Impact of the Patient Torso Model on the Solution of the Inverse Problem of Electrocardiography

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Abstract. *Cardiac diagnostics based on a solution of the inverse problem of electrocardiography offers new tools for visual assessment of cardiac ischemia. The accuracy of the inverse solution is influenced by fidelity of the patient torso model. As optimum, an individual torso model with real heart shape and position obtained from CT or MRI is desirable. However, imaging is not always available in clinical practice, hence we investigated, if a generic torso shape individually adjusted according to patient's chest dimensions, with a simplified heart model placed to a vertical position obtained from inverse localization of the early ventricular activation can result in an inverse solution close to the result obtained with an accurate torso model. Simulated inverse localization of 18 ischemic lesions for 9 subjects showed that the use of individually adjusted generic torso instead of real torso shape led to an acceptable increase of the lesion localization error from 0.7±0.7 cm to 1.1±0.7 cm when accurate heart model was used. However, if simplified heart model was used and placed in a vertical position according to the V2 lead level, the lesion localization error increased to 3.5±0.9 cm. Moving the simplified heart model to a position estimated by the inverse solution decreased the vertical heart positioning error from 1.6±2.3 cm to 0.2±1.2 cm but without adjusting the heart shape and rotation the lesion localization error did not improve and reached 3.7±1.0 cm.*

Keywords

Individual torso shape model, inverse problem of electrocardiography, inversely estimated heart position.

1. Introduction

Solution of the inverse problem of electrocardiography and topographical visualization of an cardiac electrical generator is promising tool for assessment of various cardiac disorders including local ischemic lesions or arrhythmogenic substrates. For an accurate inverse solution it is necessary to have an individual torso model with internal structures representing at least the main electrical inhomogeneities, such as lungs and ventricular cavities filled with blood [1], [2]. Another important issue discussed in the literature is the large variability of the heart position that can vary by several centimeters, namely in the vertical direction [3], [4]. Missing information on the exact heart position can strongly influence the result of the inverse solution [5]. As optimum, the real heart position should be used in the torso model rather than usually assumed position relatively to anatomical landmarks, such as the fourth intercostal space.

To obtain a faithful model of the patient torso, the use of computed tomography (CT) or magnetic resonance imaging (MRI) technique is preferable. However, in clinical practice these techniques are not always available for cardiac patients. Hence it is desirable to search other methods how to create enough accurate patient specific torso model without the need of imaging techniques.

In this simulation study an approach based on the use of a generic model of the human torso containing simplified model of the ventricular myocardium was attempted. Using several anthropometric measures the torso shape was adjusted to match with the torso of an individual subject. To estimate the vertical heart position, measured ECG data were used for solving a simplified inverse problem and finding the location of early ventricular activation that was supposed in the upper part of the septum. The aim of the study was to verify whether the use of the individually adjusted generic torso model and a simplified heart model placed in the estimated vertical position allow inverse solution with sufficient accuracy.

2. Methods and Material

2.1. Simulation of Body Surface Potentials

A simplified model of ventricular myocardium was used to simulate normal ventricular activation and activation in ventricles containing single ischemic region with changed repolarization [8], [9]. The geometry of the model was defined using several ellipsoids and its volume consisted of 1×1×1 mm cubic elements. Each model element was assigned realistically shaped action potential (AP) and the ventricular activation process was simulated by a cellular automaton. In each time step of the activation, elementary dipole moments were computed from the differences between APs of adjacent model elements, thus the equivalent cardiac electrical generator was represented by a multiple-dipole model. Using the boundary element method, body surface potentials (BSPs) $\mathbf{p}(t)$ were computed in points representing electrode positions on the surface of an inhomogeneous torso model:

$$\mathbf{p}(t) = \mathbf{A}\,\mathbf{s}(t), \tag{1}$$

where $s(t)$ is a multiple dipole source in the ventricular myocardium model and matrix $\mathbf{A}$ represents the influence of the torso as an inhomogeneous volume conductor.

From the simulated BSP maps the QRST integral map (IM) $\mathbf{i}$ was computed using the formula

$$\mathbf{i} = \int_{QRST} \mathbf{p}(t)\mathrm{d}t = \int_{QRST} \mathbf{A}\,\mathbf{s}(t)\mathrm{d}t = \mathbf{A} \int_{QRST} \mathbf{s}(t)\mathrm{d}t = \mathbf{A}\,\mathbf{s}, \tag{2}$$

where $\mathbf{i}$ is the vector of integrals of BSPs and s is an integral of multiple dipole source of the cardiac electrical field.

To mimic the local repolarization changes in the ischemic lesions, 18 small areas were modeled in the ventricular myocardium, one at a time. They were formed as spherical caps with varying diameter and height, and placed in 3 typical regions supplied by the main coronary arteries: anterior - in the region supplied by the left descending artery, posterior – in the region supplied by the left circumflex artery, and inferior - in the region supplied by the right coronary artery. In each region, 3 endocardial and 3 epicardial lesions of different sizes were modeled. In the model elements within the ischemic lesions, the AP was shortened by 20 % to simulate the changed repolarization.

For each ischemic lesion the difference QRST integral map (DIM) $\mathbf{\Delta i}$ was calculated by subtracting the IM computed for the normal activation from the IM computed in the presence of the particular lesion as

$$\mathbf{\Delta i} = \mathbf{i_i} - \mathbf{i_n} = \mathbf{A s_i} - \mathbf{A s_n} = \mathbf{A}(\mathbf{s_i} - \mathbf{s_n}) = \mathbf{A \Delta s}, \tag{3}$$

where $\mathbf{i_i}$ and $\mathbf{i_n}$ represent the vectors of QRST integrals of BSPs in case of ischemia and during normal activation, $\mathbf{\Delta s}$ represents the difference between the integral multiple dipole source under normal conditions and during ischemia. The DIM thus represents the topographical changes in the surface cardiac electrical field due to the local ischemia.

2.2. Inverse Localization of an Ischemic Lesion

To identify the ischemic lesion by an inverse solution, equivalent integral generator representing the original multiple dipole generator $\mathbf{\Delta s}$ should be determined. Because this inverse problem is generally ill-posed, additional constraints are needed for its unique solution. The constraint used in this study was the assumption that the equivalent integral generator representing the small ischemic area can be represented by a single dipole. The magnitude, orientation and position of the dipole can be searched as parameters of a "moving dipole", what yields a nonlinear problem. In this study another approach was used: only dipole magnitude and orientation were determined for dipoles in predefined possible positions. In this way the problem was converted to a linear one, however, the parameters of an equivalent integral dipole (EID) had to be computed for many positions within the ventricular myocardium and then the proper position had to be selected. To achieve sufficient resolution for the dipole localization, the mean distance between the neighboring possible dipole positions less than 1 cm was selected. For every predefined position j, the dipole moment $\mathbf{d}_j$ was computed as

$$\mathbf{d}_j = \mathbf{A}_j^{+} \mathbf{\Delta i}, \tag{4}$$

where $\mathbf{A}_j^{+}$ is the pseudo-inverse of a submatrix $\mathbf{A}_j$ of the matrix $\mathbf{A}$ that represents the relation between the EID placed in the position j and the DIM. The equation (4) is overdetermined and its unique solution exists for each position of the EID. To compute the pseudo-inverse, singular value decomposition was applied to the submatrix $\mathbf{A}_j$.

To find the best representative generator of the lesion, for each position j the surface map $\mathbf{q}_i$ was computed using corresponding EID as the generator. This map was compared with the input DIM using the relative root mean square difference $RMSDIF_j$:

$$RMSDIF_j = \sqrt{\sum_k (\mathbf{q}_{j,k} - \mathbf{\Delta i}_k)^2} \Big/ \sqrt{\sum_k (\mathbf{\Delta i}_k)^2}, \tag{5}$$

where k is the number of electrodes on the torso surface. The EID in a position that produced the map with smallest $RMSDIF_j$ was selected as the best representative of the lesion.

The distance between the selected EID position and the gravity center of the simulated lesion was defined as the lesion localization error (LE) and was used to evaluate the accuracy of the inverse lesion localization.

2.3. Vertical Heart Position Estimation

From the observed high variability of the vertical heart position relatively to the anatomically fixed electrode positions (Fig. 1) it is apparent that adjustment of the vertical heart position is highly desirable.

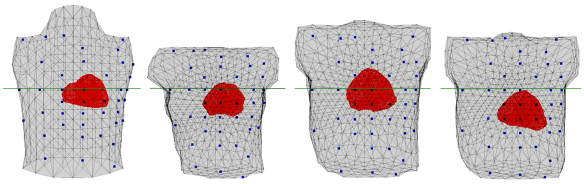

Fig. 1: Generic torso model (left) and 3 examples of real chest models of subjects used in the study. Dots indicate electrode positions, vertical position of ECG lead V2 is marked by a horizontal line.

In Fig. 1 the inter-individual variability of the vertical distance between the heart position and the level of ECG lead V2 defined in the 4th intercostal space is demonstrated. If the same generic torso and heart model (Fig. 1 left) is used for all subjects, this vertical distance is assumed to be zero what apparently may not be correct.

The possibility to estimate the individual vertical position of the heart by inverse localization of the early ventricular activation was studied using real ECG signals measured in 9 subjects (7 men, 2 women) published in [3]. The ECG signals in each subject were recorded by 62 leads of the Amsterdam lead system. Realistic torso models, as well as the electrode positions for these subjects, were obtained from MRI scans. For each subject ECG signals were recorded for 10 seconds with a sampling rate of 1000 Hz. Low-pass filter with 50 Hz stop-band was applied and the signals were time averaged to create representative signal for one heart cycle in each lead [11]. Finally, the baseline of averaged signals was adjusted by setting the mean potential of the PQ interval to zero. The time instant of the QRS onset was set manually from rms signal computed from all measured leads.

The integral map (IM) for the first 20 ms of the ventricular depolarization (from the QRS onset) was computed for each subject and used as the representative of the cardiac electrical generator during the early ventricular activation that normally occurs in the upper part of the left endocardial septum [11].

Site of the initial ventricular depolarization was estimated from the IM using the inverse solution in homogeneous torso model. Similar approach as described in section 2.2. was applied. The region activated during the early depolarization was assumed to be small enough to be represented by single EID that was searched in the whole modeled ventricular myocardium volume in predefined positions placed in regular 3 mm grid. For each subject the position j of the early activated area was determined as the site in which the RMSDIFj between the IM and the map generated by the EID was minimal. The heart model was then vertically shifted so that the site of the early ventricular activation vertically coincided with the anatomically determined area in the upper part of the left endocardial septum. The vertical errors between the real heart position and the inversely estimated heart position, as well as the standard heart position (representing the situation with no individual information about the heart position), were then evaluated.

The described method for inverse estimation of the vertical heart position was used in this study to create one type of the individual torso models for each subject.

2.4. Torso Models Used in the Study

Torso models of 9 healthy subjects introduced in section 2.3. obtained from MRI scans and described by triangulated surfaces of torso, lungs and ventricular myocardium were used in the study. The positions of 62 ECG electrodes were also included in the torso models.

Modified Dalhousie torso [6] containing the simplified ventricular myocardium model [8] described in section 2.1. and placed in anatomically defined standard position was used as the generic model of a human torso.

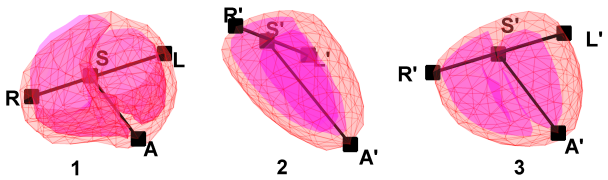

Fig. 2: Individual adjustment of the simplified heart model geometry to the heart of a particular subject (frontal view): 1 – subject's heart geometry, 2 – simplified heart model in standard position, 3 – simplified heart model rotated along the long (A-S) and short (L-R) axis and scaled along the long axis for best correspondence to the subject's heart geometry.

To have comparable heart anatomy for forward simulations in all subjects, the simplified ventricular my-

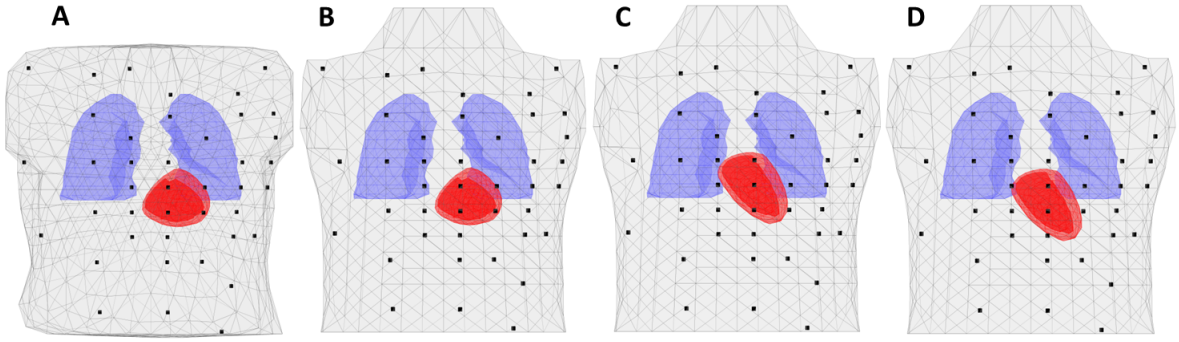

Fig. 3: Four types of torso models used for each subject in the study: A - original torso model obtained from MRI with a simplified model of ventricles placed, rotated and scaled to correspond to the subject's real heart. B - generic torso model adjusted for best match with subject's torso shape with the ventricles as in case A. C – adjusted generic torso shape as in case B but with a simplified model of ventricles in standard position, D – adjusted generic torso shape as in cases B and C but with a simplified model of ventricles vertically shifted to a vertical position based on the inversely estimated site of early ventricular activation.

ocardium model described in section 2.1. but adjusted to the heart geometry of each subject was used (Fig. 2). For each subject the long heart axis (from apex A to point S in the septum) and short heart axis (from point L in the left ventricular free wall to point R in the right ventricular free wall) were defined. Then the simplified ventricular model was positioned so that its axes coincided with the axes in the heart of the real subject and was properly scaled along its long axis.

For all 9 subjects body surface potential maps (BSPMs) corresponding to normal activation as well as to activation in case of 18 modeled ischemic lesions were simulated and single DIM was computed for each case. Realistic inhomogeneous torso models based on subjects' MRI scans, containing lungs and heart cavities filled with blood were used in the simulations. Individually adjusted simplified geometrical models of the ventricles described above were inserted into each torso. Respective electrical conductivities assigned to the lungs and heart cavities were 4 times lower and 3 times higher than the average conductivity of the rest of the torso. The DIMs were computed from 62 simulated leads placed on the torso surface according the Amsterdam lead system and used as input for the inverse solutions.

To study the impact of the torso model shape and heart position on the noninvasive inverse localization of ischemic lesions, several types of torso models were used in the inverse computations (Fig. 3).

Model A – the same torso model as used in the forward simulations. It consists of realistic outer torso shape and electrode positions based on MRI scan, lungs, and individually adjusted simplified heart model that was placed, oriented and scaled for best correspondence with the subject's heart model obtained from MRI.

Model B – torso shape created from the generic torso model by adjusting its shape according to 10 anthropometric measures of the subject (see Fig. 4) as proposed in [7]. The same individually adjusted simplified heart model as in torso model A was used.

Model C – the same adjusted generic torso shape with electrodes as in model B but with a generic heart model placed and oriented in a standard way – as if no knowledge about the heart position, orientation and size was available. The vertical position of the heart model is in the level of the standard ECG lead V2.

Model D – the same adjusted generic torso shape with electrodes as in models B and C but with a generic heart model vertically shifted according to the result of the inverse estimation of the vertical heart position.

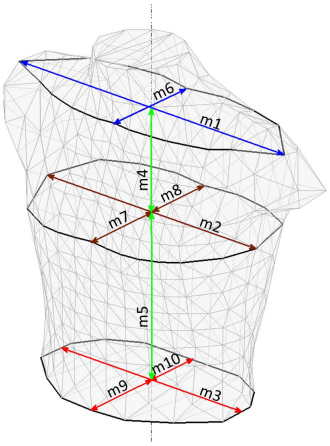

Fig. 4: Selected 10 anthropometric measures for subject-specific adjustment of the generic torso shape.

The errors of the inverse localization of all 18 modeled lesions were evaluated for each of the 9 subjects and each type of the torso model. The results for different torso model types were compared.

3. Results

3.1. Vertical Heart Position Estimation

The inversely estimated sites of early ventricular depolarization were found in the upper septal area for all 9 investigated subjects. Their positions (transformed to a single standard simplified ventricular model) are depicted in Fig. 5 together with their mean position (larger marker) computed as the gravity center of the results for individual subjects. The average spatial distance between the individual positions of the early depolarization sites from their mean position was 1.6±0.6 cm and the standard deviation of the vertical position of results for individual subjects was ±1.3 cm. These numbers indicate the possible error range when assuming that the early activation site should serve as a reference point for adjustment of the vertical heart position.

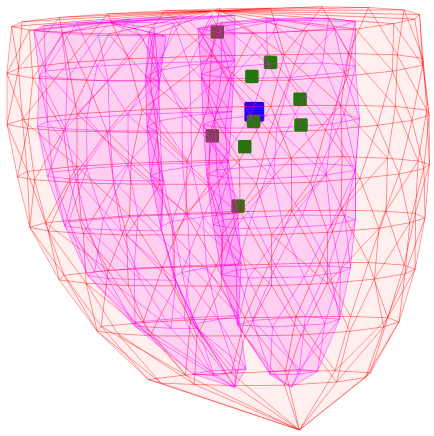

Fig. 5: The estimated sites of early ventricular depolarization for 9 studied subjects (small markers) and the gravity center of the positions (large marker) depicted in standard simplified ventricular model.

While the average vertical error (for all 9 subjects) between the real position of the heart ventricles and position of the standard ventricular model (marked as "stand. pos.") was 1.6±2.3 cm, the average vertical error between the real position of the ventricles and the position of ventricles estimated from the site of early ventricular activation (marked as "early dep.") dropped to 0.2±1.2 cm. The results for all 9 subjects are shown in Fig. 6. These results indicate that despite the vague definition of the site of the early ventricular activation as a reference point, its inverse estimation can improve the vertical positioning of the heart model if no other information on the heart position in the torso is available.

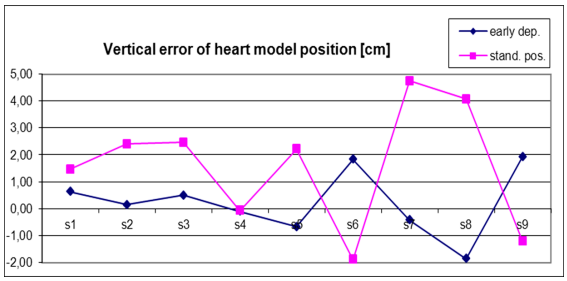

Fig. 6: Errors of vertical position of the heart models for all studied subjects: Diamonds – errors between real positions of ventricles and positions estimated from the early ventricular activation. Squares - errors between real position of ventricles and the position of standard simplified ventricular model.

3.2. Impact of Approximate Torso Shape on the Inverse Solution

To study the impact of the use of an approximate torso shape created by adjusting a generic torso shape according 10 anthropometric parameters of the subject, results of the inverse solutions with torso models A and B were compared. As it can be seen in Fig. 7, the LE values obtained with approximate torso model B (from 0.6±0.4 cm to 1.6±1.1 cm) with the mean LE for all subjects of 1.1±0.7 cm were only slightly worse than the LE values obtained with the torso model A created from MRI scans (from 0.5±0.3 cm to 0.8±0.9 cm) with mean LE for all subjects of 0.7±0.7 cm. In all subjects, the mean LE was slightly worse when torso model B was used, with the exception of subject s7, where the values were equal.

This result suggests that the use of individually adjusted generic torso shape in the inverse solution can be acceptable if no imaging data are available.

3.3. Impact of the Vertical Heart Position Estimation on the Inverse Solution

To study the impact of the vertical positioning of the heart model, results of the inverse lesion localization with torso models C and D were evaluated and compared also with results with torso model B. In all these models individually adjusted generic torso shape was used. When the torso model C with the heart model

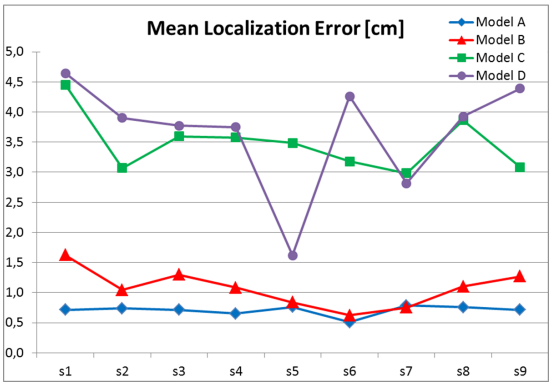

Fig. 7: Mean errors of the inverse lesion localization computed for all 18 modeled lesions in 9 studied subjects (s1–s9) and using 4 torso and heart model configurations (models A, B, C, D).

located in standard vertical position given by the level of ECG lead V2 was used in the inverse solution, the mean LE values varied from 3.0±0.5 cm to 4.5±0.7 cm, with the mean LE for all subjects of 3.5±0.9 cm. For torso model D, where the heart model was vertically moved to the inversely estimated position, the LE values ranged from 2.8±1.2 cm to 4.6±0.5 cm, with a noticeably lower value of 1.6±1.4 cm for the subject s5. The LE averaged for all subjects was 3.7±1.0 cm.

These results show, that despite the improved vertical positioning of the heart model in torso model D in comparison with torso model C, in all but two subjects (s5 and s7) the results with model D were even worse than those with torso model C. Comparison with much better results obtained with torso model B indicates that merely positioning of the heart model without its proper rotation and scaling does not yield acceptable errors of the inverse lesion localization.

4. Discussion

The experimental inverse localization of the early ventricular activation in 9 subjects indicated that the site of the initial activation can be estimated within about 1.6±0.6 cm. In all studied subjects the found positions were in agreement with Durrer's findings [11] that the ventricular activation in healthy subjects starts in endocardial areas of the left ventricular cavity near septum. Although the achieved average error of the estimated vertical position was only 0.2 cm, its standard deviation of ±1.2 cm is quite large and errors of almost 2 cm were found in subjects s6, s8 and s9 (Fig. 6). In spite of this, the method generally improved the vertical positioning of the heart model in comparison with the error of 1.6±2.3 cm in a situation when no information on the heart position was used and the ventricular model was positioned with the use of anatomical landmarks. The reason for the remaining inaccuracy of the estimated vertical heart position could be the individual variability of the normal ventricular activation sequence as well as neglect of torso inhomogeneities in the inverse computations. However, serious limitation of this method is the imperative of normal initial ventricular depolarization.

From the results with the torso model B in the second part of the study it implies that adjustment of a generic torso shape according to individual anthropometric measures of the subject and maintaining real electrode positions is a promising way how to obtain subject-specific torso geometry accurate enough for the inverse solution. However, from the results obtained with torso models C and D the great impact of the used heart model on precision of the inverse solution is also apparent.

In the third part of the study the method for individual assessment of the vertical heart position using the inverse localization of early ventricular activation was used in 9 subjects to create their individual torso models (model D). From the graph for model D in Fig. 7 it is apparent that the improvement of vertical position of the heart, without its additional adjustment by proper rotation and scaling did not decrease the lesion localization error in the inverse solution. Individual positioning of the heart model in 3D space along all three coordinates based on the estimated site of the early ventricular activation was not used because it was not always possible to fit the heart model in the torso without additional heart scaling.

The importance ofinformation about heart size and rotation suggests the necessity of some heart imaging (e.g. USG, CT or MRI) even if the whole torso imaging is not available. This issue should be studied further.

The limitation of the forward simulations used in the study was the simplified model of the heart activation and cardiac electrical generator. However, it was sufficient to demonstrate the importance of individually adjusted torso and heart models used in the inverse solution for each examined subject.

The principal limitation of the presented inverse method is the need of BSPMs measured during the ischemia (with changed repolarization phase of the myocytes AP) and also in a situation without the ischemia manifestation. Both measurements in the same subject are necessary for computation of the DIM that is used as the input for the inverse solution. To have such data for a patient admitted with acute myocardial infarction would be extremely difficult. However, such data can be obtained when ischemia is evoked in controlled conditions, e.g. before and after the exercise stress test or by repeated examinations. Another possible application of the inverse method could be in revealing of regions responsible for transient beat-to-beat changes

in ECG, e.g. those expressed as changes of the non-dipolarity index in integral BSPMs reported in [12].

5. Conclusion

From the results obtained in this study it is apparent that the use of a generic torso model with patient-specifically adjusted torso shape and with electrode positions defined in accordance with their real placement allows acceptable inverse localization of pathological cardiac events based on a dipole model of the cardiac electric generator. Accuracy of the solution is only slightly worse than that obtained with individual torso model created from MRI scans. However, the use of reasonably accurate heart model is still necessary.

The use of information from measured ECG signals can improve the individual positioning of the heart model in the torso in comparison to the standard heart position based on the ECG lead V2 level. However, in spite of this result, such information without proper rotation and scaling of the heart model does not lead to improved accuracy of the inverse solution. Hence some heart imaging allowing the creation of a patient/specific heart model seems unavoidable even if the whole torso imaging is not available.

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