The Effects of Inhomogeneities and Anisotropies on Electrocardiographic Fields: A Three-Dimensional Finite Element Study

Ruth Nicholson Klepfer
Department of Bioengineering
rklepfer@cs.utah.edu

Christopher R. Johnson
Department of Computer Science
crj@cs.utah.edu

Robert S. MacLeod
CVRTI
macleod@cvrti.utah.edu

University of Utah, Salt Lake City, UT 84112

Abstract

In this paper we provide quantitative information on the effects of selected inhomogeneities and anisotropies on the finite element solution of the forward electrocardiographic field problem. The geometry of the model is based on the Utah Torso model and includes epicardial fatpads, cardiac chambers, major arteries and veins, sternum, ribs, spine and clavicles. Experimentally measured epicardial potentials were used for the electrical source and solutions were computed using the finite element (FE) method. Simulations were performed varying the conductivities assigned to the different structures in the model including anisotropic configurations of the skeletal muscle. Results showed the anisotropic skeletal muscle had a significant impact on the distribution of electrocardiographic fields with an average of 13% percent difference from the homogeneous isotropic case.

Introduction

Finite element models have increasingly been used to study ECG fields, pacing and defibrillation [1, 2]. The complexity of most physiological systems far outstrips current modeling capabilities. As such, models used to study electrocardiology must be limited to a subset of thoracic tissues. Currently, little quantitative data is available on the effects of including or excluding a tissue's conductivity from a realistic model. This study provides quantitative data on the effects of inhomogeneities and anisotropies on computed body surface potential maps (BSPMs) from a large-scale model of the human thorax.

Methods

Problem Formulation The electric fields produced in the human thorax can be mathematically described by Maxwell's equations, which in the quasistatic limit can be reduced to Laplace's equation

$$\nabla \cdot \sigma(\nabla \Phi) = 0 \text{ in } \Omega. \tag{1}$$

Here, σ is the conductivity tensor, Φ is the unknown vector of potentials, and Ω is the solution domain. Solving the forward ECG problem involves calculating the potential and current density distributions throughout the torso given the potentials on the epicardium. This requires solving (1) with boundary conditions $\Phi = \Phi_0$ on Γ_H and $(\sigma \nabla \Phi) \cdot \mathbf{n} = 0$ on Γ_T, where Γ_H is the epicardium, Γ_T is the torso surface and $(\sigma \nabla \Phi) \cdot \mathbf{n}$ is the current flux normal to Γ_T. The FE method was used to approximate the potentials and current density distributions.

Geometry The Utah Torso model contains detailed anatomical structures of bone, including the sternum, 12 ribs on both sides, the spine and the left and right clavicle. The blood vessels in the vicinity of the heart are also modeled including the ascending and descending aorta, the pulmonary arteries and veins, the superior and inferior vena cava, the innominate and azygous veins and the left and right subclavian veins. The left and right ventricles and atria are specified, allowing separate assignment of conductivities for the myocardium and the blood filled chambers of the heart. The model also includes lungs, a skeletal muscle region and a subcutaneous fat region. The full computational model consists of a mesh of approximately 1 million tetrahedra (168,000 nodes).

Simulations Two sets of simulations were performed, all with the same boundary conditions of experimentally measured epicardial potentials. The first simulations measured the effect on computed BSPMs of adding a single inhomogeneity to a homogeneous, isotropic version of the Utah Torso Model. In the homogeneous isotropic control, every region in the model was assigned the same conductivity tensor with equal values in all directions. For each simulation in this set, the conductivity of a single region or group of regions was assigned according to values cited in the literature for the applicable tissue [3]. The skeletal muscle was modeled both as an isotropic and anisotropic region. In modeling the anisotropy, the skeletal muscle region was divided into 12 equal subregions of 30 degrees of rotation. The conductivity tensor was constructed with a higher conductivity value perpendicular to the bisector of the 30 degree subregion and a smaller value in the radial direction. The anisotropy is described by the ratio of the circumferential to radial conductivity. Ratios of 3:1, 7:1, 10:1 and 15:1 were simulated. The 7:1 ratio approximates

Time (ms)	159	167	175	183	Ave.
Iso	0.00%	0.00%	0.00%	0.00%	**0.00%**
Aniso 3:1	7.89%	10.80%	17.40%	10.66%	**11.69%**
Aniso 7:1	8.36%	9.04%	17.35%	18.51%	**13.3%**
Aniso 10:1	9.12%	9.64%	20.14%	24.08%	**15.74%**
Aniso 15:1	10.62%	12.59%	26.08%	31.54%	**20.21%**
Fat	8.06%	12.62%	15.08%	10.41%	**11.54%**
Bones	0.10%	0.36%	0.49%	0.37%	**0.33%**
Lungs	2.17%	6.76%	10.12%	19.66%	**9.68%**
Heart	0.41%	0.99%	6.42%	11.46%	**4.82%**
Myc.	0.55%	0.20%	1.76%	3.15%	**1.42%**
Fatpads	0.50%	3.62%	4.85%	7.30%	**4.07%**
Vessels	0.64%	1.52%	6.48%	15.52%	**6.04%**

Table 1: Percent RE between BSPMs of the homogeneous isotropic case and adding the inhomogeneity listed for selected times during the cardiac cycle.

the values closest to those reported in the literature [3].

The second set of simulations measured the effect of removing one inhomogeneity from an inhomogeneous, anisotropic model. The 'control' simulation had every region in the model assigned its specific conductivity tensor with skeletal muscle modeled at a 7:1 anisotropy ratio. In each simulation, then, one region or group of regions was assigned the homogeneous isotropic conductivity.

Analysis Potential and current density were computed at each node throughout the torso. To compare the results of different scenarios, BSPMs were produced from the potentials calculated for the torso surface. Correlation coefficient (CC), root mean squared error (RMS) and the relative error (RE) were calculated for each simulation.

Results and Discussion

Table 1 shows the % RE in BSPMs found for the simulations where a single inhomogeneity was added to a homogeneous isotropic torso. From the table, it appears that the bones and the isotropic representation of skeletal muscle add little information to the solution. The isotropic skeletal muscle was nearly identical to the homogeneous case with a difference in RE of less than .001%. Our results show anisotropic skeletal muscle having the most significant average impact on the BSPM with the size of the % RE increasing with an increasing anisotropy ratio. In general, the structures closer to the torso surface tended to have a greater impact on the BSPM.

Table 2 shows the % RE in BSPM's between the full model and the full model without the inhomogeneity listed. The trend seen in the first set of simulations' results does not occur in Table 2. In this table, we see the structures closest to the heart (including the heart itself) have a larger impact on the BSPM than regions closer to the torso surface such as the skeletal muscle and the subcutaneous fat. Unlike the Table 1 results, the bones had a relatively large % RE when taken out of the full model. It is noteworthy that the range of values for this table is much smaller than in Table 2 with all but two of the averages falling within 2% of one another.

Time (ms)	159	167	175	183	Ave.
Muscle	7.41%	11.87%	18.61%	13.21%	**12.8%**
Fat	8.41%	12.03%	23.74%	32.14%	**19.1%**
Bones	18.37%	22.32%	36.52%	34.12%	**27.8%**
Lungs	16.53%	22.96%	36.01%	30.82%	**26.6%**
Heart	19.42%	23.06%	36.18%	32.37%	**27.8%**
H Chmbrs	19.60%	22.88%	36.43%	32.21%	**27.8%**
Fatpads	18.09%	21.09%	35.37%	34.49%	**27.3%**
Vessels	20.18%	22.96%	36.69%	34.05%	**28.5%**

Table 2: Percent RE between BSPMs of full model and the full model without the inhomogeneity listed.

The results of the first set of simulations should be analyzed further to determine if any significant localized effects might be occuring in the vicinity of the heart that are not apparent when considering the full torso surface. The difference in the trends of the two sets of simulations may indicate that the homogeneous isotropic conductivity is a poor representation of the full model. Additional research is necessary to examine this issue, as well as how the specific implementation of the skeletal muscle anisotropy may affect the results and how it might be effectively modeled.

References

[1] C.R. Johnson, R.S. MacLeod, and M.A. Matheson. Computer simultions reveal complexity of electrical activity in the human thorax. *Comp. in Physics*, 6(3):230–237, May/June 1992.

[2] C.E. Miller and C.S. Henriquez. Finite element analysis of bioelectric phenomena. *Crit. Rev. in Biomed. Eng.*, 18:181–205, 1990.

[3] K.R. Foster and H.P. Schwan. Dielectric properties of tissues and biological materials: a critical review. *Crit. Rev. in Biomed. Eng.*, 17:25–104, 1989.

Acknowledgements

This research was supported in part by awards from the NIH, NSF, and the Whitaker Foundation. Special thanks to J. Schmidt for his suggestions and assistance.