Computing Ischemic Regions in the Heart: On the Use of Internal Electrodes

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Abstract

In order to better locate ischemic regions in the heart using electrical measurements and inverse solutions, we explore the possibility for supplementing BSPM data sets with additional internal electrodes in the esophagus. We investigated whether such internal electrodes closer to the heart's surface could significantly improve the ability to pinpoint ischemic regions. A framework based on exercise ECG testing and a mathematical model for identifying ischemic regions from ECG measurements was implemented to test the effect of potential internal electrodes. This method identifies areas with abnormal perfusion by minimizing the difference between recorded and simulated ECGs. To investigate the effect of the extra electrodes in the esophagus, we computed the location of the ischemic zones with and without the internal electrodes for both synthetic data and using clinically obtained BSPMs. Computations based on pure synthetic data illuminate that, if an ischemic region is close to an electrode in the esophagus, then the use of internal electrodes might improve the result significantly. However, the simulations also indicate that ischemic areas further away from the internal electrodes are not better recovered with the use of such additional ECGs. This study indicates that the use internal electrodes, along with standard BSPMs, might improve the accuracy of the inverse ECG technology.

1. Introduction

Ischemia, the reduction of local blood perfusion, can often lead to damage or dysfunction when it occurs in the rapidly beating myocardium. Accurately determining the extent and location of ischemia is important for effectively planning treatment. Although techniques such as scintigraphy can provide information on cardiac perfusion, improving methods that do not rely on radioisotopes could give better diagnostic value. As electrical measurements collected on the body surface arise due to the underlying cardiac function, methods have been developed that can give reconstructions of the electrical activity of the heart using inverse solutions [1–3]. In order to improve these reconstructions, with the advent of more routine transesophageal procedures, the idea of using ECG leads within the esophagus may be possible. The close proximity to the heart could provide measurements that could allow greater precision from using inverse solutions to detect ischemia.

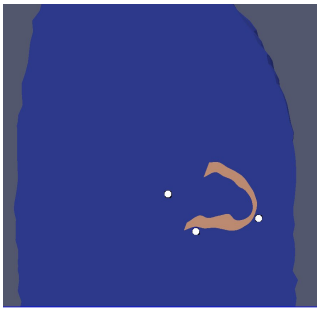

Figure 1. A 2D slice (sagittal view) of the 3D geometry that is used in this study. The three white dots indicate the position of the potential esophagus electrodes while the heart muscle is visualised in brown.

2. Mathematical framework

Let H denote the domain occupied by the heart and consider the stationary model (a simplification of the bidomain model [4, 5])

$$\nabla \cdot ((M_i + M_e)\nabla u) = -\nabla \cdot (M_i \nabla v) \quad \text{in } H, \qquad (1)$$

where M_i and M_e are the intra- and extra-cellular conductivities tensors, whereas v and u represent the transmembrane and extracellular potentials, respectively. In general, both v and u are unknown functions, but according to lab measurements, the transmembrane potential v can

be approximated by certian constant values during the PR[1] and ST[2] segments of the the heart cycle. Furthermore, during these two time segments, the values for v depend upon whether a supply/demand imbalance does occur, i.e. whether ischemic tissue is present or not [6]. Typical values for v during relaxation and at maximum workload are given in Table 1 and in Table 2, respectively. Below, we let D denote the ischemic region in the heart.

Table 1. Approximate values for v before exercise.

Segment	Potential
PR	$v(x,t_1) \approx -96mV, \quad x \in H$
ST	$v(x,t_2) \approx 0mV, \quad x \in H$

Table 2. Approximate values for v at maxmium workload.

Segment	Potential
PR	$v(x,t_3) \approx \begin{cases} -96mV, & x \in H \setminus D \\ -60mV, & x \in D \end{cases}$
ST	$v(x,t_4) \approx \begin{cases} 0mV, & x \in H \setminus D \\ -20mV, & x \in D \end{cases}$

Since myocardial ischemia results whenever there is a transient imbalance between coronary blood flow and myocardial work, we are interested in an approximation for the shift h in the transmembrane potential, i.e. the transmebrane potentials at maximum workload *relative* to the potentials during relaxation

$$h(x) = \underbrace{\overbrace{v(x,t_4)}^{\text{ST}} - \overbrace{v(x,t_3)}^{\text{PR}}}_{\text{maximum workload}} - \big[\underbrace{\overbrace{v(x,t_2)}^{\text{ST}} - \overbrace{v(x,t_1)}^{\text{PR}}}_{\text{relaxation}}\big] \quad (2)$$

The associated shift r in the extracellular potential u is defined as

$$r(x) = \underbrace{\overbrace{u(x,t_4)}^{\text{ST}} - \overbrace{u(x,t_3)}^{\text{PR}}}_{\text{maximum workload}} - \big[\underbrace{\overbrace{u(x,t_2)}^{\text{ST}} - \overbrace{u(x,t_1)}^{\text{PR}}}_{\text{relaxation}}\big]. \quad (3)$$

Since (1) must hold for all t, and thereby for $t = \{t_1, t_2, t_3, t_4\}$ for all $x \in H$, it follows from (1), (2) and (3) that

$$\nabla \cdot ((M_i + M_e)\nabla r) = -\nabla \cdot (M_i \nabla h) \quad \text{in } H. \quad (4)$$

Further, by using (2) and the values in Table 1 and Table 2, an approximation for h is given as

$$h(x) \approx \begin{cases} 0mV, & x \in H \setminus D \\ -56mV, & x \in D. \end{cases} \quad (5)$$

[1]The PR segment is assumed to occur 1-25 ms before onset of the QRS complex.

[2]The ST segment is measured 60-80 ms after the end of the QRS complex.

In this study, the electrical potentials are recorded on the surface of the body and in the esophagus. Therefore, an equation representing r also outside the heart must be included in our model. Outside the heart there are no sources, and thus r is governed by a standard homogeneous potential equation:

$$\nabla \cdot ((M_o \nabla r) = 0 \quad \text{in } T, \quad (6)$$

where M_o represents the conductivity in T, and T is defined as the domain surrounding the heart H. Details concerning suitable interface and boundary conditions for model (4) and (6) can be found in [1].

2.1. Inverse solution

The approximate recovery of h from ECG data d is accomplished by dividing the left ventricle into 60 subunits and assigning a basis function to each of these units:

$$N_1(x),\ N_2(x), \ldots,\ N_{60}(x),$$

where

$$N_j(x) \approx \begin{cases} 0\text{mV}, & x \text{ outside subunit } j, \\ -56\text{mV}, & x \text{ inside subunit } j, \end{cases} \quad (7)$$

for $j = 1, 2, \ldots, 60$. In this paper we have only studied subendocardial ischemic regions. Therefore, the support of N_j was restricted from endocardium and 3/5 of the endocardium-epicardium distance into the heart wall.

The shift in the transmembrane potential was discretized by putting

$$h(x) = \sum_{j=1}^{60} p_j N_j(x). \quad (8)$$

Our scheme for identifying ischemic zones is based on the output least squares approach. More specifically, assuming that we have e electrodes, we suggest to recover such regions by minimizing the deviation between the ECG data $d = (d_1, d_2, \ldots, d_e)$ and the simulated ST shift on the body surface and in the esophagus, i.e.

$$\min_{p_1, p_2, \ldots, p_{60}} \frac{1}{2} \left\{ \sum_{k=1}^{e} [r(y_k) - d_k]^2 + \alpha \sum_{j=1}^{60} p_j^2 \right\} \quad (9)$$

subject to

$$\nabla \cdot ((M_i + M_e)\nabla r) = -\nabla \cdot (M_i \sum_{j=1}^{60} p_j \nabla N_j) \quad \text{in } H, \quad (10)$$

$$\nabla \cdot ((M_o \nabla r) = 0 \quad \text{in } T, \quad (11)$$

$$0 \le p_j \le 1 \quad \text{for } j = 1, 2, \ldots, 60. \quad (12)$$

Here, $\alpha > 0$ is a regularization parameter and $y_1, y_2, \ldots, y_e$ are the positions of the electrodes.

2.2. Simulations

A description for genereting patient specific 3D grids can be found in [3]. Using these grids, we performed computations based on *pure synthetic data* and involving a combination of *clinical and synthetic data*. For both cases, synthetic ECG data d has to be generated at points where clinicians indicated that esophageal probes could be located (Figure 1). This synthetic data is created by first defining an ischemic region D in (5). The model (4)-(6) is thereafter used to calculate the shift r. To make the examples realistic and to reduce the effect of inverse crimes, these "measurements" can not be used directly in (9), but must be corrupted by some sort of noise:

- In examples 1-2 a set of small random numbers $n_1, n_2, \ldots, n_e$ is generated, and the ECG data is produced by $d_k = r(y_k) + n_k$, for $k = 1, 2, \ldots, e$.
- In example 3, clinical BSPMs recorded at Oslo University Hospital (typically 72 measurements) are used. However, for the three esophagus electrodes such clinical data is not available. Therefor the ECG data is given by

$$d_k = \begin{cases} r(y_k + m_k), & k = 1, 2, 3 \\ BSPM_k, & k = 4, 5, \ldots, e \end{cases} \tag{13}$$

where m_1, m_2 and m_3 are vectors with small norm, i.e. $y_1 + m_1, y_2 + m_2$ and $y_3 + m_3$ are small perturbations of y_1, y_2 and y_3, respectively. To produce realistic esophagus measurements, D is chosen as the hashed region in Figure 4 (a) when solving (4)-(6).

3. Results

Using purely synthetic data, our results indicate that if there is ischemia near the location of a transesophageal ECG probe, having internal electrical information could significantly improve the resolution of the resulting inverse solution (Figure 2). However, if the area of ischemia is located far from the lead, this effect may be minimal (Figure 3).

When tested against a combination of clinical data with synthetic esophageal ECG data, our simulations qualitatively demonstrate a better resolution of ischemia with the use of internal electrodes, when compared to scintigraphy results (Figure 4).

4. Conclusions

While further study is needed to determine whether these synthetic studies will carry over to experimental or clinical results, our initial simulations indicate that there could be some benefit of internal electrodes in reconstructing the electrical activity of the heart by inverse solutions. Such internal electrodes are becoming possible through more spread use of transesophageal imaging, and electrodes placed through the esophagus could be quite close to the heart and improve on the signal from any ischemia. Not surprisingly, the ability to improve resolution of ischemia diminishes the further the ischemia is away from the internal electrodes.

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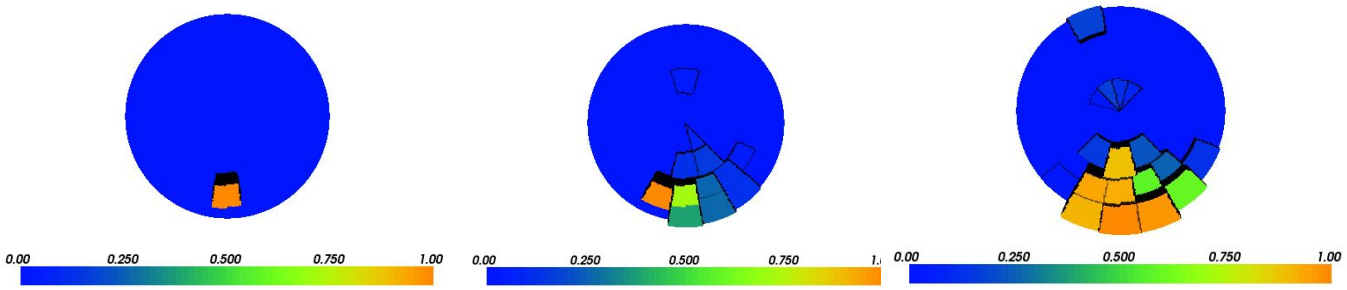
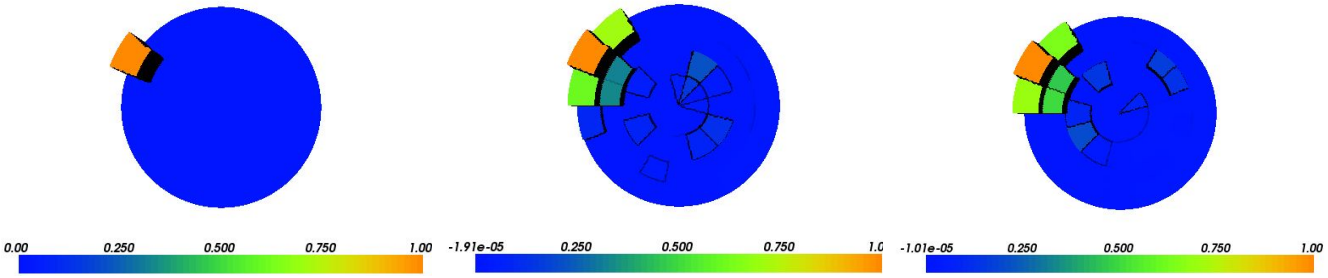


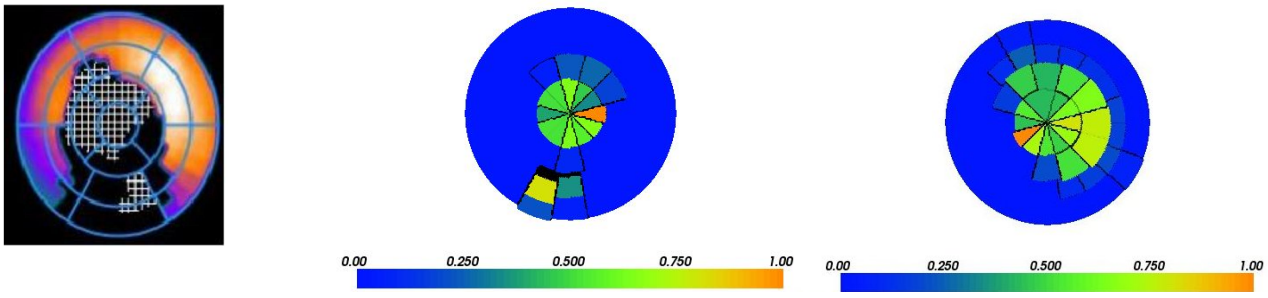
(a) Ischemic region in the inferior wall (b) Result using BSPMs + esophagus electrodes (c) Result using only BSPMs

Figure 2. The inverse problem was solved using synthetic BSPM and synthetic esophagus measurements. The synthetic ECG data d contains 1.3 % noise. Please note that the ischemic region is relatively close to an esophagus electrode in this example. By comparing (b) and (c) with (a) we see that including esophagus electrodes clearly improve the result with respect to size.



(a) Ischemic region in the anterior/septum wall (b) Result using BSPMs + esophagus electrodes (c) Result using only BSPMs

Figure 3. The inverse problem was solved using synthetic BSPM and synthetic esophagus measurements. The synthetic ECG data d contains 1.6 % noise. Please note that the ischemic region is relatively far away from an esophagus electrode in this example. By comparing (b) and (c) with (a) we see that including esophagus electrodes do not improve the result neither with respect to position or size.



(a) SPEC myocardial scintigraphy (b) Result using BSPMs + esophagus electrodes (c) Result using only BSPMs

Figure 4. The inverse problem was solved using clinical BSPMs recorded at Oslo University Hospital and synthetic esophagus measurements. By comparing (b) and (c) with the hashed region in (a) this test indicates that the use of internal electrodes, along with standard BSPMs, might improve the accuracy of the inverse ECG technology.