NONLINEAR DYNAMICS OF HEART RHYTHMS

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Abstract. *Rhythmic phenomena represent one of the most striking manifestations of dynamic behavior in biological systems. Understanding the mechanisms responsible for biological rhythms is crucial for the comprehension of the dynamics of life. Natural rhythms could be either periodic or irregular over time and space. Each kind of dynamical behavior related to biomedical systems may be related with both normal and pathological physiological functioning. The cardiac conducting system can be treated as a network of self-excitatory elements. Since these elements exhibit oscillatory behavior, they can be modeled as nonlinear oscillators. This article proposes a mathematical model to describe heart rhythms. Three modified Van der Pol oscillators are connected considering time delay coupling in order to reproduce electrocardiogram (ECG) signals. Numerical simulations are carried out reproducing normal and pathological rhythms represented by ECG signals.*

Keywords*: Chaos, heart, natural rhythms, nonlinear dynamics.*

1. INTRODUCTION

Natural rhythms could be either periodic or irregular over time and space. Each kind of dynamical behavior related to biomedical systems may be related to both normal and pathological physiological functioning. Extremely regular dynamics may be associated with diseases including periodic breathing, certain abnormally heart rhythms, cyclical blood diseases, epilepsy, neurological tics and tremors. On the other hand, there are phenomena where regular dynamics reflect healthy behavior as sleep-wake cycle and menstrual rhythms. Moreover, irregular rhythms can also reflect disease: cardiac arrhythmias, such as fibrillation, and different neurological disorders (Savi, 2006, 2005; Glass, 2001; Ferriere & Fox, 1995).

Rhythmic changes of blood pressure, heart rate and other cardiovascular measures indicate the importance of dynamical aspects in the comprehension of cardiovascular rhythms. Several studies are pointing to the fact that certain cardiac arrhythmias are instances of chaos (Witkowski *et al.*, 1995; Radhakrishna *et al.*, 2000). This is important because it may suggest different therapeutic strategies, changing classical approaches. Among cardiac arrhythmia, one can cite premature beats, atrial fibrillation, bradycardia, tachycardia and ventricular arrhythmias, as fibrillations and tachycardia, which are the most severe and life-threatening arrhythmias being the cause of many deaths.

There are different forms to evaluate the heart functioning by the measurement of some signal. An electrocardiogram (ECG) records the electrical activity of the heart being used to measure the rate and regularity of heartbeats as well as the size and position of the chambers. The electrical impulses related to heart functioning are recorded in the form of waves, which represents the electrical current in different areas of heart.

The clinical arrhythmias that have the greatest potential for therapeutic applications of chaos theory are the aperiodic tachyarrhythmias, including atrial and ventricular fibrillation. Garfinkel *et al.* (1992) and Garfinkel *et al.* (1995) discuss the application of chaos control techniques in order to avoid heart arrhythmic responses. This approach may be incorporated into pacemakers, avoiding ventricular fibrillation, for example. Since chaotic responses may be controlled by an efficient way using OGY method or its variants, this may inspire some interesting approaches in order to stabilize unstable orbits associated with the normal heart rhythm.

This article proposes a mathematical model to describe heart rhythms. Since the qualitative features of the heart actuation potential is close related to the dynamical response of the classical Van der Pol (VdP) oscillator, this oscillator may be considered as the starting point of this modeling. Actually, the classical article due to Van der Pol & Van der Mark (1926) describes the heart behavior from coupled VdP oscillators. Afterwards, Hodkin & Huxley (1952) describes the heartbeat behavior by considering different models. Recently, Grudzinski & Zebrowski (2004) proposes a variation of the classical Van der Pol oscillator in order to capture some important aspects regarding the heart action potential. Santos *et al.* (2004) use coupled oscillators in order to describe general aspects of heartbeat rhythms discussing different coupling terms. Campbell & Wang (1998) presents a discussion dealing with some coupling characteristics of these oscillators highlighting the importance to consider time delay coupling. Here, three modified VdP oscillators are connected considering time delay coupling in order to reproduce ECG signals. Normal and pathological rhythms are reproduced altering coupling terms.

2. HEART AND ITS ELECTRICAL ACTIVITY

The heart walls are composed of cardiac muscle, called myocardium, and also striations that is similar to skeletal muscle. Basically, the heart consists of four compartments: the right and left atria (upper part) and the right and left ventricles (lower part) (Figure 1). The blood returns from the systemic circulation to the right atrium and from there goes to the right ventricle. It is ejected from the right ventricle to the lungs. Oxygenated blood returns from the lungs to the left atrium, and from there to the left ventricle. Finally blood is pumped through the aortic valve to the aorta and to the systemic circulation.

The heart muscle cell (myocyte) has an electric activation that takes place by means of the inflow of sodium ions across the cell membrane. A plateau phase follows cardiac depolarization, and thereafter repolarization takes place, which is a consequence of the outflow of potassium ions. The mechanical contraction of the heart is associated with the electric activation of cardiac muscle cell.

An important distinction between cardiac muscle tissue and skeletal muscle is that the activation of the cardiac muscle can propagate from one cell to another in any direction. Therefore, the activation wave-fronts are of rather complex shape. The only exception is the boundary between the atria and ventricles, where the activation wave usually cannot cross except along a special conduction system, since there is a nonconducting barrier of fibrous tissue.

All heart rhythms are governed by activation potentials controlled by specialized cells. The sinus node (sinoatrial or SA node) is located in the right atrium at the superior vena cava (Figure 1). The SA nodal cells are self-excitatory, pacemaker cells, which generate an action potential at the rate of about 70 pulses per minute. Activation propagates from the SA node throughout the atria, but cannot propagate directly across the boundary between atria and ventricles. The atrioventricular node (AV node) is located at the boundary between the atria and ventricles (Figure 1). It has an intrinsic frequency about 50 pulses/min, however, this node follows higher frequencies when it is triggered. In a normal heart, the AV node provides the only conducting path from atria to ventricles. Therefore, under normal conditions, ventricles can be excited only by pulses that propagate through it. Propagation from the AV node to the ventricles is provided by a specialized conduction system. Proximally, this system is composed of a common bundle, called the bundle of His. More distally, it separates into two bundle branches propagating along each side of the septum, constituting the right and left bundle branches. Even more distally the bundles ramify into Purkinje fibers that diverge to the inner sides of the ventricular walls.

Because the intrinsic rate of the SA node is the highest, it sets the activation frequency of the whole heart. If the connection from the atria to the AV node fails, the AV node adopts its intrinsic frequency. If the conduction system fails at the bundle of His, the ventricles will beat at the rate determined by their own region that has the highest intrinsic frequency.

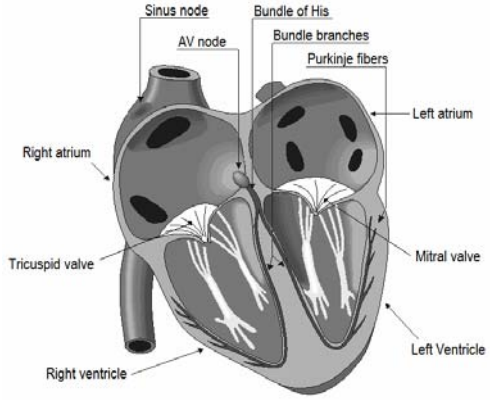

Figure 1 – Schematic picture of the heart.

2.1. Activation Potential

The heart electric activity is closely related to cellular rhythms known as Na-K pump. The concentration of sodium ions (Na+) is about 10 times higher outside the cellular membrane than inside, whereas the concentration of the potassium (K+) ions is about 30 times higher inside as compared to outside. When the membrane is stimulated the transmembrane potential rises about 20mV and reaches the threshold – that is, when the membrane voltage changes from −70 mV to about −50 mV – the sodium and potassium ionic permeability of the membrane change. Figure 2 presents a schematic picture of the ionic flow that generates the activation potential. The sodium ion permeability increases very rapidly at first, allowing sodium ions to flow from outside to inside, increasing the inside positive potential, reaching about +20 mV. After that, there is a slowly increasing of potassium ion permeability allowing potassium ions to flow from inside to outside, causing the return of the intracellular potential to its resting value. The

maximum excursion of the membrane voltage during activation is about 100mV; the duration of the impulse is around 300ms. While at rest, following activation, the Na-K pump restores the ion concentrations inside and outside the membrane to their original values.

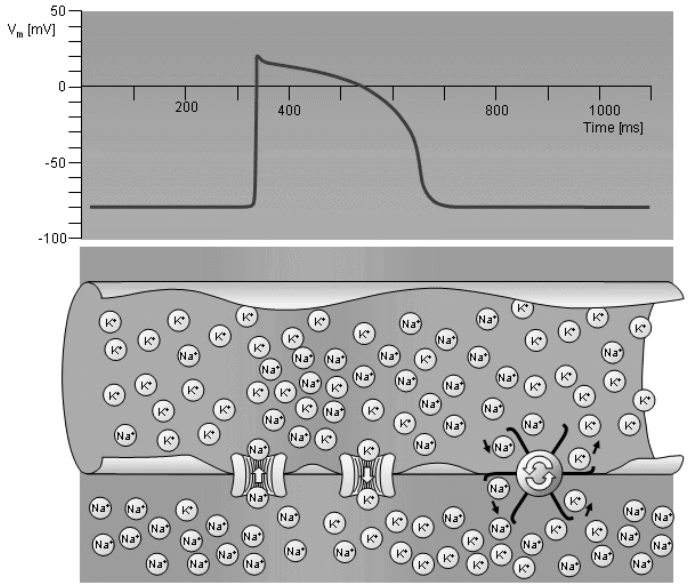

Figure 2 – The Na-K pump and the activation potential (Malmivuo & Plonsey, 1995).

Once activation has been initiated, the membrane is insensitive to new stimuli, no matter how large the magnitude. This phase is called the absolute refractory period. The activation process encompasses certain characteristics such as currents, potentials, conductivities, concentrations and ion flows. The term action impulse describes the whole process. The bioelectric measurements focus on the electric potential difference across the membrane. Therefore, the electric measurement of the action impulse is called the action potential that describes the behavior of the membrane potential during the activation.

Different groups of cells present different configurations of ionic channels, which lead to different action impulse velocities. Therefore, cells are classified as *slow* or *fast* response action potential cells, which present different shapes for the action potential signal, represented in Figure 3. The first group (slow response) includes the atrial and ventricular muscle, His bundle and Purkinje fibers while the second (fast response) are the SA and AV nodes.

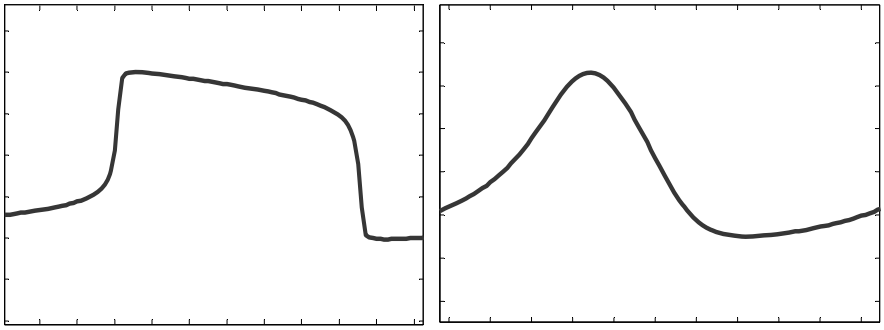

Figure 3 – Wave shape action potential response: slow response (left side) and fast response (right side).

The cellular rhythms produces electric activity that is related to depolarization and repolarization waves. The sodium influx is triggered by the very large and rapid rise in sodium permeability. This current is confined to a zone called the depolarization zone. The current outflow is the "local circuit" current that initially depolarizes the resting tissue, and which is advancing in the direction of propagation. When a cell depolarizes, it produces an electric field which triggers the depolarization phenomenon in another cell close to it, which then depolarizes. Therefore, the depolarization may be understood as a propagating wave within cardiac tissue. The nature of the repolarization wave is different from that of the depolarization wave. Unlike depolarization, the repolarization is not a propagating phenomenon, however, by

examining the location of repolarizing cells at consecutive time instances, it is possible to approximate the repolarization with a proceeding wave phenomenon.

3. ELECTROCARDIOGRAM (ECG)

Cardiac electric signals on an intracellular level may be recorded with a microelectrode, which is inserted inside a cardiac muscle cell. On the other hand, it is possible to record the electric potential generated by the heart electrical activity on the surface of the thorax. The electrocardiogram (ECG) is a measure of the extra-cellular electric behavior of the cardiac muscle tissue.

The propagation wave-front of the cardiac electrical signal through the body presents a very complicated shape due to different conductivity values of the tissues composing the human body and their complex spatial distribution. Besides, as the depolarization and repolarization phenomena propagate through heart conducting system, the electric field has a periodic response.

Many cardiac signal measurement systems are already proposed, where leads are used to measure potential difference between points on the body surface. Actually, the measured signal is the projection of the dipole along the line defined by the leads. Therefore, depending on the leads configuration, different measurements are obtained but, in general, the signal contains the following waves:

- *P-Wave*: It is the first wave registered in the ECG, representing the atrium activation just after the sinus stimulation. It normally lasts between 60 and 90 ms in adults, has a round shape with maximal amplitude between 0.25 and 0.30mV.

- *PR-Interval*: measured from the start of the P-wave to the start of the QRS-Complex and lasts 90ms.

- *QRS-Complex*: It corresponds to the ventricular activation and is measured from the start of the first wave (no matter if it is Q or R-wave), to the last wave (R or S-wave). In normal adults, the complex lasts about 80 ms and presents a sharp shape, because of the high frequencies of the signal; however its shape varies a lot, depending on the lead system used.

- *ST-Interval*: It lasts from the end of the QRS-complex to the star of the T-wave and corresponds to part of the ventricular re-polarization process.

- *T-Wave*: It represents the ventricular activation, has a round shape with amplitude about 0.60mV.

Some characteristics of the electric cardiac events and their related waves are summarized in the Table 1. From such characteristics it is possible to state the shape of each wave, and their combination result in the amplitude signal of the dipole, as shown in Figure 4. The ECG is the projection of such signal according to a variable angle (the dipole orientation).

Table 1 – Electric Events in the Heart

<table>
<tr><th colspan="2">Location in the heart</th><th>Event</th><th>Time [ms]</th><th>ECG Terminology</th><th>Conduction velocity [m/s]</th></tr>
<tr><td colspan="2">SA node</td><td>impulse generated</td><td>0</td><td></td><td>0.05</td></tr>
<tr><td colspan="2">Right atrium</td><td>depolarization</td><td>5</td><td rowspan="2">P</td><td>0.8-1.0</td></tr>
<tr><td colspan="2">Left atrium</td><td>depolarization</td><td>85</td><td>0.8-1.0</td></tr>
<tr><td colspan="2">AV node</td><td>arrival of impulse</td><td>50</td><td rowspan="2">P-Q interval</td><td rowspan="2">0.02-0.05</td></tr>
<tr><td colspan="2">AV node</td><td>departure of impulse</td><td>125</td></tr>
<tr><td colspan="2">Bundle of His</td><td>activated</td><td>130</td><td rowspan="3"></td><td>1.0-1.5</td></tr>
<tr><td colspan="2">Bundle Branches</td><td>activated</td><td>145</td><td>1.0-1.5</td></tr>
<tr><td colspan="2">Purkinje Fibers</td><td>activated</td><td>150</td><td>3.0-3.5</td></tr>
<tr><td rowspan="2">Endocardium</td><td>Septum</td><td>depolarization</td><td>175</td><td rowspan="3">QRS</td><td rowspan="2">0.3</td></tr>
<tr><td>Left Ventricle</td><td>depolarization</td><td>190</td></tr>
<tr><td rowspan="4">Epicardium</td><td>Left Ventricle</td><td>depolarization</td><td>225</td><td rowspan="2">0.8</td></tr>
<tr><td>Right Ventricle</td><td>depolarization</td><td>250</td></tr>
<tr><td>Left Ventricle</td><td>repolarization</td><td rowspan="2">400</td><td rowspan="3">T</td><td rowspan="3">0.5</td></tr>
<tr><td>Right Ventricle</td><td>repolarization</td></tr>
<tr><td>Endocardium</td><td>Left Ventricle</td><td>repolarization</td><td>600</td></tr>
</table>

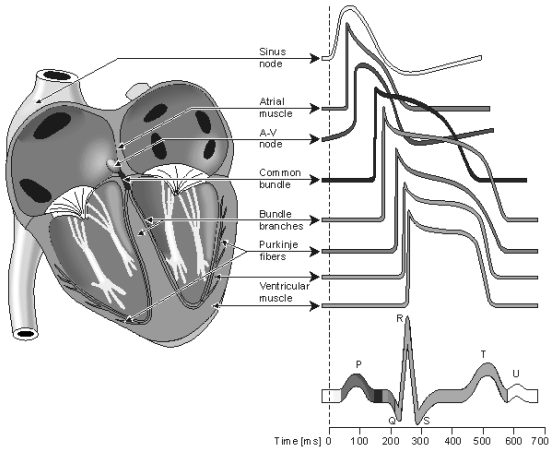

Figure 4 – Different waveforms for each of the specialized cells found in the heart (Malmivuo & Plonsey, 1995).

4. MATHEMATICAL MODELLING

The Van der Pol (VdP) equation was originally employed to describe relaxation oscillators in electronic circuits, and has been frequently used in theoretical models of the cardiac rhythm. This equation is very useful in the phenomenological modeling of natural systems, especially the heartbeat, since it displays many of those features supposed to occur in the biological setting as limit cycle, synchronization and chaotic behavior. The general form of this equation is presented below:

$$\ddot{x} + a\left(1 - bx^2\right)\dot{x} + cx = \Gamma(t) \tag{1}$$

where a, b and c are system parameters and $\Gamma(t)$ is an external forcing.

The qualitative features of an isolated VdP oscillator present a close similarity to the features of the heart actuation potential. Both kinds of actuation potential response (slow and fast) can be easily reproduced by VdP oscillator. Grudzinski & Zebrowski (2004) proposed a modified oscillator in order to simulate important physiological features of the action potentials. In general, the new equation has two fixed points and a damping term asymmetric with respect to the voltage:

$$\ddot{x} + a(x - w_1)(x - w_2)\dot{x} - \frac{x(x+d)}{ed} = \Gamma(t) \tag{2}$$

here a, d and e are system parameters and $\Gamma(t)$ is an external forcing.

As already mentioned, the normal cardiac rhythm is primarily generated by the SA node, which is considered the normal pacemaker. In addition, there is another pacemaker, the AV node. Each of these instances presents an actuation potential, which is fundamental to the heart dynamics, but not necessarily the most expressive to compose the ECG signal. Each activation (depolarization followed by repolarization) corresponds to a different region of the heart and therefore to a different tissue mass generating currents of different magnitudes. Therefore, the combination of activation waves coming from each region of the heart is responsible for the ECG form and is not exactly as sketched in Figure 3. Some of these signals may be preponderant in this composition, like the waves originated in the atrium and ventricle. On the other hand, as these regions follow very close the activation of the SA and AV nodes, their signature on the ECG signal is representative of the pacemakers' signals, and it is possible to associate these signals atrium and ventricle, respectively.

Therefore, it is expected that coupled oscillators, each one representing a different heart region signal, may represent the general heartbeat dynamics. Usually, two oscillators are considered representing the SA and AV nodes, however, it is observed that these two oscillators are not enough to reproduce the ECG signal. This is because the signal of the first oscillator corresponds to the activation of the SA node and atrium, and the signal of the second oscillator corresponds just to the ventricle depolarization. Under this assumption, it is possible to reproduce the P-curve but not the QRS-complex, because this interval mainly corresponds to the ventricle repolarization.

This observation motivate the inclusion of a third oscillator that represent the pulse propagation through the ventricles, which physiologically represents the His-Purkinje complex, composed by the His bundle and the Purkinje fibers. Figure 5 presents the conceptual model showing either the oscillators or the coupling among them. In order to

build a general model, bidirectional asymmetric couplings are assumed among all oscillators. Moreover, an external pacemaker is incorporated to the system, considering a periodic driving term on the oscillators, $\Gamma(t)$.

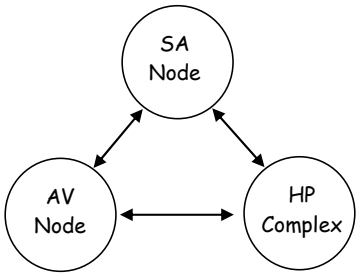

Figure 5 – Conceptual model with three coupled oscillators.

$$
\begin{aligned}
\dot{x}_1 &= x_2 \\
\dot{x}_2 &= -a_{SA}x_2(x_1 - w_{SA_1})(x_1 - w_{SA_2}) - x_1(x_1 + d_{SA})(x_1 + e_{SA}) + \rho_{SA}\sin(\omega_{SA}t) + k_{SA-AV}(x_1 - x_3) + k_{SA-HP}(x_1 - x_5) \\
\dot{x}_3 &= x_4 \\
\dot{x}_4 &= -a_{AV}x_4(x_3 - w_{AV_1})(x_3 - w_{AV_2}) - x_3(x_3 + d_{AV})(x_3 + e_{AV}) + \rho_{AV}\sin(\omega_{AV}t) + k_{AV-SA}(x_3 - x_1) + k_{AV-HP}(x_3 - x_5) \\
\dot{x}_5 &= x_6 \\
\dot{x}_6 &= -a_{HP}x_6(x_5 - w_{HP_1})(x_5 - w_{HP_2}) - x_5(x_5 + d_{HP})(x_5 + e_{HP}) + \rho_{HP}\sin(\omega_{HP}t) + k_{HP-SA}(x_5 - x_1) + k_{HP-AV}(x_5 - x_3)
\end{aligned}
\tag{3}
$$

These governing equations may be rewritten in a compact form as follows:

$$
\ddot{y} = f(y, \dot{y}) + \Gamma(t) + Ky \tag{4}
$$

$$
\begin{aligned}
y &= \begin{bmatrix} x_1 & x_3 & x_5 \end{bmatrix}^T \qquad \dot{y} = \begin{bmatrix} x_2 & x_4 & x_6 \end{bmatrix}^T \\
\Gamma &= \begin{bmatrix} \rho_{SA}\sin(\omega_{SA}t) & \rho_{AV}\sin(\omega_{AV}t) & \rho_{HP}\sin(\omega_{HP}t) \end{bmatrix}^T \\
K &= \begin{bmatrix} k_{SA-AV} + k_{SA-HP} & -k_{SA-AV} & -k_{SA-HP} \\ -k_{AV-SA} & k_{AV-SA} + k_{AV-HP} & -k_{AV-HP} \\ -k_{HP-SA} & -k_{HP-AV} & k_{HP-SA} + k_{HP-AV} \end{bmatrix}
\end{aligned}
\tag{5}
$$

Notice that the governing equations have a general form $f(y, \dot{y})$ (related to the modified VdP oscillator), $\Gamma(t)$ is the forcing term and K represents the coupling matrix.

Time delays in signal transmission are unavoidable and, since even small delays may alter the system dynamics, it is necessary to understand how conduction delays change the behavior of coupled oscillators. The inclusion of time delays in differential equations can cause drastic changes and can make chaos emerges in a system that would otherwise be described by a regular behavior (Campbell & Wang, 1998). On this basis, the proposed mathematical model is changed in order to consider delay aspects in coupling terms. Therefore, governing equations are changed as follows:

$$
\begin{aligned}
\dot{x}_1 &= x_2 \\
\dot{x}_2 &= -a_{SA}x_2(x_1 - w_{SA_1})(x_1 - w_{SA_2}) - x_1(x_1 + d_{SA})(x_1 + e_{SA}) + \rho_{SA}\sin(\omega_{SA}t) + k_{SA-AV}(x_1 - x_3^{\tau_{SA-AV}}) + k_{SA-HP}(x_1 - x_5^{\tau_{SA-HP}}) \\
\dot{x}_3 &= x_4 \\
\dot{x}_4 &= -a_{AV}x_4(x_3 - w_{AV_1})(x_3 - w_{AV_2}) - x_3(x_3 + d_{AV})(x_3 + e_{AV}) + \rho_{AV}\sin(\omega_{AV}t) + k_{AV-SA}(x_3 - x_1^{\tau_{AV-SA}}) + k_{AV-HP}(x_3 - x_5^{\tau_{AV-HP}}) \\
\dot{x}_5 &= x_6 \\
\dot{x}_6 &= -a_{HP}x_6(x_5 - w_{HP_1})(x_5 - w_{HP_2}) - x_5(x_5 + d_{HP})(x_5 + e_{HP}) + \rho_{HP}\sin(\omega_{HP}t) + k_{HP-SA}(x_5 - x_1^{\tau_{HP-SA}}) + k_{HP-AV}(x_5 - x_3^{\tau_{HP-AV}})
\end{aligned}
\tag{7}
$$

where $x_i^{\tau} = x_i(t - \tau)$ and τ represents the time delay. Notice that, actually, there are different delays depending on the connection type.

The classical fourth order Runge-Kutta method is adapted in order to deal with difference differential equation. Basically, it is assumed a function equivalent to the delayed system and, for time instants $t < \tau$, the function y_0 provides values corresponding to time delays prior to the initial instant of observation (Boucekkine *et al.*, 1997):

$$y_0(t) = y(t-\tau) \tag{8}$$

The determination of $y_0(t)$ may be done using Taylor's series, approximating the exact solution (Cunningham, 1954):

$$y(t-\tau) \cong y(t) - \tau\,\dot{y}(t) + \frac{\tau^2}{2}\ddot{y}(t) \tag{9}$$

Under this assumption, it is possible to obtain approximated values for $y(t-\tau)$ in the time interval $t < \tau$. After this, it is possible to use the integrated values in order to construct the time delayed function.

5. NUMERICAL SIMULATIONS

This section considers numerical simulations performed with the proposed heart model. Parameter values suggested by Grudzinski & Zebrowski (2004) are used as reference values for the normal ECG. The other parameters are adjusted in oder to qualitatively match the normal ECG. The following parameters are adopted to represent the normal heart functioning: $a_{SA} = 3$, $w_{SA_1} = 0.2$, $w_{SA_2} = -1.9$, $d_{SA} = 3$, $e_{SA} = 6$; $a_{AV} = 3$, $w_{AV_1} = 0.1$, $w_{AV_2} = -0.1$, $d_{AV} = 3$, $e_{AV} = 3$; $a_{HP} = 5$, $w_{HP_1} = 1$, $w_{HP_2} = -1$, $d_{HP} = 3$, $e_{HP} = 7$. Concerning coupling aspects, it is assumed that the normal heart has a unidirectional coupling from SA to AV nodes and also from AV to HP. Therefore, $k_{SA\text{-}AV} = -5$ and $k_{AV\text{-}HP} = -20$ are non-vanishing terms and all others coupling vanishes. Moreover, it is necessary to establish proper coupling time delay parameters. If there is no coupling, the time delay does not matter, since it represents the elapsed time when the signal travels from a pacemaker node to another. Therefore, the following parameters are assumed: $\tau_{SA\text{-}AV} = 0.8$ and $\tau_{AS\text{-}HP} = 0.1$, vanishing all others. Under this condition, the ECG simulation is presented in Figure 6. As it is shown in its enlargement, the simulated ECG captures its general characteristics, presenting the important waves: P, QRS, T.

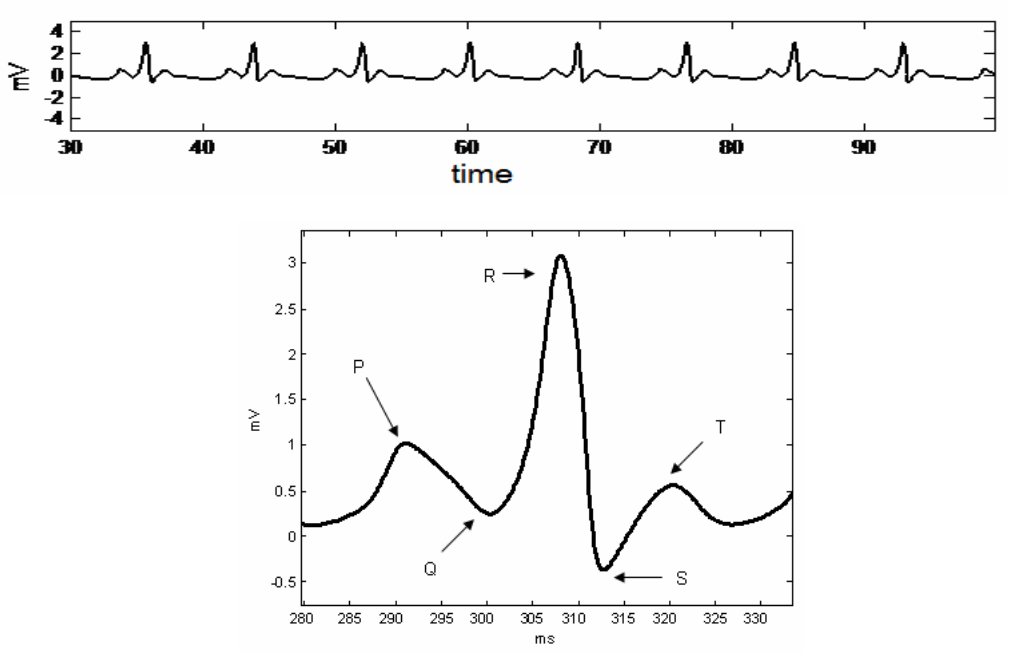

Figure 6 – Normal ECG.

The forthcoming analysis treats different coupling terms in order to simulate some heart pathologies identified from ECG. At first, it is considered the case where there is no communication between the SA and AV nodes, what is simulated by eliminating the coupling between the first and second oscillators ($k_{AV\text{-}SA} = 0$). Under this condition, the system is driven by the AV node, presenting a higher frequency rhythm when compared with the normal ECG. This pathology is named "ventricular flutter" presenting a typical ECG form shown in Figure 7.

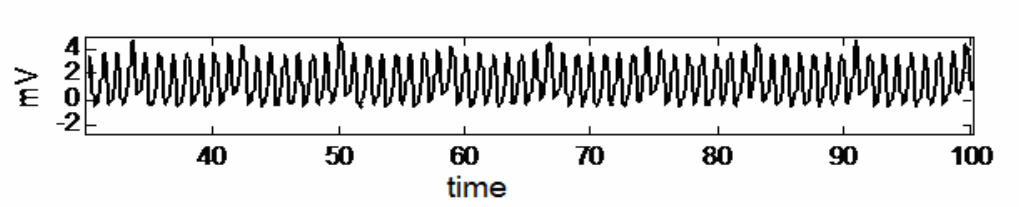

Figure 7 – ECG without SA-AV coupling (ventricular flutter).

The AV-HP coupling is now in focus, assuming $k_{HP\text{-}AV} = 0$. Under this condition, the contribution of the HP oscillator vanishes and the ECG signal is related to the SA and AV activation, which means that ventricles do not perform their function. Figure 8 presents the ECG of this pathological functioning corresponding to the atrial activation.

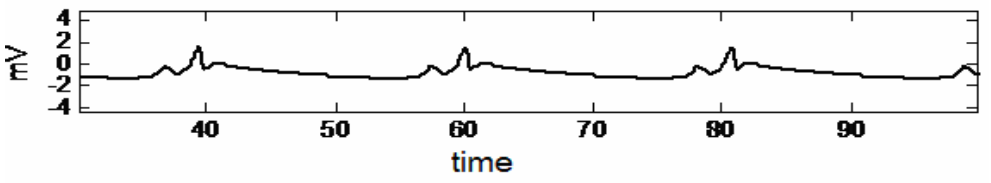

Figure 8 – ECG without AV-HP coupling (sinus bradycardia).

At this point, the normal ECG is considered again, however, an external pacemaker is exciting the heartbeat. Therefore, it is considered the same parameters related to Figure 6 together with the following forcing parameters: $\rho_{SA} = 1$, $\rho_{AV} = 1$, $\rho_{HP} = 20$, $\omega_{SA} = \omega_{AV} = \omega_{HP} = 2\pi/(60/70)$. Under this condition, a "ventricular fibrillation" is induced. This pathological response is caused by different ventricle stimulation being characterized by irregular ECG with fast QRS response.

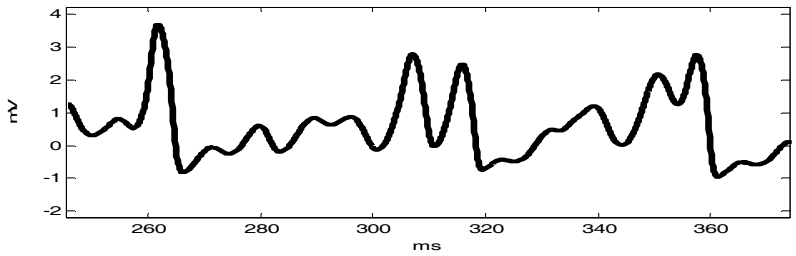

Figure 9 – ECG with external pacemaker excitation (ventricular fibrillation).

6. CONCLUSIONS

A mathematical modeling of heart rhythms is developed considering three coupled modified Van der Pol oscillators. Each oscillator represents one of the main important heart activation potentials: sinoatrial node (SA), atrioventricular node (AV) and His-Purkinje complex (HP). Numerical simulations of the proposed model shown that it is capable to capture the general heartbeat dynamics, representing the general ECG form with P, QRS and T curves. Afterwards, some pathological rhythms are of concern by establishing different coupling situations. Basically, it is assumed some communication interruption in the heart electric system. Under this condition, it is possible to simulate cardiopatologies as ventricular flutter. Moreover, external pacemaker excitation is of concern representing ventricular fibrillation. These results may encourage the identification of different pathological rhythms.

7. ACKNOWLEDGEMENTS

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8. REFERENCES

Boucekkine, R., Licandro, O. & Paul, C. (1997), "Differential-difference equations in economics: On the numerical solution of vintage capital growth models", *Journal of Economic Dynamics and Control*, v.21, pp. 347-362.

Campbell, S.R. & Wang, D. (1998), "Relaxation oscillators with time delay coupling", *Physica D*, v.111, pp.151-178.

Cunningham, W.J. (1954), "A Nonlinear Differential-Difference Equation of Growth" *Proceedings of the National Academy of Sciences of the United States of America*, v.40, n.8, pp.708-713.

Dubin, D. (1996), "*Interpretação Rápida do ECG*", Editora de Publições Biomédicas – EPUB, Rio de Janeiro.

Ferriere, R. & Fox, G. A. (1995), "Chaos and evolution", *Trends in Ecology and Evolution*, v.10, n.12, pp.480-485.

Garfinkel, A., Spano, M. L., Ditto, W. L. & Weiss, J. N. (1992), "Controlling cardiac chaos", *Science*, v.257, pp.1230-1235.

Garfinkel, A., Weiss, J. N., Ditto, W. L. & Spano, M. L. (1992), "Chaos control of cardiac arrhythmias", *Trends in Cardiovascular Medicine*, v.5, n.2, pp.76-80.

Glass, L. (2001), "Synchronization and Rhythmic Processes in Physiology", *Nature*, v.410, March, pp.277-284.

Hodkin, A. L. & Huxley, A. F. (1952). "A quantitative description of membrane current and its application to conduction and excitation in nerve", *J. Physiology,*v.117, pp.500-544.

Malmivuo, J. & Plonsey, R. (1995), "*Bioelectromagnetism - Principles and applications of bioelectric and biomagnetic fields*", Oxford University Press, New York.

Moffa, P.J. & Sanches, P.C.R. (2001) " *Eletrocardiograma Normal e Patológico*", Editora Roca.

Radhakrishna, R. K. A., Dutt, D. N., Yeragani, V. K. (2000), "Nonlinear measures of heart rate time series: Influence of posture and controlled breathing", *Autonomic Neuroscience: Basic and Clinical*, v.83, pp.148–158.

Santos, A.M., Lopes, S.R. & Viana, R.L. (2004), "Rhythm synchronization and chaotic modulation of coupled Van der Pol oscillators in a model for the heartbeat", *Physica A*, v.338, pp.335-355.

Savi, M.A. (2005), "Chaos and order in biomedical rhythms", *Journal of the Brazilian Society of Mechanical Sciences and Engineering*, v.XXVII, n.2, pp.157-169.

Savi, M.A. (2006), "*Nonlinear dynamics and chaos*", Editora E-papers (in portuguese).

Van Der Pol, B. (1926), "On relaxation oscillations", *Philosophical Magazine*, v.2, pp.978.

Van Der Pol, B. & Van Der Mark, J. (1928), "The heartbeat considered as a relaxation oscillator, and an electrical model of the heart", *Philosophical Magazine*, v.6 suppl., pp.763.

Witkowski, F. X., Kavanagh, K. M., Penkoske, P. A., Plonsey, R., Spano, M. L., Ditto, W. L. & Kaplan, D. T. (1995), "Evidence for determinism in ventricular fibrillation", *Physical Review Letters*, v.75, n.6, pp.1230-1233.

Witkowski, F. X., Leon, L. J., Penkoske, P. A., Giles, W. R., Spano, M. L., Ditto, W. L. & Winfree, A. T. (1998), "Spatiotemporal evolution of ventricular fibrillation", *Nature*, v.392, March, pp.78-82.

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