Projection of Local Changes of Ventricular Action Potential to Surface Distribution of Activation-Recovery Intervals.

[1]Tyšler M., [1]Turzová M., [2]Szathmáry V.

[1]Institute of Measurement Science and [2]Institute of Normal and Pathological Physiology SAS, Bratislava, Slovakia

Introduction

Increased risk of ventricular arrhythmia (VA) is closely connected with inhomogeneity of myocardium repolarization [1,2]. However, identification of cardiac patients threatened by VA by direct evaluation of local refractory periods [3] has limited applicability to clinics and reliable non-invasive methods for determining of repolarization characteristics have to be searched for. One of possible methods is based on activation-recovery interval (ARI) measured in the surface ECG as interval between the most negative slope in QRS complex and the most positive slope in T wave [4]. Under simplified conditions, this interval can be considered some representation of the action potential duration (APD) in the underlying myocardium, projected on the body surface [5].

The aim of our model study was to test the possibility of non-invasive assessment of local repolarization properties from spatial distributions of ARI obtained from multiple ECG leads.

Methods

Surface ECG signals corresponding to normal depolarization - repolarization sequence and to sequences with local changes of APD were simulated to estimate the ability of ARI mapping method to identify ventricular areas with changed repolarization properties. Finite element isotropic model of heart ventricles with analytically defined shape was used [6]. Thin layer with three times increased conduction velocity was defined on a part of the endocardial surface to simulate Purkinje fibers and several layers with different APD (81 to 132 ms), decreasing from endo- to epicardium were defined in ventricular walls and in the septum. Starting points of activation were selected in agreement with experimentally observed early - activated areas in normal human heart. Activation spread was governed by Huygens' principle.

To simulate repolarization changes, areas with changed APD were defined and their position, size, conduction velocity and severity of APD change were varied. In this study, local depolarization disorders were simulated by APD changes ±25% from the normal value. Two typical positions (Fig.1) of regions with changed repolarization were examined. The first one (AR) was located anteriorly in the left ventricle and septum near the apex and was close to the torso surface. The second one (PR) was located in postero-lateral part of the left ventricle, close to the heart base and was relatively far from the torso surface. Changed areas with diameters of 16, 32 or 48 millimeters represented 1% to 16% of the ventricular volume.

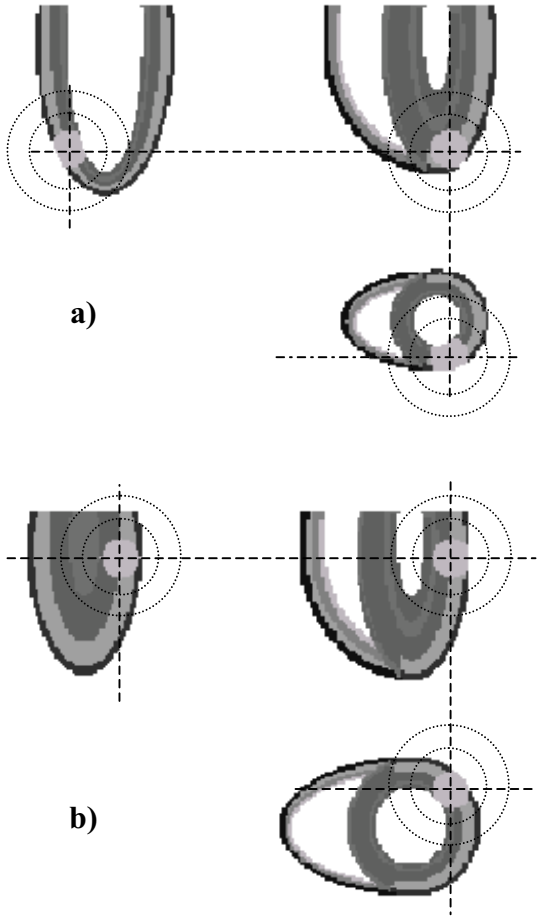

Fig.1. Model of heart ventricles with two regions of changed APD: a) anterior near the apex (AR), b) postero-lateral near to heart base (PR). Dashed circles mark borders of regions of different size. Three orthogonal cuts through the centers of the regions are shown.

Equivalent multiple dipole (MD) representation of the cardiac electric generator with 168 dipoles was used for computation of potentials on the surface of an inhomogeneous realistic torso. From the simulated potentials, ECG signals were derived in 192 points of a 16x12 mapping grid, were digitally filtered and used for computation of ARIs. Results were displayed as surface isochronal maps and further analyzed. Intervals from signals with multi-phasic or negative T waves in the upper right anterior and posterior torso were not evaluated.

Results

Simulated surface ARI maps with masked areas of negative and multi-phasic T wave are shown in Fig.2. All ARI values (in milliseconds) are represented by the same gray scale. Surface ARI map representing normal repolarization-depolarization sequence was in good agreement with measured maps and is shown in Fig.2a).
Changes of APD in small AR and PR regions representing 1.2% to 1.4% of the ventricular volume (spheres with diameter of 16 mm) could hardly be observed for both, prolonged and shortened APD. In AR representing 5.8% or 14.5% of the ventricular volume (spheres with diameters of 32 or 48 mm) APD shortening was clearly projected in the left antero-lateral superior part of the torso (in the middle of ARI maps above the transversal level) as shorter ARIs as illustrated in Fig.2 b) and c) and APD prolongation was manifested even stronger as longer ARIs in the same map area. When the APD changes were located in PR, APD shortenings could not be reliably identified in surface ARI maps, while APD prolongation in regions representing 7.4% or 16.1% of the ventricular volume (spheres with diameters of 32 or 48 mm) could be recognized in the inferior part of posterior torso (right part of ARI maps below the transversal level) as areas of longer ARIs as illustrated in Fig.2d) and e).
For quantitative evaluation of differences between ARI maps corresponding to normal activation and activations with changed APD, correlation coefficients and relative root mean square deviations from normal ARI map were used. Obtained characteristics are presented in Table 1. With regards to the possible intra-individual variability of the ARI values in real measurements [7], only differences represented by correlations less than 0.7 and relative rms deviations greater than 7% were considered significant and are shaded in the table. In anterior myocardium, prolongation of APD was stronger reflected in rms deviations, while APD shortening in correlation coefficients corresponding to changed ARI maps patterns. In postero-lateral myocardium, only APD prolongation in area with diameter of 48 mm brought significant differences in ARI map.

Table 1. Comparison of ARI maps corresponding to local changes of APD with a normal ARI map. Correlation coefficients and relative rms deviations for different positions and sizes of region with changed APD are shown.

Position of the region	**Size** ø[mm]	**APD + 25%**		**APD – 25%**	
		correl.	rel.dev [%]	correl.	rel.dev [%]
Anterior	16	0.98	1.1	0.72	4.1
	32	0.64	7.7	0.39	6.2
	48	0.50	14.6	0.09	7.8
Postero-lateral	16	0.99	0.6	0.98	1.0
	32	0.95	2.1	0.93	2.2
	48	0.49	8.7	0.71	4.4

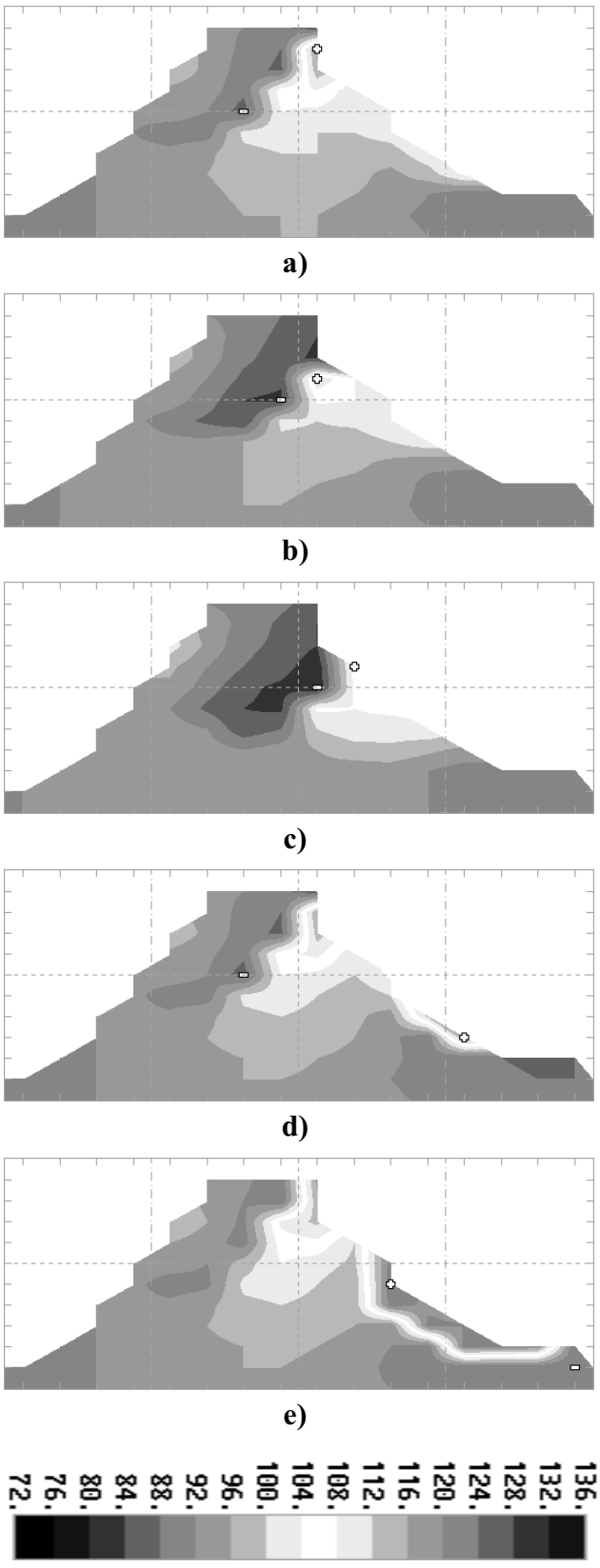

Fig.2. Simulated surface ARI maps.
Gray scale corresponds to ARI values in milliseconds.
a) normal depolarization – repolarization,
b) APD shortening in anterior region (5.8% volume),
c) APD shortening in anterior region (14.5% volume),
d) APD prolongation in postero-lateral region (7.4%),
e) APD prolongation in postero-lateral region (16.1%)

Discussion

To check the reliability and reproducibility of real ARI maps, body surface potential maps were measured by ProCardio mapping system and surface ARI maps were computed from both, single beat and averaged ECG data. In contrast to more often used QT intervals, obtained surface distribution of ARI values exhibited good intra-individual stability with correlation between ARI maps above 0.7, depending on the number of measured leads and used ECG processing methods. Comparison of results obtained from single beat and averaged maps showed that also single beat data of good quality could be used for ARI analysis. Reconstruction of ARI maps from limited number of leads was checked using several lead sets and results from 63 or more leads seem acceptable.

In normal subjects, patterns with shorter intervals mainly on the inferior and left lateral chest surface were observed, while longer intervals were present in the superior part of the right anterior and posterior chest. ARI interval distribution in post-MI patients deviated from the normal pattern and could reflect functional and morphological disturbances. Simulated normal ARI maps used in this study were in good agreement with measured ARI maps.

Although the resolution of body surface potential mapping is in principle limited by the smoothing effect of torso, results of our simulations suggest that changes of APD in larger myocardium regions (representing more than 6% of the ventricular volume), particularly in regions underlying the anterior chest, can be recognized in surface ARI maps. On the other hand, changes of APD can be hidden in ARI artifacts arising from body surface areas with more complex ECG signals where the evaluation of ARI is problematic.

In our presentation, body surface areas with negative or multi-phasic T wave over the upper right anterior and posterior torso surface reflecting the endocardial activation (and also atrial activation in real measurements) were excluded from the analysis (Fig.3). Only transmural lesions were introduced and only the action potential duration of the myocardial cells was changed without changing the resting value or the amplitude of the action potential. Inclusion of all these factors will lead to additional changes in surface ECG, namely to ST segment elevation or depression and to changed polarity of the T wave. Possibility to recognize APD changes under these circumstances and in presence of noise in real ECG signals needs therefore further analysis. Because of the more complex ST-T morphology, modification of the ARI evaluation that would account for the changed morphology can be expected.

Despite of the simplifications of the used model, results of our simulations suggest that ARI maps may give some insight into the underlying repolarization process of the myocardium and may help in recognition and localization of the tissue with changed APD, namely if the lesions are in heart segments close to the anterior chest surface.

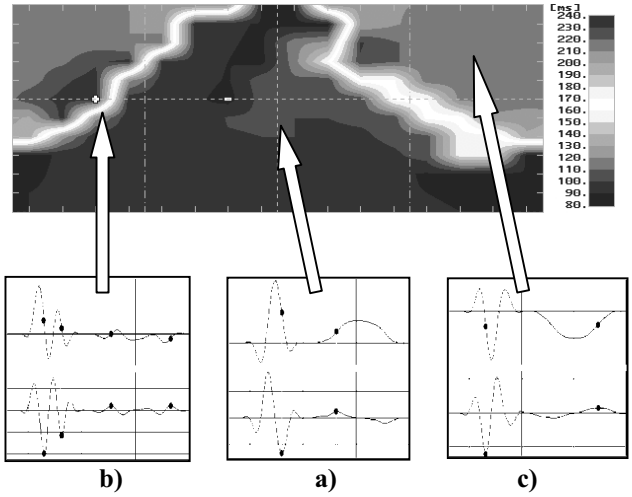

Fig.3. Normal simulated surface ARI map. Areas of different morphologies of ECG curves can be recognized: (ECG signals and their derivatives with marked possible activation and recovery time instants are shown)

a) 'regular' ECG shape included in ARI analysis,
b) 'transition' ECG shape reflecting several activation events, excluded from ARI analysis
c) 'inverted' ECG shape reflecting also endocardial activation, excluded from ARI analysis

References

1. Burgess M.J.: Relation of ventricular repolarization to electrocardiographic T-vawe form and and arrhytmia vulnerability. Am J Physiol, 1979, 236: H391-H402.
2. Kuo C.S., Munakata K., Reddy C.P., et al: Characteristics and possible mechanism of ventricular arrhythmia dependent on the dispersion of action potential durations. Circulation 1983, 67: 1356-1367.
3. Franz M.R.: Bridging the gap between basic and clinical electrophysiology: What can be learned from monophasic action potential recordings? J Cardiovasc Electrophysiol, 1994, 5: 699-710.
4. Mirvis D.M.: Electrocardiography. A physiologic Approach. St.Louis, Mosby, 1993.
5. Sandor G: New signal processing methods of the electrocardiology with special regards to assessment of the repolarization. Dissertation thesis, Budapest, 1999.
6. Szathmáry V., Ruttkay-Nedecký I.: Model study of effects of different repolarization patterns in the left and right ventricle on the resultant cardiac vectors. In.: Abstract book, XXVIIth international Congress on Electrocardiology, University of Milan, 2001, 74.
7. Tyšler M., Turzová M., Filipová S.: Spatial distribution of QT-intervals in body surface potential maps from limited leads. In.: Electrocardiology 2000, Proceedings of the XXVIIth international Congress on Electrocardiology, University of Milan, 2001 (in press).

Acknowledgements

This work was supported by grants 2/1135/21 and 2/7234/21 from the VEGA grant agency.