fluctuation function at these scales by \( \tilde{F}_1(s-1), \tilde{F}_1(s) \) and \( \tilde{F}_1(s+1), \) respectively.

3. In each segment, compute the dynamic scaling exponent \( \alpha(t,s) \) by the finite difference approximation

\[
\alpha(t,s) \approx [h_-^2 \tilde{F}_1(s + 1) + (h_+^2 - h_-^2) \tilde{F}_1(s) - h_-^2 \tilde{F}_1(s - 1)] / [h_- h_+ (h_+ + h_-)],
\]

where \( h_- = \log(s) - \log(s - 1) \) and \( h_+ = \log(s + 1) - \log(s) \) are the logarithmic backward and forward differences. Fluctuation functions computed with maximally overlapping windows are empirically found to be smooth enough to permit the direct application of the finite difference scheme.

2. Numerical Validation

Fractional Brownian motion (fBm) and its increments, fractional Gaussian noise (fGn), are commonly utilized for benchmarking DFA (see, e.g., Ref. [47] and references therein). These processes are characterized by the Hurst parameter \( 0 \leq H < 1 \), and exhibit long-range correlations with the asymptotic (large-scale) scaling exponents \( \alpha = H \) for fGn and \( \alpha = H + 1 \) for fBm [30][31]. Deviations from the asymptotic behavior occur at shorter scales due to the finite length of the samples and the intrinsic bias in DFA due to the detrending. It is, however, possible to compute the exact theoretical scale-dependent scaling exponent \( \alpha(s) \) for these processes, as described in Appendix A.

We validate the dynamic DFA (DDFA) method by applying it to simulated fGn and fBm, and comparing the results to the theoretically expected values. We utilize the Davies–Harte method, which is an efficient method for simulating these processes with their exact covariance structure [19][50]. We generate \( 10^5 \) samples of fGn and fBm of length \( 10^5 \) for each value of the Hurst parameter \( H \). From these simulated time series, we compute the dynamic scaling exponent \( \alpha(t,s) \) in non-overlapping dynamic segments with various dynamic segment length factors \( a \). The mean difference between the DDFA exponent \( \alpha(t,s) \) and the theoretically expected DFA exponent \( \alpha(s) \) is illustrated in Fig. 1. The general trend is that the limited sample size results in underestimation of the scaling exponents: the shorter the dynamic segments, the greater the bias. Similarly, processes with larger asymptotic \( \alpha \) suffer from larger bias, with a weak discontinuity at \( \alpha = 1 \) (when the process changes from fGn to fBm). This tendency can be understood as arising from the increased abundance and length of streaks in more correlated time series. This effect is not fully captured by the relatively short segments. However, for the shortest scales (5 and 6), particularly in the anti-correlated region, the exponent is slightly overestimated instead. We also observe that the contour lines for the bias in \( \alpha(t,s) \) have nearly converged to a constant value of the asymptotic \( \alpha \) already at the scale \( s = 40 \).

The standard deviation of the DDFA estimation of the exponent \( \alpha \) is shown in Fig. 2 for segment lengths 4, 5, 7, and 10 in (a-d), respectively. The deviation consistently decreases with the increasing segment length. On the other hand, the deviation reduced as the DFA-1 exponents approach the limits \( \alpha = 0 \) and \( \alpha = 2 \), since at these boundaries there is less room for variations. In particular, as \( \alpha \) is reduced from 1/2 towards zero (corresponding to the anticitrelated regime) the deviations are strongly reduced. The local reduction in the deviation just above \( \alpha = 1 \) is due to the most anticorrelated
Asymptotic of $f_{Gn}$ and $f_{Bm}$

FIG. 1. Bias of the dynamic detrended fluctuation analysis (DDFA) estimate of $\alpha(t, s)$ relative to its theoretically expected value for fractional Gaussian noise (fGn) and fractional Brownian motion (fBm). Characterized by the Hurst parameter $0 \leq H < 1$, the asymptotic $\alpha$ for these processes is $\alpha = H$ and $\alpha = H + 1$ respectively. Appendix A provides details on how to compute the theoretical scale-dependent DFA exponent $\alpha(s)$. The bias is defined as the observed $\alpha(t, s)$ minus its theoretically expected value $\alpha(s)$. The dynamic segment length factors $a$ are 4, 5, 7, and 10 in (a-d), respectively.

increments in this regime (see Table III).

Generally, the bias and the standard deviation of the DDFA method are found to be acceptable for our purposes, especially in view of the fact that we have particular interest in the anticorrelated region as underlined below in the results. All our DDFA computations below are performed with the dynamic segment length factor $a = 5$ corresponding to Fig. 2(b). This was found to be a good compromise between the accuracy of the DDFA method and the dynamical resolution requiring a sufficiently small segment size.

E. Dynamic Partial Autocorrelation Function (DPACF)

In order to obtain a local estimate of the partial autocorrelation function $C(\tau)$ we compute it using an approach similar to that of the DDFA algorithm. The steps of this approach can be summarized as follows:

1. Perform dynamic segmentation for each lag $\tau$. The value of $a = 10$ was found to be an acceptable value for the dynamic length factor, which is utilized in all of our DPACF calculations.

2. In each segment $S_{\tau,t}$, perform polynomial detrending of order $m$.

3. For each segment, compute $C(\tau)$ by, for example, solving the Yule-Walkers equations with the Levinson-Durbin recursive scheme [51]. Choose each time for the maximum lag the parameter for which we are estimating the partial autocorrelation function. Denote this dynamic PACF by $C(t, \tau)$.

Resorting to the Central Limit Theorem, it is a known result that the partial autocorrelation function is approximately non-zero at 5% significance level if $|C(t, \tau)| < 1.96/\sqrt{n(\tau)}$. The evaluation of this significance band is statistically valid only if $\ell(\tau) > 30$ and therefore if $\tau > 3$ for $a = 10$.

Notice that in DPACF the detrending is applied to the original time series in contrast to the integrated series in DDFA. Therefore, the results would be expected to be qualitatively similar when the DPACF detrending order is one smaller than the DDFA detrending order $n$. This explanation is complicated by, e.g., the removal of linear correlations in PACF. However, the relationship $m \approx n - 1$ is supported by empirical observations.

IV. RESULTS

A. Scaling of Heartbeat Fluctuations during Exercise

Figure 3 demonstrates our methods applied to one subject (M1) of the marathon (M) group. The color-coded value of the scale dependent exponent $\alpha(s)$ is shown in the first row as function of running time $t$ and also as function of binned heart rate HR. Over the studied scales $s$ from 5 to 5000 heart beats, the scaling exponent $\alpha(s)$ tends to decrease from values around one and larger (fractional Brownian motion) on large time scales to clearly below 1/2 on shorter time scales. These remarkably small values for $\alpha(s)$ indicate anticorrelated RR intervals that are clearly visible up to the scales of 20...30 heartbeats.
FIG. 3. Beat-to-beat (RR) interval correlations for the Marathon race of subject M1. Note that the upper-left and upper-right color bars refer to (a,c) and (b,d), respectively. (a) Color-coded dynamic (DDFA-1) scaling exponent $\alpha(t,s)$ on different scales $s$ (y-axis) as a function of binned HR (x-axis). Here $\alpha(t,s)$ is averaged over those dynamic segments, whose average HR falls within 0.1 BPM wide bins. The values for empty bins are linearly interpolated if the gap does not exceed 0.5 BPM. The black solid line shows the mean together with the standard deviation (thin lines) and the standard error of the mean (thick lines, barely visible) of the conventional short-scale (4–16 RRIs) scaling exponent $\alpha_1$. The exponent is computed in moving windows of size 50 RRI’s in HR bins of 2 BPM. (b) Color-coded partial autocorrelation functions (DPACF-0) $C(t,\tau)$ with different lags $\tau$ (y-axis) as a function of the binned HR. (c) Similar to (a) but as a function of time during the marathon race. The instantaneous heart rate is overlaid on the data. (d) Similar to (b) but as a function of time. The values that do not pass the non-zero significance test as described in the text are shown in white. (e) Probability density histogram for $\alpha(t,s)$ for different scales $s$ as a function of the HR. (f) Probability density histogram of the DPACF-0 for different lags $\tau$ as a function of the HR. The histograms in (e-f) consist of 31-by-31 bins, and the probability densities are separately normalized for each HR bin, so that they better depict the distributions as a function of the HR instead of measuring the prevalence of different HR regions. Furthermore, the color bar is capped at the 99.5th percentile to avoid outliers dominating the color scale.

In contrast, a typical 24-hour RR tachogram of a healthy subject displays on long times scales (or low frequencies $\lesssim 0.05$ Hz) usually 1/f or pink noise, corresponding to $\alpha = 1$, and larger values for $\alpha$ at higher frequencies.

The black curve in the left plot of the first row in Fig. 3 corresponds to the conventional DFA exponent $\alpha_1$ over scales from 4 to 16 heart beats. It also shows a very drastic decrease to values around 1/2 and below when the DDFA exponent $\alpha(s)$ displays multiscale anticorrelated behavior. The scale $s$ on which these anticorrelations appear changes with heart rate and hence exercise intensity. This can be seen from a comparison of the heart beat rate (black curve in the right panel of the first row of Fig. 3) and a clear shift of the anticorrelated (blue) region to longer scales $s$ with increasing beat rate.

To explore the scale dependence of the anticorrelations in more detail, we show in the third row of Fig. 3 the probability density for the values of $\alpha(s)$ for six different scales $s$ from 5 to 20 heart beats as a function of the binned heart rate. On all six scales, the probability density is maximum for $\alpha < 1/2$ with a heart rate dependent modulation and an absence of anticorrelations on the two
largest scales \( s = 15, 20 \) for lower beat rates.

In order to understand the relevant time scales of the physiological processes behind the observed anticorrelated beat intervals, we have also performed a DPACF analysis. The result is shown for lags between 1 and 20 heart beats in the second row of Fig. 2. The PACF reveals direct anticorrelations (negative values) after a time lag of 1 and 2 beats, starting at low exercise intensities, and additional anticorrelations up to about 10 beats beyond a certain exercise intensity. The latter branch of anticorrelations corresponds to the DDFA exponents \( \alpha(s) < 1/2 \) in the upper row. The probability density of the DPACF values for lags between 1 and 6 beats, shown in the last row of Fig. 2, confirm dominant direct anticorrelations on the shortest time scales of 1 to 2 beats, and 4 beats for high exercise intensities (here \( \text{HR} \geq 170 \)).

Our findings are qualitatively similar for all the subjects in group M. Figure 3 displays the aggregated DDFA and DPACF results for \( \alpha(s) \) for all members of group M as a function of absolute (left) and relative (right) HR. Anticorrelated RR intervals gradually appear beyond exercise intensities of around 80\% of the maximum HR, and become more clearly visible at approximately 90\%. In the same range, the conventional DFA exponent \( \alpha_1 \) computed for the scale of 4...16 beats (black curve in Fig. 1) drops below 1/2, confirming the anticorrelated behavior. There is a shift of the anticorrelations to larger scales of up to 20...30 beats at about 87\% and particularly at about 95\% of the maximum HR. In the absolute scale (a) this feature is visible at about \( \geq 175 \) BPM. Naturally, the aggregated results should be interpreted with care as they represent an average result over all the samples. Secondly, there is uncertainty in the maximum HR values of the subjects. The individual DDFA and DPACF results for all subjects of group M are shown in Fig. 3 in Appendix B as a function of both the relative HR and running time.

In order to study the correlations of RRI's over a wider range of exercise durations and intensities, we performed the same analysis for subjects in group T. It is instructive to consider first a single exercise of one subject which is shown in Fig. 5. It consists of six intervals of high-intensity running, each interval lasting about 160 seconds. As a function of exercise time \( t \) the DFA exponent \( \alpha(s) \) and the PACF consistently reveal strong anticorrelations of RR intervals that develop rapidly after the start of the intense interval and shift to larger time scales with increasing heart rate. At rest between the intervals the anticorrelations vanish rapidly. PACF shows the existence of anticorrelations over time lags up to 10 beats with strongest anticorrelations within 1 to 2 beats, as observed in group M. As a function of heart rate, the anticorrelated behavior develops rapidly after an intensity threshold (see the left panel in the first row of Fig. 5).

Next, we study the typical behavior of RRI correlations when averaged over many running exercises of different intensity and duration. The corresponding aggregated results from DDFA and DPACF analysis for one subject (T07) of group T are shown in Fig. 6, representing a total running distance of 1889 km. For this large data set, we obtain very good statistics for the aggregated data and expect them to provide a reliable representation of the typical RRI correlations as function of the exercise intensity. Indeed, both DDFA exponent \( \alpha(s) \) and DPACF show very clearly two distinct bands of anticorrelated RRI's (see first row of Fig. 4). Both bands shift to larger scales \( s \) or lags \( \tau \) with increasing exercise intensity (heart rate). This observation confirms our findings during individual marathon competitions analyzed above (see Figs. 3 and 4). The conventional DFA exponent \( \alpha_1 \) (black curve in Fig. 6) displays non-monotonous behavior, reflecting its limitation to the scale of 4...16 beats.

The last two rows of Fig. 6 show the probability density plots of \( \alpha(s) \) and PACF values for different scales \( s \) and lags \( \tau \). The existence of two regions with anticorrelated RRI's is clearly visible. They are separated by a region with positive correlations (or \( \alpha > 1/2 \)). The aggregated data for all subjects of group T are shown in Fig. 5 in Appendix B. They are consistent with the findings discussed here for the average behavior of DDFA, DPACF, and the probability densities.

We assessed the suitability of higher order detrending for our analysis and decided to employ DDFA-1 due to the following reasons: i) The qualitative behavior remains the same at the shortest scales, which is the most interesting region for dynamic exercise intensity analysis, ii) the short-scale bias in DFA is larger and crossover scales are shifted with higher order methods, iii) higher orders of DFA appear to require longer dynamic segments for similar statistical accuracy and have increased computational cost.

Finally we point out that it is important to check the reliability of our DDFA and DPACF methods with respect to trends. Hence, we have filtered the data of subject T07 according to the condition that the standard deviation of the heart rate within the dynamic segments is smaller than the values for certain quantiles. The result of this analysis for six different choices of quantiles is shown in Fig. 10 of Appendix B. It shows that the observation of the bands with anticorrelations is very robust and independent of the choice of the quantile filter.

B. Relation between Heartbeat Dynamics, Hemodynamics and Exercise Intensity

In the previous section we have seen that both during marathon running and also during running exercise of various durations and intensity, the RR intervals become anticorrelated beyond a certain threshold of exercise intensity over time scales that vary with exercise intensity between a few beats and about 20 beats. It is interesting to explore possible physiological mechanisms that could generate such dynamics. Based on the available data for RR intervals only, we obviously cannot examine other physiological variables. Hence, we can only de-
velop some simple arguments that could be relevant for explaining the observed RR interval dynamics.

First, we point out that there are three physiologically relevant time scales that fall into the range over which the anticorrelations occur: (i) the stride frequency which is typically around 85 strides per leg and per minute [52], (ii) the respiration frequency which fluctuates around 1/(4 beats), and (iii) the blood pressure fluctuations, i.e., the so-called Mayer waves, which result from an oscillation of sympathetic vasomotor tone and is of the order of 10 seconds [53].

All three processes can influence the hemodynamics and in particular the venous return (VR) of blood to the heart. The peripheral vascular resistance decreases substantially with increasing exercise intensity [8]. As VR is determined by the ratio of the pressure difference between peripheral veins and the right atrium and the vascular resistance, the return increases with exercise intensity. Moreover, the decreased resistance makes the venous return also more sensitive to small fluctuations in pressure differences. Transient differences between cardiac output and VR must be compensated by a rapid
adjustment of heart beat frequency. To maintain mechanical efficiency of the heart muscle, the stroke volume remains relatively stable beyond a low-intensity threshold. Instead of increasing the stroke volume further, the Bainbridge reflex responds to an increased VR by a sympathetic stimulation of the heart rate. Rapid fluctuations in systemic VR are hence expected to induce rapid, periodic modulations of RR intervals.

Transient changes in the VR can be induced by muscle contraction during running, since they promote the venous return. The frequency for leg muscle contractions is set by the stride frequency. This suggests VR fluctuations on the scale of 170/min, which is at high intensity of the order of the heart rate. This could explain the anticorrelations on the shortest scale with lag $\tau = 1$ beat. The VR is also modulated by respiratory inspiration (expiration) due to a decreased (increased) right atrial pressure. The corresponding modulation with a periodicity of about 4 heart beats suggests anticorrelations with a lag of about $\tau = 2$ beats, which are clearly visible in our DPACF results. Another source of modulations of VR are Mayer waves in arterial blood pressure with a periodicity of about 10 sec, corresponding to a periodicity of about 23 to 33 beats for heart rates between 140 and 200 beats/min. This frequency band is consistent with the observed regions with RR anticorrelations in our DDFA and DPACF results, on a scale $s \sim 10$ to 20 beats. Mayer waves are believed to be a transient oscillatory responses to hemodynamic perturbations and their amplitude depends both on the strength of these triggering perturbations and on the baroreflex gain [53]. Their amplitude has been presumed to measure vascular sympathetic activity since they are strongest in response to sympathetic stimuli and are strongly attenuated or even abolished after acute $\alpha$-adrenoceptor blockade [54]. As sympathetic activation occurs at moderate and higher exercise intensities in humans, the Mayer wave amplitude must be increasingly enhanced with raising exercise intensity. This could be the reason why we observe anticorrelations at larger scales of 10 to 20 beats only beyond a certain threshold for exercise intensity. At lower intensities, the Mayer wave modulations are present but are
previously too small to sufficiently influence the systemic vein dynamics for activating the Bainbridge reflex via VR modulations.

V. SUMMARY AND CONCLUSIONS

Our main result is the discovery of anticorrelated RR intervals during exercises of various durations and intensities. The anticorrelations have a dynamical structure that depends on the scale and the exercise intensity. The characteristics of the dynamical structure is revealed by our methodology, in particular the dynamic detrended fluctuation analysis and dynamic partial autocorrelation functions, which are here developed, optimized, and numerically validated.

The observed anticorrelations are persistent on short scales (a few beats) at low and moderate exercise intensities, but move to larger scales (up to dozens of beats) at higher intensities. At rest, e.g., between running intervals, the anticorrelations rapidly vanish, and appear immediately when the intensity is increased. The change in the scaling is in fact quicker than the change in the heart rate itself as shown in, e.g., the right panel of Fig. 5. Hence, the presented methodology may provide a new tool to exercise assessment in sport monitors and watches.

We propose an explanation for our findings in terms of exercise induced enhancement of Mayer waves and the Bainbridge reflex but a number of challenges remain for future work. It is highly desirable to develop a theoretical model for the complex dynamics of the cardiovascular feedback loops during high-intensity exercise load that can explain the observed time scales for the anticorrelated RR intervals. Clearly, a more systematic study with subjects performing specific exercise protocol should be performed to verify our observations. Besides, a thorough validation and calibration of our results with data collected from, e.g., VO2max tests is a natural next step for our study.

We expect that the reported RR interval correlations are suitable to represent a dynamical “fingerprint” of the exercise-induced cardiovascular load. Hence, our methodology – which is readily applicable to the present devices on the market – could potentially become a new tool in real-time exercise monitoring without previous knowledge of maximal thresholds such as the maximum hearth rate and lactate or ventilatory thresholds.

Appendix A: Additional remarks on DFA for the validation of DDFA

In this section we provide some known theoretical results for the conventional DFA algorithm. They are used to supplement the validation procedure presented in Sec. III D 2.

In DFA, the range of detectable exponents is determined by the degree of detrending, n, and is given by $0 \leq \alpha \leq n + 1$ [25]. While the existence of values $\alpha > 1$ may be criticized as a failure of the detrending procedure [55], they may also be understood as an advantage of the method for allowing meaningful quantification of non-stationary processes [31, 32]. The detrending may be considered successful if it achieves the statistical equivalence over the DFA windows, so that the fluctuation function $F(s)$ does not depend on the window [31, 32]. This condition is fulfilled for DFA $n$ with time series exhibiting polynomial trends of degree $n - 1$. In general, for two uncorrelated signals $X_A(t)$, $X_B(t)$ (random processes or trends), a superposition principle holds, stating that the squared fluctuation function of the sum $X_{A+B}(t) = X_A(t) + X_B(t)$ is given by $F^2_{A+B}(s) = F^2_A(s) + F^2_B(s)$ [26].

For stationary processes and for non-stationary processes with stationary increments the fluctuation function $F_s$ does not depend on the window and may be analytically computed [29, 30]. Its squared value is determined as the weighted sum of the autocovariance function $C(\tau) = \langle [X(\tau_0)X(\tau_0 + \tau) - \langle X \rangle^2 \rangle$ in the former case, and that of the variogram $S(\tau) = \langle [X(\tau_0 + \tau) - X(\tau_0)]^2 \rangle$ in the latter case,

$$F^2_s = \sum_{j=-s+1}^{s-1} G(j,s) \hat{C}(j)$$

$$F^2_s = \sum_{j=1}^{s-1} G(j,s) S(j)$$

with the weight function $G(j,s)$ given by [56]

$$G(j,s) = \frac{1}{s} \sum_{k=1}^{s-|j|} a_{k,k+|j|},$$

where $a_{k,k'}$ are the elements of the matrix

$$A = D^\top \left[ I - B^\top (BB^\top)^{-1} B \right] D,$$

where the elements $d_{i,j}$ of the matrix $D$ are unity if $i \geq j$ and zero otherwise [26, 30]. The effect of detrending is incorporated into the so-called design matrix $B$ of least squares regression, which for DFA-1 is given by [30]

$$B = \begin{bmatrix} 1 & 1 & \cdots & 1 \\ 1 & 2 & \cdots & s \end{bmatrix}.$$  

This matrix $A$ describes an operator for constructing the squared fluctuations $F_s = X_{t,w}^\top A X_{s,w}$ from the values of the time series $X_{s,w}$ in window $w$ at the scale $s$ [30].

The autocovariance function for fractional Gaussian noise (fGn) and the variogram for fractional Brownian motion (fBm) with Hurst parameter $H$ are known to be

$$\hat{C}_{fGn}(\tau) = \frac{\sigma^2}{2} \left( |\tau + 1|^{2H} - 2|\tau|^{2H} + |\tau - 1|^{2H} \right),$$

$$S_{fBm}^2(\tau) = \sigma^2 |\tau|^{2H},$$

where $\sigma^2$ is the variance of the white noise process.
Asymptotic of $f_{Gn}$ and $f_{Bm}$

Deviation from asymptotic

FIG. 7. Theoretical deviation of the DFA estimate $\alpha(s)$ from asymptotic scaling exponent $\alpha$ for $f_{Gn}$ and $f_{Bm}$ as a function of the scale $s$. The deviation is defined as the theoretical scale-dependent exponent $\alpha(s)$ minus the asymptotic scaling exponent $\alpha$. Note the quasi-logarithmic scale for the deviation.

where $\sigma^2$ corresponds to the variance of ordinary Gaussian noise $[48]$. From these correlations and by using Eqs. (A1) and (A2), the theoretical fluctuation function may be computed for these processes. The spectrum of the scaling exponent $\alpha(s)$ is then obtained from Eq. (3). These theoretical results may be utilized for studying the behavior of the DFA method as a function of the scale $s$. For example, the deviation between the DFA estimate for $\alpha(s)$ and the asymptotic large scale exponent $\alpha$ is visualized in Fig. 7 for $f_{Gn}$ and $f_{Bm}$. The well-known overestimation of the scaling exponent at the shortest scales is clearly visible, and it is most pronounced in the anticorrelated region with $\alpha < 1/2$. Around the asymptotic value of $\alpha = 1$ there is an abrupt qualitative change as the scaling exponent is suddenly underestimated for an extended range of scales. This has been observed previously $[30]$.

In general, the short scale behavior depends on the details of the underlying process, and hence can be different for other processes such as an autoregressive model AR($p$). For more details on the scale dependence of the deviation between asymptotic $\alpha$ and $\alpha(s)$, please see also Ref. $[57]$.

Appendix B: Additional Heartbeat Correlation Plots

Here we present beat-to-beat (RR) interval (RRI) correlations for all the subjects in the study. In Fig. 8 we illustrate the average RRI correlation results as a function of the heart rate aggregated over all the runs for each subject of Group T. The relative heart rate is utilized to better facilitate the comparison between different individuals. Similar correlation plots for the marathons of Group M are shown in Fig. 9 along with the correlation landscapes as a function of time during the marathon races. Additionally, we establish the consistency of the anticorrelated bands in the presence of possible trends in Fig. 10. We demonstrate this by limiting the analysis to subsets of data where the heart rate within the dynamic segments exhibits subsequently lower and lower standard deviation. The analysis is performed for subject T07, who has the most data.
FIG. 8. Aggregate RRI correlation results for each subject of Group T. For each subject the average DDFA-1 scaling exponents $\alpha(t, s)$ (upper panels) and DPACF-0 correlations $C(t, \tau)$ (lower panels) as a function of binned relative heart rate. For a detailed explanation about how the data is computed, please see the caption for Fig. 3.
FIG. 9. Overview of all the marathons in Group M. Left: Average RRI correlation results as a function of binned relative heart rate for each subject of Group M. Right: RRI correlation landscapes of the Marathon runs. For each subject the DDFA-1 scaling exponents $\alpha(t, s)$ (upper panels) and DPACF-0 correlations $C(t, \tau)$ (lower panels) are shown. For a detailed explanation about how the data is computed, please see the caption for Fig. [3]
FIG. 10. Consistency of the results with respect to trends. Top row: different quantiles $Q$ of the mean and standard deviation of the heart rate within the dynamic segments for all the data of subject T07. For DPACF-0 and DDFA-1 the dynamic segment length factor $a$ has values of 10 and 5, respectively, and for conventional DFA the short-range (4–16 beats) scaling exponent is computed in moving windows consisting of 50 RRIs. Lower panels: the average RRI correlation results as a function of the heart rate when the data is limited to dynamic segments with the heart rate standard deviation less than the value for the specified quantiles $Q$. For a more detailed explanation of these kinds of plots, please see the caption for Fig. 3.
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