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A NEW THEORETICAL MODEL FOR ANALYZING ELECTROCARDIOGRAPHY SIGNALS THROUGH A RICCATI GAUGE

BY

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Abstract. We develop a non-linear theoretical model for analyzing Electrocardiography (ECG) signals. For this purpose, a non-linear Lagrangean with a Riccati-type gauge for studying signals corresponding to transitions from normal heart functioning to arrhythmias has been used. Our results show that a specific attractor in the states space can be associated to this transition. In such context, the patterns dynamics (attractors) can be employed for the early detection of arrhythmias in pregnant women, offering the possibility of rapid and specific management of the mother and fetus.

Keywords: Non-linear Lagrangean; Riccati-type gauge; attractor; heart functioning; cardiac arrhythmia.

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1. Introduction

The heart, its functions and properties have long been studied by physicians, biologists, chemists and physicists. In the following we want to remind the main physiological properties of the heart (Iaizzo, 2015):

i) excitability – the property to fully respond to stimuli that reach or exceed a threshold value. The heart is excitable only in the relaxation phase (diastole), while in the systole phase it can be found in an absolute refractory state and does not respond to stimuli;

ii) automatism – the property of the nodal tissue to rhythmically self-excite. The normal heart rate, usually 70-80 beats/minute, is determined by the sinoatrial node and can be modified by external stimuli;

iii) conductivity – the property to propagate the excitation along all the heart's fibers. The nodal tissue generates and conducts impulses, and the myocardial tissue responds to contractions;

iv) contractility – the property to respond to stimuli through changes in dimension and tension.

The cardiac cycle is accompanied by acoustic, mechanic, and electric manifestations, their analysis allowing the evaluation of the (normal) functioning of the heart (Pandele, 2007):

i) acoustic manifestations – the exterior signs of heart activity are represented by cardiac noise, which can be directly heard with a stethoscope, or graphically registered on a phonocardiogram. Heart noise is caused by changes in blood flow velocity and by consecutive vibrations of atrioventricular and sigmoid valves. Normally, two distinct main cardiac noises can be distinguished: the systolic noise and the diastolic noise;

ii) mechanic manifestations – each ventricular contraction is followed by blood expulsion into the aorta, generating a pressure wave which propagates along the aorta and its branches, called pulse. The graphical representation is called a sphygmogram. The pulse frequency is the same as the heart frequency, this being an accessible parameter for heart activity;

iii) electric manifestations – in the myocardial tissue, the excitation is initiated in a point and rapidly propagates from one cell to another. The boundary between the activated portion (electronegative) and the non-activated one (still electropositive) is a spatial surface. This moves along the direction of the excitation wave. On either side of this surface an electric field exists, giving rise to an electric dipole.

In the following we want to present examples of abnormal health functioning cases.

Cardiac arrhythmia represents the alteration of heart beats and/or the ratio between the atrial and ventricular rhythms (Walraven, 2010). There are multiple causes for arrhythmia: ischemia, hypoxia, acidosis, ionic imbalance (hypopotassemia), catecholamines excess, mechanical factors (dilation or

hypertrophy), various drugs etc. Arrhythmia is a pathological situation in which the heart is not in a normal sinus rhythm, determined by the sinus node automatism. In this way, impulses generation/formation and their conduction/propagation can be altered.

Atrial fibrillation (AFIB) represents an abnormal heart rhythm, characterized by rapid and irregular beats. Paroxysmal AFIB is a form of tachycardia, manifesting in the case in which the ventricular electric activity is disorganized and very rapid. The ventricles have very rapid contractions, which makes them inefficient (Camm and Yap, 2014). In this way, a “pulse deficit” can occur, a discrepancy between the central and peripheral pulses, the later being lower. In Fig. 1 an electrocardiogram (ECG) corresponding to an atrial fibrillation is presented.



Fig. 1 – Electrocardiogram corresponding to an atrial fibrillation.

The atrial flutter (AFL) is an abnormal heart rhythm, which begins in the ventricular chambers (Sawhney *et al.*, 2009). When it first appears, it is often associated with a high cardiac frequency (over 100 beats/minute), being classified as a type of supraventricular tachycardia. There are two types of AFL: type I and type II. Type I AFL has a rate of 240-340 beats/minute. Type II AFL, more rarely encountered, is usually between 340-440 beats/minute. In Fig. 2 we present an ECG corresponding to an atrial flutter.

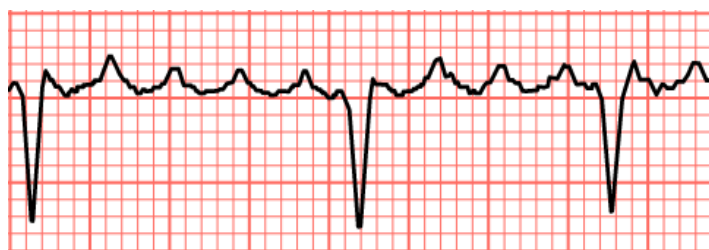


Fig. 2 – Electrocardiogram corresponding to an atrial flutter.

Ventricular fibrillation (VF) is caused by the presence of multiple entry circuits in the ventricular muscle, and the ventricular electrical activity is completely chaotic (Rajskina, 1999). The ventricular mechanical activity is also unorganized, ventricular contractions are inefficient from a hemodynamic point of view, leading to cardiac arrest. VF is the most common cause of sudden death. In Fig. 3 an ECG corresponding to a ventricular fibrillation is shown.



Fig. 3 – Electrocardiogram corresponding to a ventricular fibrillation.

Since the fundamental discoveries made by Einthoven, electrocardiography has been widely employed to visualize, record, and analyze the changes in the electric potential that accompany the heart's activity. An ECG can provide information about heart rate, impulse origin and its propagation, heart position, heart attacks, electrolytes concentration influence on heart properties, drug effects on the heart, artificial pacemakers functioning etc. (Camm *et al.*, 2009).

2. Methods

The analyzed electrocardiograms were obtained from the PhysioNet database (<https://archive.physionet.org/physiobank/>). The PhysioBank is a free collection of physiological signals, coming from various patients. Dedicated software for viewing and analyzing them EEGs is also available. This data bank was created by the National Institute of General Medical Science (NIGMS) and the National Institute of Biomedical Imaging and Bioengineering (NIBIB). The free access obeys the rules of the ODC Public Domain Dedication and License v1.0. PhysioNet is widely used to stimulate biomedical and physiological signals current research.

It is known that, in the one-dimensional case, the propagation equation of the electrical component of the biological field can be obtained using the non-linear Lagrangean, in \dot{Q} and Q coordinates:

$$L(\dot{Q}, Q, t) = \frac{1}{2} A \exp\left(2\frac{B}{A}t\right) \left(\dot{Q} + \frac{w}{A}Q\right)^2 \quad (1)$$

This Lagrangean satisfies the following differential equation (of a Riccati-type):

$$A\dot{w} - w^2 + 2Bw + AC = 0 \quad (2)$$

Let us note that there is a connection between the Riccati-type Eq. (2) and the Hamiltonian dynamics. We find in this situation that the matrix system has the expression:

$$\begin{pmatrix} \dot{\eta} \\ \dot{\xi} \end{pmatrix} = \begin{pmatrix} -\frac{B}{A} & \frac{C}{A} \\ -1 & \frac{B}{A} \end{pmatrix} \begin{pmatrix} \eta \\ \xi \end{pmatrix} \quad (3)$$

with $w = \frac{\eta}{\xi}$, which rigorously represents a Hamiltonian system. Therefore, variables η and ξ must be identified with phase space coordinates.

$$P(w) = w^2 - 2Bw - AC \quad (4)$$

can be written as

$$w_1 \equiv B + iA\Omega, \quad w_2 \equiv B - iA\Omega, \quad \omega^2 = \frac{C}{A} - \left(\frac{B}{A}\right)^2 \quad (5)$$

Through the homographic transformation

$$z = \frac{w - w_1}{w - w_2} \quad (6)$$

it results that z is a solution of the linear and homogenous first order equation

$$\dot{z} = 2i\omega z \quad (7)$$

which allows the solution

$$z(t) = z(0)e^{2i\omega t} \quad (8)$$

Therefore, if the initial condition $z(0)$ is conveniently expressed, the general solution of Eq. (2) can be found, by writing transformation (6) as:

$$w = \frac{w_1 + re^{2i\omega(t-t_r)}w_2}{1 + re^{2i\omega(t-t_r)}} \quad (9)$$

where r and t_r are two real constants which characterize the solution. By using relations (5) we can write this solution with real terms, as

$$z = B + A\omega \left(\frac{\frac{2r \sin[2\omega(t-t_r)]}{1+r^2+2r \cos[2\omega(t-t_r)]} + i \frac{1-r^2}{1+r^2+2r \cos[2\omega(t-t_r)]}}{\quad} \right) \quad (10)$$

which highlights a self-modulation of the pulsation-type characteristic ω known as the Stoler transformation (Stoler, 1970, 1971), implying a complex form for this parameter. In Fig. 4 we present this self-modulation phenomenon through $\text{Re}z$ time dependences, for various values of r and ω . The dependences of $\text{Re}z$ on r and ω (3D and contour dependences) at various scale resolutions are shown in Fig. 5.

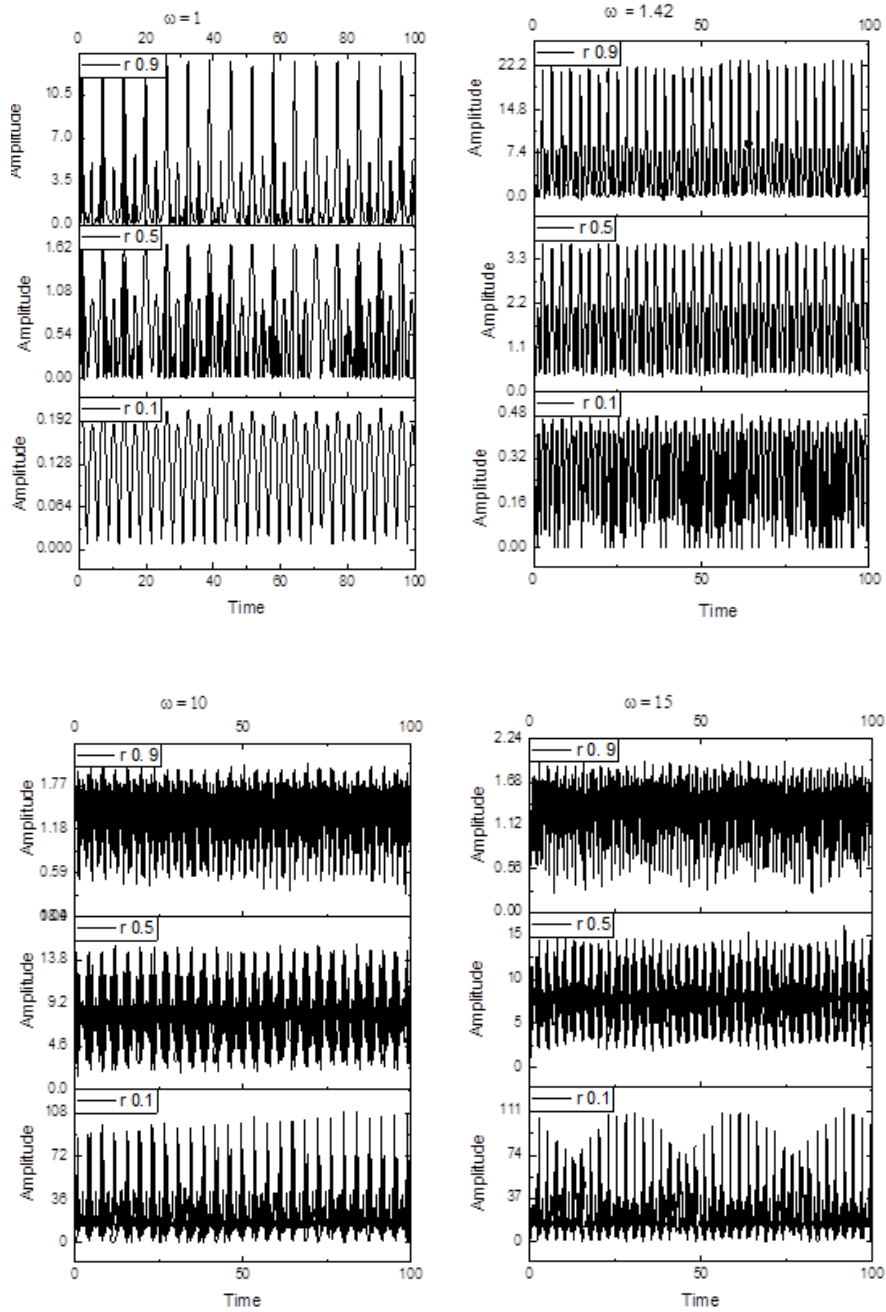


Fig. 4 – Amplitude variation with time of the Rez solution for four different values of the (pulsation-type) characteristic (1, 1.42, 10, and 15).

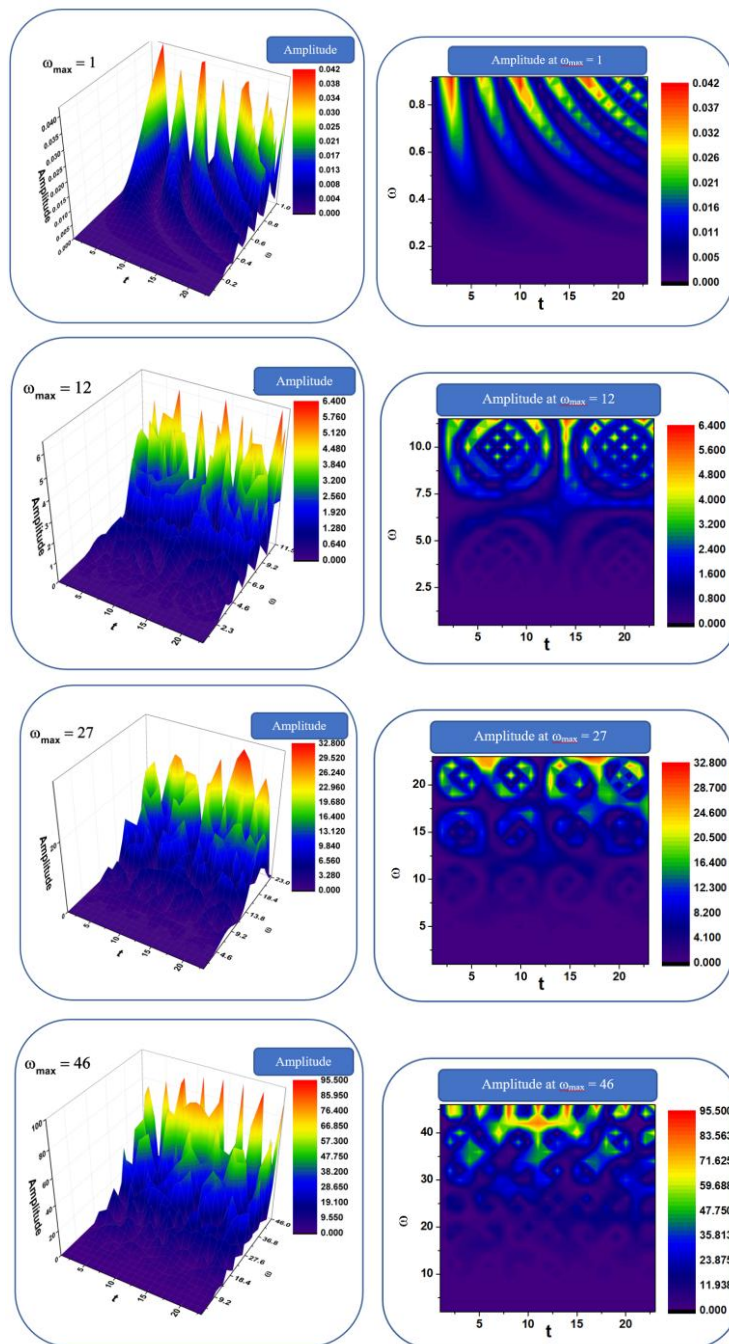
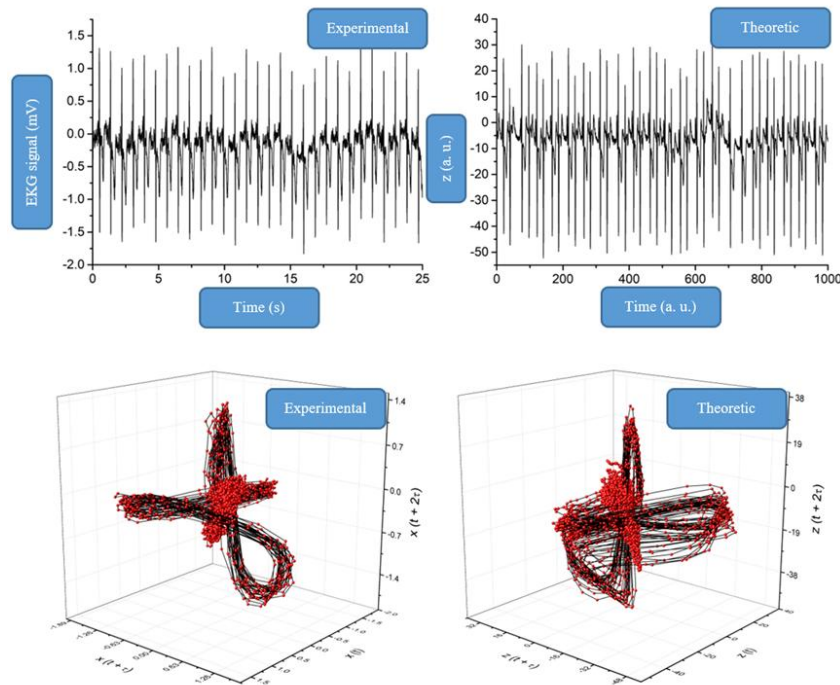


Fig. 5 – 3D and 2D representation of solution Rez at various scale resolutions given by the maximal value of the (pulsation-type) characteristic (1, 12, 27 and 46). Self-modulation of the signal can be observed.

3. Results and Discussions

From the above-presented facts we can see that the temporal stochasticization aiming to “transform” a system’s dissipative dynamics into non-dissipative dynamics implies a Riccati-type “gauge” (the dynamic variables must satisfy a Riccati-type differential equation). The explication of such a situation can be found in a Stoler-type self-modulation (described by a Stoler transformation) of any signal. It can be thus observed that the temporal stochasticization processes of any biostructure entities dynamics through a Riccati-type gauge impose Stoler-type self-modulations of the signals that describe their dynamics.

Can there be correspondences in such a context between our theoretical model and experimental data (in vivo or in vitro)? In order to answer this question, let us compare ECG signals registered through electrocardiography (and their associated attractors from the states space), corresponding to heart transitions from normal activity to arrhythmias (fibrillations), and the signals (and their associated attractors) obtained by means of our theoretical model – see Fig. 6.



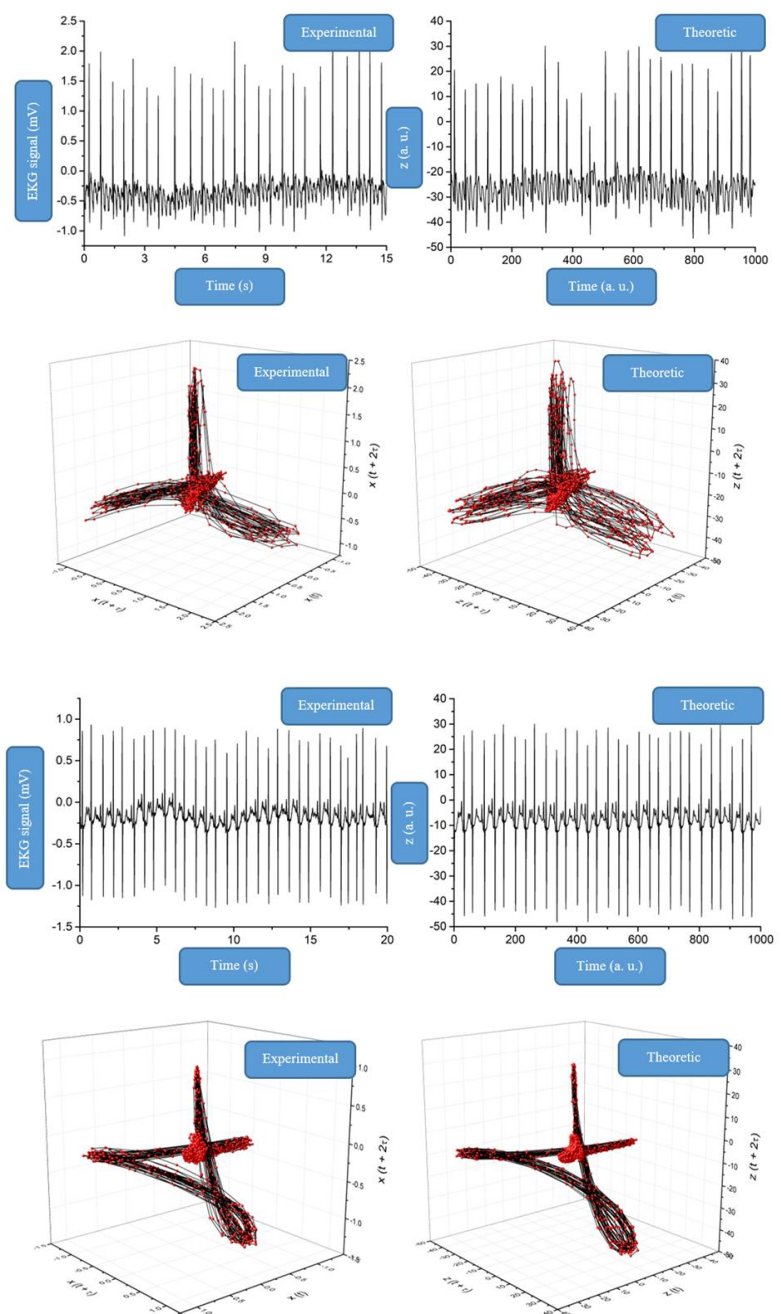


Fig. 6 – ECG signals and their reconstructed attractors from the states space comparison: recorded signals (left) and theoretical model (right). The attractors from such spaces were obtained through a time delay method.

Let us note that the interaction process with the biological medium mimics a characteristic modulation process. More precisely, this process is in fact a “calibration” of the difference between the kinetic and potential energies of a biostructure, resulting in a perfect square. The physical meaning of the perfect square Lagrangean is that it can describe the behavior of a fundamental biostructure inside a more complex biostructure (*eg.* the coupling of a cell to a tissue, the coupling of a tissue to an organ, the coupling of an organ to an organism etc.).

4. Conclusions

In this paper a non-linear model for analyzing ECG signals has been developed. To this end, we employed a non-linear Lagrangean with a Riccati-type gauge for analyzing signals corresponding to transitions from normal hearth functioning to arrhythmias (fibrillations). In this context we show that a specific attractor in the states space can be associated to this transition. Therefore, in our opinion, any organ dysfunctionality can be characterized through a specific attractor.

Cardiac arrhythmias represent very spread cardiac complications that may occur during the pregnancy. Sometimes the pregnancy due to its physiological changes during the pregnancy may complicate a pre-existing arrhythmia whilst in other situations could trigger off the condition. The promptitude of the maternal arrhythmias diagnostic using our non-linear model is feasible for patients with history of arrhythmia in order to prevent hemodynamic compromise or maternal and fetal risk. Our model could also possibly be applied for the detection of fetal arrhythmias, improving the fetal survival rate by increasing the fetal cardiac time intervals captured by echocardiographic techniques.

Non-linearity and fractality can be used for describing different physical phenomena (Agop *et al.*, 2008; Colotin *et al.*, 2009), thus paving the way for developing new theoretical models for biophysical processes.

Authors contributions: All the authors have contributed equally to this paper.

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UN NOU MODEL TEORETIC PENTRU ANALIZA
SEMNALELOR ELECTROCARDIOGRAFIILOR FOLOSIND UN
ETALON RICCATI

(Rezumat)

În această lucrare am dezvoltat un model teoretic neliniar pentru analiza semnalelor ECG. În acest scop am utilizat un Lagrangean neliniar cu un etalon Riccati pentru a studia semnalele ce corespund tranzițiilor de la o funcționare normală a inimii la aritmii. Rezultatele noastre au arătat că acestei tranziții i se poate asocia un atractor specific din spațiul stărilor.

