Biol. Cybern. 67, 57–65 (1992)

Biological Cybernetics

Complex dynamics underlying the human electrocardiogram

Flavia Ravelli [1] **and Renzo Antolini** [1,2]

[1] Istituto per la Ricerca Scientifica e Tecnologica, Trento, Italy
[2] Dipartimento di Fisica, Università degli Studi di Trento, Italy

Received October 2, 1990/Accepted in revised form December 2, 1991

Abstract. Sequences of different human cardiac rhythms terminating in ventricular fibrillation have been studied, both qualitatively and quantitively, with methods of nonlinear dynamics. The analysis has been applied to ECG epochs belonging to rhythms of increasing electrocardiographic irregularity: from sinus rhythm to prefibrillatory rhythms and then to ventricular fibrillation. The phase portraits of these rhythms have been reconstructed from the ECG recording with the time-delay technique, and their correlation dimensions have been estimated with the algorithm of Grassberger and Procaccia (1983a, b). Different cardiac rhythms exhibit different correlation dimensions that describe the corresponding degrees of complexity. The correlation dimension increases as one proceeds from sinus rhythm to fully developed ventricular fibrillation via intermediate rhythms. The fully developed ventricular fibrillation shows the highest degree of complexity. The dimensional analysis supports the existence of complex dynamics underlying different cardiac rhythms and reveals an increase in dimensional complexity corresponding to an increase in electrocardiographic irregularity. Our results indicate that nonlinear dynamics may be used to assess various dynamic states of the heart and may offer a non-invasive tool to investigate the complex dynamic phenomena occuring during arrhythmia.

1 Introduction

Since the pioneering work of Einthoven (1903), the recording of cardiac electrical activity by means of electrodes placed on the surface of the body – the electrocardiogram (ECG) – has become the most common clinical tool for cardiac diagnoses, particularly diagnoses of rhythm disturbances. A great variety of rhythms, with different degrees of regularity, have been documented and classified (Sandøe and Sigurd 1984). Yet a full understanding of the time evolution of such rhythms is still a major problem in cardiac physiology, with important clinical consequences in the management of arrhythmic patients. Could the study of rhythm dynamics help to distinguish benign arrhythmias from rhythm cascade towards life threatening arrhythmias, like ventricular fibrillation? An appreciation of the actuality of this issue may be found in the vast literature generated by recent large scale clinical trials aimed at testing the hypothesis that drug arrhythmia suppression would reduce the incidence of sudden cardiac death (Task Force of the Working Group on Arrhythmias of the European Society of Cardiology 1990).

Technically, accurate long-term ECG monitoring (Holter ECG) can be readily obtained by means of small portable tape recorders (Wenger et al. 1981) and permits showing a marked spontaneous variability in both heart rate and arrhythmia frequency (Michelson and Morganroth 1980; Southall et al. 1981). However, conventional analysis of such records is mainly oriented to characterize mean heart rate and range, presence and frequency of abnormal ECG complexes, whereas the dynamic aspects of the rhythm are generally ignored.

This paper deals with the study of cardiac rhythm evolution in human subjects, using nonlinear dynamics methods. Evidence that concepts from nonlinear dynamics are relevant in cardiac physiology comes from both experimental and theoretical studies. Experimental investigations on cardiac preparations of increasing complexity – from single cells to perfused whole hearts – have shown, in response to electrical stimulation, the generation of complex patterns, typical of nonlinear systems, such as phase locking, period-doubling bifurcations and chaotic activity (Glass et al. 1983; Chialvo and Jalife 1987; Savino et al. 1989; Zbilut et al. 1989; Chialvo et al. 1990). Complex temporal or spatio-temporal patterns have also been described by computer simulations based on either the equations of membrane dynamics (Jensen et al. 1984), or a formalized model of excitable medium (Moe et al. 1964; Krinsky 1968; Smith and Cohen 1984; Chee et al. 1988). We would like to test the hypothesis that nonlinear dynamics may also play a role in the genesis of human cardiac arrhythmia.

Indeed, great caution is necessary when transferring experimental results to clinical situations, due to the

Correspondence to: F. Ravelli, Area di Biofisica Medica IRST, I-38050 Treuto, Italy

enormous increase in complexity and to the presence of pathologies not easily reproducible in the experimental models. Nevertheless, preliminary applications of nonlinear dynamics methods to human data, obtained during clinical electrophysiological investigations (Shrier et al. 1987) and routine ECG recordings (Babloyantz and Destexhe 1988; Mayer-Kress et al. 1988; Zbilut et al. 1988; Courtemanche et al. 1989; Kaplan et al. 1991) is encouraging. In particular, the ECG analysis may draw advantages from recent techniques developed to study irregular signals through the identification of attractors in the dynamics of the underlying system (Ekmann and Ruelle 1985; Holden 1986).

Several dynamic invariants have been introduced for this purpose. Among these, the correlation dimension computed with the algorithm of Grassberger and Procaccia (1983a, b) is the most widely used in the analysis of experimental data. Convincing results have been obtained with long time series of high quality relative to well controlled experiments on various physical and chemical systems, and the presence of low dimensional attractors has been demonstrated (Mayer-Kress 1986; Abraham et al. 1989).

Attempts to use the same methods to analyse complicated time evolutions, such as those observed in biological systems, have encountered more difficulties (see Ruelle (1990) for a discussion on this point). As far as human ECG is concerned, Babloyantz and Destexhe (1988) have shown, in a population of healthy subjects, that the rhythm generated by the normal cardiac oscillator is not periodic but follows a deterministic dynamics of chaotic nature. Moreover, we have recently analysed the extremely irregular ECG recorded from subjects in ventricular fibrillation (Ravelli and Antolini 1991), the most dangerous arrhythmia that has been modelled as a turbulent propagation of the excitation waves in the cardiac tissues (Moe et al. 1964, Krinsky 1968). The analysis was made when the fibrillation was fully developed, and we were not able to strictly demonstrate the presence of an attractor in the highly complex dynamics underlying the arrhythmia. This behaviour is not surprising, since it is generally observed in the fully turbulent systems of different nature, including those experimentally well controlled. In these cases, however, knowing that the time evolution at the onset of turbulence is chaotic, one may confidently infer that chaos and unpredictability are also present for fully developed turbulence, even if the experimental verification is not practically possible (Ruelle 1990).

In the present study, following the above suggestion we examined rhythms of increasing electrocardiographic complexity in good quality Holter ECG tapes of patients who had an episode of ventricular fibrillation during the recording. The tapes display sequences of different cardiac rhythms representing common patterns terminating in a low voltage ventricular fibrillation. The ECG signals of each rhythm have been reinterpreted as geometrical objects (phase portraits) in multidimensional embedding spaces, and a geometrical property of these objects – the correlation dimension – has been used to characterize the rhythms.

2 Material and methods

Holter ECG signals were obtained from the American Heart Association (AHA) Database of ventricular arrhythmias. We considered only the least half hour of each tape because it had been identified and annotated beat to beat by three experienced electrocardiographers. From the 10 tapes containing an episode of ventricular fibrillation, we first focused the analysis on one tape reaching the best compromise between signal quality and number of different rhythms, stable for at least one minute during the last half hour.

The sequence of rhythms displayed by this tape involves 24 min of sinus rhythm followed by occurrences of ventricular extrasystoles falling on the antecedent T wave (the electrocardiographic R-on-T phenomena). After about one minute, this phenomenon precipitates into ventricular fibrillation. Initially, the fibrillation shows large-amplitude oscillations (coarse fibrillation), which deteriorate into low-amplitude

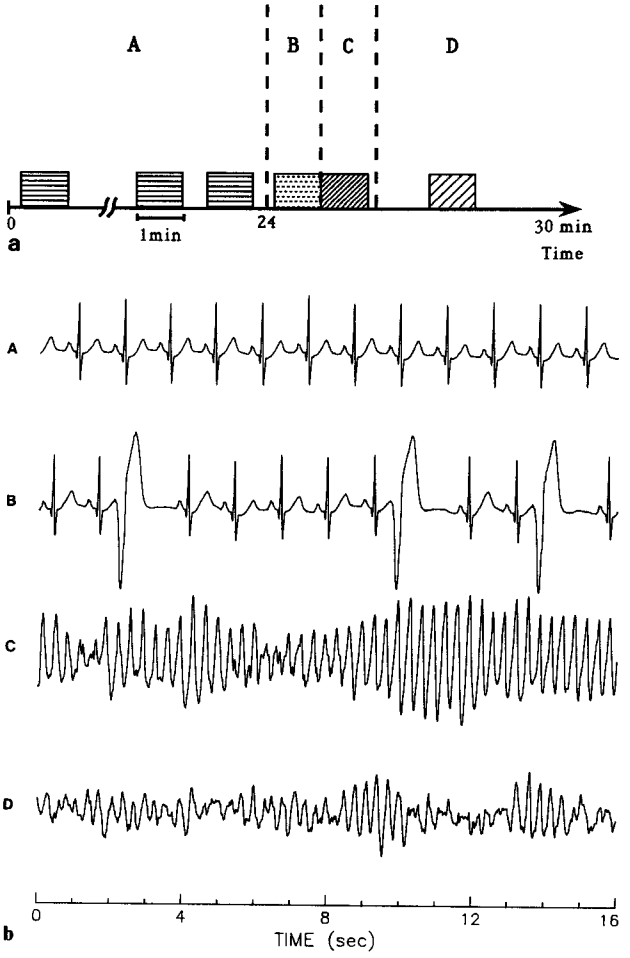

Fig. 1. **a** Schematic illustration of the ECG recording showing the location of the analysed epochs 1 min long. **b** Short ECG stretches of the four different cardiac rhythms of the recording: sinus rhythm (*A*), ventricular extrasystoles during sinus rhythm (*B*), coarse ventricular fibrillation (*C*), fine ventricular fibrillation (*D*). The four ECGs (identically scaled) are sampled at 250 Hz with 12-bit precision

waves (fine fibrillation) when the fibrillation is fully developed. This pattern of rhythms, illustrated in Fig. 1, represents a typical evolution towards ventricular fibrillation caused by the occurrence of ventricular extrasystoles during the vulnerable period of the ventricle.

The reproducibility of the results was tested in two additional tapes displaying a sinus rhythm evolving through an intermediate rhythm towards ventricular fibrillation. In one case the intermediate rhythm was a ventricular tachycardia, in the other case it was a ventricular bigeminy.

The ECG signal was sampled at 250 Hz with 12-bit precision, then segmented into stationary one-minute epochs. Figure 1a shows the location of the six epochs on the ECG record of the first case analysed. Each epoch has been processed in two steps: first by reconstructing their phase portraits, and then by estimating the respective correlation dimensions. The phase portrait of each experimental data series $\{V_i: i = 1, \ldots, N\}$ has been obtained by the time-delay technique (Takens 1981). In short, and n-dimensional vector

$$\{V_i^{(n)}\} = \{V_i, V_{i+\tau}, \ldots, V_{i+(n-1)\tau}\} \tag{1}$$

is constructed by introducing a time lag τ between the scalar data V_i. In this way, a state of the underlying system is represented as a point $\{V_i^{(n)}\}$ in an abstract n-dimensional phase space, whereas the whole time-series defines a trajectory that constitutes the phase portrait of the system dynamics. In case of dissipative systems, the trajectory is asymptotically confined within a well defined subregion of the phase space: the attractor. Since Takens (1981) has shown that the space spanned by the reconstructed variables is at least topologically equivalent to the original phase space, the geometrical reconstruction may provide qualitative information of the system dynamics. In particular, deterministic chaos, related to "strange attractors", could be distinguished from random fluctuations, corresponding to an unstructured cloud.

The next step is a quantitative characterization of the attractors obtained by computing a geometrical invariant: the dimension. Calculation follows a method proposed by Grassberger and Procaccia (1983a, b) and estimates the correlation dimension $D2$, which is a lower bound for both the Hausdorff and the information dimensions. The algorithm involves the computation of the correlation integral:

$$C_n(R) = N^{-2} \sum_{\substack{i,j=1 \\ i \neq j}}^{N} \Theta(R - \| V_i^{(n)} - V_j^{(n)} \| \tag{2}$$

where Θ is the Heaviside function and n the embedding dimension. The integral measures the spatial correlation of the points on the attractors and should scale as $C_n(R) \sim R^{D2(n)}$. The correlation dimension $D2$ is then estimated as:

$$D2 = \lim_{\substack{R \to 0 \\ n \to \infty}} \frac{\text{Log } C_n(R)}{\text{Log}(R)} \tag{3}$$

In practice, $C_n(R)$ could be approximated by averaging in (2) on a limited number K of origins V_i instead of N. For our calculation, we used 1000 equally spaced reference points, the total number of data being $N = 15000$. A test of the independence of $C_n(R)$ from K has been performed. For the estimation of $D2(n)$, we first plot Log $C_n(R)$ as a function of Log R, then we look for a linear region and we compute the slope with a least square fit. To better identify a linear region, we also report plots of the slope d Log $C_n(R)/d$ Log R as a function of Log $C_n(R)$. To reduce the fluctuations from one point to the others the slope has been estimated as:

$$\frac{(\text{Log } C_n(R_{i+1}) - \text{Log } C_n(R_{i-1}))}{(\text{Log } R_{i+1} - \text{Log } R_{i-1})} \tag{4}$$

$D2$ has been evaluated by calculating $D2(n)$ at successively higher values of the embedding dimension n. If $D2(n)$ reaches a saturation value, the system represented by the time series should possess an attractor, and the saturation value $D2$ is an estimation of the attractor dimension.

3 Results

Some qualitative information on the dynamics of the system underlying the ECGs are given by the trajectories generated from the delayed vectors (1). Figure 2 shows the two-dimensional phase portraits of the four different rhythms: sinus rhythm, ventricular extrasystoles during sinus rhythm, coarse fibrillation, fine

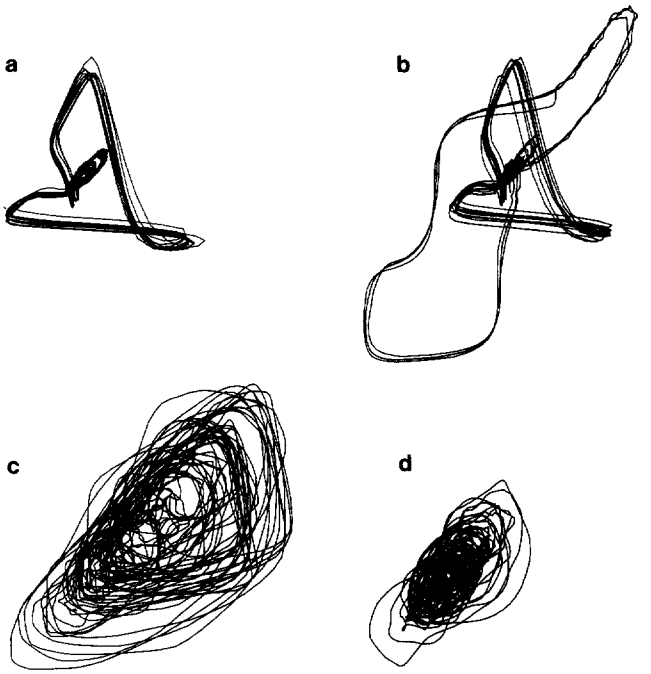

Fig. 2. Two-dimensional phase-portraits $V_{i+\tau}$ vs V_i (identically scaled) of the four different cardiac rhythms displayed in Fig. 1 (sinus rhythm (**a**), ventricular extrasystoles during sinus rhythm (**b**), coarse ventricular fibrillation (**c**), fine ventricular fibrillation (**d**)). The four portraits are generated by 16 s of recording with τ equal to 40 ms

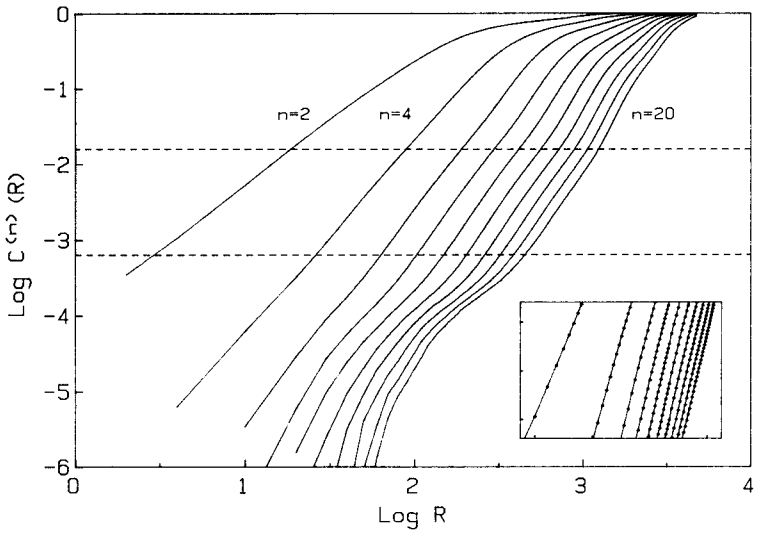

Fig. 3. Log-Log plot of the correlation integrals ($n = 2, 4, 6, \ldots, 20$) for the ECG recording displaying ventricular extrasystoles during sinus rhythm. The two dotted lines define the range of linearity of the correlation integrals. The linear zone is expanded in the insert. This calculation is based on 15000 data points sampled at 250 Hz

fibrillation. The protraits show a tendency towards more complicate structures going from the sinus rhythm to the fully developed fibrillation. In fact, the trajectory of the sinus rhythm follows a well defined path with a high degree of coherence, although it does not describe a single close curve which would represent a perfect periodic oscillator. The phase portrait of the second rhythm is more complicated than the first one, since another closing loop – corresponding to the ventricular extrasystoles – has been introduced. During fibrillation, the definite paths break off showing trajectories without a visible structure. Nevertheless, the phase portrait in the first minute of fibrillation occupies a larger portion of the phase space than during fully developed fibrillation. In this final stage, the phase portrait is densely filled, showing the minimum coherence.

The preliminary observations about the features of the ECG phase portraits can be expressed quantitatively by the correlation dimension. Figure 3 displays the logarithmic plot of the correlation integrals at increasing n for the ECG recording containing ventricular extrasystoles. It should be noted that only part of the correlation integral can be used for the calculation of the dimension. In fact, at small values of R the correlation curves are spoiled by statistical fluctuations and by a distortion due to the oversampling of the data (Theiler 1986), whereas at large values of R there are evident nonlinearities. The range of linearity, delimited by two dotted lines and expanded in the insert of Fig. 3, is then limited to intermediate values of R.

Due to these problems, the characteristics of the correlation curves are better evidenced by displaying their slopes. Figure 4 shows the slopes, at a fixed

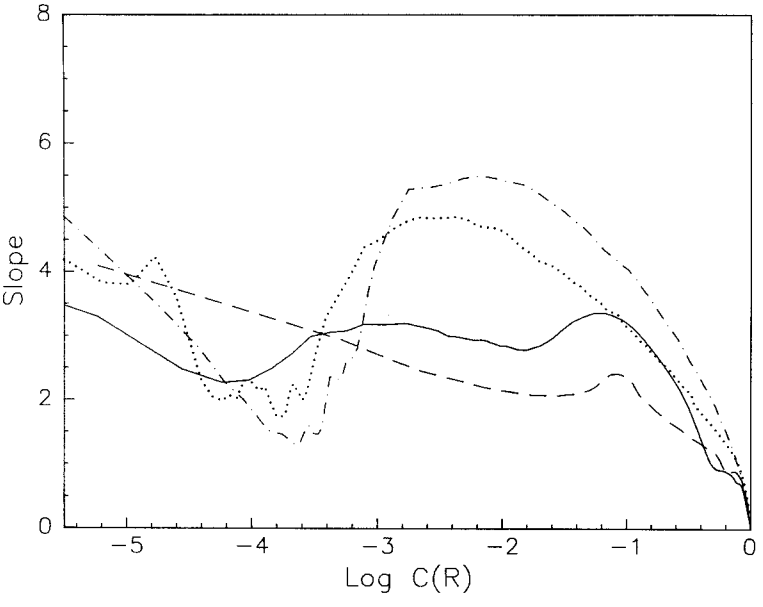

Fig. 4. The slopes of the correlation integrals as a function of Log $C_n(R)$ at fixed embedding dimension ($n = 8$) for the four different cardiac rhythms (– – sinus rhythm, —— ventricular extrasystoles during sinus rhythm, · · · · coarse fibrillation, – · – fine fibrillation)

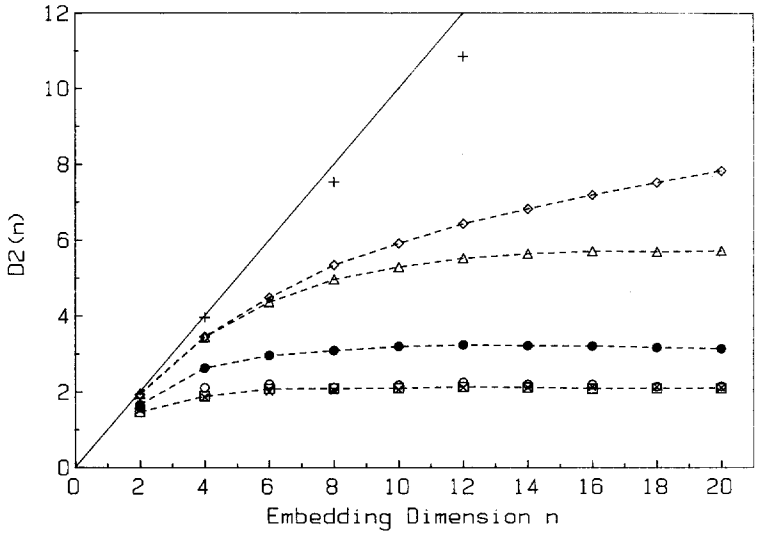

Fig. 5. Correlation dimension vs embedding dimension n for the six analysed ECG epoches (□, ×, ○: first, second and third epoch of sinus rhythm; ●: ventricular extrasystoles during sinus rhythm; △: coarse ventricular fibrillation, ◇: fine ventricular fibrillation) and for a signal generated by a gaussian random process (+)

embedding dimension ($n = 8$), for the four different rhythms. The four curves are not well distinguishable for Log $C(R) < -3$, although the distortion due to oversampling is more marked for those obtained from ventricular fibrillation ECGs. In fact, the sources of corruption of the slope at small values of R (noise and excessive digitalization rate) are characteristic of the whole ECG recording and therefore are common to the four ECGs. On the contrary, the slopes in the linear region display well distinct values corresponding to the four different rhythms. Moreover, when the rhythms become more complex – going from sinus rhythm to ventricular fibrillation – the slope shows an evident increase. The results are summarized in Fig. 5, where the $D2(n)$ are displayed as a function of the embedding dimension n for the six ECG epochs.

It should be noted that the correlation dimensions corresponding to the four different cardiac rhythms have well distinct values at each embedding dimension, forming four different curves. By contrast, the correlation dimensions for the tree epochs of the sinus rhythm recording are superimposed. These results suggest the existence of a characteristic correlation dimension for each analysed cardiac rhythm. The curves for the sinus rhythm, the ventricular extrasystoles during sinus rhythm, and the first minute of fibrillation show saturation at increasing embedding dimension, while the curve corresponding to the fully developed fibrillation does not display a complete saturation, although it stays largely below the one calculated for a pure random process. $D2$ for the sinus rhythm is equal to 2.1. This value increases to 3.2 when ventricular extrasystoles occur. When the ventricular fibrillation appears, there is a further jump in the dimensionality, and $D2$ for the coarse fibrillation is 5.7. The value of $D2$ can not be evaluated for the fine fibrillation since a small increase of the dimension is still observed at embedding dimension equal to 20 where the correlation dimension reaches a value of 7.8. All best-fit values of the slopes have an overall estimated error of ± 0.1.

It should also be noted that the correlation dimensions of each rhythm – except for the fully developed fibrillation – stays well below the upper bound $\text{Log}_{10}N^2$ of the Grassberger-Procaccia algorithm proposed by Ruelle (1990). More exactly, since we did not use all the distances between the N data points but only those between K reference points and the N data, the upper limit should be $\text{Log}_{10}KN$ instead of $\text{Log}_{10}N^2$. In our case, such a limit is 7.1. Only the fully developed fibrillation violate this condition, indicating first of all that the slope has been calculated over less than a decade, and moreover that this arrhythmia cannot be associated with a low-dimensional chaotic dynamical system.

Calculation of the correlation dimension on physiological data requires a careful check of the influence of the parameters considered in the computation algorithm on the results. An important point in the evaluation of the correlation dimension is the choice of the value of time delay τ. Theoretically, for an infinite time series the value of τ may be chosen arbitrarily. However, in the experimental setting – where the number of data points is finite – only some values of τ give reliable results. The first zero of the autocorrelation function, or the first minimum in mutual information content, have been proposed to obtain the optimal time delay (Fraser and Swinney 1986). In all the computation, we have used the time of the first zero crossing of the autocorrelation function as a value of τ. The values of τ for the six ECG epochs range from 40 ms to 224 ms. To check for the validity of the choice of τ, we have calculated the dependence of the correlation dimension as a function of τ. For each rhythm we have found a range of τ with a size of about 200 ms, where the results are reproducible and the times of the first zero of the autocorrelation function are included in the range.

The choice of the number of points to be used to obtain consistent results also has been investigated. Since for small data sets there is a substantial underesti-

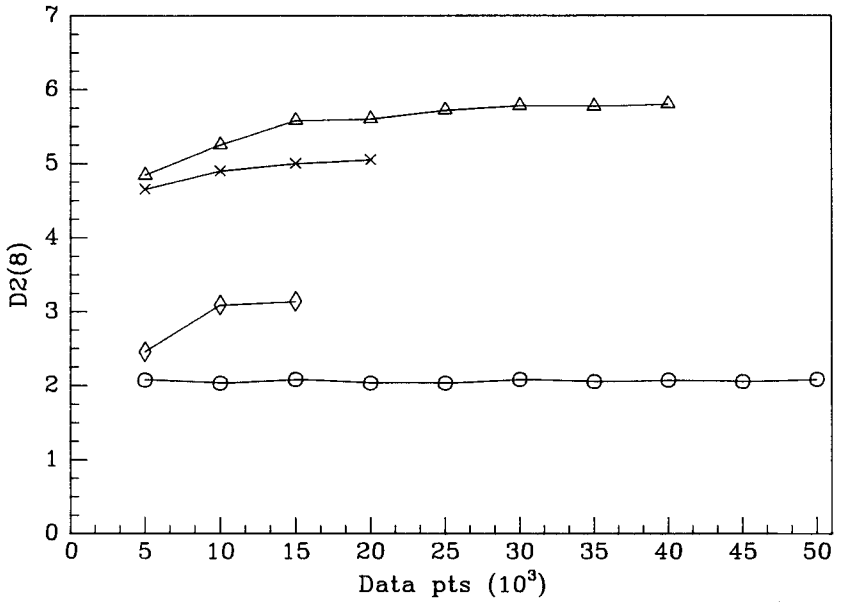

Fig. 6. Correlation dimension as a function of the number of data points at fixed embedding dimension ($n = 8$) for the sinus rhythm (○), ventricular extrasystoles during sinus rhythm (◇), coarse fibrillation (×) and fine fibrillation (△)

mation of the dimension, the analysis of the correlation dimension requires long time series. On the other hand too long sequences of physiological data can include non-stationary phenomena. Thus the choice of the number of data is a critical point. The convergence of the value of the correlation dimension at increasing number of points has been studied. Figure 6 shows the variation of the correlation dimension with the number of points for the four analysed rhythms. The maximum number of points used for each ECG is limited by the duration of each rhythm and by the necessity of using stationary data sets. In our case the longest data set was obtained for the sinus rhythm. By considering the whole set of rhythms the values of the correlation dimension converge for $N \geqslant 15000$. For the sinus rhythm smaller data sets can be used.

The choice of the sampling rate requires further attention. Too high a sampling rate will include noise and produce distortion in the correlation integral, while too low a sampling rate may lose the fine structure of the trajectories. We decreased the original sampling rate of the ECG from 250 Hz to 125 Hz and then to 50 Hz. The most evident effect of the undersampling was the disappearance of the distortion of the correlation integral well evident in Figs. 3 and 4 at small values of R. Figure 7 gives the correlation dimension as a function of the embedding dimension for the three values of the sampling rate. Although the range of linear region changes, the value of the correlation dimension doesn't change substantially with the variation of the sampling rate.

To verify that changes in cardiac rhythm are coupled to a parallel variation in the correlation dimension,

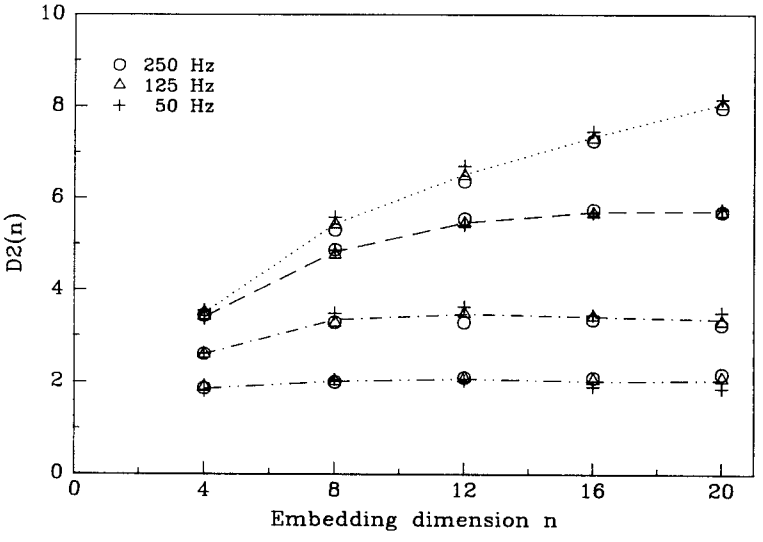

Fig. 7. Correlation dimensions vs embedding dimension n for the sinus rhythm (– · · –), ventricular extrasystoles during sinus rhythm (– · –), coarse fibrillation (– –) and fine fibrillation (· · · ·) calculated at three different sampling rates: 250 Hz, 125 Hz, 50 Hz. The same 60 s ECG were used for the three attempts

two additional cases of evolution from sinus rhythm to ventricular fibrillation have been studied. In one case the sequence of rhythms was a sinus rhythm followed by a ventricular tachycardia which deteriorated into ventricular fibrillation. In the second case the sinus rhythm was followed by a ventricular bigeminy that precipitated into ventricular fibrillation. The analysis has been applied to these additional cases following the procedure described above. In the first additional case the correlation dimension of the sinus rhythm is 3.2. This value increases to 5.3 when ventricular tachycardia occurs, then to 6.7 when ventricular fibrillation appears. In the second case the correlation dimension increases from 4.7, the value for the sinus rhythm, to 5.8 for ventricular bigeminy to 7.1 for ventricular fibrillation.

4 Discussion

Our concern was to investigate the cardiac rhythm evolution with methods of nonlinear dynamics by reconstructing the phase portrait of each rhythm and estimating its correlation dimension. The rationale for the dimensional analysis of ECGs relies on a large collection of experimental and simulation results supporting the modelling of cardiac electrical activity as a nonlinear system (Glass et al. 1983; Jensen et al. 1984; Chialvo and Jalife 1987; Chee et al. 1988; Savino et al. 1989; Zbilut et al. 1989). This approach offers a promising explanation to the intriguing contradiction between the sudden changes in rhythm observed on the ECG and the relatively slow variations in the overall cardiac state. Within this framework, based on a model of nonlinear system with more than one basin of attraction, the transition to fibrillation may be considered as the switch from one basin to another induced by a proper disturbance (Kaplan et al. 1988).

The experimental analysis was carried out principally on a good quality ambulatory ECG from a patient who had an episode of ventricular fibrillation during the recording. The correlation dimension $D2$ calculated for the sinus rhythm is 2.1 and remains constant during the 24 min considered, also in the presence of a 30% reduction of the mean cardiac rate. The sudden change in rhythm due to the appearance of ventricular extrasystoles on the ECG is clearly detected, both qualitatively, by an evident modification of the phase-portrait, and quantitatively by an increase of $D2$ from 2.1 to 3.2. It should be noted that the two attractors here considered are inhomogeneous like the majority of the physiological attractors. Since the correlation dimension in principle describes homogeneous attractors the use of $D2$ to characterize inhomogeneous attractors has to be considered an approximation. Nevertheless, at present, in the absence of other robust methods able to characterize inhomogeneous attractors, the correlation dimension, largely applied to physiological data series, can constitute a good approximation of the degree of complexity of the system dynamic.

After the appearance of ventricular extrasystoles the successive sudden change in the ECG rhythm marks the transition to ventricular fibrillation that first appears in coarse form and then degenerates into fine form after about 1 min. Also in this case, the change in rhythm of the ECG determines a gross modification in the phase portrait that loses any clear structure and produces a jump in the correlation dimension $D2$ from 3.2 to 5.7. $D2$ saturates as a function of the embedding dimension and stays well under both the values calculated from a gaussian random process and the limits proposed by Ruelle (1990).

The last rhythm, the fine ventricular fibrillation, produces a phase portrait densely filled, corresponding to a correlation dimension that does not completely saturate as a function of the embedding dimension, although it largely stays below the values corresponding to a guassian random process. In this case, due to the lack of complete saturation, we cannot estimate $D2$, but – purely for comparative purposes – we can compare the values of the correlation dimension at fixed embedding dimension for the fine ventricular fibrillation to the corresponding values calculated for the coarse ventricular fibrillation. It can be seen that $D2(n)$ for fine fibrillation is at each embedding dimension higher than the dimensions calculated for the coarse fibrillation. Also in this more complex case, an increase in the disorganization of the ECG corresponds to an increase in the complexity parameter.

The evolution towards ventricular fibrillation on which we focused our analysis is only one possible route to ventricular fibrillation, although quite typical. In the clinical setting other sequences of rhythms terminating in ventricular fibrillation have been observed. In this study we have extended the analysis to two additional cases of evolution towards ventricular fibrillation. In one case the intermediate rhythm between sinus rhythm and ventricular fibrillation was a ventricular tachycardia, in the other case a ventricular bigeminy. The analysis of these two sequences of cardiac rhythms confirmed the results previously obtained: the correlation dimension increases as one proceeds from sinus rhythm to ventricular fibrillation via intermediate rhythms. The ventricular fibrillation shows the highest degree of complexity.

Our results relative to sinus rhythm in patients undergoing ventricular fibrillation may be compared with those of Babloyantz and Destexhe (1988); Mayer-Kress et al. (1988); and Kaplan et al. (1991), obtained from normal subjects, and with the results of Zbilut et al. (1988) obtained from transplanted hearts. Qualitatively, all of these results are in agreement and show that the sinus rhythm follows chaotic dynamics. As concerns the estimate of the correlation dimensions on the ECG signal our results show a value of $D2$ ranging from 2.1 to 4.7 for the three cases of sinus rhythm. These values are in agreement with the results of a similar analysis performed on the entire ECG of healthy subjects and described in the literature (Babloyantz and Destexhe 1988).

The fact that ventricular fibrillation displays the highest dimensionality is in agreement with the current physiological view of fibrillation, describing the arrhythmia as the most disordered rhythm. Such a description is supported by the very complex spatio-temporal pattern of fibrillation mapped in real hearts (Allessie et al. 1985) or described in mathematical models (Moe et al. 1964; Krinsky 1968; Smith and Cohen 1984) and by recent studies that, using the coherence spectrum calculated from the electrograms recorded in two intracardiac sites, discriminate fibrillatory and nonfibrillatory rhythms (Ropella et al. 1989).

The results here presented support the existence of chaotic dynamics underlying different cardiac rhythms. Nevertheless since real-word data such as ECG signals are noise prone and since the presence of noise can corrupt the values of the correlation dimension some cautions are in order. The application of other more recent approaches to the phase space reconstruction (Broomhead and King 1986) and the calculation of other dynamical quantities such as Lyapunov exponents and Kolmogorov entropy could support further the chaos hypothesis. Nevertheless the calculation of the correlation dimension, independently from the chaos test, can lead to the estimation of an index of complexity of the rhythm in examination. However, such estimations can not at present be considered as absolute values since the cardiac system is not uniquely defined. The same rhythm in different subjects can have its own peculiarity and therefore it is probable that the extension of the analysis to a large population does lead to a characteristic dimension for each rhythm but with a large variance. Given the difficulty of estimating the absolute value of dimensions in biological systems, we indicated the use of the dimensional analysis in a comparative sense as in the case of EEG analysis (Babloyantz and Destexhe 1987). Our results support the conjecture that the longitudinal study applied to single hearts switching between different cardiac states can provide a valuable tool for detecting relative changes in the complexity of the system dynamics.

The application of nonlinear dynamic methods to the analysis of ECG is only at the beginning and therefore at present this new approach can not offer the interpretational assurance of the traditional methods of analysis, such as spectral analysis. However all these new methods, which are basically different from the classical approaches assuming the signal is generated by the superposition of periodic oscillations, can reveal dynamical aspects unrecognized by traditional analysis. In particular, although this study does not lead to the formulation of the mathematical models underlying cardiac activity, it can help in the construction of a model. In any case the evaluation of the correlation dimension may be used to assess various dynamic states of the heart and may constitute a non-invasive tool for analysis of the complex dynamic phenomena occurring during arrhythmias.

Acknowledgements. We gratefully acknowledge financial support from Istituto Trentino di Cultura, Consiglio Nazionale delle Ricerche (CNR-GNCB) and Ministero per l'Università e la Ricerca Scientifica e Tecnologica (40% Biofisica).

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