

Electrocardiographic Imaging of Myocardial Infarction Using Heart Vector Analysis

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Abstract

Hypothesis/Objective: The aim of this study is to characterize the location and extent of moderate to large, relatively compact infarcts using ECG evidence.

Method: In this paper, we proposed a method on the basis of vectorcardiography which assumes that heart vector is proportional to relevant active depolarization area(s). To examine our ideas, we used the normal VCG which includes the information of location, amplitude, and direction of heart vector at any instant. The model based comparison of cases under study and relevant normal VCGs gives region i.e. segment(s) and depth i.e. extent of myocardial infarction.

Results and Conclusion: We evaluated the method on CinC/Physionet Challenge 2007 database. In our final entry the scores of EPD equal to 32 (ranked 3rd), SO equal to 0.933 (ranked 3rd) and CED equal to 1 (ranked 1st) are achieved. It also ranked the third among the other methods proposed to CinC/Physionet Challenge 2007.

1. Introduction

Despite the reported decline in the incidence of acute myocardial infarction, in the western world cardiovascular disease is still the number one killer. Consequently, there is an ongoing need to discover new treatment modalities as well as better diagnostic and prognostic algorithms. The Scandinavian Cardiovascular Journal Jensen and co-workers report how quantitative vectorcardiographic analysis can help risk stratify patients in-hospital with the prognosis predicted up to five years follow-up [1]. In this regard, lots of works have done and several methods of myocardial infarction prognosis and detection have proposed.

L Wolff (1955) used Spatial vectorcardiography to study initial, early, and terminal forces related to ventricular depolarization and concluded that changes in the early forces is related to infarction of the free wall of the left ventricle [2]. In 1959, G. Howitt et al. analyzed

the vectorcardiograms of patients with myocardial infarction and normal subjects and the problems of analysis, qualitatively or quantitatively, system of recording and the merits of electrocardiography and vectorcardiography were discussed [3]. In another study, H Abramson (1962) discussed several cases of inferior and anterior wall myocardial infarction in which the vectorcardiogram was proved superior to the electrocardiogram as a diagnostic aid [4]. Later in 1983, M Sederholm et al. used continuous recordings of the X, Y and Z Frank leads to follow ST and QRS vector changes that accompany acute myocardial infarction [5]. Recently, RA Warner et al. (1997) proposed a new method for calculation of the direction of the instantaneous vectors of heart electrical forces and used that to develop some useful criteria for healed inferior myocardial infarction (IMI) [6]. Also, SV Eriksson (1999) used VCG monitoring to identify myocardial reperfusion at an early stage and valuable prognostic information was offered in patients with unstable angina and acute myocardial infarction [7]. In the more recent study, SM Jensen et al. (2003) monitored patients with rigorous continuous ischemia using computerized vectorcardiography and introduced a new electrocardiographic method called STC-VM [1].

The aim of this study is to probe the myocardial infarction in two stands, first to determine the location and center of the infarcted area, and second to determine the extent of infarction. Our method is a model based approach using vectorcardiography analysis which is discussed briefly in the following sections.

2. Materials and methods

In order to demonstrate heart vector during systolic depolarization of ventricles, a normal model is used. This model is based on the normal ranges of heart vector reported by HW Draper et al. [8]. The model includes three cases with minimum, average and maximum amplitudes of heart vector (Figure 1).

The model is then compared and adjusted to the

subject under consideration. By knowing the actual VCG and the relevant VCG model, the location, extent, and centroid of the myocardial infarction evaluated which is briefly described below.

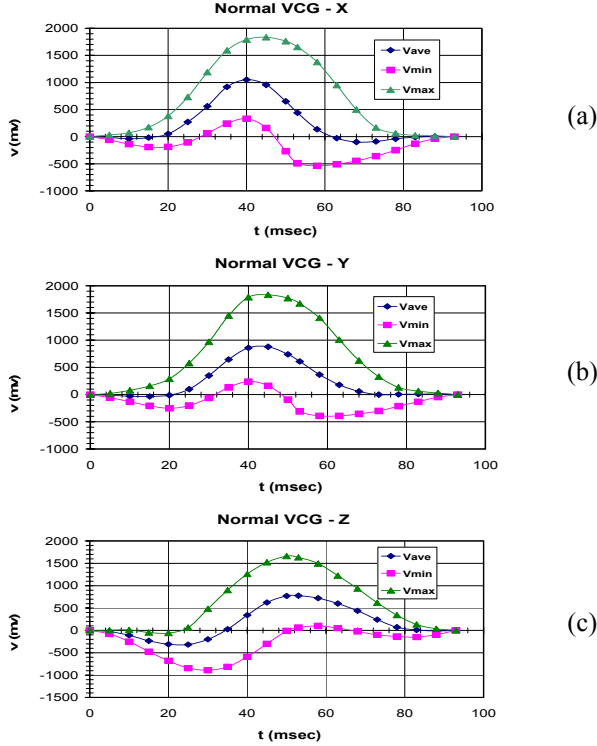


Figure 1. Normal VCG for three cases with maximum, average and minimum amplitudes in the standard XYZ coordination of the body; a) X-axis, b) Y-axis, c) Z-axis

2.1. A model of electrical propagation through ventricles

The propagation of the electrical potential and the relevant current depolarization area can be simply related by a coefficient. This assumption is highly true while the action potentials of myocardial cells are similar, and any area of depolarization includes rather the same concentration of myocardial cells. Thus, we can write:

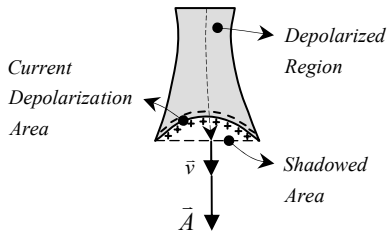


Figure 2. The relation between heart vector and depolarized area in normal cases

$$\vec{v} = \alpha \vec{A} \quad (1)$$

Where \vec{v} is spatial heart vector, \vec{A} is the shadowed area of depolarization which is parallel to \vec{v} , and α is a coefficient relating spatial heart vector to the area of depolarization (Figure 2).

2.2. Effects of myocardial infarction on heart vector

Equation 1 not only describes the relation between total heart vector and current depolarization area, but also exhibits the infarcted area of the heart beforehand. As is shown in Figure 3, the infarcted area forms a bipolar area changing heart vector in both direction and amplitude. The infarcted area also reduces current depolarization area i.e. $A_1 < A^*$, where A^* is the normal depolarization area.

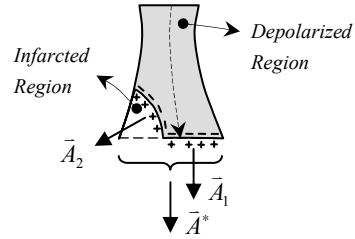


Figure 3. Change in heart vector and relevant active depolarization area caused by MI

Therefore, the depolarization area at any time instant can be formulated as given by Equation 2:

$$\vec{A} = \vec{A}_1 + \vec{A}_2 \quad (2)$$

Where \vec{A}_1 is the area of depolarization at tangential direction defined by \vec{A}^* , \vec{A}_2 is the bipolar area beforehand. Also, the propagation of infarction through myocardial, \vec{A}_3 can be determined as by Equation 3.

$$\vec{A}_3 = \vec{A}^* - \vec{A}_1 \quad (3)$$

In order to determine region and extent of MI \vec{A}_2 and \vec{A}_3 are needed. However, the exact determination of these areas is not possible while just only \vec{A}^* , \vec{A} , and the directions of \vec{A}_1 and \vec{A}_3 are given. But, an approximation can be made assuming that \vec{A}_1 is perpendicular to $d\vec{A}_2$. Therefore, we can write:

$$\vec{A}(y) = \vec{A}_2(y - dy) + dA_2(y) \cdot \hat{e}_n + A_1(y) \cdot \hat{e}_t \quad (4)$$

Where y is the tangential distance from the initial

depolarization point (i.e. septum in normal cases) to the centroid of current depolarization area, \hat{e}_t is a unit vector parallel to the normal heart vector, and \hat{e}_n is a unit vector perpendicular to \hat{e}_t (Figure 4). The boundary condition is assumed to be the same as of the normal heart, i.e. $\bar{A}(0) = \bar{A}^*(0)$, or $\bar{A}_2(0) = \bar{A}_3(0) = 0$. Note that $d\bar{A}_2$ is not necessarily parallel to \bar{A}_2 .

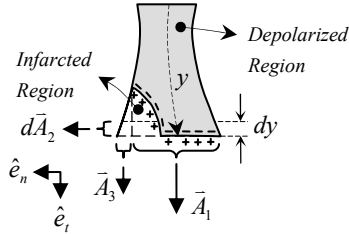


Figure 4. MI region approximated locally by areas of $d\bar{A}_2$ and \bar{A}_3

2.3. Extent of myocardial infarction

The local extent of MI is specified by \bar{A}_3 and the total extent of MI could be evaluated by using Equation 5:

$$Extent = \frac{1}{V} \int_0^S A_3(y) dy \times 100\% \quad (5)$$

Where V is the total Myocardial Volume, and S is the total average distance crossed while depolarization of ventricles. The value of V is not reported in the literature and we implemented an approximation using the local thickness of myocardial [9] which is about 31.5 cm^2 . We assume that S is identical for normal and infarcted cases. On the other hand, the normal propagation velocity of heart vector reported by J Malmivuo et al. [10] is 0.3 m/s for endocardium and is 0.8 m/s for epicardium. Moreover, it takes about 50 msec that epicardium starts to depolarize. Therefore, we can evaluate S by using Equation 6 which is about 5 cm .

$$S = \int_0^T \dot{y}(t) dt \quad (6)$$

Where T is the average temporal duration of depolarization of ventricles which is about 93 msec [8].

The value of coefficient α in Equation 1 can be calculated now:

$$\alpha = \frac{\int_0^T v(t) \dot{y}(t) dt}{\int_0^S A(y) dy} = \frac{1}{V} \int_0^T v(t) \dot{y}(t) dt \quad (7)$$

2.4. Location of myocardial infarction

$d\bar{A}_2$ expresses the local infarcted area on myocardial surface of ventricles. Generally, we know depolarization region at any time instant [10, 11]. Moreover, the direction of $d\bar{A}_2$ can be used for specifying special segment(s) beyond some candidate segments of MI. For instance, existing MI at apex significantly turns the heart vector toward the Y axis or the opposite direction.

3. Results

We evaluated the method on CinC/PhysioNet Challenge 2007 database. There are four subjects, which the first two are for training. In Figure 5 the results of $d\bar{A}_2$ for subjects #3 and #4 are depicted.

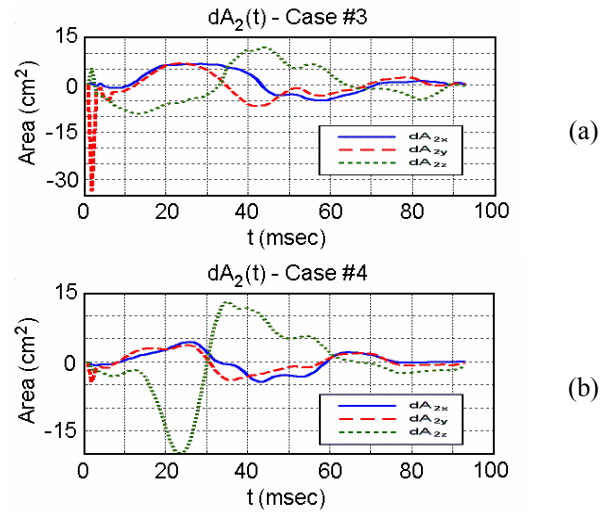


Figure 5. The results of $d\bar{A}_2$ for subjects a) #3, b) #4

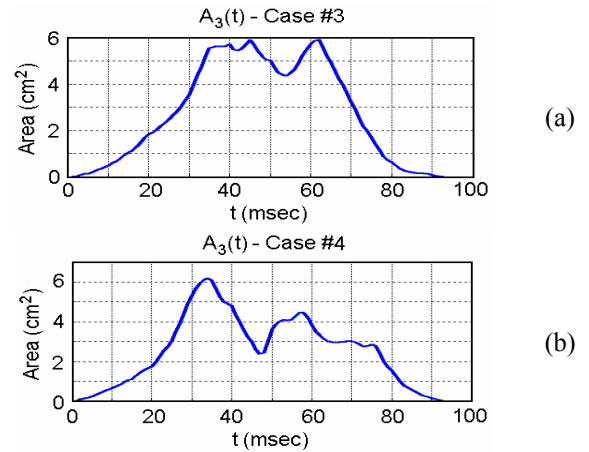


Figure 6. The results of $|\bar{A}_3|$ for subjects a) #3, b) #4

In Figure 6, the results of $|\bar{A}_3|$ for subjects #3 and #4 are depicted.

The Final evaluation of locations and centroids of MI evaluated on the basis of analysis of $d\bar{A}_2$ and the results of percentage extents of MI on the basis of analysis of $|\bar{A}_3|$ are depicted in Table 1. For addressing the locations, the standard segmentation of Figure 7 is used.

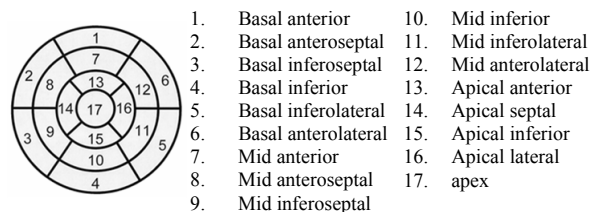


Figure 7. Left ventricular segmentation

Table 1. Result of the method for subjects #3 & 4

Case #	Locations	Centroid	Extent %
3	4-5-6-10-11-15-16	11	41
4	3-4-9-10-14-15	9	35

4. Discussion and conclusions

In this study, we proposed a method of electrocardiographic imaging of myocardial infarction using vectorcardiography. We assessed the method on CinC/Physionet Challenge 2007 database and submitted to entries. The scoring of the results are performed by three component which are EPD (0 to 200; lower is better), SO (0 to 2; higher is better), CED (0 to 8; lower is better), where EPD, SO, and CED stand for Extent Percentage Discrepancy, Segment Overlap, and Centroid Estimated Distance respectively. Our final entry achieved EPD equal to 32 which is ranked 3rd, SO equal to 0.933 which is ranked 3rd, and CED equal to 1 which is ranked tied for 1st. The method also achieved overall rank of 3rd as for event 4.

The individual and total ranks address the proposed method as a reliable and stable one, even though high EPD and low SO are achieved. These errors might be because of approximations made for calculation of $d\bar{A}_2$ and $|\bar{A}_3|$. However, the error reduces near the centroid of MI which causes low COD.

On the other hand, fast implementation and simplicity

of the method are two of its major advantages which made it as one of the best methods proposed. Finally, it should be noted that the method is not applicable for cases with rhythmic abnormalities, bundle branch block, and hypertrophy.

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