ASSESSMENT OF HEART REPOLARIZATION PROPERTIES FROM BODY SURFACE POTENTIAL MAPS

M. Tyšler, M. Turzová, *V. Szathmáry

Institute of Measurement Science and *Institute of Normal and Pathological Physiology,
Slovak Academy of Sciences, Bratislava, Email: umertysl@savba.sk

Abstract

Use of activation-recovery interval (ARI) in surface ECG leads as possible indicator of changed repolarization in the underlying myocardium was studied. Model ECG data representing normal and pathological myocardium repolarization were simulated on surface of realistic inhomogeneous torso. Results of simulations suggest that shortening and prolongation of action potentials in anterior myocardial regions representing more than 6% of the heart volume can be visible in surface ARI maps while only prolongation in postero-lateral regions can be observed. Reproducibility of ARI was checked on real measurements using 32 to 192 ECG leads. Obtained ARI maps exhibited good intra-individual stability with correlation 0.89 to 0.94. From the model and experimental results it has been concluded that analysis of ARI may give some insight into the myocardium repolarization and can help to recognize tissue with changed properties namely in heart regions underlying the anterior chest.

1. Introduction

Ventricular arrhythmias are major cause of sudden cardiac death in many industrial countries. Identification of persons threatened by ventricular arrhythmias plays an important role in improving the health care of cardiac patients. It is widely accepted that increased risk of ventricular arrhythmias is closely connected with inhomogeneity of myocardium repolarization. However, arrhythmic indications that could be obtained by direct evaluation of local refractory periods of myocardial cells, e.g. by measurement of monophasic action potential have limited applicability to clinics and reliable non-invasive methods for assessment of repolarization characteristics have to be searched for.

Several non-invasive methods are based on measurement of time intervals between selected features in surface ECG signals that denote tissue depolarization and recovery. One of these methods [1] is based on relation between action potentials duration (APD) and so-called activation-recovery interval (ARI) that is determined as time interval between the most negative slope in QRS complex and the most positive slope of T wave in each surface ECG signal. Under simplified conditions, this interval can be regarded as some representation of APD in the underlying myocardium, projected on the body surface. It was experimentally shown [1] that despite the smoothing effect of torso volume conductor, there was a high correlation between epicardially recorded APD and superficially measured ARI.

The aim of our model study was to test the possibility of non-invasive assessment of local repolarization properties from spatial distributions of ARI measured in surface ECG leads used for body surface potential mapping. On real data we also checked the reproducibility of measured ARI distributions and the influence of the number of measured ECG leads and applied signal processing methods.

2. Method and Material

To estimate resolution of ARI mapping method, surface ECG signals corresponding to normal activation-repolarization sequence and to sequences with local changes of APD were simulated. Finite element model of heart ventricles with elements of $1mm^3$ was used to simulate normal and pathological cardiac sources. Geometry of ventricles was defined analytically and was based on several ellipsoids. Properties of myocardial cells, such as conduction velocity, action potential (AP) amplitude and duration (APD) were defined for all elements. Five layers with different APD (from 81 to 132 ms), decreasing from endocardium to epicardium, were defined in ventricular walls and in septum. Layer with 3 times increased

conduction velocity was defined on the endocardial surface to simulate Purkinje fibers. Starting points of ventricular activation were set in the endocardial layer with increased conduction velocity and their positions were in agreement with experimentally observed early activated areas in normal human heart. Activation spread was governed by Huygens' principle, isotropic myocardial tissue was supposed [2].

Besides normal heart model, also local repolarization changes with APD shortened or prolonged by 25% from the normal value were simulated. Two typical regions of changed APD were defined in the left ventricle as shown in Fig.1. The first one (a) was located anteriorly near apex, the second one (b) postero-laterally closer to heart base. Diameters of the regions were 16, 32 and 48 mm (marked by dashed spheres in Fig.1) and represented 1.2 to 16.1% of the myocardial volume.

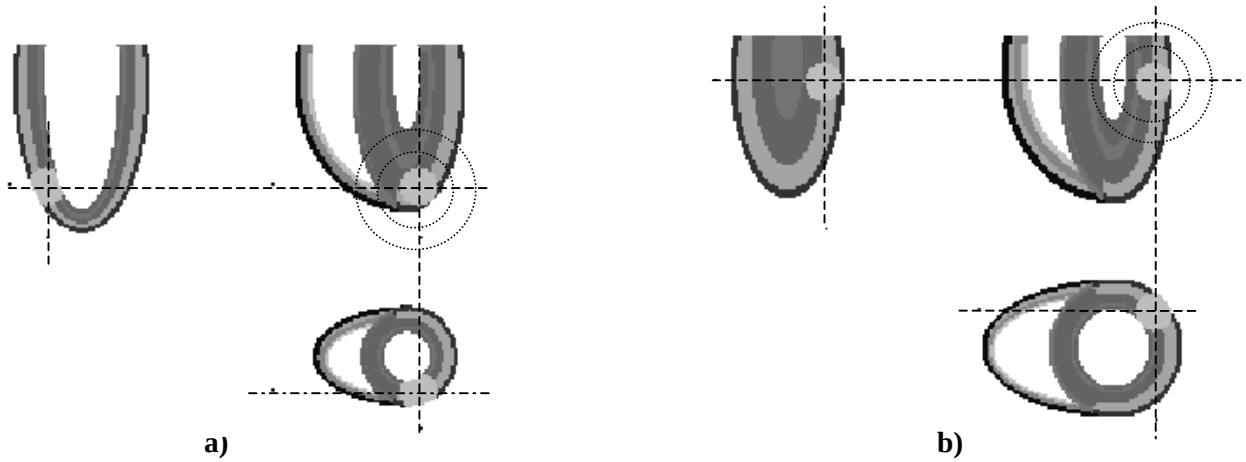


Fig. 1. Myocardium model with two regions of changed repolarization (APD ±25%). Dashed spheres mark borders of analyzed regions of different size. Three orthogonal cuts through the center of each changed region are shown.

a) - anterior region near apex, b) - postero-lateral region close to heart base

Equivalent multiple dipole (MD) representation of the cardiac electric generator was used for computation of electric potentials on the surface of torso volume conductor. Myocardium was divided into 168 segments and activation of each segment was represented by a dipole placed in the weight center of that segment. Each segmental dipole moment was obtained as the sum of dipole moments of elements belonging to the particular segment.

Cardiac generator was placed into a realistic torso model with basic inhomogeneities. Boundary element method was used for computation of potential distribution. Potentials corresponding to particular depolarization-repolarization sequence were computed in 198 points on the model torso surface [3]. ECG signals in 192 points of a 16x12 mapping grid were obtained by linear approximation, digitally filtered and used to compute surface ARI distribution. In each ECG signal, ARI was determined as interval between the most negative derivative in the QRS complex and the most positive derivative in the T wave. Intervals obtained by automated algorithms were manually corrected if necessary. Spatial distributions of ARI were displayed as surface isochronal maps and results were visually analyzed.

To check reproducibility of ARI computations, real distributions of ARI in a group of normal patients were measured using body surface potential mapping (BSPM) techniques implemented in the ProCardio mapping system [4]. 32 leads (set Lux-32a) and 192 leads (16x12 grid, measured in 4 groups of 48 leads) were used. If only 32 leads were measured, potentials in the full mapping grid were approximated first by using specific algorithm for that limited lead set. ARI were then calculated at each mapped site. Changes of ARI surface distribution for several MI patients were also observed.

3. Results

Simulated normal patterns of surface ARI maps were in good agreement with measured data. Changes of APD simulated in anterior regions (AR) were projected mainly in the left antero-lateral superior part of the

torso (in the middle of ARI maps above the transversal level), while APD changes in postero-lateral regions (PR) were projected in the inferior part of posterior torso (right part of ARI maps under the transversal level). Changes of APD in AR caused greater changes in ARI maps. The APD prolongation was demonstrated more clearly then APD shortening.

Example of simulated normal ARI map and maps for regions with prolonged and shortened APD of different sizes and positions are shown in Fig.2. ARI values are represented in gray scale (Fig. 2a), right).

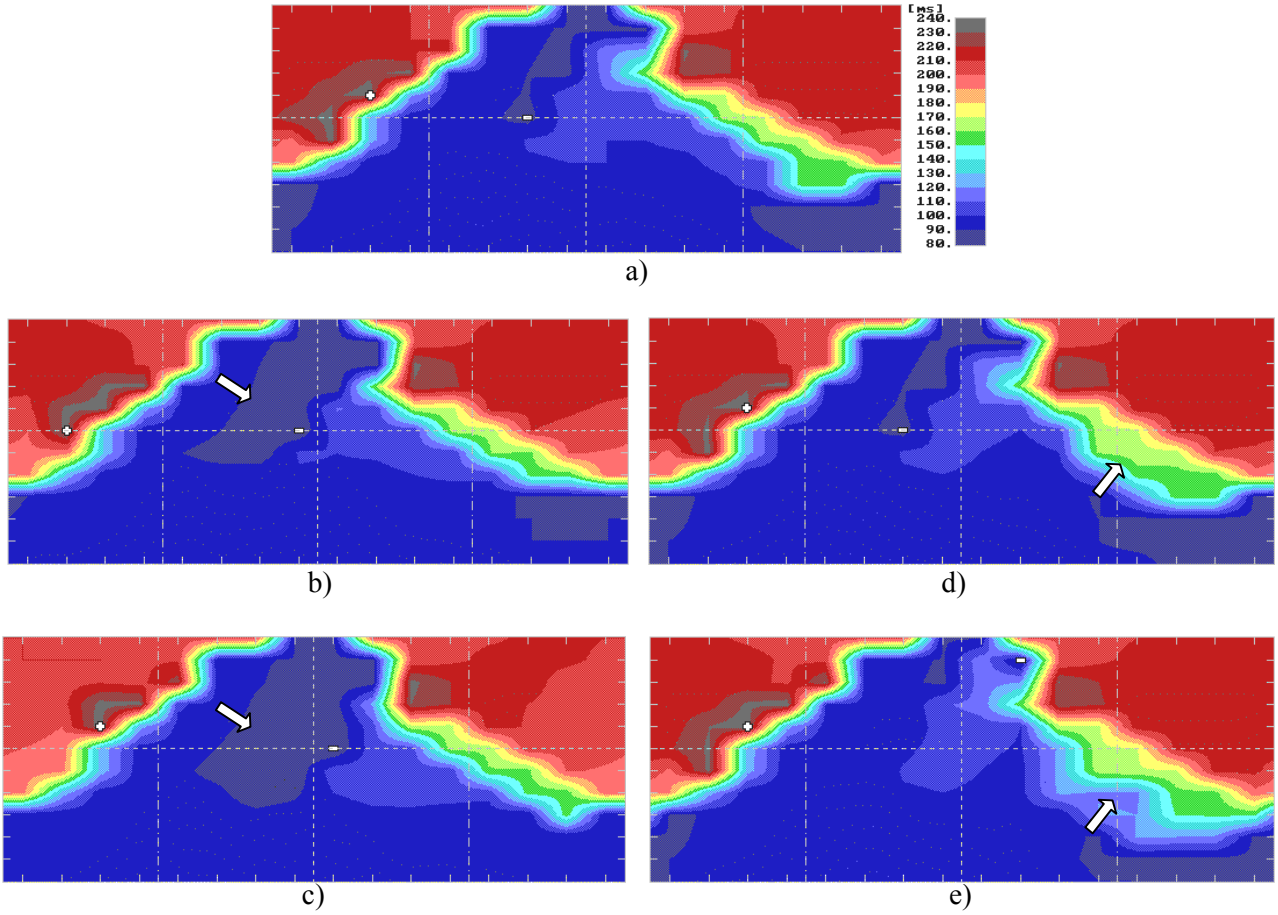
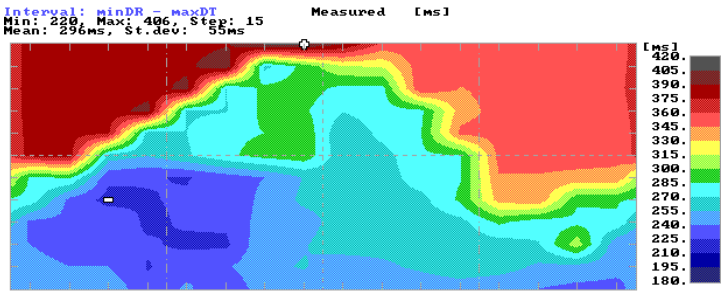


Fig.2. Simulated surface ARI maps: a) normal activation with scale,
b), c) projected APD shortening in anterior regions with diameter of 32 and 48mm,
d), e) projected APD prolongation in postero-lateral regions with diameter of 32 and 48 mm.

Changes of ARI in small regions representing about 1.2% - 1.4% of the myocardium volume (spheres with diameter of 16 mm) could hardly be observed even for prolonged APD. APD shortening in AR representing 5.8% or 14.5% of the myocardial volume (spheres with diameters of 32 or 48 mm) was clearly projected in the ARI maps as illustrated in Fig.2a), b) and APD prolongation was manifested even stronger. When the APD changes were located in PR, APD shortenings could not be observed in surface ARI maps, while APD prolongation could be recognized for regions representing 7.4% or 16.1% of the myocardial volume (spheres with diameters of 32 or 48 mm) as demonstrated in Fig.2d), e).

Maps of ARI intervals on the body surface were computed and observed also for real ECG measurements. To check the reliability and reproducibility of real ARI maps, both, single beat and averaged ECG data (60 seconds) were used. In normal subjects, ARI map patterns with shorter intervals recorded mainly in the inferior and left lateral chest were observed. Longer intervals were present in the right anterior and posterior superior chest (Fig.3, left). ARI interval distribution in post-MI patients deviated from the normal pattern, displaying rise in ARI duration over specific areas, which could reflect functional and

morphological disturbances. Comparison of results obtained from single beat and averaged maps showed that single beat maps of good quality could be used for ARI analysis (Fig.3, right).



measured ECG	correlation	rms error [ms]
192 leads, single beat	0.94	22
32 leads, averaged	0.90	26
32 leads, single beat	0.89	31

Fig.3. Normal ARI map obtained from 192 measured ECG leads averaged over 60 seconds (left) and correspondence (correlation and rms deviation) to maps from single beat and limited lead measurements (right).

4. Discussion and Conclusions

Although the resolution of surface mapping is in principle limited by the smoothing effect of torso, results of our simulations suggest that changes of APD in larger myocardium regions, particularly in regions underlying the anterior chest, can be recognized in surface ARI maps. However, the possibility to recognize APD changes, especially in combination with changed amplitudes of AP and in presence of noise in real ECG signals, needs further analysis. More detailed analysis of T wave morphology and of spatial smoothness of the ARI parameter could allow better identification of local repolarization changes.

Reconstruction of ARI maps from limited number of leads and their reproducibility was checked using several lead sets and both single beat and averaged ECG signals and the results seem fully acceptable. Limitations of the experimental part of our study are the small number of experimental cases and possible methodological errors caused by sequential measurement of the full grid ECG data (4 groups of 48 leads).

From the obtained results we have concluded that ARI maps can be useful for characterization of local variations of cardiac repolarization and may help in recognition and localization of possible arrhythmogenic foci in myocardial tissue.

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