Proceedings of the 29th Annual International
Conference of the IEEE EMBS
Cité Internationale, Lyon, France
August 23-26, 2007.

Contribution of the Left Anterior Myocardium to the Body Surface Potentials in Case of Apical Ectopic Beat

Juho Väisänen, Jesús Requena-Carrión, Felipe Alonso-Atienza,
José Luis Rojo-Álvarez, Jaakko Malmivuo and Jari Hyttinen

Abstract— **The present paper describes a study where effects of anterior myocardium on body surface potentials were investigated. The study combines numerical lead field analysis combined with cardiac automata model. Electric fields are calculated with finite difference method in a 3-D model of male thorax. The cardiac activation applied in the study is an ectopic beat originating in the apex. The correlations and mean differences between signal generated by anterior segments of left ventricle and signal generated by both ventricles were analysed for 117 leads. The results show that there are leads which have high correlation (>0.9) with low the relative mean difference (<0.2) between the signals generated by anterior segments and signals generated by whole ventricles. These electrode locations would be optimal to monitor the activation of anterior segments when ectopic beats originate in apex.**

I. INTRODUCTION

Various electrode systems are applied in measuring the electrical activity of myocardium in different clinical and non-clinical situations. The number of electrodes varies from two applied in implantable Holter electrocardiograph (ECG) monitors up to tens and even hundreds applied in body surface potential maps (BSPM). The properties and benefits of these systems have been studied widely. Studies have tried to find the answer to the questions; How many leads are needed for different measurement purposes and where should they be located. Trägårdh and colleagues [1] have recently published a good review of these clinical studies. The BSPM studies have been concentrating on finding the optimal electrode locations for different clinical cases such as detection of infarctions and ischemia [2, 3]. These studies suggest electrode locations specific to measure and indicate changes in the activation of different parts of myocardium such as anterior segments of left ventricle.

True origin of measured electrophysiological phenomenon is of interest in many cases. Electrocardiographic imaging with inverse solution and experimental setups has been applied in studying the effects of spatial and temporal characteristics of cardiac activation on the body surface potentials [4-6]. For example MacLeod and colleagues found in [7] that some locations of infarcts are more visible in some body surface potentials than in others. They also reported so called silent changes in cardiac activation which are not projected to the body surface. These studies suggest that there are electrode locations which are sensitive to activation arising in certain areas of myocardium. The studies of myocardial activation in terms of electrocardiographic imaging and inverse solution are based on the BSPMs recorded or modeled with even hundreds of electrodes [4, 8]. In clinical practice high amount of electrodes is not practical and thus it is valuable to study if there are electrode locations in the body surface which are sensitive and specific to activation arising in certain section of myocardium. This is especially of interest when monitoring different abnormalities in cardiac activation such as infarctions, ectopic beats and other arrhythmias.

In this paper we study the contribution of anterior myocardium to the body surface potentials of 117 leads. The present study combines sensitivity distribution modeling with dynamic source model of cardiac activation in studying the origins of the body surface potentials. The sensitivity distributions i.e. lead fields, were calculated in a realistic model of human thorax by applying finite difference method (FDM) and the principle of reciprocity. We simulated activation of an ectopic beat which originates at apex and conducts through both ventricles. Signals of 117 surface leads were solved by combining the lead fields and source distributions. The contribution of anterior myocardium was studied by calculating the correlation and relative mean difference between signals generated in anterior segments and whole ventricles. This analysis was applied to all leads and thus we can estimate which of the electrodes is most optimal to measure the activation of anterior myocardium during apical ectopic beat.

II. METHODS

A. Model data

Finite difference method (FDM) was applied in modeling the electric fields in the volume conductor. In the FDM the segmented volume data, e.g. from an MRI dataset, are divided into cubic elements forming a resistive network. The FDM is based on the Poisson's equation that can be used to describe the bioelectric quasistatic source-field problems [9]. The FDM allows the implementation of complex anatomic geometries from image data, and resulting potentials and

Manuscript received April 2, 2007. This work was supported in part by the Finnish Cultural Foundation, Ragnar Granit Foundation and .

J. Väisänen is with the Ragnar Granit Institute, Tampere University of Technology, PO. Box 692, 33101, Tampere, Finland (phone: +358-3-3115-2117; e-mail: juho.vaisanen@ tut.fi).

J. Requena-Carrión, F. Alonso-Atienza and J. L. Rojo-Álvarez are with Teoría de la Señal y Comunicaciones, Universidad Rey Juan Carlos, Fuenlabrada, Spain

J. Malmivuo and J. Hyttinen are with Ragnar Granit Institute, Tampere University of Technology.

currents can be calculated within the whole volume conductor model [9]. In the present study we applied a FDM model of the 3D male thorax based on the Visible Human Man dataset (VHM) [10]. The applied dataset represents data on 95 segmented slices containing altogether 2.7 million nodes with 2.6 million elements. The model applied contains over 20 different organ and tissue types with corresponding resistivities which are listed in [11].

B. Lead Field and Reciprocity

Lead fields were applied to describe and study the measurement of the electric fields in the volume conductor. The lead current density vectors define the relationship between the measured potential in the lead and the current sources in the volume conductor following Equation 1. The measured lead voltage is dependent on the magnitudes and directions of the lead and source current [12].

$$z[n] = \sum_{V} \frac{1}{\sigma} \frac{1}{I_r} \bar{J}_L \bullet \bar{J}^i[n] \qquad (1)$$

Where *z[n]* is the lead voltage as a function of time [n], $\bar{J}_L$ is the lead current density vector [A/cm^2] , I_r is the applied reciprocal current [A], $\bar{J}^i_{[n]}$ is the current source density vector [A/cm^2] as a function of time, σ is the conductivity [1/Ωcm] of the source location in the volume conductor and *V* is the source volume.

The lead field in the volume conductor can be established by applying the principle of reciprocity. The current field in the volume conductor rose by the reciprocal unit current (I_r=1 A) applied to the measurement electrodes corresponds to the lead current density and hence to the lead field. The sensitivity of a measurement lead at all source locations in the volume conductor can be calculated with a single calculation.

C. Cardiac Activation Model

As a cardiac activation model we adopted a state machine approach defined in [13]. This model of cardiac electric activity reproduces electric restitution of both action potential duration (APD) and conduction velocity (CV), as well as curvature effects. Cardiac tissue is modeled as a grid of discrete elements characterized by three discrete states, namely, *Rest*, *Refractory*1 and *Refractory*2, and transitions among them. The excitation of an element, i.e. the transition from *Rest* to *Refractory*1 is interpreted as a probabilistic event, depending on the amount of excitation in its neighborhood, and the excitability of the element, that can be accessed through the restitution curve of CV. Transitions from *Refractory*1 to *Rest* through *Refractory*2 depend on the current of APD. Additionally, a membrane voltage is assigned at every time instant, by temporarily scaling a standard ventricular one. Finally, non-conservative sources, $\bar{J}^i[n]$, at each time *n* and location *i* are solved based on the voltage differences and conductivities between neighboring elements.

D. Calculations

Lead fields, $\bar{J}_L$, were calculated in a realistic model of the male thorax for Dalhousie lead system containing 117 electrodes (Figure 1). The calculations were executed with bioelectric field software which applies the Incomplete Cholesky Preconditioner and Conjugate Gradient in solving linear equations [14].

We studied the contributions of the sources of anterior segments to the signals measured with 117 leads. The left ventricle was divided into 12 segments based on the standard 12 segment left ventricular subdivision recommended by the Committee on Nomenclature of Myocardial Wall Segments of the International Society of Computerized Electrocardiography [15]. The anterior segments applied are the same as the anterior infarction volume containing segments 1-7, 10 and 11 which are supplied by left descending artery or its branches [15].

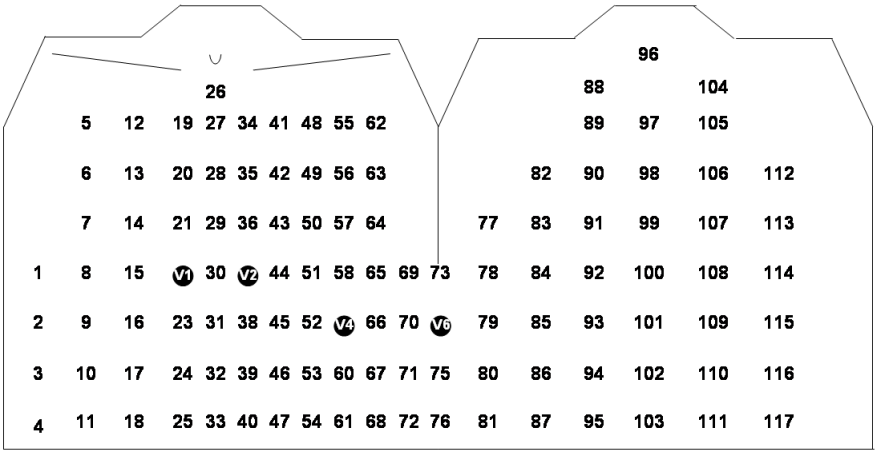

Figure 1. 117 Body surface leads

Applied source distribution $\bar{J}^i[n]$ presents the activation starting on the apex and conducting through both ventricles over the 0.7 second. The source distribution was combined with the lead field, $\bar{J}_L$,of the surface electrode to solve the measured potentials in the surface leads as described by (1). For each lead we calculated signals originating from anterior segments (source volume V_o) and the signal generated by ventricles (source volume V).

E. Analysis

We studied the contribution of anterior segments to the signal in surface lead by calculating the mean square difference (MSD) between the signal generated by V_o and signal generated by V. From (1), we can express that the ECG signal *z[n]* is generated by V and $z_o[n]$ is generated by the source in V_o. If we measure the similarity between *z[n]* and $z_o[n]$ based on MSD, we obtain:

$$MSD\{V_o\} = E\left[(z[n] - z_o[n])^2\right] \quad (2)$$

Furthermore, by normalizing MSD by the mean power of whole source volume *z[n]*, P_{zz}, we can obtain a more convenient measurement of similarity relative to the mean power of *z[n]*:

$$MSD_n\{V_o\} = \frac{MSD\{V_o\}}{P_{zz}} \quad (3)$$

The MSD_n describes only the mean difference in signal amplitudes but in order to analyze contribution of anterior activation to the total signal more deeply we calculated correlation coefficients between V_o and V.

III. Results

Figure 2 presents examples of signals calculated for two leads 92(A) and 22(B). The MSD_n and correlation of lead 92 were 0.12 and 0.95, respectively. The MSD_n and correlation of lead 22 were 1.93 and -0.47, respectively. Figure 3 present contour presentation of MSD_ns (A) and correlations (B) for surface electrodes. The lower the MSD and the higher the correlation are the higher is the contribution of segments to the measured signal. The anterior segments have high contribution to the measured signal in leads which have both high correlation and low MSD_n. There exist also lead regions which have low correlation and high MSD_n. The anterior segments have low contributions to these leads. Overall it can be noticed that areas which have better MSDs values have also better correlation values and vice versa areas with poor MSDs have poor correlations.

IV. Discussion

The present study introduces a modeling based analysis of cardiac activation. It has been recognized that lead field analysis together with cardiac automata applies fast and effective method to analyze different electrode setups and activation models. The sensitivity of measurement lead to all locations in the volume conductor can be achieved with one calculation by adopting the reciprocal lead field approach. The methods were applied in analyzing the contributions of anterior segments to the surface potentials. The analysis was based on the mean differences and correlation between signals generated by anterior segments and whole ventricles. The method presented is based on the modeling of the electric fields in the realistic volume conductor. The model was based on the anatomy of a single human subject and the segmentation of the tissues might also have some shortcomings. Furthermore, the model applied is isotropic. Anisotropic conductivities, especially of the cardiac muscle would improve the quality of state machine approach. The model also lacks of conduction pathways such as Purkinje fibers. The effect of these will be studied in the future with higher resolution models containing anisotropy and pathways.

Results of the study show that lower and higher left anterior, higher posterior regions have high correlation and low MSDs. These regions are most specific to the activation of anterior segments. The higher anterior leads are specific

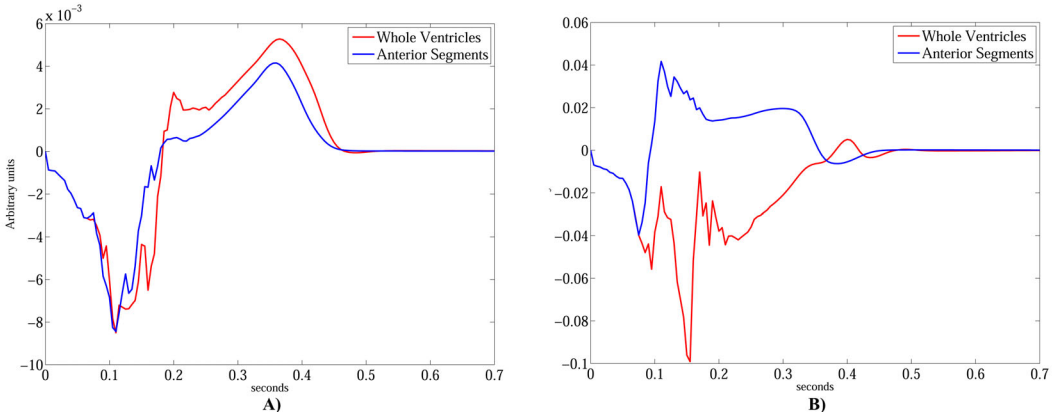

Figure 2 Signals produced by whole ventricles (red) and anterior segments (blue) in leads 92 (A) and 22 (B). Lead 92 has MSD_n=0.12 and correlation=0.95. Lead 22 has MSD_n=1.93 and correlation= -0.47.

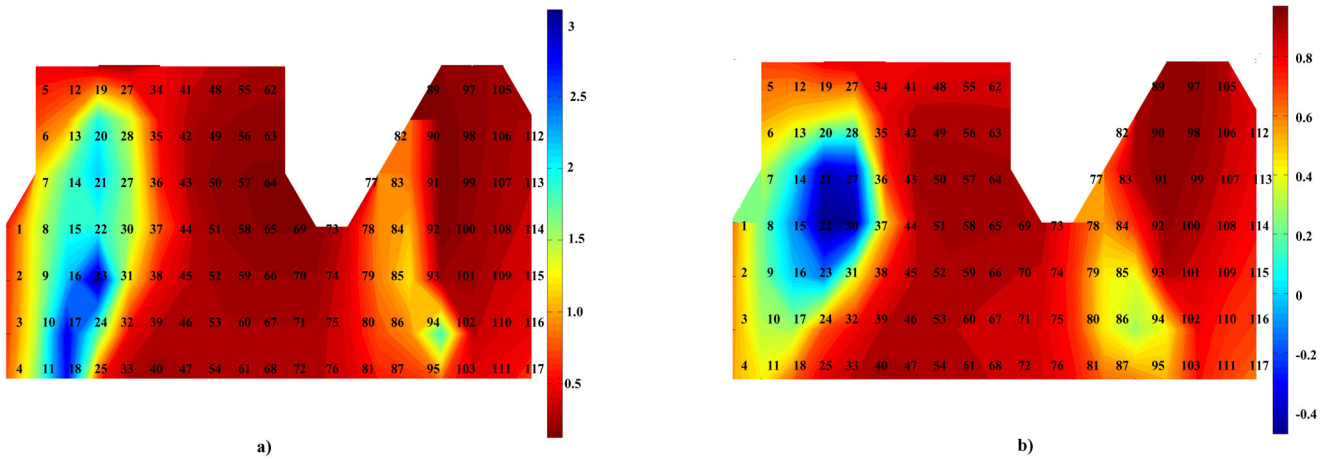

Figure 3. The MSD_ns (A) and correlations (B) for surface electrodes. Red regions have low MSDs and high correlations while blue regions have high MSDs and low correlations.

because they are closest to the sources. Reason for specificity of posterior and low anterior leads is not as evident. One possibility might be simply volume conductor effects of different organs and tissues.

Kornreich and colleagues found in [3] that the optimal leads to detect anterior infarction are located in the left thorax around leads 50 and 69. Also leads on the back around lead 100 were found to be sensitive. Similar kinds of results are obtained in this study. The leads in mid-left thorax have both high correlation and low mean difference between signals generated by anterior segments and whole ventricle. It should be noticed that model applied here does not contain properties of infarction in the anterior segments. These propeties, such as higher resistivity and lower conduction velocity, should be taken into account in future studies when results are more deeply compared to clinical studies.

In the present study one type of activation model was applied. The activation model has impact on the results and in the future we will apply multiple models to study e.g. the effect of starting point of ectopic beat on the sensitivities of the leads. We well also study activation of different myocardial regions such as inferior and posterior. In the future this method will be also applied in studying and designing of implantable ECG monitors as well as analyzing the origins of signals measured with implantable cardiac defibrillators, pacemakers and other cardiac measurements.

As conclusion the results of the study indicate that the anterior segments have major effect on some of the surface leads when measuring apical ectopic beats. In these leads the signal generated by anterior myocardium has high correlation and low mean difference when compared to the total signal generated by the ventricles.

REFERENCES

[1] E. Tragardh, H. Engblom, and O. Pahlm, "How many ECG leads do we need?," *Cardiology Clinics*, vol. 24, pp. 317-330, 2006.

[2] B.M. Horacek, J.W. Warren, C.J. Penney, R.S. MacLeod, L.M. Title, M.J. Gardner, and C.L. Feldman, "Optimal electrocardiographic leads for detecting acute myocardial ischemia," *J Electrocardiol*, vol. 34 Suppl, pp. 97-111, 2001.

[3] F. Kornreich, T.J. Montague, P.M. Rautaharju, P. Block, J.W. Warren, and M.B. Horacek, "Identification of best electrocardiographic leads for diagnosing anterior and inferior myocardial infarction by statistical analysis of body surface potential maps," *Am J Cardiol*, vol. 58, pp. 863-71, 1986.

[4] H.S. Oster, B. Taccardi, R.L. Lux, P.R. Ershler, and Y. Rudy, "Electrocardiographic Imaging : Noninvasive Characterization of Intramural Myocardial Activation From Inverse-Reconstructed Epicardial Potentials and Electrograms," *Circulation*, vol. 97, pp. 1496-1507, 1998.

[5] B. He, G. Li, and X. Zhang, "Noninvasive imaging of cardiac transmembrane potentials within three-dimensional myocardium by means of a realistic geometry anisotropic heart model," *IEEE Trans Biomed Eng*, vol. 50, pp. 1190-202, 2003.

[6] D.H. Brooks and R.S. MacLeod, "Electrical imaging of the heart," *Signal Processing Magazine, IEEE*, vol. 14, pp. 24-42, 1997.

[7] R.S. MacLeod, R.L. Lux, and B. Taccardi, "A possible mechanism for electrocardiographically silent changes in cardiac repolarization," *J Electrocardiol*, vol. 30 Suppl, pp. 114-21, 1998.

[8] J.E. Burnes, B. Taccardi, R.S. MacLeod, and Y. Rudy, "Noninvasive ECG imaging of electrophysiologically abnormal substrates in infarcted hearts : A model study," *Circulation*, vol. 101, pp. 533-40, 2000.

[9] C.R. Johnson, "Computational and numerical methods for bioelectric field problems," *Crit Rev Biomed Eng*, vol. 25, pp. 1-81, 1997.

[10] M.J. Ackerman, "The Visible Human Project," *J Biocommun*, vol. 18, pp. 14, 1991.

[11] P. Kauppinen, J. Hyttinen, P. Laarne, and J. Malmivuo, "A software implementation for detailed volume conductor modelling in electrophysiology using finite difference method," *Comput Methods Programs Biomed*, vol. 58, pp. 191-203, 1999.

[12] R. McFee and F.D. Johnston, "Electrocardiographic leads. I. Introduction," *Circulation*, vol. 8, pp. 554-68, 1953.

[13] F.A. Atienza, J.R. Carrion, A.G. Alberola, J.L.R. Alvarez, J.J.S. Munoz, J.M. Sanchez, and M.V. Chavarri, "A probabilistic model of cardiac electrical activity based on a cellular automata system," *Revista Espanola De Cardiologia*, vol. 58, pp. 41-47, 2005.

[14] N. Takano, *Reduction of ECG Leads and Equivalent Sources Using Orthogonalization and Clustering Techniques*, in *Ragnar Granit Institute*. Tampere University of Technology: Tampere, 2002, p. 302.

[15] R.H. Startt/Selvester, G.S. Wagner, and R.E. Ideker, *Myocardial Infaction*, in *Comprehensive Electrocardiology: Theory and Practice in Health and Disease*, T.D.V. Lawrie, Editor. 1989, Pergamon Press. p. 565-629.