Research paper

Quasiperiodicity route to chaos in cardiac conduction model

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A R T I C L E   I N F O

A B S T R A C T

It has been suggested that cardiac arrhythmias are instances of chaos. In particular that the ventricular fibrillation is a form of spatio-temporal chaos that arises from normal rhythm through a quasi-periodicity or Ruelle-Takens-Newhouse route to chaos. In this work, we modify the heterogeneous oscillator model of cardiac conduction system proposed in Ref. [Ryzhii E, Ryzhii M. A heterogeneous coupled oscillator model for simulation of ECG signals. Comput Meth Prog Bio 2014;117(1):40–49. doi:10.1016/j.cmpb.2014.04.009.], by including an ectopic pacemaker that stimulates the ventricular muscle to model arrhythmias. With this modification, the transition from normal rhythm to ventricular fibrillation is controlled by a single parameter. We show that this transition follows the so-called torus of quasi-periodic route to chaos, as verified by using numerical tools such as power spectrum and largest Lyapunov exponent.

1. Introduction

Experimental and numerical studies have shown that chaos is a phenomenon that appear in a wide variety of chemical, physical and biological systems [2,3]. The universality of chaotic dynamics has renewed the application of nonlinear analysis, particularly in biology where the chaos theory has been used to explain phenomena such as population dynamics [4], epidemiology of some infectious diseases [5], metabolism and intra-cellular rhythms [6] and cardiac rhythms [7].

Concerning cardiac rhythms, the study of its abnormalities, usually called arrhythmias [8], is a major concern. Premature beats, bradycardia, tachycardia, atrial and ventricular fibrillation (VF) are the most common arrhythmias. Particularly, the VF is of the greatest interest because it is the most severe cardiac rhythm disturbance characterized by uncoordinated and ineffective contraction of ventricular muscles. The VF without well-timed treatment leads to sudden cardiac death in few minutes [9]. Despite its importance, yet it is not clear how VF occurs. Since during VF the electrical activity becomes highly disordered VF has been traditionally treated as random or irregular process and it is now accepted that general features of arrhythmias can only be fully understood in the context of nonlinear and stochastic dynamics (for a recent review see Ref. [10]). Still in this context however, there are different approaches without strong evidence.

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The arrhythmias are manifested in the variability of features of the electrocardiogram (ECG) signal, which is a record of the potential differences between pairs of electrodes located at predefined points on the chest surface. One of the existing approaches based on real ECG studies considers arrhythmias as instances of chaos [11–14] or at least resulting from a nonlinear deterministic dynamics amenable to chaotic control techniques [15–17]. In Ref. [18] three stationary forms of fibrillation were studied, namely, human chronic atrial fibrillation, a stabilized form of canine VF, and fibrillation like activity in thin sheets of canine and human ventricular tissue in vitro. In all these studies, evidences indicated that fibrillation is a form of spatio-temporal chaos. Even more, it has been suggested that the route to chaos the heart follows from a normal rhythm to VF is a turbulent process that results from the so-called torus bifurcation, quasi-periodicity or Ruelle-Takens-Newhouse scenario [19]. This route is basically the birth and evolution of a torus in the phase space and goes through three steps before chaos appears: a Hopf bifurcation or oscillating behavior (which is in fact the natural state of the heart), with frequency w_1; a second Hopf bifurcation but now with a frequency w_2, such that w_1/w_2 is irrational, that is the motion is quasiperiodic, the trajectory in the phase space never closes and fills a torus; finally small perturbations of this torus lead to chaos [19,20].

From a biological point of view, it is believed that there are two possible mechanisms responsible for the appearance (genesis) and maintenance of VF: multiple self-sustained electrical wavelets in myocardium (multiple wavelets VF) and mother rotor driven by a single dominant source of reentrant electrical excitation (mother rotor VF) [21]. These reentrant electrical sources produce rapidly expanded activation wave fronts, which either cause block or allow conduction propagation in the surrounding tissue due to heterogeneity in refractory periods [22]. As a result, multiple small wave breaks and irregular activation patterns are generated [23]. From a clinical point of view, an important challenge is to design experiments to understand how reentrant arrhythmias are initiated, in order to prevent their formation, and how to stop them once formed. The verification of the mechanisms of VF origin may suggest new therapeutic strategies. In this sense, mathematical or computer modeling of cardiac electrophysiology is recognized as a useful tool to understand real processes taking place in healthy and malfunctioning hearts. Given the lack of strong clinical and experimental evidences in favor of one of the proposed mechanisms, computer simulations of VF have been also the subject of many research efforts (see Section. 2). In this work we use the model developed by E. and M. Ryzhii [1] which reproduces clinically comparable ECG waveforms. Assuming that VF is deterministic chaos [24], we extend the model by the inclusion of an ectopic pacemaker (EP) that stimulates ventricular muscles, as it is known that such type of reentrant arrhythmias can be initiated intentionally by a proper application of point stimuli [8]. With this addition, chaotic responses associated with VF can be produced by the variation of a single control parameter. Standard numerical tests are applied to show that with this new model, the transition from regular to chaotic oscillations follows the torus or quasi-periodic route to chaos. This result constitutes evidence in favour of the chaotic approach.

The outline of the paper is as follows. In Section. 2, the main computational and mathematical models to generate ECG signals are discussed. In Section. 3 the model [1] is presented in some detail since in this work we develop an extension of this model to produce VF. This extension is presented in Section. 4 and the numerical results in Section. 5. Finally, in Section. 6 we draw conclusions on this work.

2. Nonlinear cardiac models

Many complex cardiac models describe heart function on the cellular level including a variety of ionic currents (see, for example, Ref. [25] and references therein). Usually, the models are accomplished by a torso model and can reproduce realistic ECG signals. This approach requires a lot of computational resources to solve huge number of differential equations with many parameters. In this paper we intentionally omitted the consideration of the models of such type. There is another simplified class of models, which describe heartbeat dynamics in general with a set of ordinary differential equations, and some of them are capable to simulate ECG. McSharry et al. [26] proposed a dynamical model based on a quasi-periodic motion of the z trajectory around a limit cycle, which consists of three coupled ordinary differential equations. The variable z is a sum of Gaussians functions with different amplitudes and widths given in such a way that the distinct points on the ECG (P, Q, R, S and T waves) are described by events corresponding to the Gaussians functions. Dabanloo et al. [27] developed a model based on a modification of the Zeeman model [28] to produce the RR-tachogram. In this model, a simple neural network is used to generate a ECG cycle and the effects of sympathetic and parasympathetic nervous system activities are included in order to generate the low and high frequency peaks in the power spectrum. Kaplan et al. [29] used coupled oscillators to generate ECG signals; each of these oscillators is known as the Wien Bridge, and is basically used as half- and full-wave rectifier. The Wien Bridge is an oscillator derived from the Van de Pol (VDP) oscillator and the authors shown that two coupled Wien Bridges are capable to generate ECG signals. Gois and Savi [30] reproduced the electrical activity of the heart by means of a mathematical model consisting of three modified VDP oscillators connected with time delay couplings. They shown that some pathological rhythms can be produced by changing some system parameters or establishing different coupling situations and external pacemaker excitations. Sadayi et al. [31] proposed an extension of the model reported in Ref. [26]; they shown that synthetic ECG signals can be generated by using separate characteristic waves such as the atrial and ventricular complexes, and they also reproduced some arrhythmias. The model in Ref. [26] was transformed into a discrete dynamical system and coupled with the circle and the standard maps by Gidea et al. [32]; the resulting discrete model exhibits intrinsic non-linearity, which attempts to mimic the self-regulatory function of the heart. Das and Maharatna [33] developed an incommensurate fractional order model, based on two VDP oscillators coupled with time delays and gains.