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Estimating Infarct Severity from the ECG using a Realistic Heart Model

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Abstract

The early phase of myocardial infarction is accompanied by changes in the ST segment of the ECG. This makes the ST segment the clinical marker for the detection of acute myocardial infarction. The determination of the infarct severity, location and size of the myocardial tissue at risk will support clinical decision making. In this study we used an inverse procedure to estimate the location and size of the infarcted heart region. The method estimates the local transmembrane amplitude based on the ECG amplitude near the J-point of the standard 12 leads signals using a patient specific volume conductor model. For the 5 available patient cases the positions as well as the size of the estimated infarct region were in accordance with results based on MRI.

1. Introduction

The early stages of myocardial infarction (AMI) are accompanied by changes in the electrocardiogram (ECG), of which the changes in the ST segment and T wave are the earliest signs. The ST segment is therefore used in many algorithms to detect AMI. Many algorithms have been developed to identify ST-elevated myocardial infarction (STEMI) and predict the culprit artery from the ECG [1-2], however still with rather varying performance. Therefore in this retrospective study a patient specific approach is used to estimate the infarct region.

In our previous research we have been using patient specific volume conductor models to estimate cardiac activation and recovery [3]. In this study a similar approach is used to estimate the local cardiac transmembrane potential based on the recorded ECG amplitude 40 ms after the end of the QRS complex (J point). The recorded ECGs were just the standard 12 leads signals, in contrast to the 64-256 signals used for the currently [3-4] available inverse procedures, to estimate the electrical activity of the heart.

2. Material

The standard 12 leads ECGs were recorded from five well documented STEMI patients (Table 1) who underwent a percutaneous coronary intervention (PCI) procedure. For each patient a cardiac MRI scan was made to determine the size and location of the myocardial infarction [5]. The scan was recorded at least two days after the PCI procedure. Electrocardiogram-gated images were acquired on a clinical 1.5 Tesla scanner during repeated breath-holds of approximately 10 seconds. The standard 12 lead ECG's were recorded at the same day of the PCI procedure. For each patient an individualized MRI-based volume conductor model was constructed, incorporating the major inhomogeneity's in the conductive properties of the thorax, *i.e.* the lungs, the blood-filled cavities and the myocardium (figure 1).

Table 1. Patient demographics and the volume of myocardial tissue, left and right ventricle combined as computed from the reconstructed heart model (see Figure 1).

Patient	sex	age	myocardial volume cm^3
A	F	60	193
B	M	63	297
C	M	63	290
D	M	66	274
E	M	47	259

3. Methods

The inverse procedure used in our research is based on the equivalent double layer (EDL) source model. The local source strength is the transmembrane potential (TMP) at the surface of the myocardial surface [6]. The amplitude of the TMP was estimated from the recorded 12 lead ECGs. In this analysis only Vr, Vl, Vf and V1-6 were taken into account, using the average of all these signals as a reference (zero mean).

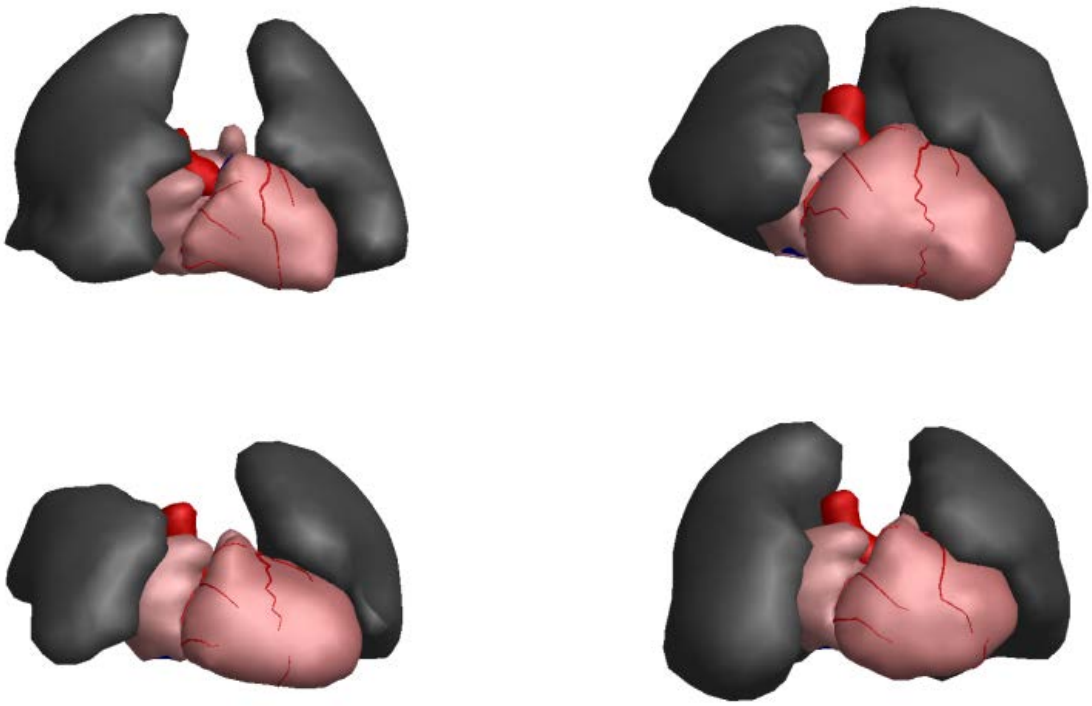

Figure 1 Four of the five patient specific volume conductor models used in this study. From left to right and top to bottom patient A – D. The models differ significantly as can be observed from the images. Especially the heart of patient A is much smaller.

The fiducial points were derived from the 12 lead signals, i.e. onset QRS, end QRS (J-point) and the end of the T wave. A linear baseline correction was applied between onset QRS and the end of the T wave to minimize the atrial contribution in the ST segment. The ECG amplitude to be matched was then taken from the ECG between the J-point + 40 ms and the J-point + 60 ms.

The problem to be solved can now be formulated as; determine the TMP amplitude ($\boldsymbol{S}$) that minimizes the amplitude difference between the measured $\boldsymbol{ECG_{st}}$ at the ST segment, or

$$\boldsymbol{argmin_S(ECG_{st} - A \cdot S)}$$

In which **A** is the transfer matrix expressing the involved volume conductor effects [7].

Rather than treat this problem as an optimization problem, we used an extensive search approach in which different "ischemic" (transmural) regions were tested by reducing the TMP amplitude. First, "ischemic" regions were generated for all nodes on the myocardial surface with the corresponding node acting as the centre of the "ischemic" region. Next the node for which the computed ECG ($\boldsymbol{A \cdot S}$) matches the measured $\boldsymbol{ECG_{st}}$ best is selected. This process is repeated until the difference between measured and simulated ECG increases again.

From the measured ECGs 8-10 beats were selected for analysis. Each analyzed beat resulted in estimated TMP per node on the heart. These TMP amplitudes were averaged over all beats. The estimated infarct location and size was retrieved from the average TMP amplitude map on the heart surface.

The area or volume at risk is derived by taking into account the nodes for which the average TMP amplitude was decreased by more than 15%. For the area only the left endocardial surface was used, because in this study only transmural ischemic regions were created. Hence, the relative surface is a percentage of the LV endocardial surface. The volume of an ischemic region was derived from the surface by multiplying the area by the computed local wall thickness derived from the heart model.

Infarct size analysis was performed by a blinded core laboratory. Segment location was defined on cine and late gadolinium enhancement (LGE) images according to the 17-segment model (figure 2) [5]. Segmental wall thickening was calculated by subtracting end-diastolic from end systolic wall thickness. Dysfunctional segments were defined as segments with systolic wall thickening of less than 3 mm. Total infarct size was calculated by summation of all slice volumes of hyperenhancement, using a standardized and predefined definition (signal intensity > 5 SD above the mean signal intensity of remote myocardium), and expressed as percentage of LV

mass. The transmural extent of infarction was calculated by dividing the hyperenhanced area by the total area of the predefined segment [9]. Segments with more than 50% hyperenhancement were considered segments with transmural infarction.

In order to be able to compare the results from the inverse ECG based method with MRI-analysis, the results were converted to the standard 17-segment bull's-eye maps (figure 3). The calculated TMP amplitudes were projected onto a cone placed along the long axis of the left ventricular chamber, which was subsequently projected on a single plane.

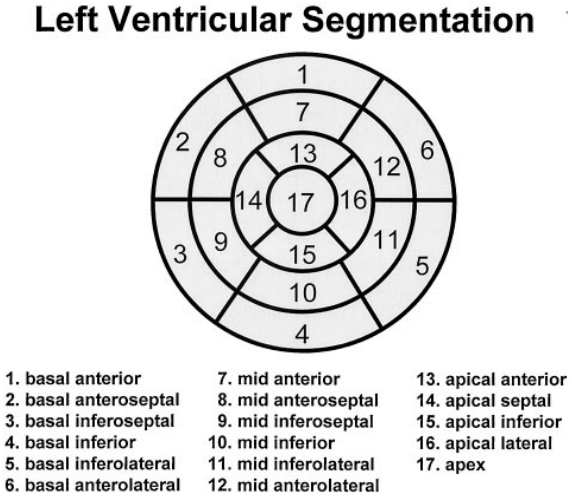

Figure 2. 17-segment bull's-eye plot as recommended by the American Heart Association. This bull's-eye plot is used to project relative decrease in TMP amplitude.

4. Results

In table 2 the ischemic segments of the bull's-eye plot as derived from the reference MRI measurements are given as well as the MRI and ECG based estimated infarcted size as a percentage of the LV wall volume.

Table 2 Left ventricular infarcted region, location, LV segments, and size as % of LV volume. Given values as derived from the reference, MRI determined, and based on new ECG based method. For the ECG based segments see Figure 3.

	MRI reference		ECG estimate	
patient	**infarcted region %**	**LV segments**	**Area %**	**Volume %**
A	8.7	4, 5 (10, 11)	15.3	15.6
B	19.1	4, 10, 15 (5)	17.1	15.6
C	14.9	8, 9, 13, 14	36.6	45.3
D	18.9	8, 9, 13, 14 (2,15)	50.3	41.2
E	9.2	none	27.9	29.3

In figure 3 the standard bull's-eye plot (right panels) for the different patients is shown. The colors indicate the relative change in regional TMP amplitude, blue is no change, red is a 40 % decrease in TMP amplitude. To be able to relate this to the cardiac anatomy the TMP amplitude change distribution is shown on the endocardial and epicardial surface of the heart. The heart is shown in a base to apex view, whereas the standard bull's-eye plot is apex to base. The base to apex bull's-eye plot is added for orientation purposes too (middle panels figure 3).

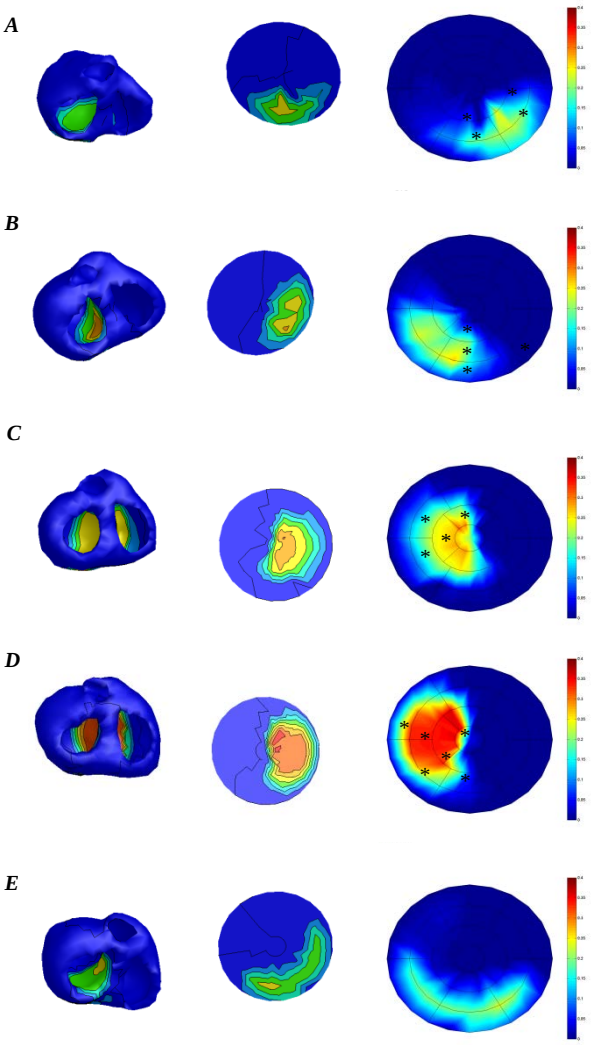

Figure 3 The 5 patient specific models used in this study. Top row is patient A, bottom row patient E. The relative decrease in TMP amplitude is indicated from blue to red, maximum indicated in red is 40 % decrease in TMP amplitude. Left panel: the heart approximately in base to apex view, with the accompanying bull's-eye plots in the middle panel. On the right the bull's-eye plot in the original apex to base view. See figure 2 for the legend of the segments. The black stars mark the ischemic segments identified by the MRI analysis. No LV segments were found for patient E.

The top row of Figure 3 shows the results of patient A with an LCx related region, i.e. the identified region as being ischemic is the inferior wall with parts in the septum. The location is a somewhat more extended into the inferior septum compared to the results from the MRI analysis (see table 2). Patient B and E suffered from an occlusion in the mid RCA. The areas identified with a rise in TMP amplitude are located in the posterior septum and parts of the posterior wall. The identified areas by the ECG method are well related with the location of the RCA, although the locations are somewhat more septal compared to the MRI identified areas. For patient C and D with mid LAD occlusions the position of the identified area matches with the MRI indicated location.

The sizes of the estimated ischemic regions, especially for the LAD cases, were exaggerated when using the endocardial surface or the derived volume (see table 3).

5. Discussion

The position of the ischemic regions based on just the standard 12 leads signals and a patient specific volume conductor model were in accordance with the analysis solely based on the MRI. The size, however, was too large compared to the MRI based results. This might be caused by the fact that the MRI was recorded several days after the PCI procedure. At that time the infarcted region might be smaller than just after the PCI procedure, the time of the ECG recordings. Furthermore the MRI analysis leaves out the apical segment (17) whereas the ECG based method incorporated this segment as well [5].

The projection of the heart geometry on the bull's-eye plot, or the bull's-eye plot derived from the MRI images may need to be adapted. The MRI analysis results seem to indicate somewhat rotated regions compared to those of the ECG based method. However, in all five patients the ECG based method identified regions associated with the occluded artery. Further analysis is required to compare the MRI and ECG based results.

The method assumed transmural ischemic regions, i.e. only transmural ischemic areas were created to match the ST segment of the ECG. In the clinical practice though often the ischemic region starts sub-endocardial and eventually spreads out to a transmural one. The search algorithm needs to be adapted to determine the right region in this highly dynamical process. This might also influence the estimated decrease in TMP amplitude, which is now very prominent for the LAD cases (up to 35 %). In these latter cases the ischemic region is proximal to leads V1-3, resulting in large ST deviations. The standard 12 lead ECG might not be optimal for the determination of ischemic regions. On the other hand the fact that all regions are correctly located indicates that incorporation of prior knowledge, in our case the accurate volume conductor models, enable the correct location of these regions just based on the standard 12 leads.

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