A New Nonlinear Model for Generating RR Tachograms

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Abstract

Using a mathematical model to produce the time series for heart rate variability (HRV) is needed to produce artificial electrocardiogram (ECG) signals. In this paper, we incorporate into our model the effects of both respiratory sinus arrhythmia (RSA) and Mayer waves. In addition, the inter-coupling between sympathetic *and* parasympathetic *systems is also included.*

1. Introduction

The dynamic response of the cardiovascular control system to physiological changes is reflected in variations of heart rate and blood pressure. The variation in the time series of consecutive heartbeats is referred as heart rate variability (HRV). The times between R-peaks of adjacent QRS-complexes give us the RR-intervals, which is referred to as an *RR tachogram*. The variability of these RR-intervals is conveniently called *heart rate variability* (HRV). Recent attention has focused on what this time series signifies in term of cardiovascular health. HRV is being investigated as a high-risk indicator for possible mortality following myocardial infraction [1]. Beat-to-beat variations of human RR-intervals display fluctuations over a number of different time scales ranging from seconds to days. Some of these fluctuations are relatively well understood and arise from: (i) the interactions between different physiological control mechanisms such as *respiratory sinus arrhythmia* (RSA) and *Mayer waves*; (ii) the amount of physical and mental activity; (iii) the circadian rhythm; and (iv) the effects of different sleep stages [2]. Producing a time series for HRV is an important factor in generating an artificial ECG signal.

Previous works have had some limitations. In [2], the authors tried to generate RR-intervals by starting from a given spectrum and then solving the inverse problem. This may be useful when generating an artificial ECG without any intuition about the origin of the signal, but we cannot generate abnormal HRV by changing the parameters in different parts of the synthesis mechanism. In [1], there is no consideration of systems with coupling between *sympathetic* and *parasympathetic* systems. In [3] for example, the SA node cannot generate any beats if its inputs are off. We will try to overcome these limitations in this paper.

The paper is organized as follows. In section 2, a summary of HRV characteristics is given. The proposed mode is presented in section 3. Simulation results will be given in section 4, and finally conclusions are in section 5.

2. Heart rate variability characteristics

The *Autonomic Nerve System* (ANS) is responsible for short-term regulation of the blood pressure. The ANS is a part of the *Central Nervous System* (CNS).The ANS uses two subsystems - the *sympathetic* and *parasympathetic* systems. The *sympathetic* system is active during stressful situations, in order to provide a higher heart rate up to 180 beat per minute (bpm). Sympathetic fibers innervate the entire heart, including the sinus node, the AV conducting pathways and the atrial and ventricular myocardium. Increased activity of the sympathetic nerves increases heart rate (HR) and force of contraction. In addition, the rate of conduction through the heart is increased and the duration of contraction is shortened. When sympathetic activity increases, there is a latent period of up to 5 seconds before there is an increase in HR, which then reaches a steady level after about 30 seconds. In contrast, the parasympathetic system is active during rest and can reduces the HR down to 60 bpm. It innervates the atriventricular conducting pathways and the atrial muscle. The latency of the response of the sinus node is very short, just in the first of second beat after its onset. There is a linear relation between decreasing the HR and the frequency of parasympathetic stimulation. Spectral analysis of HRV is typically used to estimate the effects of sympathetic and parasympathetic modulation of RR-intervals. The main frequency bands are referred to as LF band (0.04-0.15 Hz) and HF band (0.15-0.4 Hz). The ratio of power in the LF band to power in the HF band

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(LF/HF) has previously been used as a measure of the balance between the effects of sympathetic and parasympathetic systems [2]. In some papers the frequency component around 0.04 Hz is called the very low frequency (VLF) and the one around 0.1 Hz called LF [3]. It has been noted that an imbalance between the competing sympathetic and parasympathetic systems is an important indicator of many cardiac disorders [1]. The HF component is in synchrony with respiration and is referred as respiratory sinus arrhythmia (RSA)[2].

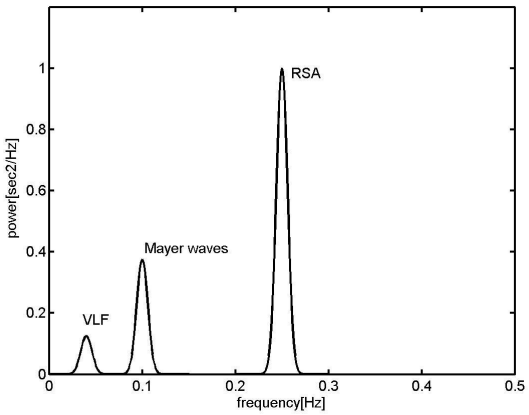

Fig. 1. A typical power spectrum of HRV

Unlike the HF component, there is still some controversy as regards the origin of the LF and VLF components. The LF component is referred to as *Mayer waves* [3]. It is believed that under normal conditions the existence of at least three oscillatory systems are responsible for the generation of the Mayer waves, namely: (i) The baroreceptor or chemoreceptor feedback system; (ii) centrogenic rhythm in the brain stem with interconnection to the respiratory oscillator; (iii) the autorhythmicity of vascular smooth muscle [3]. Fig 1 shows a typical power spectrum of HRV.

3. Our proposed model for HRV

Our model is based on the following hypothesizes:

1. Each controlling influence acts as an sinusoidal oscillator.

2. The *Sinus Atrial* (SA) node has an oscillation between 100-120 bpm, when there is no activation of sympathetic and parasympathetic systems. However, this SA node oscillation frequency changes very slowly, and so this frequency may be considered constant, for the purposes of our model.

3. The most important contribution to changes in HRV is the effect of RSA, which is believed to be produced by fluctuations of vagal-cardiac nerve activity. It produces the HF component of the HRV power spectrum. The HR accelerates during inspiration and decelerates during expiration, and the magnitude of this response depends on the rate and depth of respiration. Because of the latent response of the sympathetic system and its low pass filtering behavior, we consider the respiration response only in the parasympathetic system.

4. The LF component which occurs around 0.1 Hz, originates from self-oscillation in the vasomotor part of the baroreflex loop as a result of negative feedback in the baroreflex. This fluctuation is synchronous with fluctuations of blood pressure, and it is known as Mayer wave. This fluctuation decreases with both parasympathetic and sympathetic blockade

5. The very low frequency (VLF) component, which is believed to arise from thermoregulatory peripheral blood flow adjustments, is caused by the sympathetic nervous system

6. The neural regulation of the circulatory function is attained mainly by the balance between the sympathetic and parasympathetic activities. In most physiological conditions, the activation of either one of these is accompanied by the inhibition of the other. Here we suppose that sympathetic and parasympathetic can inhibit each other after reaching pre-defined thresholds.

7. The sympatho-vagal balance is affected by the interaction of many factors, such as: central neural integration, peripheral inhibitory reflex mechanisms (with negative feedback characteristics), and peripheral excitatory reflex mechanisms (with positive feedback characteristics). But here we will externally control this balance by use of the coupling factors (without using these feedbacks)

A very simple (but adequate) model for SA node is the Integral Pulse Frequency Modulation model (IPFM) - see Fig. 2. It was first suggested as a model for a cardiac pacemaker by Hyndman and Mohn [4]. The IPFM model integrates the input signal until it reaches a threshold (here we assume that $Th = 1$ in (1b) and Fig. 2). Then it generates a pulse as a heartbeat. After that, the integrator is reset to zero [1]. So we can write:

$$s(t) = \sum_k \delta(t - t_k) \tag{1a}$$

$$Th = \int_{t_k}^{t_{k+1}} (m_0 + m(t))dt. \tag{1b}$$

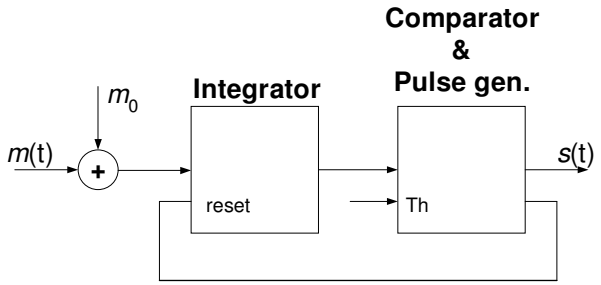

Fig. 2. IPFM block diagram

where $m(t)$ is the input signal representing autonomic nervous activity, and t_k is the time of the k^{th} R wave. When the input signal is zero the IPFM model generates a heartbeat with an interval equal to the HR [5]. Of course $m_0 + m(t)$ should be positive. Note that the timing of $s(t)$ relates to the artificial R wave.

Now, consider the inter-coupled differential equations in (2). Here the x_1, x_2, y_1 and y_2 produce outputs of the sympathetic and parasympathetic systems respectively. The internal signal of the SA node is produced by z; ω_1 is the angular velocity corresponding to the thermoregulatory peripheral blood flow adjustment system; ω_2 ,corresponds to the vasomotor part of the baroreflex loop, as a result of negative feedback in the baroreflex from sympathetic and parasympathetic systems; ω_3 is the angular velocity corresponding to respiratory sinus arrhythmia (RSA); and finally ω_4 corresponds to the angular velocity of internal variations of the SA node. The t_1 and t_2 thresholds in (2) are used to adjust the influence of sympathetic and parasympathetic activity, where the α and β parameters reflect the strengths of their respective influences. Fig. 3 shows the proposed model where sgn represents the signum function .Now we can apply the output of the proposed dynamical system to the IPFM in Fig. 3.

$$\begin{aligned}
\frac{d^2x_1}{dt^2} &= -\omega_1^2\left(x_1 + \alpha\left(\mathrm{sgn}(y_1 + y_2 - t_2) + 1\right)\right) \\
\frac{d^2x_2}{dt^2} &= -\omega_2^2\left(x_2 + \alpha\left(\mathrm{sgn}(y_1 + y_2 - t_2) + 1\right)\right) \\
\frac{d^2y_1}{dt^2} &= -\omega_2^2\left(y_1 + \beta\left(\mathrm{sgn}(x_1 + x_2 - t_1) + 1\right)\right) \\
\frac{d^2y_2}{dt^2} &= -\omega_3^2\left(y_2 + \beta\left(sgn(x_1 + x_2 - t_1) + 1\right)\right) \\
\frac{d^2z}{dt^2} &= -\omega_4^2 z.
\end{aligned} \tag{2}$$

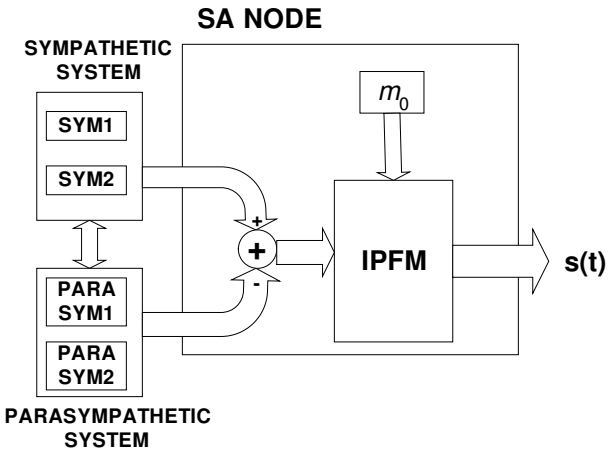

Fig. 3. The proposed model

$$\begin{aligned}
&m(t) = k_1\left(x_1(t) + 1\right) + k_2\left(x_2(t) + 1\right) - \\
&k_3\left(y_1(t) + 1\right) - k_4\left(y_2(t) + 1\right) \\
&m_0 = k_5\left(k_6 z(t) + 1\right) \\
&\int_{t_k}^{t_{k+1}} \left(m(t) + m_0\right) dt = Th \\
&s(t) = \sum_k \delta(t - t_k).
\end{aligned} \tag{3}$$

Now $s(t)$ in (3) gives us the heart beats.

4. Simulation results

From knowledge of the characterization of the real HRV, and from experimentation, we have considered the values of the parameters in (3) as follows:

$t_1 = 0.0000, t_1 = 0.0000, \alpha = 0.5000, \beta = 0.5000, \omega_1 = 0.2236, \omega_2 = 0.4123,$
$\omega_3 = 1.0000, \omega_4 = 0.4123, k_1 = 0.0200, k_2 = 0.0700, k_3 = 0.0800, k_4 = 0.0200,$
$k_5 = 1.3150, k_6 = 1.0000.$

We start a discrete-time simulation of the model in Fig. 3, and Fig. 4 shows the simulated HRV for a healthy human. Here the average HR was 78 beats per minute. Figs 5 shows the power spectrum of the simulations for HRV with respect to parasympathetic blockade, high sympathetic balance, and high parasympathetic balance,

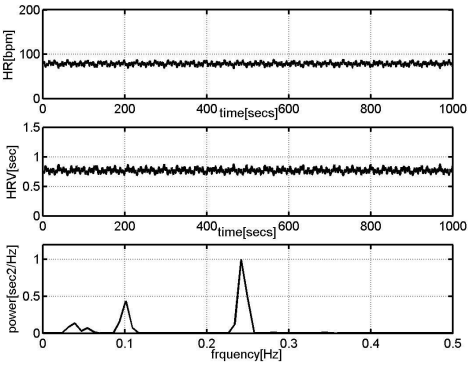

Fig. 4. From top to bottom: the simulated heart rate, HRV (RR-intervals), and power spectrum of HRV for a healthy person

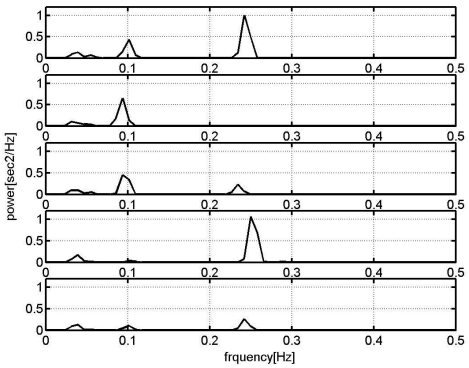

Fig. 5 Normalized power spectrum of simulated HRV for a healthy case and four different sicknesses (by changing parameters k_1 to k_6), as generated by the proposed model in (3) and (4). Top to bottom: *healthy*, *parasympathetic blockade*, *high sympathetic balance*, *high parasympathetic balance* and *CAN sickness*.

and cardiovascular autonomy neuropathy (CAN) in a diabetic person respectively. In these simulations we generated data for 1000 seconds.

Because of using nonlinear coupling, if we try to change the effect of each of the inputs of IPFM, we can see changes in the center frequency of each of the spectral components. This effect needs to be investigated more in future work.

5. Conclusion

A model for generating artificial RR-intervals has been presented. Our model can provide RR-intervals for both healthy and sick cases. We get the very low frequency and Mayer waves components, as well as the RSA component. We got good results in simulation of parasympathetic blockade, high sympathetic balance, and high parasympathetic balance. We also simulated the RR-intervals relating to cardiovascular autonomy neuropathy (CAN) sickness. Future work will be concerned with more accurately modeling m_0 in the SA node.

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