

Evidence of phase transitions in heart period dynamics

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Abstract. Complexity measures of non-linear dynamics are a useful tool for quantifying observed stretching, folding, scaling and mixing processes in the Takens-reconstructed state space of heart period dynamics. Although such measures are not suited to provide evidence of deterministic chaos or to estimate true fractal dimensions and Lyapunov spectra in heart period time series, they allow the classification of RR dynamics and the identification of changes in RR complexity (RRC). The aim of this study was to develop appropriate measures and examine their utility in identifying the physiological effect of changes between the sleeping and waking state. Twenty-four hour electrocardiography (ECG) recordings and diaries noting their waking/sleeping period were obtained from 78 healthy subjects, aged 20 to 55 years. The approximate information dimension ($ApD1$) and the approximate Kolmogorov entropy ($ApEn$), introduced by Pincus, Kaplan and others, were modified in order to allow the calculation of strictly local values. That is, the local or pointwise dimensions and entropies were calculated for each reference vector with respect to its symmetric neighbourhood in time. For each subject the values for the local measures were averaged for 10-min periods, resulting in 144 global values over 24 h. Similarly, low- and high-frequency spectral parameters were calculated. All measures were examined and compared for the waking and the sleeping periods. All complexity measures as well as to a lesser degree high-frequency power showed a linear dependency on mean RR interval with a large individual variation. For the RRC measures this linear correlation was separated into two different clusters corresponding to the sleeping and waking periods. In almost all cases the correlation was greater in the waking period. In particular, in many cases no correlation was observed in the sleeping period. However, the r values for LF were appreciably lower and indicated solely a weak relationship to the RR interval in the waking period. Analysis of variance combining mean RR interval with RRC or spectral parameters singly and in couples revealed that the best separation with respect to physiological state could be achieved with the complexity measures, in particular with $ApEn$. The results show evidence of at least two dynamical

regimes (phases) of heart period dynamics and a close but different functional relationship within the phases between RR interval and RR complexity. The separation between these regimes and the relatively sudden shift from one regime to the other suggest the existence of a phase transition with respect to waking and sleeping periods in terms of synergetics.

1 Introduction

Measures from non-linear dynamic system theory to time series data have been applied to the investigation of heart rhythms and initial studies in the mid-1980s (Babloyantz 1988) were among the first physiological applications of chaos theory. With the initial enthusiasm, these novel analytic tools were envisioned as opening up possibilities for new diagnostic and prognostic procedures. Ten years on, we are still far from this goal.

Cardiac regulation is revealed most obviously in the sequence of heart beat periods. A beat period is generally defined as the time duration between successive R waves in the electrocardiogram (ECG) and referred to as the RR interval. In modern ECG data acquisition and analysis systems, subtle methods have been developed to detect the QRS complex with high precision. The series of RR intervals thus produced is called the RR tachogram. It is the raw material for the RR complexity (RRC) analysis, which is performed in the reconstructed state space of the RR dynamics. This is in contrast to the clinically applied methods of RR variability analysis, which apply classic time series analysis in the time and frequency domain (for an overview, see Malik et al. 1996).

Measures derived from the theory of non-linear dynamics quantify the processes of expansion, contraction and folding which, according to the principle of the baker transformation, lead to a mixing in state space and produce fractal structures. Expansion and contraction are measured by Lyapunov exponents, whereas the scaling properties of the fractal which ensues in state space after prolonged observation of the dynamic system are quantified by dimension measures.

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All dimension measures can be ordered in a sequence of generalized dimensions (Hentschel and Procaccia 1983), of which the fractal (or similarity) dimension, the information dimension as well as the correlation dimension are usually calculated.

In the 1980s the application of non-linear dynamic analysis to ECG time series focussed primarily either on attempts to demonstrate the existence of a deterministic generator of cardiac rhythms or on comparisons between experimental and model data. In contrast, the work at the onset of the following decade became more pragmatic and oriented toward clinical use (for comprehensive literature, see Bettermann 1996). Kaplan and co-workers (1990, 1991) created the concept of approximate complexity by doing without the unrealistically high number of samples required by theory as well as by avoiding the assumption of strict self-similarity which is rarely found in nature. These authors prefer a more probabilistic approach in their methods and explicitly take the dependency of complexity measures on the data structure (number of data points, resolution, noise) into consideration. In 1990 they introduced a number of complexity measures for the analysis of heart periods and blood pressure and gave recommendations for the parameter settings for their approximate procedures. More recently, Pincus and co-workers (Pincus 1991, 1994, 1995; Pincus et al. 1991; Pincus and Viscarello 1992) have advocated the use of approximate entropy (*ApEn*) which they described as a ‘regularity statistic’, thereby emphasizing that it represents a statistical measure for the regularity or order in a time series. *ApEn* has been systematically applied in clinical studies: it has been used to calculate heart rate complexity in postoperative ventricular dysfunction (Fleisher et al. 1993), prior to sudden infant death (Pincus et al. 1993), and in fetal heart period analysis (Pincus and Viscarello 1992; Van Leeuwen et al. 1996). In non-cardiac applications, changes in hormone levels have been investigated (Pincus and Keefe 1992). It was also compared with power spectral indices (Sapoznikov et al. 1995).

Similarly to Kaplan, Pincus and others, in our previous work we approximated the correlation dimension (apparent dimension D_A) globally for fixed data windows (Bettermann and Van Leeuwen 1992; Van Leeuwen et al. 1995). The calculation of D_A places particular emphasis on the assumption of stationarity. As stationarity is rarely met in RR tachograms, this method of dimension calculation is more or less arbitrary: depending on the position in the data window, a data point will be related in varying degrees to the past or the future. Thus the closer an RR interval is to the beginning of a data window, the more predominantly it will be correlated with future RR intervals and the less its relationship to the past is taken into consideration. This problem is avoided by the use of local complexity measures. The pointwise or local approximation of complexity was introduced by Mayer-Kress, who has consistently applied the pointwise dimension (Mayer-Kress 1994). He emphasizes that the variance of this measure, which reflects the inhomogeneity of the dynamics, has high explanatory power. In pointwise measures, the local RRC is calculated for each RR interval in a symmetric neighbourhood of RR intervals in state space. The local approximation has the advantage that the course of the local complexity can be represented beat to beat, continually over long periods of time in the 24-h ECG, whereby each

beat is considered in like manner. Furthermore, global complexity measures can be formed by averaging according to need. A comprehensive theoretical basis dealing with local and global complexity of invariant measures can be found in the publication of Grassberger and co-workers (1988).

In previous work we were able to show that, in a majority of subjects, the apparent dimension D_A was significantly higher for nighttime values (Van Leeuwen et al. 1995). As there was also a concomitant increase in heart period at night, the question remains as to what degree the observed differences in day and nighttime D_A values were a result of changes in heart rate. It is not clear whether different values of D_A , as a global measure, represent physiological changes or whether this is due to a systematic bias resulting from limitations in the temporal resolution of RR intervals. A further interesting question from the point of view of synergetics is: do the day-night differences in RRC result solely from a linear correlation between D_A and RR interval or does a phase transition exist between the RR dynamics of the sleeping and waking states?

In an attempt to answer these questions, we turned to the methods of Pincus, Kaplan and others and approximated the information dimension (*ApD1*) and the Kolmogorov entropy (*ApEn*). Both are global complexity measures and characterize the scaling (static) properties of the distribution in state space as well as the expansion behaviour (dynamic properties) of the generating dynamics. However, in contrast to the original definitions, we use a strictly local approach for the approximation of these global measures. By strictly local, we mean the computation of a local or pointwise dimension and entropy for each reference vector with respect to its symmetric neighbourhood in time. These local measures are then averaged to global measures over 10-min periods.

2 Methods

2.1 RR complexity analysis

Non-linear time series analysis in state space is based on Takens’ theorems which guarantee a topological equivalence between original state space and the reconstructed state space by using time delay coordinates. Without loss of generalization, we interpret the RR tachogram as a ‘time’ series with a one beat delay. Thus, k successive RR intervals form a vector in the reconstructed state space indexed by the beat number of the leading RR interval:

$$\vec{RR}_i = (RR_i, RR_{i+1}, \dots, RR_{i+k-1}) \in \text{reconstructed state space} \quad (1)$$

A data window with length N (RR intervals), which provides $N - k + 1$ k -dimensional reconstructable vectors, was continuously moved over the whole RR tachogram. The k data values in the centre of the window form the reference vector (see Fig. 1).

In our approach the quantification of local scaling properties starts with the calculation of the local correlation integrals $C_i^k(l)$ for each data window. $C_i^k(l)$ is defined as the relative number of embedded data vectors \vec{RR}_j from the data window within a l -sphere around the reference vector \vec{RR}_i as a function of the distance l :

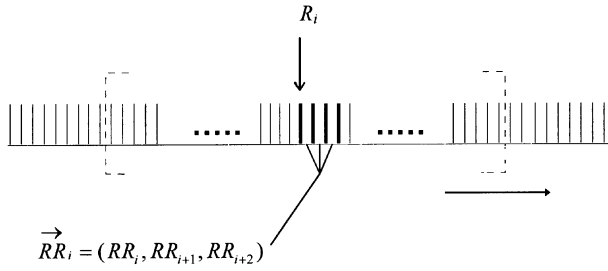


Fig. 1. Moving data window for the local complexity approximation. R_i , i th R wave (reference beat); RR_i , i th RR interval; \vec{RR}_i , i th k -dimensional RR pattern (reference vector, $k = 3$)

$$C_i^k(l) = \frac{1}{N - k + 1} \{ \text{number of vectors } \vec{RR}_j \text{ with } |\vec{RR}_i - \vec{RR}_j| < l \} \quad (2)$$

$$j \in [i - \text{int}(N/2) + 1, i + \text{int}(N/2) - k + 1]$$

Next, define

$$\begin{aligned} \Phi^k(N_{\text{Ref}}, l) &= N_{\text{Ref}}^{-1} \sum_{i=1}^{N_{\text{Ref}}} \log C_i^k(l) \\ &= N_{\text{Ref}}^{-1} \sum_{i=1}^{N_{\text{Ref}}} \Phi_i^k(l) \end{aligned} \quad (3)$$

and the approximate entropy (Pincus 1994)

$$ApEn(N_{\text{Ref}}, k, l) = \Phi^{k-1}(N_{\text{Ref}}, l) - \Phi^k(N_{\text{Ref}}, l) \quad (4)$$

$ApEn$ could be interpreted as ‘the negative logarithmic likelihood that runs of patterns that are close for $k - 1$ observations remain close on next incremental comparison with tolerance l ’ (Pincus 1994). In our case $ApEn$ could also be interpreted as an average over N_{Ref} local or pointwise approximate entropies ($ApPE$):

$$\begin{aligned} ApEn(N_{\text{Ref}}, k, l) &= N_{\text{Ref}}^{-1} \sum_{i=1}^{N_{\text{Ref}}} (\Phi_i^{k-1}(l) - \Phi_i^k(l)) \\ &= N_{\text{Ref}}^{-1} \sum_{i=1}^{N_{\text{Ref}}} ApPE(k, l, i) \end{aligned} \quad (5)$$

The local approximate dimension may be defined as the mean slope of the local correlation dimension between the two length scale values l_a and l_b :

$$ApPD(k, i) = \frac{\Phi_i^k(l_a) - \Phi_i^k(l_b)}{\log l_a - \log l_b} \quad (6)$$

l_a and l_b are chosen separately for each local approximation such that $\log_{10} C_i^k(l_a) = L$ and $\log_{10} C_i^k(l_b) = U$. By visual inspection of a large number of randomly selected local correlation integrals, L and U are fixed such that they represent as best as possible the lower and the upper bounds of the linear scaling range in the double logarithmic plots: $\log C_i^k(\log l)$. In our case, linear scaling is not a necessary condition for approximation of the dimension, but should be taken into account if present. The approximate information dimension is then defined as the mean of the local approximate dimensions:

$$ApD1(N_{\text{Ref}}, k) = N_{\text{Ref}}^{-1} \sum_{i=1}^{N_{\text{Ref}}} ApPD(k, i) \quad (7)$$

2.2 Parameter settings

In accordance with recommendations made in the literature (Kaplan et al. 1990) and our analysis of previous data (Van Leeuwen et al. 1995), we chose a three-dimensional embedding ($k = 3$). The data window encompassed 1000 RR intervals ($N = 1000$). The local complexity measures were averaged for the reference beats within 10-min intervals. As a result N_{Ref} was variable. $ApEn$ was calculated for a tolerance radius of 20 ms ($l = l_t = 20$ ms), and the scaling range for each $ApPD$ was set to $L = -2.0$ and $U = -0.5$.

2.3 Subjects

Seventy-eight healthy clinic employees with no history of cardiovascular or pulmonary disease served as subjects. After examination of their 24-h ECGs, 6 subjects were excluded because of the presence of a high number of ectopic beats or suspected autonomic neuropathy. The remaining 72 subjects (24 men) were aged between 20 and 55 years (mean \pm SD 31 ± 9 years), and 33 were regular smokers. All subjects kept a diary in which, in particular, the sleeping and waking times were noted. This permitted a differentiation of the data not only according to time of day but also with respect to sleeping and waking periods.

2.4 ECG recording and ECG analysis

The 24-h ECGs were recorded with Oxford Medilog FD2 solid-state recorders with a maximum sampling rate of 1024-Hz during the QRS complex. Computer-supported automatic evaluation was performed with an Oxford Excel ECG analyser using template analysis as follows. Beats are identified using a correlation algorithm, classes of beats are created on the basis of different wave forms (templates), and all identified beats are accordingly classified. Subsequently, medical personnel examined the initial beats in each template group to ensure proper classification. Artifacts marked as beats or extrasystoles falsely identified as sinus rhythm beats were reclassified. A data file containing beat type and time of day for all beats of the 24-h ECG was then exported to a Pentium 90 PC for the complexity analysis (ca. 2 h computing time/24-h ECG).

2.5 Ectopic beat filter

The main purpose of the ectopic beat filter was to restrict the reference vectors to those within normal physiological limits and to avoid interpolating or extrapolating beats. In the calculation of complexity measures, only the times between templates which had been classified as normal were considered. Other intervals were marked with the value 9999 ms. This served to place these vectors outside the area of the

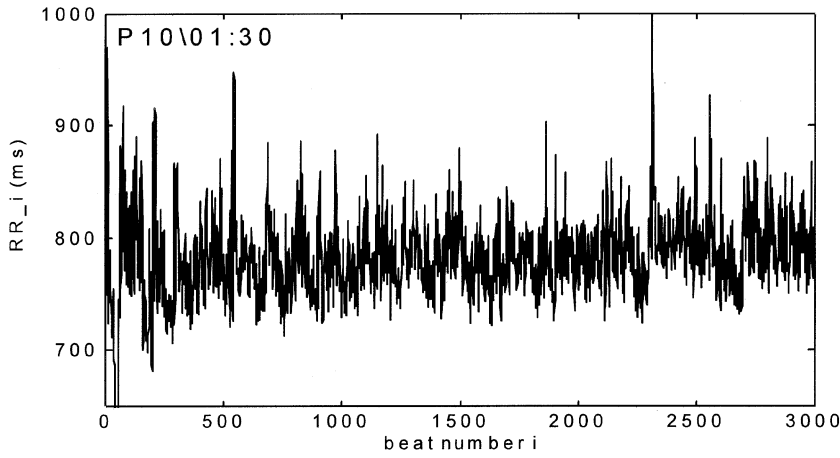


Fig. 2. RR tachogram of subject P10 from 1:30:08 to 2:09:08

reconstructed state space under consideration. As the scaling properties were only examined in the neighbourhood of the reference vectors ($l < 200$ ms), ectopic beats and artifacts are thus not considered in the calculation of $ApEn$ and $ApD1$.

2.6 Data analysis

As previously described, $ApEn$ and $ApD1$ as well as mean RR interval (mRR) were calculated for each 10-min interval over the 24 h of acquired data. Furthermore, in order to compare the results to commonly used heart rate variability parameters in the frequency domain, spectral analysis using fast Fourier transformation (FFT) was performed in each 10-min interval. The resulting spectral power density function was integrated in the low-frequency band (0.04–0.15 Hz, LF) and the high-frequency band (0.15–0.40 Hz, HF). LF and HF power (LF , HF) were computed in milliseconds such that they correspond to the standard deviation of the LF and HF band-passed RR interval series. The spectral analysis was performed according to the methods of Rottman and co-workers (1990) and is described in detail elsewhere (Betermann 1996).

The results were examined visually by plotting the complexity measures as well as the spectral measures against mean RR interval while distinguishing between values obtained in the waking or the sleeping period. We refer to these plots as complexity and variability state space diagrams, respectively. The sleeping and the waking period were defined according to the subjects' reports in their diaries. As such, they do not differentiate between varying waking states or sleep stages but simply identify the basic physiological condition of normal arousal and unconscious rest. In order to avoid possible ambiguity resulting from the transition between states as well as inaccuracies in the subjects' diary, values obtained in a 20-min period before and after the reported times were appropriately marked. The relationship between mean RR interval and the complexity and spectral measures was examined for both periods on the basis of correlation coefficients (r).

It is well-known that the RR interval changes between physiological states. We therefore attempted to quantify the possible augmentation in information with respect to the separation of waking and sleeping periods provided by each of

the measures presented here. To this end, Wilk's lambda (Λ) was calculated as a measure of phase coincidence for different projections in complexity and variability state space. Λ is defined as follows:

$$\Lambda = \det \left(T^{-1} \cdot W \right)$$

with

$$T = X_{W+S}^t \cdot X_{W+S} \quad (\text{total scatter matrix}) \quad (8)$$

and

$$W = X_W^t \cdot X_W + X_S^t \cdot X_S \quad (\text{within groups scatter matrix})$$

Λ quantifies the relationship between the variance within clusters and the total variance; X_W represents the complexity (or spectral) data matrix in the waking period, X_S that of the sleeping period, and X_{W+S} is composed of the values of all time intervals. Each line in the matrix is composed of the complexity or variability values of a 10-min interval. Low Λ values (near zero) indicate a separation of the parameters examined, and conversely, high Λ values (near 1) imply coincidence. Λ is used here as a descriptive and not as a confirmative statistic. It must further be noted that the value of Λ is dependent on the relative orientation of the clusters. The more the angle between the clusters approaches 90° , the larger the value of Λ . Nonetheless, small values of Λ indicate greater group separation.

3 Results

The following example serves to demonstrate the complexity analysis. Three thousand heartbeats with a mean beat period of 783 ms were obtained from subject P10 in the time between 1:30:08 to 2:09:08. The RR tachogram and the RR state space portrait are shown in Figs. 2 and 3, respectively. The local logarithmic correlation integrals ($k = 3$) in the scaling range for the first 100 reference beats are shown in Fig. 4. On the basis of the subject's diary the RR interval, $ApEn$, $ApD1$, LF and HF were averaged for the waking and sleeping periods (667 ms and 818 ms, 0.42 and 0.52, 1.90 and 2.26, 26.6 ms and 26.5 ms, 14.7 ms and 20.3 ms, respectively). These values are more or less representative of the overall results.

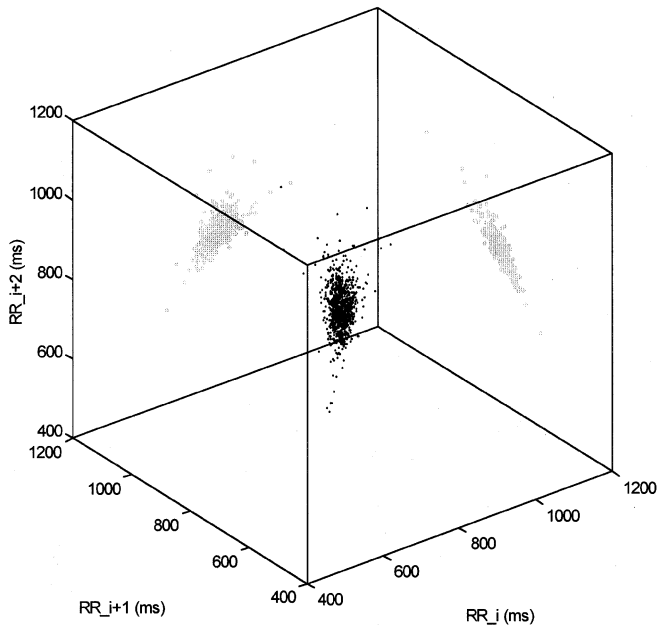


Fig. 3. RR state space portrait of the data in Fig. 2

Table 1. mRR , $ApEn$, $ApD1$, LF and HF during waking and sleeping periods as well as the difference sleeping-waking (mean \pm SD over all subjects)

	Waking	Sleeping	Sleeping-waking
mRR (ms)	729 \pm 67	976 \pm 116	247 \pm 85
$ApEn$	0.44 \pm 0.07	0.52 \pm 0.08	0.08 \pm 0.09
$ApD1$	1.86 \pm 0.11	2.19 \pm 0.13	0.33 \pm 0.15
LF (ms)	35.0 \pm 9.7	40.0 \pm 15.2	5.0 \pm 10.5
HF (ms)	18.7 \pm 7.4	32.5 \pm 17.0	13.8 \pm 12.3

The mean results for all subjects showed that all five measures were higher in the sleeping period, indicating an overall decrease in heart rate, an increase in complexity of heart period dynamics and an increase in heart rate variability (see Table 1). Only 9 (13%) $ApEn$ values, and 2 (3%) $ApD1$ values were lower during sleep. For the frequency domain, 24 (33%) of the LF values and 3 (4%) of the HF values were lower during sleep.

In Fig. 5 the 10-min values for $ApEn$ and $ApD1$ as well as those for the spectral variables, LF and HF , are plotted against mRR separately for the sleeping and waking periods for six selected subjects. These examples were chosen to demonstrate the various separation patterns between the two periods with respect to both the parameters and the subjects.

Examination of the relationship between mean RR interval and RRC values confirmed the linear correlation previously observed. However, the r values differed between the two periods, the strength of the relationship being greater in the waking period (Table 2). In particular, in many cases no correlation was observed in the sleeping period. Overall similar results were obtained for HF . However, the r values for LF were appreciably lower and indicated solely a weak relationship to mRR in the waking period.

The varying degree of correlation between the two periods raises the question in what way the relationship between the measures and RR interval differ between sleeping and

Table 2. r values for $ApEn$, $ApD1$, LF and HF with respect to mRR during waking and sleeping periods (mean \pm SD over all subjects)

	Waking	Sleeping
$ApEn$	0.79 \pm 0.21	0.37 \pm 0.44
$ApD1$	0.73 \pm 0.12	0.40 \pm 0.31
LF (ms)	0.37 \pm 0.27	0.11 \pm 0.32
HF (ms)	0.65 \pm 0.19	0.41 \pm 0.30

waking. Visual examination of the data shows that, for the RRC values, the sleeping and waking data clusters are often clearly disjunct and shifted along both axes relative to each other; e.g. the $ApEn$ waking data for subject P10 (Fig. 5) are displaced to the left and downwards from the sleeping data, the division being so clear that a straight line can be drawn to separate them completely. As the linear relationship does not run parallel to either axis, a univariate projection onto either axis results in an overlap of the sleeping and waking data. Only the bivariate representation allows the isolation of the two physiological states. In varying degrees, the same is true for the other subjects who differ (a) in the orientation of the clusters (see subjects P18 and P19), (b) in the strength of the correlation within the clusters and (c) in the transition values between the clusters. In some cases the clusters do not correspond to the waking and sleeping period completely (P04, P18). The clustering with respect to physiological period is more pronounced with $ApEn$, whereas $ApD1$ shows a more uniform linear correlation over the whole 24-h period. An exception to this can be found in P06. Examination of the spectral data reveals no similar form of clustering: on the whole, the shift between sleeping and waking data points, in particular for the LF band, can be seen as resulting from the change in RR interval. The higher r values for the HF band can be understood as a mean increase in vagal tone corresponding to the decrease in heart rate (Malik et al. 1996).

The observed clustering of the RRC values with respect to physiological period, accompanied by functional relationships in the respective clusters between mean RR interval and the complexity measures, allows us to speak of complexity phases and of phase transitions between waking and sleeping. From a systems theory standpoint, it seems likely that physiological systems go through a number of phases during 24 h, each phase having its own dynamic regulation.

Analysis of variance showed that the single variable that best explains the waking-sleeping clustering is the mean RR interval with the lowest Λ value of 0.36 (Table 3). Combining mRR with each of the other parameters lowers Λ further, whereby the greatest increase in explanatory power resulted from the addition of $ApEn$ ($\Lambda = 0.27$). In the three-dimensional combination, the addition $ApD1$ results in only a minimal further change in Λ . This combination is, however, much more efficient than linking mRR with the frequency domain parameters HF and LF .

4 Discussion

It is a well-known fact that changes in physiological state are often accompanied by changes in heart rate. Heart rate itself is mediated by various physical, chemical and neu-

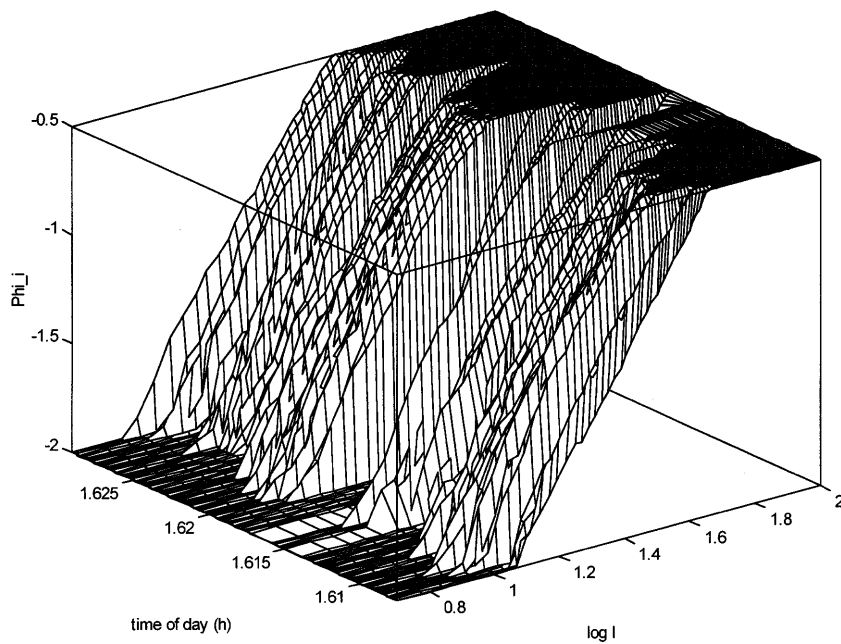


Fig. 4. Local logarithmic correlation integrals in the scaling range for the first 100 reference beats shown in Fig. 2

Table 3. Λ for *mRR*, *ApEn*, *ApD1*, *LF* and *HF*, singly and in two- and three-dimensional combinations (mean, SD, minimum and maximum over all subjects)

	<i>mRR</i>	<i>ApEn</i>	<i>ApD1</i>	<i>LF</i>	<i>HF</i>	<i>mRR</i> <i>ApEn</i>	<i>mRR</i> <i>ApD1</i>	<i>mRR</i> <i>LF</i>	<i>mRR</i> <i>HF</i>	<i>mRR</i> <i>ApEn</i> <i>ApD1</i>	<i>mRR</i> <i>LF</i> <i>HF</i>
Mean	0.36	0.81	0.74	0.87	0.69	0.27	0.33	0.33	0.34	0.26	0.31
SD	0.15	0.17	0.15	0.14	0.23	0.14	0.15	0.13	0.14	0.13	0.12
Min	0.12	0.40	0.35	0.43	0.23	0.09	0.11	0.12	0.12	0.08	0.11
Max	0.69	1.00	1.00	1.00	1.00	0.62	0.68	0.63	0.66	0.59	0.62

ral processes that take place in different, linked organs and organ systems. Thus, the description of the heart rate and its higher order behaviour, i.e. heart rate variability, permits the identification of the normal and pathological states of the organism. In order to increase the accuracy of heart rate parameters, a number of methods have been developed in the time and frequency domain to examine the changes in the variance as well as in the periodicity of the heart rate under a variety of conditions. More recently, studies have been undertaken to determine whether aperiodic but deterministic time structures can be identified. Pragmatic concerns have led to the development of parameters which permit a quantification of temporal complexity. In this study we examined whether such parameters of complexity do indeed augment information on physiological state or whether they reflect a simple correlation to the underlying heartbeat period.

The results show that the RRC measures examined here are not a simple function of the RR interval. The fact that the orientation and strength of the linear correlation between RRC measures and mean RR interval are different in the waking and sleeping periods under identical data acquisition and data evaluation conditions indicates clearly that the correlative interdependency is related to the physiological state. The qualitative examination of the results for single subjects on the basis of the figures strongly suggests evidence for the existence of dynamic phases in heart period dynamics. The change between regimes, which is often abrupt, may

be interpreted as phase transitions. The quantitative phase characterization on the basis of the cluster analysis further supports this interpretation. It also demonstrates the advantage of the complexity analysis over clinically applied heart rate variability analysis in the frequency domain. The differences in the patterns found in the complexity state space of the RR dynamics reflect the interindividual range in circadian rhythms and sleep-waking patterns (Cherepanova and Putilov 1993).

The discovery of phase transitions in the RR dynamics has important consequences. In the examination of 24-h heart rate regulation, many investigators using harmonic analysis concentrate on the search for continuous circadian rhythms produced by a central circadian oscillator which controls the dynamics in clock-like fashion. Phase transitions and bifurcations indicated by complexity shifts between sleeping and waking states cannot be explained as a product of a linear central oscillator but may occur in non-linear oscillating systems. In the latter, the internal zeitgeber of the organism itself is a chaotic attractor which, under special conditions, can also produce strictly harmonic circadian patterns. Lloyd and Lloyd (1994) assume the presence of high-dimensional chaotic dynamics which are controlled and stabilized by feedback mechanisms. Such systems are able to produce a wide spectrum of dynamic behaviour, whereby harmonic circadian rhythms play only a subordinate role and linear analyses can consequently represent only a small por-

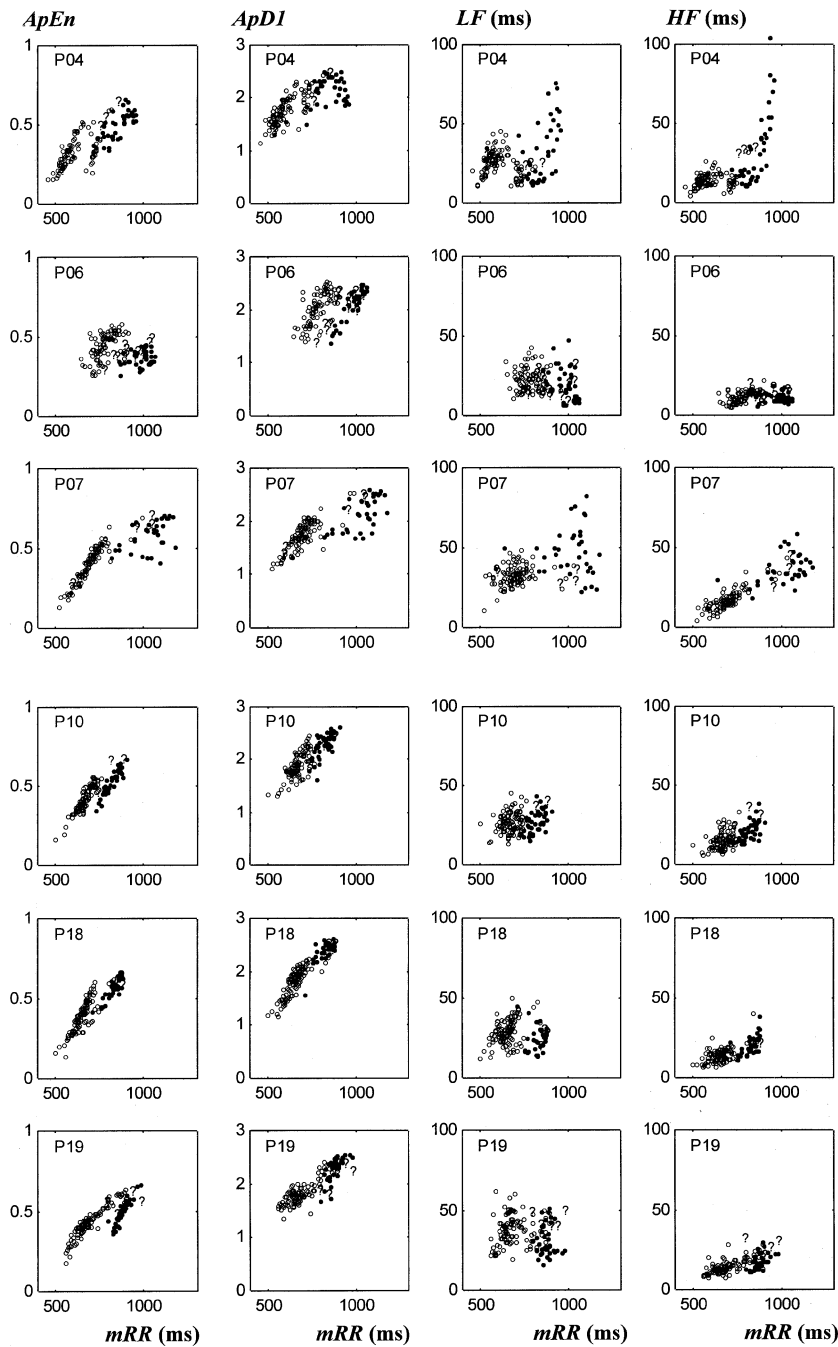


Fig. 5. Dependency of *ApEn*, *ApDI*, *LF* and *HF* on *mRR* for six selected subjects: ● waking values, ○ sleeping values, ? transition values

tion of the controlling dynamics. For comprehensive investigation of such systems, non-linear analyses are required.

The phenomenon of complexity phase transitions has been discussed previously by Y. Aizawa (1986) who refers to them as chaos-chaos phase transitions. Such transitions can produce a change of order in state space and can lead to a large inhomogeneity of the attractors. They occur preferentially in grown state chaotic systems and are seldom observed in germinal chaotic systems (routes to chaos). Aizawa also describes the possibility of identifying structural changes on the basis of fluctuations in local Lyapunov exponents or local dimensions. He describes two kinds of phase transitions: the fusion type, where several attractive

basins merge, and the entrainment type, which leads to the dominance of certain internal modes.

Physical phase transitions, such as those between thermodynamic equilibria, are generally fast dynamic processes with a high degree of unpredictability. In this context unpredictability is the means to the end and not the goal of the transition dynamics. One may image a wheel rolling down a hill: it is quite certain that the wheel will arrive at the bottom of the valley, but the route it takes can seldom be predicted. Normal physiological systems may behave in a similar fashion: the physiological landscape is rough but generally does not contain an abyss which would lead the transitional dynamics to disaster. The transition from waking to sleeping or vice versa is usually not dangerous. On the other hand,

pathology in heart rate regulation would then be characterized by a local rift in which the transition dynamics can, but need not, be caught. The above is no doubt a simplification, but it does give a synergetic explanation for phenomena which may be observed during the process of falling asleep or waking up.

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