A modified Zeeman model for producing HRV signals and its application to ECG signal generation

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Abstract

Developing a mathematical model for the artificial generation of electrocardiogram (ECG) signals is a subject that has been widely investigated. One of the challenges is to generate ECG signals with a wide range of waveforms, power spectra and variations in heart rate variability (HRV)—all of which are important indexes of human heart functions. In this paper we present a comprehensive model for generating such artificial ECG signals. We incorporate into our model the effects of respiratory sinus arrhythmia, Mayer waves and the important very low-frequency component in the power spectrum of HRV. We use a new modified Zeeman model for generating the time series for HRV, and a single cycle of ECG is produced by using a simple neural network. The importance of the work is the model's ability to produce artificial ECG signals that resemble experimental recordings under various physiological conditions. As such the model provides a useful tool to simulate and analyse the main characteristics of ECG, such as its power spectrum and HRV under different conditions. Potential applications of this model include using the generated ECG as a flexible signal source to assess the effectiveness of a diagnostic ECG signal-processing device.

Keywords: ECG; Heart rate variability; Dynamical systems; Neural networks

1. Introduction

The electrocardiogram (ECG) signal is one of the most obvious effects of the human heart operation. The oscillation between systole and diastole states of the heart is reflected in the heart rate (HR). The surface ECG is the recorded potential difference between two electrodes placed on the surface of the skin at pre-defined points. The largest amplitude of a single cycle of the normal ECG is referred to as the R-wave manifesting the depolarization process of the ventricle. The time between successive R-waves is referred to as an RR-interval, and an RR-tachogram is then a series of RR-intervals. Variability in this time series has been widely used as a measure of heart function, and this helps to identify patients at risk for a cardiovascular event or death (McSharry et al., 2002). Analysis of variations in this time series is known as heart rate variability (HRV) analysis (Malik and Camm, 1995; Braunwald et al., 2004).

The development of a dynamical model for the generation of ECG signals with appropriate HRV spectra is a subject that has been widely investigated. Such a model will provide a useful tool to analyse the effects of various physiological conditions on the profiles of the ECG. The model-generated ECG signals with various characteristics can also be used as signal sources for the assessment of diagnostic ECG signal processing devices. Now, in
constructing a comprehensive model for generating ECG signals there are two steps. Step one is producing the artificial RR-tachogram with HRV spectrum similar to experimental data—the RR-tachogram shows where the R-waves of the ECG are actually placed. And step two is constructing the actual shape of the ECG.

The dynamic response of the cardiovascular control system to physiological changes is reflected in HRV and blood pressure (Braunwald et al., 2004). Recent attention has focused on what HRV signifies in term of cardiovascular health, and HRV is being investigated as a high-risk indicator for possible mortality following myocardial infarction (Brennan et al., 1998). 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 (Malik and Camm, 1995; De Boer et al., 1987; Lavie, 1996; McSharry et al., 2002). So, being able to produce a time series for HRV is an important aspect in generating an artificial ECG signal.

The motivation for using nonlinear methods in modeling HRV is that the source of the mechanism for generating the ECG (i.e. the propagation of electrical activities in myocardium) is nonlinear. In 1972, Zeeman presented an important set of nonlinear dynamical equations for heartbeat modeling (Zeeman, 1972a, b; Suckley and Biktashev, 2003), based on the Van der Pol-Lienard equation, but this model did not entirely satisfy the biologists’ understanding of HRV generation (Tu Pierre, 1994). The reason was the lack of consideration given to important biological parameters such as sympathetic and parasympathetic modulations.2

Here, we develop a new model based on modifying the original Zeeman model to produce the RR-tachogram signal, which now incorporates the effects of sympathetic and parasympathetic activities to generate the appropriate significant peaks in the power spectrum of the HRV. By using a neural network approach based upon a modified McSharry model, the actual shape of the ECG in a single cycle can be successfully re-produced by using our model-generated power spectrum of RR time intervals.

So this paper is organized as follows. In Section 2, a summary of ECG and HRV morphology is given. The proposed model is developed in Section 3. Simulation results will be given in Section 4, and finally conclusions/discussions are presented in Section 5.

In any heart operation there are a number of important events. The successive atrial depolarization/repolarization and ventricular depolarization/repolarization occurs with every heartbeat. These are associated with the peaks and valleys of the ECG signal, which are traditionally labeled P, Q, R, S, and T (see Fig. 1). The P-wave is caused by depolarization of the atrium prior to atrial contraction. The QRS-complex is caused by ventricular depolarization prior to ventricular contraction. The largest amplitude signal (i.e. R-wave) is located here. The T-wave is caused by ventricular repolarization which lets the heart be prepared for the next cycle. Atrial repolarization occurs within ventricular depolarization, but its waveform is masked by the large amplitude QRS-complex.

The HR, which is the inverse of the RR-interval, directly affects the blood pressure. 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 HR may be increased by sympathetic activity or decreased by parasympathetic (vagal) activity. The balance between the effects of the sympathetic and parasympathetic systems is referred to as the sympathovagal balance and is believed to be reflected in the beat-to-beat changes of the cardiac cycle (McSharry et al., 2002).

Spectral analysis of HRV is a useful method to investigate the effects of sympathetic and parasympathetic activities on heart rate (HR). The afferent nerves provide the feedback information to the CNS. The sympathetic system is active during stressful conditions, which can increase the HR up to 180 beats per minute (bpm). When sympathetic activity increases after a latent period of up to 5 s, a linearly dependent increment in HR begins and reaches its steady state after about 30 s. This affects the low frequency (LF) component (0.04–0.15 Hz) in the power

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2In the brain, neural impulses are generated whose rhythmic temporal intensities reflect the degrees of autonomic tones. These impulses are transmitted either by the vagus nerves (parasympathetic) or the spinal column (sympathetic). Parasympathetic impulses act on the intrinsic cardiac pacemaker (sino-atrial node) to slow the heat beat, whereas sympathetic impulses act on the intrinsic cardiac pacemaker to increase the heartbeat.

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Fig. 1. A single cycle of a typical ECG signal with the important points labeled—i.e. P, Q, R, S and T.
effects of sympathetic (Zhang et al., 2001) and parasympathetic systems (McSharry et al., 2003). Fig. 2 shows the typical HRV power spectrum of sympathetic and parasympathetic systems (McSharry et al., 2003). Now catastrophe theory is one branch of applied mathematics that was developed in order to describe certain biological processes and has been applied by researchers, especially by Christopher Zeeman—see Zeeman (1977) for a full collection of his works.

In Zeeman’s original paper on the heartbeat study (see Zeeman, 1972a, b) he analysed two sorts of biological excitable systems (nerve and heart). As regards heart beat generation, we will begin with Zeeman’s “nerve” model that is a nonlinear dynamic system of three inter-coupled differential equations as follows:

\[ \begin{align*}
    \dot{x} &= -(x^3 + ax + b), \\
    \dot{a} &= -2x - 2a, \\
    \dot{b} &= -a - 1,
\end{align*} \]  \hspace{1cm} (1)

where \( x \) (which can be negative) is related to the length of the heart muscle fiber, \( \varepsilon \) is a positive scalar, \( b \) is a parameter representing an electrochemical control, and parameter \( a \) is related to the tension in the muscle fiber. The equilibrium state of the system can be obtained by solving the following equations:

\[ \begin{align*}
    - (x^3 + ax + b) &= 0, \\
    - 2x - 2a &= 0, \\
    - a - 1 &= 0.
\end{align*} \]  \hspace{1cm} (2)

So there is an equilibrium point at \( A(1, -1, 0) \). Now we want to find a linear approximation of the system in neighborhood of \( A \), and so we can write (Zeeman, 1972a)

\[ \begin{align*}
    \frac{\dot{x}}{2/\varepsilon} &\approx \frac{-1/\varepsilon - 1/x}{0}, \\
    \frac{\dot{a}}{2} &\approx -2 - 2a, \\
    \frac{\dot{b}}{1} &\approx -1 - b.
\end{align*} \]  \hspace{1cm} (3)

The \( A \) matrix in (3) has eigenvalues \((-1 \pm \sqrt{\varepsilon})/2\) and \((-2)/\varepsilon\), where \( \varepsilon = \sqrt{-1} \). Thus the point \( A(1, -1, 0) \) is a stable equilibrium point. Now, because \( \varepsilon \) is very small the ‘fast’ eigenvalue (with direction parallel to \( x \)-axis) is \(-2/\varepsilon\). As \((-1 \pm \sqrt{\varepsilon})/2\) is small (compared to \((-2)/\varepsilon\), the two complex eigenvalues indicate a steady slow direction toward point \( A \). Fig. 3 shows the trajectory (in \( x-a-b \) phase space) of the above model (parameterized by time).

3. Model development

Mathematical models (see Jones and Sleeman, 2003) are extremely important for understanding biological processes. As regards describing biological excitable systems—particularly nerve and heart tissues—mathematical models are historically the first, and so far the best, in terms of a quantitative description of biological phenomena (Suckley and Biktashev, 2003). Now catastrophe theory is one branch of applied mathematics that was developed in order to describe certain biological processes and has been
Now we can also consider the variation of $-x$ versus time (see Fig. 4) as a potential action which repeats in time as heart beating, because it has a stable oscillation with different phases in its path (like an action potential). Note the rapid jumps from B to C (like the depolarization process from diastole to systole state) and the slow return from C to A (like the repolarization process from systole to diastole state), (Tu Pierre, 1994). Note that we do not use the actual shape of this potential action in producing the ECG waveform. We only use the time series corresponding to the occurrence of these potential actions, and then employ our neural network in the modified McSharry model to produce the actual waveform of the ECG signals.

In order to control the cycle length of the heartbeat in the model (i.e., the time interval between two successive action potentials of solution x in (1)), we propose to add an additional control parameter (i.e. $\delta$) and get the following system:

$$\dot{x} = -(x^3 + ax + b),$$
$$\dot{a} = -2x - 2a,$$
$$\dot{b} = -a - 1.$$  \quad (4)

It is easy to see that the frequency of the oscillation in this model now depends upon the value of $\delta$. This relationship is more or less linear—see Fig. 5.

Now we consider the chronotropic modulations of HR by relating the parameter $\delta$ to the four states of sympathetic and parasympathetic activity levels, e.g., $s_1$, $s_2$, $p_1$ and $p_2$. For simplicity, we assume that the states of sympathetic and parasympathetic activities are sinusoidal and can be modeled by the equations given below:

$$\dot{s}_1 = -\omega_1^2 s_1,$$
$$\dot{s}_2 = -\omega_2^2 s_2,$$
$$\dot{p}_1 = -\omega_3^2 p_1,$$
$$\dot{p}_2 = -\omega_4^2 p_2.$$  \quad (5)

Now we can relate the parameter $\delta$ to the states of sympathetic and parasympathetic activity by the following equations:

$$q_1 = c_1(s_1 + 1) + c_2(s_2 + 1),$$
$$q_2 = c_3(p_1 + 1) + c_4(p_2 + 1),$$
$$q = q_1 - q_2,$$
$$\delta = h(q).$$  \quad (6)

The parameter $\delta$ determines the HR, and the function $h$ and the coupling factors ($c_1$, $c_2$, $c_3$ and $c_4$) determine how the sympathetic and parasympathetic activities alter the HR. Although the function $h(q)$ (in (6)) is nonlinear, it can be approximated with either piecewise linear modules (as we will do in this paper) or with a neural network (currently research in progress by the authors). Finally, the parameters $\omega_1$, $\omega_2$, $\omega_3$ and $\omega_4$ are the angular frequencies for the sinusoidal variations of the sympathetic and parasympathetic activities. In order to determine the values of $\omega_1$, $\omega_2$, $\omega_3$ and $\omega_4$, we provide the following hypothesis:

(i) The most important contribution to changes in HRV is the effect of RSA, which is believed to be produced by fluctuations of cardiac vagal–nerve activity. It produces the HF component of the HRV power spectrum. The HR decelerates during inspiration and accelerates during expiration, and the magnitude of this response depends upon 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. So $\omega_4$ will be related to the frequency of respiration.

(ii) 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 the Mayer wave. This fluctuation decreases with both parasympathetic and sympathetic blockade. So \( f_2 \) and \( f_1 \) are related to 0.1 Hz, where \( \omega_i = 2\pi f_i \).

(iii) The very low-frequency (VLF) component, which is believed to arise from thermoregulatory peripheral blood flow adjustments, is caused by the sympathetic nervous system. So \( \omega_2 \) is related to this frequency.

(iv) We assume that there is no inter-coupling between sympathetic and parasympathetic activities.

So, the timing of \((-x)\) gives us the RR-intervals—see Fig. 4. This is an approximation to the time series of RR time intervals (i.e. \( T_{RR}(t) \)), and so let us call it \( \hat{T}_{RR}(t) \). We will then have

\[
\omega(t) = \frac{2\pi}{\hat{T}_{RR}(t)}. \tag{7}
\]

Note that other influences like mechano-electrical feedback, emotions, hormones and intrinsic cardiac ganglia innervations can have an effect on the function \( h(q) \) in (6). These have not been considered here and will be a subject for further study. The next step is to use \( \omega(t) \) obtained from Eq. (7) and simulate one cycle of ECG signal and then a complete time series of ECG by using Eq. (8) as described below.

The ECG signal will be simulated by a system of three inter-coupled differential equations of \( x_1, x_2, x_3 \) as described by Eq. (8). In the phase-space, the solution of the equation shows a stable limit cycle on the projection of the \( x_1-x_2 \) plane. However, the trajectory of \( x_3 \) (i.e., \( x_3 \) against time) represents the simulated ECG signal \( f(t) \), where \( f(t) \) is the original ECG signal over one cycle—see Fig. 6.

Eq. (8) is based on the modification of the dynamic equations from McSharry et al. (2002):

\[
\begin{align*}
\dot{x}_1 &= \omega(t)x_2 + x_1(1 - x_1^2 - x_2^2), \\
\dot{x}_2 &= -\omega(t)x_1 + x_2(1 - x_1^2 - x_2^2), \\
\dot{x}_3 &= \hat{g}(t) - x_3,
\end{align*} \tag{8}
\]

where \( \omega(t) \) corresponds to the angular velocity of the limit cycle in the \( x_1-x_2 \) plane, and \( \theta = \angle(x_1 + ix_2)/2\pi, \ 0 \leq \theta < 1 \). To use Eq. (8) to simulate a complete set of the time series of the ECG based upon a given single cycle of a real ECG signal \( f(t) \), the most important step is to determine \( \hat{g}(t) \).

\( \hat{g}(t) \) is determined by a radial basis expansion as described by Eq. (9). Consider a single cycle of a normal or abnormal real ECG signal \( f(t) \) with a (normalized) period of 1 s. Note that \( t \) and \( \theta \) are interchangeable over one cycle of \( f(t) \) (the original ECG). Then \( \hat{g}(t) \) is an approximation to \( g(t) \), and \( g(t) = Tf(t)/dt \) (see (10) later):

\[
\hat{g}(t) = \sum_{i=1}^{N} w_i \exp(-((\theta - t_i)^2/(2\sigma_i^2))). \tag{9}
\]

We estimate the unknown parameters \( \{w_i, t_i, \sigma_i\} \) in (9) using a neural network approach. Suppose we take \( m \) samples of \( f(t) \). We then use a low-pass digital filter to attenuate any noise. The next step is to calculate the (scaled) derivative of \( f(t) \), by using the backward difference approximation, \( g(t) \):

\[
f = [f(0), f(T), ..., f(m-1)T)].
g(jT) = f((j-1)T) - f((j-2)T). \tag{10}
\]

By considering \( m \) samples of \( \theta \), with the \( j \)th element equal to \( jT \), we wish to get \( \hat{g}(\theta) \approx g(T) \), while the centers \( \{t_i\} \) in (9) can be assigned directly based on our ECG knowledge, or with an unsupervised algorithm. We have decided here to estimate all the parameters \( \{w_i, t_i, \sigma_i\} \) with a gradient-descent procedure (Jafarnia-Dabanloo et al., 2004).

As is already known, the radial basis function (RBF) neural network has a fast adaptation, and because of its nonlinear kernels it can easily approximate our proposed nonlinear dynamic. The ability of an RBF neural network to approximate any nonlinear function has been proven by Park and Sandberg (1991). But it is slower than the backpropagation approach in the recall phase.

4. Simulation results

First of all, we are interested in modeling certain syndromes where the ECG will appear different from the normal (as in Fig. 7) PQRST ECG cycle. These three syndromes are as follows.

**Wolf-Parkinson-White (WPW) syndrome** is best described as the presence of an accessory electrical pathway between the atrium and ventricle in the heart. Due to its electrical properties, usually (but not always) it will conduct the sinus node discharge (at the beginning of the heart beat) to the ventricle faster than usual AV-node conduction. Therefore, the typical ECG will demonstrate a shorter PR distance (faster conduction) and notched QRS complex (time for depolarization of the ventricles). The

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Footnote:

1For \( f(t) \) we obtained real ECG data at: http://physionet.caregroup.harvard.edu/physiobank/database.
importance of WPW is that it can lead to very fast tachycardia (abnormal rapid heart beating), at times leading to VF (ventricular fibrillation) and sudden cardiac death. Hyperkalemia manifests itself as tall T waves in the ECG. But note that tall T waves are not pathognomic (i.e., not distinctive) for hyperkalemia, and can be seen in other illnesses—a challenge for any ECG model. Finally, for ventricular tachycardia/flutter, there is no P wave. One of the characteristics is A–V dissociation—i.e., the absence of a relationship between atria and ventricle. If sustained and not treated, this condition may lead to collapse and death; so now for the simulation setup.

In the off-line phase of the simulation, the RBF neural network (with one hidden layer) produces \( g(\theta) \) an approximation to \( g(\theta) \). At first, we filter the derivative \( g(t) \) of the desired ECG signal \( f(t) \), in order to reduce the required number of neurons. We then feed the data into the learning process of the RBF neural network, and obtain the required parameters \( \{ w_j, t_j, a_j \} \)—i.e., weights, centers and spread factors. So from this method we can obtain the parameters for the normal ECG, ECG with WPW, ECG with hyperkalemia and finally ECG with ventricular flutter.

Then in the on-line phase, we start a discrete-time simulation of the model in (8), with arbitrary initial values for \( x_1, x_2, \) and \( x_3 \) with the generated RR-intervals. Of course for the four different ECG signals, we simply supply the model in (8) with the required parameters and the model generates the artificial ECG.

One cycle of both the simulated normal ECG and the original ECG is shown in Fig. 7. The error measure, which determines how closely we actually simulate the required ECG \( f(t) \), is the total error per cycle \( \text{TEPC} \) in (11), where \( \hat{f} \) is the actual output of the model:

\[
\text{TEPC} = \frac{100}{\text{max}(f)} \sqrt{\frac{\sum_{j=0}^{m-1} (f(j) - \hat{f}(j))^2}{m}}. \tag{11}
\]

Table 1

<table>
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<tr>
<th>N</th>
<th>TEPC1</th>
<th>TEPC2</th>
<th>TEPC3</th>
<th>TEPC4</th>
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<td>3.8720</td>
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<td>0.9260</td>
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</tbody>
</table>

The result of plotting \( \text{TEPC} \) in (11) versus different numbers \( N \) of neurons is shown in Table 1 for the four different ECGs associated with normal ECG (TEPC1), WPW syndrome (TEPC2), hyperkalemia (TEPC3) and ventricular flutter (TEPC4).

The results for the TEPC1 versus \( N \) for the normal ECG in Table 1 are given in more detail in Fig. 8. In addition, the simulated abnormal ECG signals are shown in Figs. 9–11, corresponding respectively to WPW syndrome, hyperkalemia and ventricular flutter. (Note that in order to more clearly visualize the ventricular flutter of Fig. 11, the simulated ventricular flutter (for 1 s) is also shown in Fig. 12.) And for a long time evaluation of the waveform generator of the model (not the HRV generator), we measured the average \( \text{TEPC} \) (i.e., \( \text{TEPC} \)) in these four cases over 100 s when using 20 neurons. These results are given in Table 2.

Now for a comparison between the McSharry model and our proposed model, Figs. 13 and 14 give the plots of \( \text{TEPC} \) versus the number of neurons (over 100 s) for normal ECG and ECG associated with WPW syndrome. We can see that the McSharry model is better in reproducing the normal ECG but our proposed model is superior for WPW syndrome.

Now Fig. 15 shows the results for HR, HRV (RR-intervals) and the HRV power spectrum. We can
now observe the required VLF, LF and HF components (as mentioned in Section 2) in the HRV power spectrum of Fig. 15. Note that using the original Zeeman model will not give us any of the expected significant peaks in the power spectrum of HRV, and this means that it does not truly reflect real heart activity.

We also simulated (in Fig. 16) the cases of parasympathetic blockade, sympathetic-dominant and parasympathetic-dominant modulations—by appropriately changing the coupling factors \( c_1-c_4 \) in (6). In all cases, the simulated time trajectory of the ECG matched well with experimental recordings.

Finally, we would like to emphasize that the \( h(q) \) in (6) is very important in order to obtain a HRV signal with low or high random variations. It can also lead to quasi-periodic or completely periodic signals.

5. Conclusions and discussion

As regards heart operation, it is well-known (see Braunwald et al., 2004) that HRV is used to evaluate vagal and sympathetic influences on the sinus node and to identify patients at risk for a cardiovascular event or death.
Both frequency- and time-domain analysis help resolve parasympathetic and sympathetic influences, and RR-variability predicts all-cause mortality. High-frequency components of RR-interval variability reflect vagal activity. Reduced RR interval variability, the marker of increased risk, indicates loss or reduction of the physiological periodic sinus node fluctuations.

So the major contribution of this study is to present an improved model that is able to produce a more comprehensive simulation of a realistic ECG than is observed in practice. So, based upon the original Zeeman model of 1972a, we have proposed a new model to generate the heart-rate time-series. In the model, we included the effects of sympathetic and parasympathetic activities on the VLF, LF and HF components in the HRV power spectrum. We then showed that this new model did indeed actually produce these important spectral components in the simulated HRV. We also proposed a new neural network approach for modeling ECG signals, and have presented simulations and measured the error performance.

The model presented here has some important advantages over existing models. Compared to the original Zeeman model, our model has the improved ability to generate signals that better resemble those recorded in practice. And importantly, is its ability to dynamically model a much wider class of abnormal ECG’s, with a low simulation error. In addition to modeling the abnormalities associated with the shape of the ECG, we showed that our HRV model also has the capability of accurately simulating important sicknesses associated with the autonomic regularity of the heart rate. While we have emphasized our results for the abnormal ECG, in the case of the normal ECG the McSharry model still exhibits better a performance (but only for the waveform, not the HRV). Unlike most ECG generators, in our ECG model both the HRV and the shape of the ECG can be defined and simultaneously altered in a very simple and quick process. And the nonlinearity in the RBF neural network allows us to model the ECG accurately with a small number of neurons (i.e. few parameters). Additionally, there is no limitation to the actual shape of ECG waveform, such is the advantage of using neural networks. Note also that this model development process is fully real-time implementable and does not need to solve any inverse problems in order to obtain the RR-series.

There are however some limitations in our model which we should highlight. For example, we have ignored the effects of mechano-electrical feedback, emotions, hormones and intrinsic cardiac ganglia innervations that can affect the cardiac depolarization and repolarization processes that determine the actual waveform of ECG. It will be necessary to incorporate these effects into a more sophisticated model in a future study, as they can have an effect on the function $h(q)$ in (6).

The significance of the present study is to provide a better and much more efficient ECG model, which can be easily implemented in an ECG simulator. An ECG simulator has large varieties of potential application. For example, the output of an ECG simulator can be employed to assess biomedical-processing techniques, which are used to compute clinical statistics from the ECG. An artificially generated ECG that corresponds to an actual ECG signal is needed for the development, test and servicing (say calibration) of such equipment. A user settable ECG generator with ability to produce ECG signals with the morphology of a particular subject’s ECG and its associated power spectrum of RR intervals will show great application in also testing the effectiveness of such equipment.

Finally, while we have considered the sympathetic and parasympathetic activity separately in the proposed model, the way is now open to consider the coupling between these
effects in any future design, and the same principles for model development could possibly also be applied to other physiological signals and activities like blood pressure, respiration, etc.

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