Effect of Respiration on the Solutions of Forward and Inverse Electrocardiographic Problems - a Simulation Study

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Abstract

The forward problem of electrocardiography aims at obtaining a better understanding of cardiac electrophysiological activities, by means of computer modeling and simulation. Whereas, the inverse electrocardiographic problem provides a direct insight of electrical sources into the heart without interventional procedures. Nowadays, the forward and inverse problems are mostly solved in static models, which do not take into account heart motion and respiration. Besides heart motion, neglecting respiration may also lead to remarkable uncertainties in both forward and inverse solutions. In the present work a dynamic lung model is developed. With this model the effect of respiration on the forward and inverse solutions is studied.

1. Introduction

Forward and inverse problems of electrocardiography are most often investigated in static thorax models, which are usually built from MRI or CT data-sets with the heart in diastolic state and the lungs in deflated state. Thus, modeling errors are introduced by neglecting the dynamics of human body, which can cause inaccuracy in forward simulation and instability in inverse solution. The effect of heart motion on the forward and inverse problems has been analyzed in [1]. The present study then focuses on respiration. A dynamic lung model is developed to study the influence of respiration on simulated ECG and inverse reconstructions. Tikhonov 0-order regularization and GMRES method are applied to solve the inverse problem for epicardial potentials in both dynamic and static models.

2. Methods

2.1. Anatomical model

In this study the Visible Human data-set is deployed [2]. The highly detailed image data are segmented and classified to more than 40 tissue classes and converted to a voxel-based model with a resolution of $2mm \times 2mm \times 2mm$. The excitation conduction system including bundle branches, fascicles and Purkinje fibers as well as the fiber orientation are built in the ventricles. From the voxel-based model a tetrahedron mesh is generated and applied in the forward and inverse computations (see Fig. 1) [3].

The volume change of lungs is implemented through mesh deformation in the tetrahedron model. The nodes that belong to lungs are shifted appropriately to reproduce the inflation and deflation of lungs. 1000 states with a step of $4ms$ during a simulated respiratory cycle, i.e., starting from the deflated state, $1.5s$ for inspiration, $1.5s$ for expiration and at last $1s$ in rest, are created. Accompanying the volume change, the variation of conductivity of lungs during inspiration and expiration is also included [4–6]. The lungs in the deflated and inflated states are shown in Fig. 1.

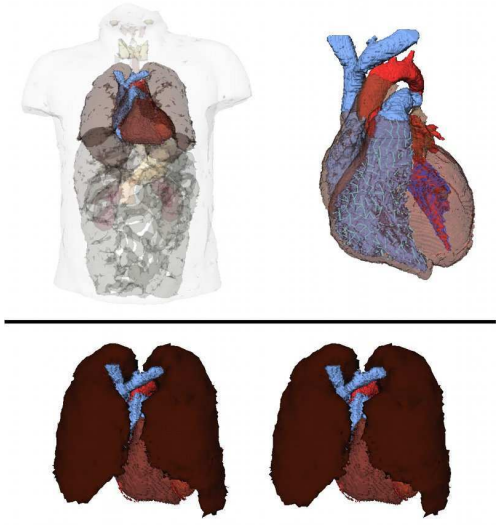

Figure 1. Visible Human thorax model (upper left), the heart model (upper right) and the lungs in the deflated state (bottom left) and in the inflated state (bottom right).

2.2. Cellular automaton

A rule-based cellular automaton is utilized to simulate the excitation propagation in the heart [7]. A voxel in myocardium can be activated by the adjacent excited voxels. After activation the state transition of this element, i.e., change of transmembrane voltage, complies with the action potential curves, which are extracted from cardiac cell model [8]. The transmural heterogeneity of myocardium is also considered. The simulation is conducted in the voxel-based heart model with a spatial resolution of $1mm \times 1mm \times 1mm$ with a time step of $4ms$.

2.3. Forward problem

The distributions of the transmembrane voltage during the cardiac cycle obtained from the cellular automaton are interpolated onto the correspondent nodes in the tetrahedron mesh. The potential distributions on the body surface are calculated by solving the bidomain equation

$$\nabla \cdot ((\sigma_i + \sigma_e)\nabla\Phi_e) = -\nabla \cdot (\sigma_i \nabla V_m) \tag{1}$$

incorporating the finite element method, where σ_i and σ_e are the intracellular and extracellular conductivity tensors; Φ_e and V_m are extracellular potentials and transmembrane voltages, respectively.

2.4. Inverse problem

In this study epicardial potentials are selected as source model. In this case the inverse problem is formulated as

$$\mathbf{Ax} = \mathbf{y}, \tag{2}$$

where $\mathbf{x}$ is the source vector (potentials on the heart surface), $\mathbf{y}$ is the measurement vector (multi-channel ECG) and $\mathbf{A}$ is called transfer matrix, which describes the relation between sources and measurement signals.

Because of the ill-posedness of the inverse problem of electrocardiography regularization techniques have to be employed in order to improve the stability of the inverse solution.

2.4.1. Tikhonov regularization

Tikhonov regularization is a classical method to solve the inverse problem [9]. It uses general information about the solution for constraints besides the $\ell 2$-norm residual. The following minimization problem is considered:

$$x_\lambda = \arg\min_x (\|\mathbf{y} - \mathbf{Ax}\|_2^2 + \lambda^2 \|\mathbf{Lx}\|_2^2), \tag{3}$$

where the regularization operator $\mathbf{L}$ is the identity matrix $\mathbf{I}$ in the case of 0-order regularization, and λ indicates the regularization parameter, controlling the weight attributed to the constraint condition $\|\mathbf{Lx}\|_2^2$. The optimal λ is determined by using the criterion of "L-curve" [10].

2.4.2. Generalized minimal residual method

GMRES method is an iterative regularization method in the category of Krylov subspace projection methods. In the iteration process the solution is sought, that satisfies

$$\mathbf{x_j} = \arg \min_{\mathbf{x} \in \mathbf{K_j}(\mathbf{A'}, \mathbf{b})} \|\mathbf{b} - \mathbf{A'} \cdot \mathbf{x}\| \tag{4}$$

where $\mathbf{K_j}$ denotes the j-th Krylov subspace, $\mathbf{A'} = \mathbf{A^T} \cdot \mathbf{A}$ and $\mathbf{b} = \mathbf{A^T} \cdot \mathbf{y}$ [11, 12].

3. Results

3.1. Forward simulation

For comparison purposes, two forward simulations are performed, without and with respiration. A respiratory cycle of $4s$ is simulated, which includes 4 normal cardiac cycles with a duration of $1s$ each. The respiration starts from the deflated state with the minimal volume of lungs. Then the lung volume reaches its maximum after $1.5s$ of inspiration. After that the expiration starts and lasts for the subsequent $1.5s$. At the end the lungs go into a resting phase for $1s$, in which the lungs are kept deflated constantly. The conductivity of lungs is set at $0.20279 S/m$ for the deflated state and $0.038904 S/m$ for the inflated state [4–6]. The conductivity values for the states in between are obtained by linear interpolation. Please note that no heart motion is considered in the present study.

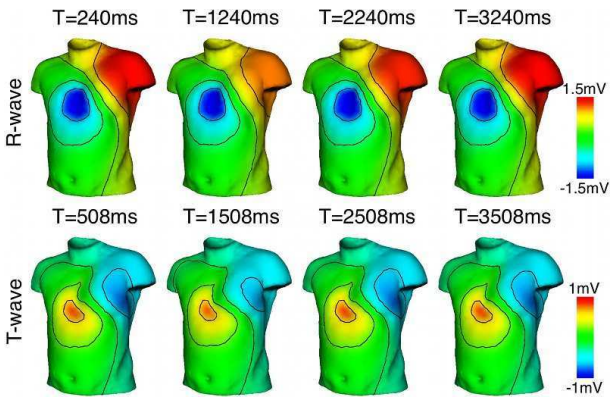

Figure 2. The BSPMs simulated with respiration at R-wave (upper) and T-wave (bottom) in different cardiac cycle during one respiratory cycle.

The simulated BSPMs with respiration are shown in Fig. 2. Because in the forth cardiac cycle the lungs are in the resting phase, which is in the same condition as the simulation without respiration, the simulation made with respiration in this cardiac cycle can be considered as the one made without respiration. In addition, 3 selected standard ECG leads for both simulations without and with respiration are shown in Fig. 3.

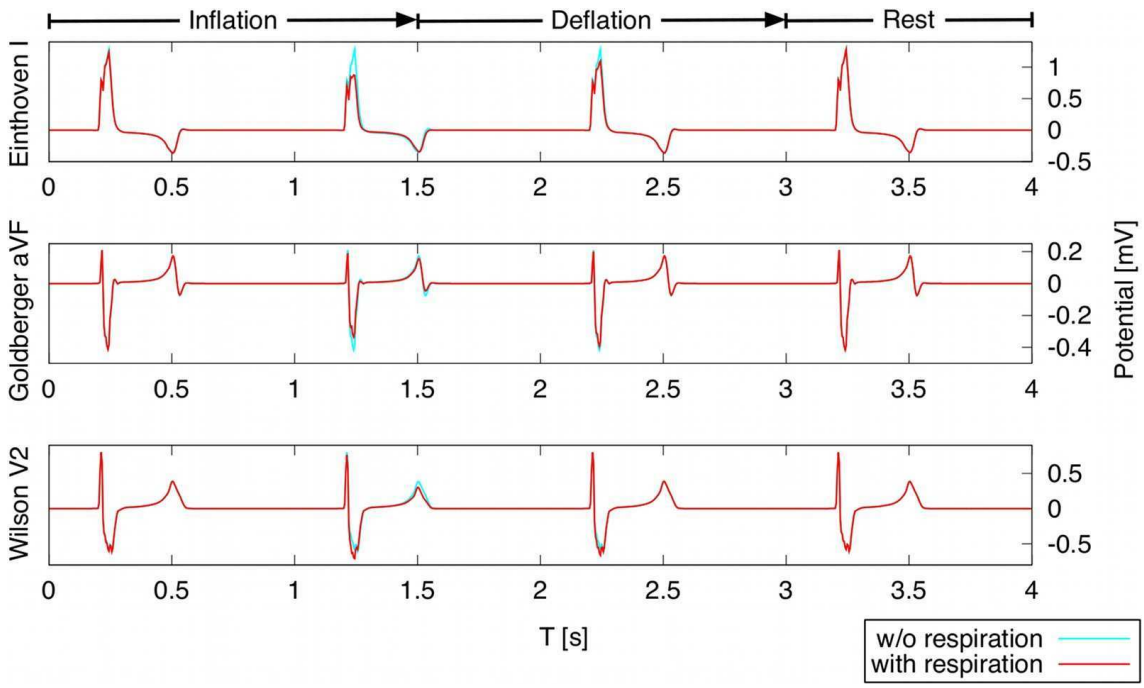

Figure 3. 3 standard ECG leads derived from the forward solutions made without and with respiration during a respiratory cycle.

3.2. Inverse solution

In order to investigate the influence of the modeling error introduced by neglecting respiration on the inverse solutions, two inverse problems are solved. In the first case, a 64 channel ECG extracted from the forward simulation made without respiration is taken as input signal and the transfer matrix is calculated using the same static model, i.e., no modeling error is introduced into the system. In the second case, the ECG is simulated using the dynamic model including respiration, but the transfer matrix is still taken from the static model as in the first case, i.e., a modeling error caused by neglecting respiration is involved in the system. The epicardial potentials are reconstructed using the Tikhonov 0-order and GMRES for both cases. The reconstructions and the simulated epicardial potentials (reference) are presented in Fig. 4. The correlations between the inverse solutions and the reference, which reflect the quality of the reconstruction, are plotted in Fig. 5.

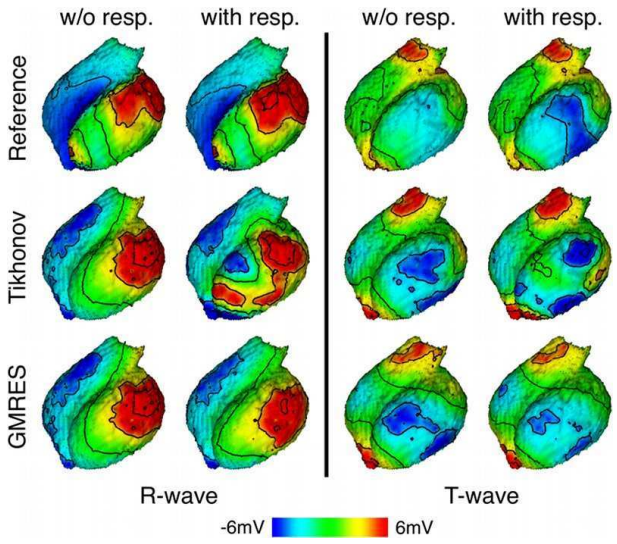

Figure 4. Comparison between the reconstructed distributions of epicardial potentials without (columns 1 and 3) and with (columns 2 and 4) respiration using Tikhonov 0-order regularization (row 2) and the GMRES method (row 3) at R-wave ($T = 1240ms$) and T-wave ($T = 1508ms$). The simulation as reference is shown in row 1.

4. Discussion and conclusions

From the BSPMs (see Fig. 2) and the ECGs (see Fig. 3), noticeable difference can only be seen in the second cardiac cycle, where the lungs approach the maximum in volume and the minimum in conductivity. Moreover, the variations caused by respiration are mainly in the signal amplitude, whereas the patterns of BSPM and ECG do not show significant change. Nevertheless, respiration should be taken into account when high accuracy in ECG simulation is desired.

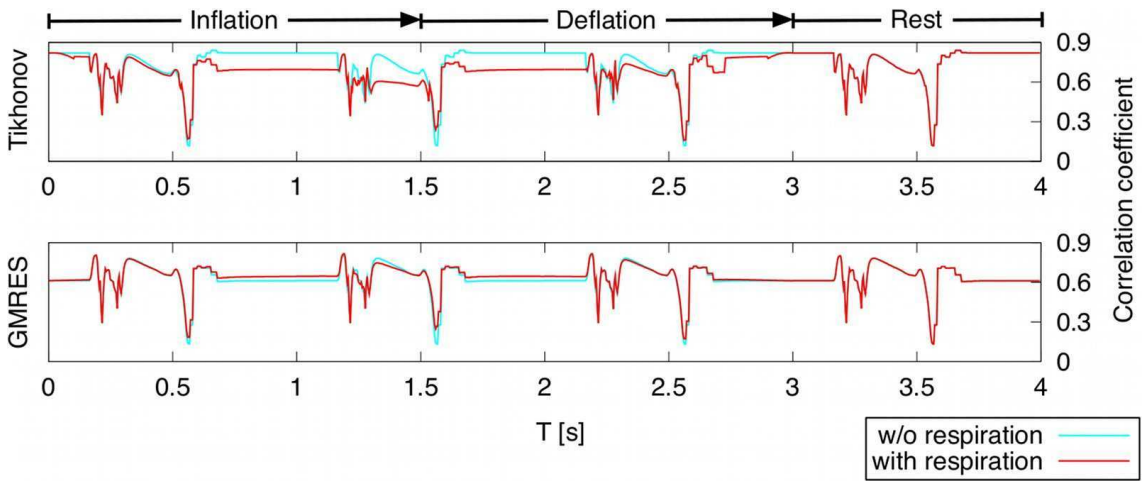

Figure 5. Correlation between the simulated reference and the inverse solutions obtained without and with respiration using Tikhonov 0-order regularization (upper) and the GMRES method (bottom).

From the point of view of inverse problem, respiration has non-negligible effect on inverse solutions. The reconstructions using Tikhonov 0-order without modeling error (introduced by neglecting respiration) are considerably better than those obtained with modeling error (see Fig. 4) and an improvement of up to 0.15 in correlation coefficient during the second cardiac cycle is shown in Fig. 5. Although Krylov subspace methods like the GMRES method are relatively stable against modeling errors, the inverse solutions can still be improved if the modeling error from disregarding respiration is eliminated (see Fig. 4 and Fig. 5). Therefore, it is suggested to incorporate the respiration into the inverse calculation according to the results in the present study.

As the next step, it is of interest to combine heart motion and respiration to create a reality-like environment for the investigation of forward and inverse electrocardiographic problems. More regularization methods and source models, e.g., MAP-based regularization and activation time reconstruction, will be tested. Furthermore, a more detailed realistic dynamic model, which will be built based on 4D MRI data-set, is planned in further work.

References

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