Epi-, Endo- and Myocardial Contributions to the Body Surface Potential

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Abstract

The contribution of the epi- and endocardial as well as the myocardial electrical activity to the body surface potentials was investigated in this study. A volume conductor model including a realistic anisotropic ventricular model was built up and the forward problem of electrocardiography was solved for four different rhythms.

The results show, that the body surface potentials depend to a high degree on the epi- and endocardial electrical activity during depolarization.

1. Introduction

The electrocardiogram (ECG) is the measurable result of the spatio-temporal distribution of the electrical cardiac sources. The responsible sources are the transmembrane voltages (historically also termed transmembrane potentials, TMPs) within the heart, which are defined as the difference between the potentials inside and outside of the cardiac muscle cells. These TMPs are in turn a result from interactively opening and closing of specific ion channels during a cardiac cycle connecting the intra- and extracellular space of the cardiac muscle cells. The contribution of the epi- and endocardial, and the myocardial portion of the heart to the ECG has so far been validated only qualitatively or at least models have been developed capable of describing the general behavior of the electrical spread of excitation, like, e.g., the double dipole layer, the oblique dipole layer or the uniform dipole layer model [1, 2]. Some of these lead to the conclusion that during the depolarization phase of the cardiac cycle the electrical behavior of the heart can be modeled applying isotropic conductivities [3, 4, 5]. The major reason for this finding is the small extension of the electrical wavefront (gradient of the transmembrane potential) during depolarization (about 1 mm) due to the high slew rate of the TMP-upstroke (about 80 to over 100 mV/ms). Introducing isotropic electrical conductivities implicates, that the major contribution to the ECG stemmed from the epi- and endocardial surfaces of the heart and that myocardial electrical activity would be negligible. Many studies – also clinically evaluated ones – showed this assumption to be acceptable [6, 7, 8]. Another consequence of employing isotropic conductivities is that the boundary element method can be used to solve the forward problem of electrocardiography reducing geometrical modeling effort and computational cost [9].

The question, whether cardiac electrical isotropic conductivities can be employed during depolarization and anisotropy has to be included for proper modeling of the electrical ventricular behavior during the repolarization, is investigated quantitatively in this study using a volume conductor model (VCM) of a patient who participated in our clinical validation study. Different ventricular rhythms – the related spatio-temporal distribution of the TMPs and their corresponding shapes – are simulated employing a three-state cellular automaton. These are entering the calculation of the body surface potentials (BSPs) on the torso surface of the VCM as input using anisotropic cardiac electrical conductivities. Thus the finite element method was applied for solving the anisotropic forward problem of electrocardiography delivering the transfer function as a result. For building up a realistic ventricular fiber structure an algorithm was implemented capable of interpolating fiber orientation in the ventricular myocardial regions according to studies performed by Taccardi and coworkers, who did experimental studies on dogs [10]. They discovered that fiber orientation rotates in a counter-clockwise manner with increasing depth between epi- to endocardium and they also showed that the fiber structure had a major impact on the measured epicardial potentials on the left ventricle when pacing at different intramural depths was performed.

2. Methods

Construction of the volume conductor model The VCM was extracted by segmentation and triangulation of the compartments ventricles, lungs, blood masses and

torso using image data of the patient, which were recorded by magnetic resonance imaging (MRI). The patient participated in a clinical study at the Department of Cardiology of the Medical University Innsbruck, Austria. Written informed consent of the patient was obtained and the study was approved by the local ethics committee. A Magnetom Vision Plus 1.5 Tesla scanner (Siemens Medical Solutions, Erlangen, Germany) was used for acquiring the ventricular geometry in ECG-gated cine mode during breath-hold (expiration, oblique short-axis scans, 4 and 6 mm spacing). The lungs and torso shape were recorded in T1 flash mode during breath-hold (expiration, axial scans, 10 mm spacing). The segmentation of the ventricular cardiac surfaces was performed using a self-developed semi-automatic extraction tool [11]. The shapes of the torso and lungs were extracted using conventional extraction algorithms. After triangulation of all compartments the surface meshes were optimized by a Voronoi-Delauny-based algorithm [12]. The optimization was necessary in order to reduce numerical errors in the forward simulation as well as to enable an optimized tetrahedral mesh generated on the basis of the high quality triangular surface mesh. For employing the finite element method the corresponding tetrahedral mesh of the VCM was created using the commercial software package Hypermesh (Altair Eng.) ensuring high quality tetrahedral meshes. The whole volumetric mesh consisted of 1,126,259 tetrahedrons and 192,638 nodes, respectively. The mean edge length of all tetrahedrons belonging to the ventricles was 2.8 mm, the mean edge length of the whole VCM was 5.4 mm.

Assignment of the ventricular fiber structure The ventricular fiber arrangement on the epi- and endocardia was chosen such, that it fitted qualitatively well with the findings described in literature [13, 14, 15, 16]. The myocardial fiber orientation was approximated employing an interpolation algorithm ensuring the counter-clockwise rotation of the fibers with increasing ventricular intramural depth (from epi- to endocardium).

Computation of cardiac activation sequences - the cellular automaton The cellular automaton (CA) used in this study was developed at the University of Technology in Graz, Austria, during the last two decades [17], modified and implemented in amiraDev 3.0$^{\text{TM}}$ [18]. Briefly, after segmentation, triangulation and tetrahedral mesh generation different types of tissue were assigned to the ventricles with corresponding parameter setting for each tissue type. The CA needs the fiber structure assigned to each node of the ventricular tetrahedral model as well as the refractory periods assigned to each tissue type. As a result the corresponding activation times as well as the transmembrane potentials of each cardiac node can be computed.

Extracellular potential computation Based on the quasi static approximation of Maxwell's equations for electromagnetic field calculation and employing the bidomain theory [19] (therefore the subheading "extracellular potential computation"), the resulting set of linear algebraic equation is:

$$\boldsymbol{\Phi} = \boldsymbol{L}\boldsymbol{\Phi}_m, \tag{1}$$

with the $(c \times n)$ matrix $\boldsymbol{\Phi}$ representing the potentials of the c torso nodes for n time instants, $\boldsymbol{L}$ is the $(c \times h)$ lead-field-matrix and $\boldsymbol{\Phi}_m$ holds the transmembrane potentials of the h cardiac source nodes. For a detailed treatment of the derivation the reader is referred to [9].

Computation of epi-, endo- and myocardial contribution Computation of the contribution stemming from the epi- and endocardial electrical activity and myocardial electrical activity is performed by dividing these volumes (areas) into epi-, endocardium (nodes belonging to the surface enclosing the ventricles) and myocardium (all other nodes belonging to the cardiac volume). Column entries of the lead field matrix $\boldsymbol{L}$ are assembled in ascending order beginning with ee nodes belonging to epi-, endocardium, and the last m nodes belong to the myocardial volume $(ee + m = h)$. The voltage in one surface node i is computed by

$$\varphi_i = \underbrace{\sum_{j=1}^{ee} l_{i,j}\varphi^{ee}_{m\ j}}_{a_i^{ee}} + \underbrace{\sum_{k=ee+1}^{m} l_{i,k}\varphi^{m}_{m\ k}}_{a_i^{m},} \tag{2}$$

with a_i^{ee} and a_i^m representing the epi-, endocardial and the myocardial electrical activity, respectively. Choosing a different resting potential level for all cardiac nodes does not effect the ECG value. It effects, however, both epi-, endo- and myocardial activities. The level of the potentials will show a proportionally increased and decreased offset, respectively. Hence, activity definitions a_i^{ee} and a_i^m are not suitable in order to describe epi-, endocardial and myocardial electrical contribution to the ECG signal, because there would occur epi-, endo- and myocardial activity, although $\boldsymbol{\varphi} = \mathbf{0}$, which corresponds to, e.g., all cardiac nodes are held at the same resting potential level.

Therefore, the derivations of all signals with respect to time, $\dot{\varphi}_i$, $\dot{a}_i^{ee}$ and $\dot{a}_i^m$, are used instead, as any change of the ECG signal in time is intrinsically related to a corresponding change of the epi-, endo- and myocardial activities.

The following algorithm is employed in order to quantify the epi-, endo- or myocardial contribution responsible for a change of the ECG signal for a discrete time step t, assuming $\dot{\varphi}_i(0) = \dot{a}_i^{ee}(0) = \dot{a}_i^m(0) = 0$ for time instant

$t = 0$ for all cases:

$$\dot{\bar{a}}_i^{ee}(t) = \frac{|\dot{a}_i^{ee}(t)|}{|\dot{a}_i^{ee}(t)| + |\dot{a}_i^{m}(t)|},$$
$$\dot{\bar{a}}_i^{m}(t) = \frac{|\dot{a}_i^{m}(t)|}{|\dot{a}_i^{ee}(t)| + |\dot{a}_i^{m}(t)|},$$
$$\text{if } \varepsilon \leq |\dot{a}_i^{ee}(t)| \vee \varepsilon \leq |\dot{a}_i^{m}(t)|, \text{ with } 0\frac{\text{nV}}{\text{ms}} \leq \varepsilon \ll 1\frac{\text{nV}}{\text{ms}}. \quad (3)$$

Computing the mean contribution of the epi-, endocardial activity for n arbitrarily chosen time steps for the whole body surface consisting of c nodes is performed by

$$\dot{a}^{ee} = \frac{1}{n \cdot c} \sum_{i=0}^{c-1} \left(\sum_{t=1}^{n} \dot{\bar{a}}_i^{ee}(t) \right), \quad (4)$$

similarly the mean myocardial contribution $\dot{a}^m$ is determined. Equation (4) describes each relative signal contribution to the BSP, but does not deliver information about the similarities between the epi-, endo-, myocardial signals and the ECG. For computation of this similarity the correlation coefficient for the epi-, endocardial CC^{ee} (and similar for the myocardial correlation coefficient CC^m) is employed according to

$$CC^{ee} = \frac{1}{n} \sum_{t=1}^{n} \frac{(\dot{\boldsymbol{a}}^{ee}(t) - <\dot{\boldsymbol{a}}^{ee}(t)>) \cdot (\dot{\boldsymbol{\varphi}}(t) - <\dot{\boldsymbol{\varphi}}(t)>)}{\| \dot{\boldsymbol{a}}^{ee}(t) - <\dot{\boldsymbol{a}}^{ee}(t)> \|_2 \, \| \dot{\boldsymbol{\varphi}}(t) - <\dot{\boldsymbol{\varphi}}(t)> \|_2}, \quad (5)$$

with the vectors $\dot{\boldsymbol{a}}^{ee}(t)$, $\dot{\boldsymbol{a}}^{m}(t)$ and $\dot{\boldsymbol{\varphi}}^{m}(t)$ holding the changes of the corresponding potentials on the nodes of the torso's surface mesh, the dot product is expressed by "$\cdot$", symbol "$\| \|_2$" represents the L_2 norm, and "$< >$" denotes the mean value among the elements of the vector in brackets.

3. Results

Investigation of the epi-, endo- and myocardial contribution to the BSPs was performed employing four different scenarios:

- ventricular sinus rhythm starting in the bundle of His (SR),
- right ventricular pacing, i.e., simulation of a ventricular extrasystole starting in the right ventricle (pRV),
- septal pacing, i.e., simulation of a ventricular extrasystole starting in the intraventricular septum (pSE), and
- sinus rhythm in combination with a Wolff-Parkinson-White syndrome with the accessory pathway assumed to be located in the left ventricle (WPW).

Each of the above mentioned rhythms was investigated with respect to epi-, endo- and myocardial activity during depolarization, repolarization and for the whole cycle length, which was set to 600 ms. The epi-, endocardial ($\dot{a}^{ee}$) and myocardial ($\dot{a}^m$) contributions as well as the correlation coefficients CC^{ee} and CC^m for each rhythm are listed in Tab. 1. Parameter ε of eq. (3) was set to $1\frac{\text{nV}}{\text{ms}}$.

Table 1. Results of epi-, endo- ($\dot{a}^{ee}$) and myocardial ($\dot{a}^m$) contributions to the body surface potentials and the corresponding correlation coefficients CC^{ee} and CC^m for four different rhythms during depolarization, repolarization and for the whole cycle length of 600 ms.

rhythm	contribution/ correlation	Depolarization	Repolarization	whole cycle
SR	$\dot{a}^{ee}$	0.65	0.55	0.58
	$\dot{a}^{m}$	0.35	0.45	0.41
	CC^{ee}	0.93	0.76	0.81
	CC^{m}	0.56	0.47	0.50
pRV	$\dot{a}^{ee}$	0.61	0.55	0.57
	$\dot{a}^{m}$	0.39	0.45	0.43
	CC^{ee}	0.95	0.84	0.89
	CC^{m}	0.77	0.62	0.69
pSE	$\dot{a}^{ee}$	0.61	0.49	0.54
	$\dot{a}^{m}$	0.38	0.51	0.46
	CC^{ee}	0.90	0.94	0.93
	CC^{m}	0.68	0.77	0.75
WPW	$\dot{a}^{ee}$	0.68	0.51	0.54
	$\dot{a}^{m}$	0.32	0.49	0.45
	CC^{ee}	0.94	0.68	0.73
	CC^{m}	0.50	0.56	0.57

4. Discussion and conclusions

The epi-, endo- and myocardial contributions to the BSPs for four different ventricular heart rhythms were investigated in this study. The related anisotropic forward problem of electrocardiography was solved employing a VCM containing ventricles with a corresponding fiber architecture. The fiber structure was chosen such that it both qualitatively and quantitatively fitted with the findings of Taccardi and coworkers, who figured out, that the epicardial potential shapes had changed with increasing point-pacing depth. This is due to the counter-clockwise rotation of the fiber orientation between epi- and endocardium.

Summing up the results for the depolarization sequences for all four rhythms, epi- and endocardial contribution to the BSPs was found to cover approximately 2/3, myocardial contribution to be about 1/3 of the cardiac activity. These contribution numbers show, that the BSPs amplitudes are more effected by epi- and endocardial than by myocardial contribution during the depolarization sequences.

In case of isotropic conductivities, the ECG is impacted

only by electrical sources occurring on the closed surface (i.e., epi- and endocardial surfaces) of the heart, which means, that only epi- and endocardial contributions have to be considered. The relationship between the time course of the ECG and the epi- and endocardial electrical activity is much better reflected by the correlation coefficient. The epi-, endocardial correlation coefficients for all types of rhythms simulated in this study exceeded 90 % during depolarization, whereas the correlation between BSPs and myocardial contribution was on an average about 63 %. This indicates, that the time course of the BSPs during depolarization is to a very high degree depending on epi- and endocardial cardiac electrical activity. The amplitudes, however, are up to 1/3 influenced by myocardial activity expressed by the relative contribution number. Thus, the corresponding BSPs would not show proper potential amplitude levels when neglecting myocardial activity.

When potential patterns of BSP-maps play an important role, then isotropy could be applied in order to describe the genesis of the BSPs. In particular NICE (Noninvasive Imaging of Cardiac Electrophysiology) – an inverse noninvasive reconstruction method for imaging of cardiac electrical activation times – is much more sensitive to errors of potential patterns than to errors of potential amplitudes [3], which could also be figured out by clinical validation studies [7, 8].

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