A Biophysical Model of ECG Signals during Atrial Fibrillation Used to Evaluate the Performance of QRST Cancellation Algorithms

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Abstract

Characterization of electrical signals during atrial fibrillation (AF) is facilitated when the ventricular electrical activity (QRST complexes) has been suppressed. However, evaluating the performance of the QRST cancellation requires knowing the atrial activity during the QRST complex. A biophysically based model of the ECG during AF was developed, in which the exact separate contributions of the atria and the ventricles is available. Abnormal electrical propagation was simulated in a 3-D model of the human atria. The atrial electrical activity on the thorax was obtained by applying the boundary element method to a compartmental torso model. The ventricular activity was incorporated as a sequence of QRST complexes extracted from the clinical ECG of a patient in sinus rhythm. The ECG obtained as the sum of the atrial and ventricular activity described above may be used as a benchmark for testing and evaluating QRST cancellation and feature extraction techniques.

1. Introduction

The standard electrocardiogram (ECG) remains the most common non-invasive tool for diagnosing atrial fibrillation (AF). While the ECG provides a global, overall impression of the atrial activity, an unfortunate consequence of the spreading out of the electric currents generated by the active atrial cells is that the electrical image that appears on the body surface is blurred. Moreover, the (relatively much larger) signals arising from the electric activity of the ventricles are mixed with the atrial signals for a major part of the duration of a heart beat.

These complications led to the development of different signal processing tools aimed at isolating the contribution of the atria to the ECG signals during AF [1–3]. In order to validate these techniques, a common approach has been to apply the methods to synthetic signals obtained,

for example, by a superposition of sine waves [4], by combining rescaled P waves [5], or by concatenating segments of ECG signals extracted within the TQ interval [1].

In this paper, we advocate the use of a more sophisticated signal generation, based on an electro-anatomical model of the atria and the application of volume conduction theory to the simulation of electrograms and ECGs. This approach is illustrated by demonstrating how the performance of QRST cancellation techniques can be evaluated using a dedicated biophysical model of AF.

2. Methods

The synthetic ECG signals are obtained as the sum of the atrial and ventricular activity computed successively as follows.

2.1. Atrial activity

A three dimensional model of human atria was constructed from magnetic resonance (MR) images [6]. Figure 1A shows the resulting atrial geometry as seen from an anterior view (on the left) and from a posterior view (on the right). The major anatomical details are indicated: the tricuspid valve (TV), the mitral valve (MV), the inferior vena cava (IVC), the superior vena cava (SVC), and the pulmonary veins (PV), the sino-atrial node (SAN), the Bachman's bundle (BB), and the left atrium appendage (LAA). The electrical propagation of the cardiac impulse was simulated using a reaction-diffusion system (monodomain formulation) based on a detailed ionic model of the cell membrane kinetics, namely the Courtemanche *et al.* model [7]. In order to create a substrate for AF, patchy heterogeneities in action potential duration were introduced by modifying the local membrane properties [8]. Simulated AF was induced by rapid pacing in the left atrium appendage.

The boundary element method was applied to a compartmental torso model including the atria, the ventricles, blood cavities and the lungs, to compute the effect of vol-
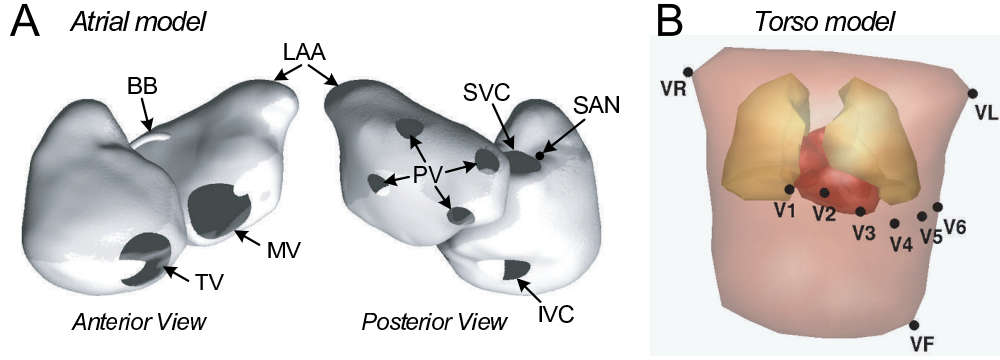
Figure 1. Geometry of the models representing the atria and the thorax.

ume conduction heterogeneity on the atrial contribution to the standard 12-lead ECG during the episode of simulated AF [8]. Figure 1B displays this torso geometry (nearly frontal view), also derived from MR images, as well as the position of the 9 ECG electrodes as in the standard 12-lead system: V1–V6 are the precordial leads; VR (right arm), VL (left arm) and VF (left foot) are the limb leads.

2.2. Ventricular activity

The ventricular electrical activity was incorporated as a sequence of QRST complexes extracted from a clinical ECG (5 minutes long) recorded during sinus rhythm in a 78-year-old patient with paroxysmal AF. Their positioning was inferred from the atrial activation times computed close to the atrioventricular (AV) node, using the model of human AV nodal function proposed by Jørgensen *et al.* [9].

This model, described in Table 1, predicts the output sequence of ventricular activations on a beat-to-beat basis given an input sequence of atrial activations. The AV node has a refractory period θ following the conduction of a cardiac impulse through the AV node to the ventricles [Eq. (3) in Table 1]. Any activation arriving at the AV node during this refractory period is blocked and prolongs the refractory period by Δ [Eq. (5)]. The conduction time through the AV node (AV delay) is assumed to be a function of the recovery time from the end of the preceding refractory period [Eq. (2) and (6)]. In total, 5 parameters are involved in the model. The values used were taken from [9]: $\theta = 114$ ms, $\Delta = 81$ ms, $AV_\infty = 70$ ms, $\alpha = 280$ ms, $\tau = 60$ ms.

The clinical QRST complexes were sorted according to the preceding RR interval. For each ventricular activation output by the Jørgensen model, a complex was selected in the database of clinical complexes according to the preceding *predicted* RR interval. The Q wave of each complex was aligned with the predicted onset of the ventricular activation.

2.3. Evaluation of QRST Cancellation

Since the exact contribution of the atria and the ventricles is available, the atrial electrical activity as obtained from the ECG after QRST cancellation can be compared to the true contribution of the atria. Then, any measure of similarity can be used to quantify the performances of the QRST cancellation technique, for instance the relative root mean square residual error. For the sake of illustration, the QRST cancellation method of Lemay *et al.* [2] is used in this paper.

Table 1.

Algorithm: *Prediction of the ventricular response* [9]

Input: A_k = activation times at AV node, $k = 1, 2, \ldots$

Output: $C_k = 1$ if a ventricular beat is initiated by stimulus k, and 0 otherwise.
V_k = timing of the onset of the ventricular activation (if $C_k = 1$).

Parameters: θ, Δ, AV_∞, α, τ

Variables: R_k = end of the refractory period

Initialization: $R_1 = -\infty$

Loop: **for** $k = 1, 2, \ldots$ **do**
    if $A_k > R_k$ **then** (conduction)

$$C_k = 1 \tag{1}$$
$$V_k = A_k + f(A_k - R_k) \tag{2}$$
$$R_{k+1} = V_k + \theta \tag{3}$$

    else (block)

$$C_k = 0 \tag{4}$$
$$R_{k+1} = R_k + \Delta \tag{5}$$

Function: $\quad f(RA) = AV_\infty + \alpha \exp(-RA/\tau) \tag{6}$

Figure 2. Construction of the synthetic ECG (lead V1): (A) Transmembrane potential computed close to the AV node; (B) reconstructed ventricular activity; (C) corresponding simulated AF activity; (D) complete ECG, *i.e.* sum of signals B and C; (E) example of a clinical ECG (also lead V1) during AF.
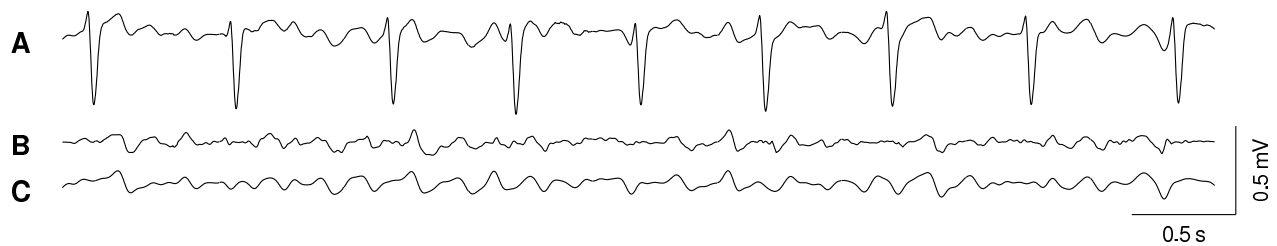

Figure 3. Example of QRST cancellation: (A) Synthetic ECG, lead V1; (B) atrial activity after using the QRST cancellation algorithm. (C) corresponding simulated AF activity, lead V1 (as in Fig. 2C);

3. Results

Figures 2 illustrates the successive steps to generate a synthetic ECG during AF. Figure 2A shows the transmembrane potential at a node close to the anatomical location of the AV node computed during simulated AF. Based on the activation sequence extracted from this signal, the 12-lead ECG signals representing the ventricular activity were assembled (Fig. 2B). The synthetic ECG (Fig. 2D) was obtained by summing the ventricular electrical activity (Fig. 2B) and the atrial electrical activity (Fig. 2C).

The mean RR interval ($\pm$ standard deviation) of the synthetic ECG is 631 ± 97 ms, and is consistent with the value 669 ± 105 ms measured in the patient (in AF) whose ECG was used by Jørgensen *et al.* [9] to estimate the parameters of the AV node model. In addition, both frequency analysis of the atrial activity and inspection of the local atrial cycle length showed a dominant frequency around 6.7 Hz. This value lays within the clinical range of 5.6 ± 1.1 Hz (min 3.1, max 8.1 Hz) measured in 31 patients with AF (lead V1 after QRST cancellation). Figure 2E displays a clinical ECG recorded during AF and demonstrates that the computed ECG has the major features of clinical ECGs.

Figure 3 illustrates the evaluation of the quality of the QRST cancellation. The ECG signal after QRST cancellation is represented on Fig. 3B and can be compared to the true atrial activity (Fig. 3C). For a detailed analysis of the performance of this QRST cancellation technique when applied to the synthetic ECG signals described in this paper, see Lemay *et al.* [2].

4. Discussion and conclusions

We have developed a tool to generate synthetic ECGs similar to those recorded during AF and in which the exact contribution of the atria is available. The resulting signals

were found to be suitable for evaluating, comparing and validating QRST cancellation techniques.

The atrial activity was derived from a biophysical model of the human atria describing the current sources within the myocardium. An important advantage of this approach is that different substrates and different volume conductor models can be simulated, leading to different F-wave morphologies. In addition, ECG signals can be generated for any lead system. However, due to the high computational load required by the large scale simulation (around one million nodes), the signal length is usually limited (20 s in this paper). Longer signals can be constructed by successive repetition of this 20-s signal. Provided that the QRST complex database is large enough, the resulting signal will not be periodic and may include a sufficient number of different types of QRST complexes for an accurate QRST template construction.

The positioning of the QRST complexes could have been selected by means of an independent random process, like in [5]. ECGs recorded in a patient in sinus rhythm (with the P waves removed) could also have been used directly. The approach presented here relies on a model of the AV node and takes into account the physiological dependency between the atrial and the ventricular activity, which may be critical to test the validity of algorithms assuming the *independence* of these signals, such as blind source separation [3].

Signal processing has always been a important tool for analyzing the outcome of computer models and comparing them with experimental data. This paper shows that computer modeling can assist in developping and validating efficient signal processing techniques.

Acknowledgements

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