On the evidence of deterministic chaos in ECG: Surrogate and predictability analysis

R. B. Govindan, K. Narayanan, and M. S. Gopinathan
Department of Chemistry, Indian Institute of Technology, Madras, Chennai, India-600 036

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The question whether the human cardiac system is chaotic or not has been an open one. Recent results in chaos theory have shown that the usual methods, such as saturation of correlation dimension D_2 or the existence of positive Lyapunov exponent, alone do not provide sufficient evidence to confirm the presence of deterministic chaos in an experimental system. The results of surrogate data analysis together with the short-term prediction analysis can be used to check whether a given time series is consistent with the hypothesis of deterministic chaos. In this work nonlinear dynamical tools such as surrogate data analysis, short-term prediction, saturation of D_2 and positive Lyapunov exponent have been applied to measured ECG data for several normal and pathological cases. The pathology presently studied are PVC (Premature Ventricular Contraction), VTA (Ventricular Tachy Arrhythmia), AV (Atrio-Ventricular) block and VF (Ventricular Fibrillation). While these results do not prove that ECG time series is definitely chaotic, they are found to be consistent with the hypothesis of chaotic dynamics. © *1998 American Institute of Physics.* [S1054-1500(98)00202-X]

It is often difficult to decide whether the dynamics of a biological system is chaotic or not. An experimental signal from such a system must therefore be put through several stringent tests to detect the signature of chaos. In this article we apply several recently developed tests to the human electrocardiogram signal. It is suggested that while no conclusive proof for chaos in an experimental system is possible, the hypothesis of chaos cannot be ruled out in the human cardiac system.

I. INTRODUCTION

Nonlinear system theory has been widely used in recent years to characterize the behavior of a dynamical system from a single experimental time series especially in the analysis of electrocardiogram (ECG) and electroencephalogram (EEG) signals.[1–8] The purpose of such studies is to determine whether dynamical indices such as correlation dimension, Lyapunov exponent and entropy can serve as clinically useful parameters.

However, the reliability of these indices has been questioned.[9,10] The basic difficulty is in ascertaining whether the experimental time series is generated by a chaotic or a linear stochastic process. It has now been realized that the usual measures like saturation of correlation dimension and existence of positive Lyapunov exponent cannot by themselves establish the chaotic behavior of the system.[11]

Errors associated with the acquisition of data like inappropriate sampling frequency, noise filtering and digitization error can lead to uncertainties in the value of correlation dimension, D_2. Even for uncorrelated random data, the correlation dimension converges at a value of $D_{2,\max} = (-2 \log N)/\log \epsilon$, where N is the number of points and ϵ is the length scale at which the slope of the correlation integral is calculated.[12] Therefore, we cannot confidently take the system behavior as chaotic based solely on the convergence of D_2.

For a time series without noise the largest Lyapunov exponent $\lambda_{\max}$ gives the exponential rate of divergence of two neighboring trajectories in the phase space. However, the existence of positive $\lambda_{\max}$ is true of stochastic dynamical systems also. Therefore, this broader sense definition of $\lambda_{\max}$ cannot be used to brand a system as chaotic or random since both the chaotic and stochastic systems can have a positive $\lambda_{\max}$.[11]

There are two possible approaches for calculation of the Lyapunov exponent, namely the Jacobian method[13,14] and the direct method.[15,16] In the Jacobian method, Lyapunov exponents are computed by multiplying Jacobian matrices along the trajectory, with the matrices computed by local linear fit and applying QR decomposition to maintain orthogonality. In the direct method $\lambda_{\max}$ is calculated directly from the divergence of pairs of trajectory segments. The numerical estimation of even the largest Lyapunov exponent can be problematic in the presence of noise.[17] Even for the linear stochastic process, there can be local expansions by sheer chance resulting in a positive exponent.[11]

Previous nonlinear dynamical studies of the ECG time series by Casseleggio *et al.*[18] and Babloyantz *et al.*[19] suggested that the cardiac system is chaotic based on the saturation of D_2 and the positive value obtained for the largest Lyapunov exponent. Values of D_2 ranging from 2.1 to 5.2 were reported. The study of D_2 and short-term predictability of interbeat intervals has also supported the hypothesis of chaos in the cardiac system.[20]

Recent work based on surrogate data sets[11,21] together with the short-term prediction[22,23] of the time series has shown that these methods can be valuable in ruling out linear stochastic processes in a time series.

In this work, we report detailed results of surrogate analysis of ECG time series with D_2 as discriminating metric and also the short-term predictability test on the ECG for several normal and pathological cases. We also report the estimation of D_2 and λ_{max} for these cases.

Our results on the evidence of presence of nonlinearity from surrogate analysis together with the results of short-term prediction with D_2 and positive λ_{max} are consistent with the hypothesis of chaos in all normal and pathological cardiac systems. It must, however, be emphasized that these tests are only suggestive and probably there is no foolproof method of conclusively establishing that a given biological or experimental signal arises from a low dimensional chaotic process.

II. SUBJECTS

The first step in the study is the acquisition of ECG time series of normal and pathological subjects. We recorded the lead II of the ECG for normal subjects at a sampling frequency of 360 Hz using volunteers. The ECG of pathological subjects such as premature ventricular contraction (PVC), ventricular tachyarrhythmia (VTA), atrio-ventricular (AV) block and ventricular fibrillation (VF) were taken from the MIT-BIH Arrhythmia Database.[24] The PVC and AV block time series are sampled at 360 Hz and VTA and VF at 250 Hz. We filtered the ECG between 0.5 to 45 Hz and down sampled the normal, PVC and AV block to 90 Hz and the rest of them to 125 Hz.

Before applying the nonlinear dynamical tools to characterize the ECG signals, we checked for the stationarity of the time series in all cases studied here by calculating the autocorrelation function and the rms deviation for every one-fifth of the time series. For example, for a normal subject, the values obtained are 70.0 ± 1.3 and 0.03 s for rms deviation and correlation time, respectively, for every one-fifth of the time series. Similar results obtained for all the cases show the stationarity of the time series.

III. CORRELATION DIMENSION AND LYAPUNOV EXPONENT

The ECG signal was processed in three steps. First by reconstructing their phase portraits, second by estimating D_2 and finally by calculating the largest Lyapunov exponent λ_{max}. The phase portrait of each experimental data series $\{x_i : i = 1, \ldots, N\}$ was obtained by the time-delay technique[25] using delay vectors: $\{X_i^n\} = \{x_i, x_{i+\tau}, \ldots, x_{i+(n-1)\tau}\}$, where n is the embedding dimension and τ is the delay time.[26] The next step is the estimation of D_2 which gives the minimum number of variables necessary to describe the state of the system at any time. We followed the method proposed by Grassberger–Procaccia[27] according to which saturation value of D_2 gives the attractor dimension. λ_{max} is calculated by following the method of Wolf *et al.*[15] in which the largest Lyapunov exponent is computed from the growth of length elements and when the length of the vector between two points becomes large, a new point is chosen near the reference trajectory, minimizing both the replacement length and the orientation change.

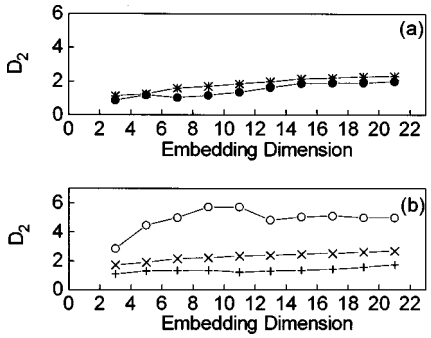

FIG. 1. Variation of correlation dimension D_2 with embedding dimension for typical subjects. (a) Normal and VTA indicated by ''•'' and ''*'' (b) VF, PVC and AV block indicated by ''○,'' ''×,'' and ''+'', respectively.

We obtained the saturation of D_2 (see Fig. 1) for all the subjects around the embedding dimension of 15. We have also obtained positive λ_{max} for all the subjects we analyzed by reconstructing the attractor at optimum embedding dimension obtained from the results of correlation dimension analysis. The optimum embedding dimension is given by the integer value of D_2 (see Table I) plus one. In all the cases we have used 10,000 data points of the down sampled ECG time series. The D_2 values for normal, PVC, VTA and AV block subjects saturates around 2–4 (Fig. 1) indicating low dimensional deterministic process. However, for the case of VF, D_2 saturates at a higher value of around 6 which shows that the dynamics underlying VF spans significantly higher dimensions [refer to Fig. 1(b)] than other pathological cases. The λ_{max} values for various cases are given in Table I.

The entries Nor1 and Nor2 in the column data of Table I, represents the data files recorded by ourselves and the rest of the data were taken from MIT-BIH Database.[24] There is no significant difference in the values of λ_{max} for normal and pathological conditions except in a few cases of VTA and AV block where the values are low as for cu03 and mit231 in Table I.

To verify whether the number of data points are sufficient for the D_2 calculation, we have carried out the D_2 estimation for a normal and pathological subject VF as a test

TABLE I. Values of correlation dimension and largest Lyapunov exponent for various subjects. Ten thousand data points were used in all the calculations.

Subject	Data	Correlation dimension (D_2)	Largest Lyapunov exponent λ_{max} bits s^{-1}
Normal	Nor1	1.91 ± 0.05	1.45 ± 0.24
	Nor2	2.23 ± 0.20	1.27 ± 0.25
PVC	mit107	2.57 ± 0.10	1.70 ± 0.13
	mit200	3.50 ± 0.10	1.63 ± 0.11
AV block	mit207	1.65 ± 0.11	0.92 ± 0.07
	mit231	2.76 ± 0.18	0.69 ± 0.15
VTA	Cu02	2.17 ± 0.08	1.12 ± 0.03
	Cu03	1.73 ± 0.05	0.56 ± 0.07
VF	Cu05	5.90 ± 0.10	1.69 ± 0.16
	Cu10	5.01 ± 0.10	1.58 ± 0.14

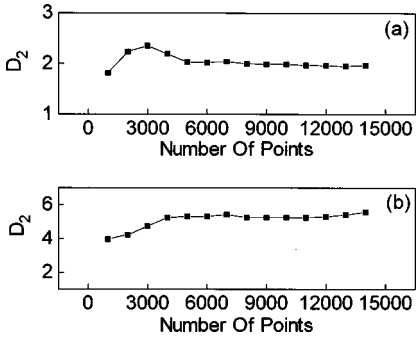

FIG. 2. Variation of correlation dimension D_2 as a function of number of data points. (a) For a normal subject. (b) For a pathological subject VF.

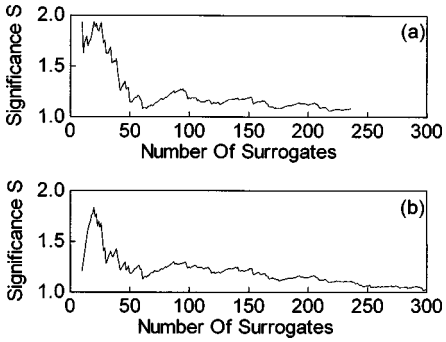

FIG. 3. Variation of the significance S for filtered noise using D_2 as discriminating metric with the number of surrogates. (a) Random phase surrogates. (b) Gaussian scaled surrogates.

case, as a function of number of data points, with a high embedding dimension of 16. This study showed no significant change in the D_2 values as we increase the number of data points beyond 6,000 indicating that the 10 000 data points used are sufficient for the D_2 calculation (Fig. 2).

We cannot confirm the presence of chaos from the saturation of D_2 and the existence of positive Lyapunov exponent alone for the reasons mentioned earlier. We now proceed to surrogate data and predictability analysis in the following sections to test the null hypothesis that the ECG time series is generated by a linear stochastic process.

IV. SURROGATE DATA ANALYSIS

The method of surrogate data analysis was developed by Theiler *et al.*[21] to detect any nonlinearity present in the time series. Since nonlinearity is the essential criteria for chaotic dynamics, the technique is widely applied[28–34] to rule out linear stochastic processes in an observed time series. Since linear correlations create many of the spurious results, the method compares the original time series with artificially generated random series, the so-called ''surrogate data'' that can mimic the linear properties of the original signal.

In this study, random phase surrogate sets and Gaussian scaled random phase surrogate sets are generated and used to test the null hypothesis that the ECG time series is generated by a linear stochastic process.

The random phase surrogate addresses to a hypothesis that the original time series is linearly correlated Gaussian noise.[32] This type of surrogates are generated by first calculating the power and phase spectrum of the original time series and then randomizing the phase information which destroys the nonlinear structure, if any, and then Fourier transforming back into the time domain. The surrogate and original time series will have the same power spectrum and therefore the same autocorrelation function.

The Gaussian scaled random phase surrogate addresses to a hypothesis that the original time series is linearly correlated noise that has been transformed by a static, monotone nonlinearity.[32] These types of surrogates are prepared by first generating a Gaussian distributed set of random numbers, followed by the reordering of the rank structure of the Gaussian data set in such a way that ranks of the Gaussian set and ranks of the original time series agree. After that, the phase randomization procedure is applied to the Gaussian data set. Finally, the original time series is reordered so that rank structure of the original time series agrees with the rank structure of the phase randomized Gaussian data set. The Gaussian scaled random phase surrogate is given by the reordered original time series. This surrogate will have the same empirical distribution as the original data and therefore the same first-order statistics like average and variance and it preserves the autocorrelation function approximately.[33]

While constructing the surrogate data sets the linear trend is subtracted out before calculating the spectrum. The trend is restored to the surrogate before computing any discriminating statistic. This is to avoid any small errors in the calculation of spectrum that can have a statistically significant deleterious effect on the surrogates.

We choose to use D_2 as the discriminating metric. All spurious effects in the calculation of D_2 due to spectral filtering, linear correlation or limited number of data points affect both the original time series and the surrogate data sets to the same extent. If the original and its surrogate behave significantly differently, for a chosen discriminating statistic, then we can conclude to a good degree of confidence, that the system under study has a nonlinear structure. However, this does not mean that the underlying dynamics is necessarily chaotic. The method of surrogate data can be used to exclude certain classes of stochastic dynamics but a definite positive conclusion of chaos in the experimental data cannot be inferred.

The significance of the difference between original time series and surrogate data can be measured by[32] $S=(\langle M_{\text{surr}}\rangle - M_{\text{org}})/\sigma$, where M_{surr} and M_{org} are the discriminating metric measure of surrogate and original time series, respectively. $\langle M_{\text{surr}}\rangle$ is the mean of M_{surr} and σ is the standard deviation of M_{surr}.

Before applying the surrogate data analysis to ECG, a test for the software used to generate surrogate was performed on the lines suggested by Rapp.[35] The artificial data (i.e., the set of uniformly distributed random numbers on the unit interval which is filtered using the procedure given in Ref. 35) is subjected to surrogate analysis and the number of surrogates were increased until the saturation of S with D_2 as the discriminating metric is observed (Fig. 3).

The saturation value of S is nearly 1.1 ± 0.1 for both random phase and Gaussian scaled surrogates which is not

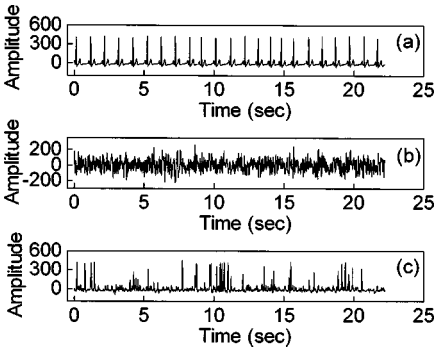

FIG. 4. Portion of signal for a typical normal subject along with its surrogates. (a) Original time series. (b) Phase randomized surrogate. (c) Gaussian scaled surrogate. Amplitude is in arbitrary units.

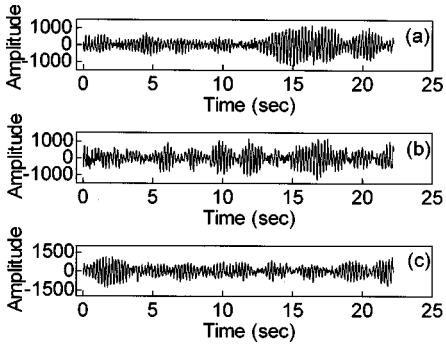

FIG. 6. Portion of signal for a typical VF subject along with its surrogates. (a) Original time series. (b) Phase randomized surrogate. (c) Gaussian scaled surrogate. Amplitude is in arbitrary units.

significant.[35] Hence we fail to reject the null hypothesis. Further, according to the Barnard-Hope criterion[32] also, we fail to reject the null hypothesis addressed by both types of surrogates since in the case of random phase surrogates, out of 236 surrogates, for 204 cases we have $D_{\text{orig}} > D_{\text{surr}}$ and in the case of Gaussian scaled surrogates out of 300 surrogates, for 255 cases $D_{\text{orig}} > D_{\text{surr}}$. The Monte Carlo probability P_M defined as

$$P_M = \frac{(\text{number of cases } D \leqslant D_{\text{orig}})}{(\text{number of cases})},$$

which gives the probability that a value of D_{surr} will be less than that of D_{orig}, is 0.86 for random phase and 0.85 for Gaussian scaled surrogates. By this criterion also we fail to reject the hypothesis addressed by both types of surrogates. This failure to reject the hypothesis shows that the software used to generate the surrogate sets is reliable.

After the check for the reliability of the software, the ECG time series of normal and pathological conditions were subjected to random phase surrogate analysis. The original ECG data along with their phase randomized and Gaussian scaled surrogates for a duration of 22 s out of ~100 s of data points are shown in Figs. 4, 5 and 6 for a typical normal, PVC and VF subjects, respectively. D_2 for both the original and their surrogate sets were calculated for normal and pathological subjects using 10,000 data points. The variation of D_2 as a function of embedding dimension for various subjects with one of their typical surrogates are shown in Figs. 7(a)–7(e).

In all the cases, the number of random phase surrogates were increased until a stable value of S was reached (Fig. 8). The significance S for a normal, PVC, VTA, AV block and

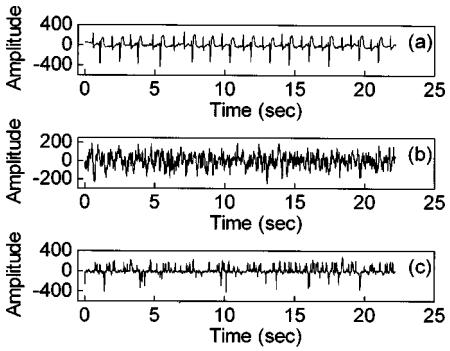

FIG. 5. Portion of signal for a typical PVC subject along with its surrogates. (a) Original time series. (b) Phase randomized surrogate. (c) Gaussian scaled surrogate. Amplitude is in arbitrary units.

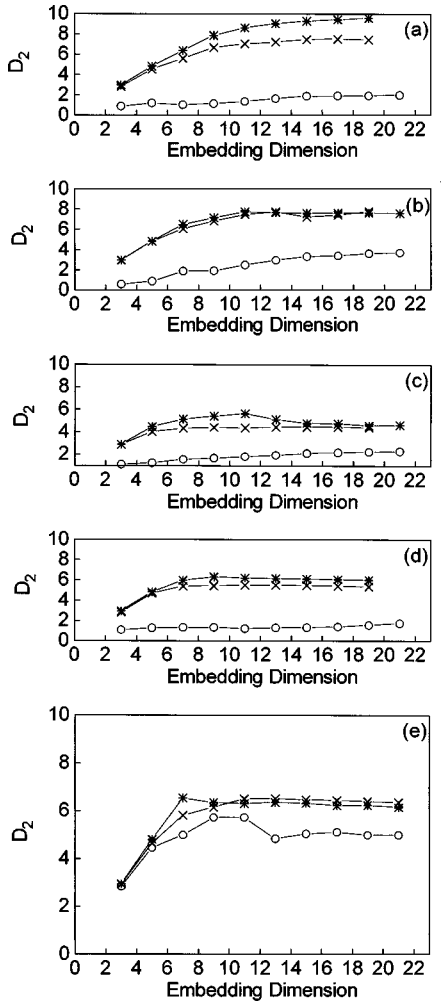

FIG. 7. The comparison of correlation dimension D_2 of different typical subjects (a) Normal. (b) PVC. (c) VTA. (d) AV block. (e) VF with their surrogates as a function of embedding dimension. Random phase, Gaussian scaled and original signal are represented by *, ×, and ○, respectively. Only one typical plot out of 200 surrogates from each type is shown.

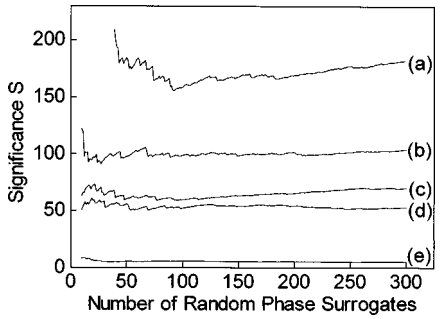

FIG. 8. Variation of significance S with number of random phase surrogates for various typical subjects. (a) PVC. (b) AV block. (c) Normal. (d) VTA. (e) VF.

VF are 65.1 ± 0.2, 181.1 ± 1.8, 52.7 ± 0.1, 100.1 ± 0.3, and 5.2 ± 0.1, respectively, and can be taken to be significant.

In all the cases, the D_{surr} values are greater than D_{orig} giving $P_M\sim0$ and the value of confidence level p less than 0.005 in the nonparametric Barnard–Hope criterion which is defined by $p=1/(N_{\text{surr}}+1)$, where N_{surr} is the number of surrogates. Hence we can confidently reject the hypothesis that the ECG is linearly correlated Gaussian noise.

Since there is a possibility that in some data where the random phase null hypothesis is rejected while the Gaussian scaled null hypothesis is not rejected,[32] it is necessary to examine the Gaussian scaled surrogates.

The ECG time series of normal and pathological condition were further subjected to Gaussian scaled surrogates as in the case of random phase surrogates. The values of S observed for normal, PVC, VTA, AV block and VF are 33.5 ± 0.2, 24.4 ± 0.1, 19.1 ± 0.1, 25.2 ± 0.1, and 5.76 ± 0.1, respectively (Fig. 9), which are once again significant even though there is a decrease in S compared to random phase surrogates. Similar to the random phase surrogates, for all the cases, all D_{surr} values are greater than D_{orig} which leads us to confidently reject the hypothesis that the ECG time series is linearly correlated noise that has been transformed by a static, monotonic nonlinear function. The value of p is less than 0.005 in the nonparametric Barnard–Hope criterion.

The above results obtained from surrogate data analysis clearly indicate the presence of nonlinear structure in the ECG time series of all the normal and pathological conditions considered here.

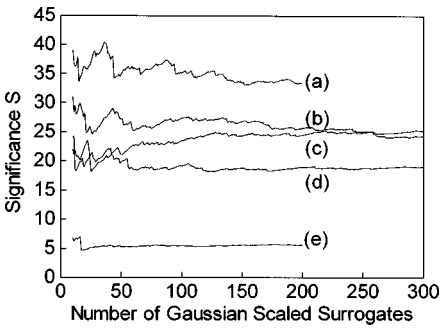

FIG. 9. Variation of significance S with number of gaussian scaled surrogates for various typical subjects. (a) Normal. (b) AV block. (c) PVC. (d) VTA. (e) VF.

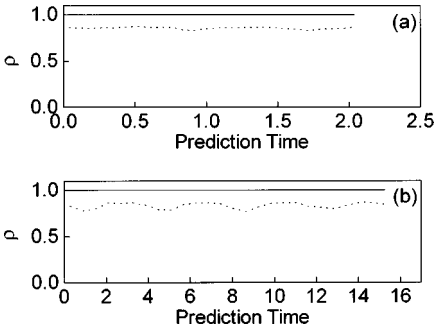

FIG. 10. Variations of correlation coefficient (ρ) with prediction time (T_p) in time units for (a) $\sin(0.5t)$, (b) van der Pol oscillator. ρ for original time series is denoted by solid line and time series with 50% Gaussian noise is denoted by dotted line.

V. PREDICTION ANALYSIS

A characteristic feature of a dynamical system exhibiting deterministic chaos is that it is possible to predict the behavior of the system with some degree of confidence in the short-term even though long-term prediction is impossible. On the other hand, if the variations are nearly random, even the short-term prediction would be impossible. Many prediction methods have been suggested[22,23,36,37] and in this study, we have followed the method proposed by Lefebvre *et al.*[20] which is a modification of the Sugihara and May method.[22] We used the first difference of the ECG signals as the time series to make predictions.

The time series is divided into equal halves of which the first half is used as the library pattern to make predictions about the behavior of the second half. We choose an n-dimensional vector X_t from the second half of the time series for which the prediction has to be made, called the predictee. The $n+1$ nearest vectors are obtained from the library patterns so that the predictee is contained in the smallest simplex formed by the $n+1$ neighbors. The prediction is obtained by following where the points in the simplex end up after p time steps.

To obtain the predicted value, we calculate where the predictee has evolved after p time steps giving weight to original distances from the corresponding neighbors. The loss of predictive power can be measured by linear correlation coefficient ρ between the original time series and the corresponding predicted values. The attractor is constructed in an embedding dimension for which ρ is maximum for a given delay time and prediction time.

First, the above method has been applied as test cases to the time series generated from a function $x_t=\sin(0.5t)$ and the van der Pol oscillator $\ddot{x}-\epsilon(1-x^2)\dot{x}+x=0$, where $\epsilon=2$. Both these cases are limit cycles. The correlation coefficient ρ was found to be constant and very close to unity as the prediction time (T_p) is increased for both these nonchaotic deterministic time series (Fig. 10). This is indeed what we expect for these two cases of limit cycles. Second, to these time series, 50% Gaussian white noise is added and

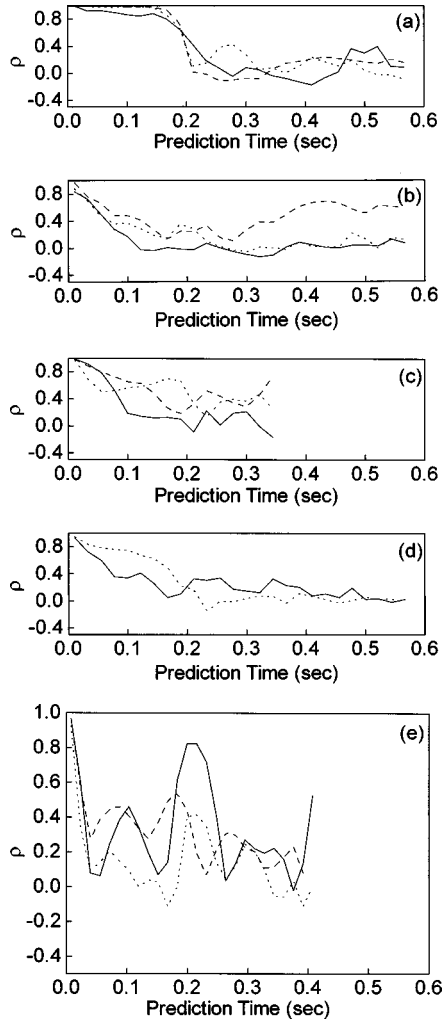

FIG. 11. The variation of correlation coefficient (ρ) with prediction time (T_p) for (a) normal, (b) PVC, (c) VTA, (d) AV block, and (e) VF. In each case three different subjects are shown except for AV block where only two cases are shown.

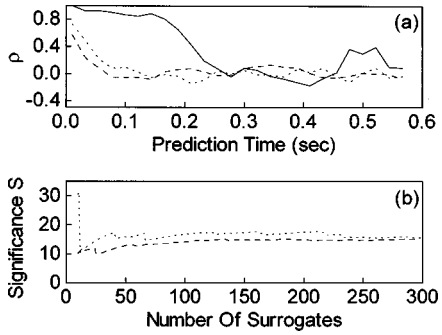

FIG. 12. (a) Decay of correlation coefficient (ρ) versus prediction time (T_p) for a typical normal subject. Original time series, random phase and Gaussian scaled are indicated by solid, broken and dotted lines, respectively. (b) The variation of significance S using T_p^0 as discriminant with number of random phase and Gaussian scaled surrogates represented by broken and dotted lines, respectively.

subjected to the prediction analysis. ρ does not decrease with increasing T_p as would be the case for a chaotic system and remains around 0.8 (Fig. 10) showing that predictions with additive noise seem to have a fixed amount of error, regardless of how far or close, one tries to predict, whereas the predictions with deterministic chaos is expected to deteriorate as T_p is increased. This has been demonstrated by Lefebvre *et al.*[20] and Sugihara and May.[22] Thus it appears that we can distinguish a noisy limit cycle from a chaotic system from the way ρ changes with T_p.

The predictability analysis was done on the ECG time series of normal and pathological conditions. In all cases, we observed a sharp decrease in the ρ with increasing T_p which is a characteristic feature of chaotic systems[20,22] (Fig. 11).

Clearly, there is short-term predictability for the normal subject of the order of 0.2 to 0.3 s within which time ρ decays to near zero. We denote this value of T_p as T_p^0. For PVC and VTA, T_p^0 is around 0.1 to 0.2 s and is 0.17 to 0.22 s in the cases of AV block. For VF, the predictability falls off quickly near 0.05 s and large oscillations are observed as the T_p is increased beyond T_p^0. This may be due to the lower periodicity of the VF time series.

We have also performed predictability test for the surrogate data sets (both random phase and Gaussian scaled) of the normal and pathological conditions using T_p^0 as the discriminant. For all the 300 surrogates, the correlation coefficient ρ goes to near zero within 0.05 s itself. The number of surrogates was increased until the saturation for the significance S is observed (see Fig. 12). For a normal subject, the value of S is typically 15 ± 0.5 and it is significant.

Figure 12(a) compares the distinction in the fall of ρ versus T_p for a typical normal subject with its surrogate and also variation of S with the number of random phase and Gaussian scaled surrogates [Fig. 12(b)]. These results on the short-term predictability of the various ECG signals enable us to reject the null hypothesis addressed by both the type of surrogates.

The sharp fall of ρ with T_p in the case of all ECG time series strongly indicates that it is not a noisy limit cycle.

VI. UNSTABLE PERIODIC ORBITS (UPO) ANALYSIS

Another indication for deterministic chaos is the possibility of describing the attractor in terms of a limited number of unstable periodic orbits UPOs.[38] Our analysis (see Refs. 39 and 40 for details) of UPOs, has shown that a normal healthy cardiac attractor is characterized generally by three or four UPOs. A typical UPO distribution for a normal subject and a pathological PVC case is shown in Fig. 13. For the cases displayed in this figure, there are three dominant UPOs of periodicity of 0.99, 1.98 and 2.97 s for normal and five for PVC with the periodicity of around 0.83, 1.74, 2.6, 4.28 and 5.1 s.

The positive Lyapunov exponents for these three UPOs of normal are 3.9, 1.7 and 1.2 bits s^{-1} and for five UPOs of PVC are 3.4, 1.8, 1.2, 0.8 and 0.6 bits s^{-1}. This is another indication of deterministic chaos in the ECG. Details of the UPO analysis of the human cardiac system for normal and several pathological subjects showing that the UPOs offer a signature of the cardiac condition will be published elsewhere.[40]

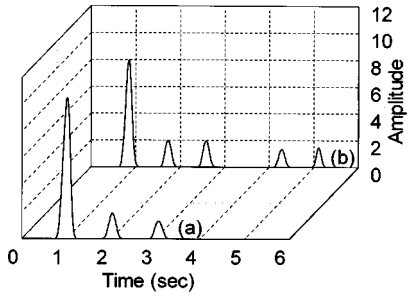

FIG. 13. UPOs of typical (a) normal healthy subject and (b) PVC. Area under the peaks are {77,13,10} and {54,14,14,9,9} for normal and PVC with increasing values of recurrence time. Amplitude is in arbitrary units.

VII. CONCLUSION

In this article, several normal and pathological ECG signals have been subjected to a variety of tests designed to detect nonlinear dynamics in the cardiac system. The usual tests such as correlation dimension D_2, largest Lyapunov exponent $\lambda_{\max}$, combined with extensive predictability and surrogate analysis using correlation dimension D_2 and prediction time T_p^0 as discriminants strongly indicate that the dynamics underlying the cardiac signals is nonlinear. Further, the sharp fall in the correlation coefficient ρ with increasing prediction time T_p and UPO analysis indicate the possibility of deterministic chaos. These results do not of course constitute a definite proof of chaos in human cardiac dynamics but only show that they are consistent with such a process. Further, it may be emphasized that we do not imply that the dynamics of the ECG signal is governed by the cardiac system alone, but could as well be the result of coupling with the control mechanisms of the body. In this study we have been concerned with the nature of the cardiac output signal and not with its physiological origin.

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