Correlation between electrical and mechanical properties of the left ventricle in patients with postinfarction ventricular tachycardia

Wojciech Wróbel, Krystian Wita, Andrzej Hoffmann, Cezary Czerwiński, Anna Maria Wnuk-Wojnar, Anna Rybicka-Musialik, Zbigniew Tabor, Artur Filipecki, Maria Trusz-Gluza

1st Department of Cardiology, Silesian Medical University, Katowice, Poland

Abstract

Background: Electroanatomical mapping allows differentiation between viable and scarred myocardium. Echocardiography is widely used to assess myocardial contractility. The relationship between electrophysiological and echocardiographic assessment of left ventricular function has not yet been well established.

Aim: To correlate mechanical and electrical function of the left ventricle in patients with postinfarction ventricular tachycardia and to assess clinical, echocardiographic and angiographic parameters affecting regional electrical function.

Methods: In 32 patients (25 males, 64±9 years old) mean unipolar (UP) and bipolar (BP) voltages were obtained with electroanatomical mapping (CARTO system) for a 12-segment model and compared with segmental wall motion function scored as normal, hypokinetic and a- or dyskinetic. UP voltage in individual groups of segments was: 7.8±4.2 mV, 6.5±4.2 mV, 4.7±2.5 mV, $p < 0.01$ and for BP voltage 2.1±1.5 mV, 1.9±1.9 mV, 1.1±1.0 mV, $p < 0.01$, respectively. Left ventricular ejection fraction ≤30%, end-diastolic diameter >56 mm, previous inferior or anterior myocardial infarction (MI), MI ≤5 years and open infarct-related artery were associated with lower voltage in normokinetic segments.

Conclusions: Segments with advanced systolic dysfunction had significantly lower uni- and bipolar voltage than normo- and hypokinetic segments. However, preserved local electrical function could be found in a/dyskinetic regions. Left ventricular remodelling, time and location of MI and patency of infarct-related artery influenced voltage in normokinetic segments.

Key words: ventricular tachycardia, echocardiography, electroanatomical mapping

Kardiol Pol 2007; 65: 24-29

Introduction

Left ventricular (LV) remodelling after myocardial infarction (MI) is associated with cavity dilatation, changes in shape, hypertrophy and deterioration in contractile function. Malignant ventricular arrhythmias are an important cause of death after MI, especially in patients with advanced LV dysfunction. It seems that LV remodelling provides an important substrate for life-threatening ventricular arrhythmias such as ventricular tachycardia (VT) and ventricular fibrillation (VF). Even in patients with an implantable cardioverter-defibrillator (ICD), recurrent VT remains a management challenge. Catheter ablative therapy offers the ability to eliminate frequent VT.

Electroanatomical mapping with a low-intensity magnetic field allows exact localisation of the electrode to register electrical activity from different places of the LV endocardium, assess activation sequence and create voltage maps. Previous studies showed that endocardial electroanatomical assessment in patients with prior MI and regional wall motion abnormalities can differentiate viable from scarred myocardium. Marchlinski et al. established bipolar voltage criteria

Address for correspondence:
Wojciech Wróbel MD, ul. Ligocka 5/16, 40-570 Katowice, tel.: +48 504 935 197, e-mail: wowr@wp.pl

Received: 04 May 2006. **Accepted:** 30 August 2006.

Acknowledgements: *The study was supported by a grant awarded by the State Committee for Scientific Research.*

of 1.5-2.0 mV for a normal signal recorded from LV endocardium, 0.5-1.5 mV for border zone and <0.5 mV for scarred myocardium [1]. Conducting channels of viable myocardium can be found in scar regions. Voltage mapping can elucidate VT reentry loops suitable for catheter ablation.

We hypothesised that there is a close relation between regional systolic function/dysfunction assessed by echocardiography and electrical function measured by the electroanatomical CARTO system in patients with LV post-MI dysfunction and VT. We also assessed some clinical, electrocardiographic, angiographic and echocardiographic indices which could affect regional electrical function, especially in the normokinetic region of postinfarction remodelling myocardium, because these relations have not been explored systematically.

Methods

Patient population

Thirty-two consecutive patients selected for catheter ablation of recurrent and drug-resistance VT were enrolled in this retrospective study. All patients had a history of at least one MI. Exclusion criteria were: inability to advance a catheter to the LV (e.g. high grade of aortic valve stenosis, dissecting aortic aneurysm) and contraindication for fluoroscopy (e.g. pregnancy). Informed consent was obtained before the procedure.

Before the ablation procedure, all patients underwent clinical, electrocardiographic and echocardiographic examination. Coronary angiography was repeated if necessary for clinical decision making.

Echocardiography

Echocardiography was performed with a 2.0-2.5 MHz transducer using a Sonos 2000 ultrasound machine (Hewlett Packard, USA; years 2001-2002) or Vivid 7 system (GE Vingmed Norway; years 2003-2005) within 1-3 days before electroanatomical mapping.

The end-diastolic (EDD) and end-systolic (ESD) diameters, end-systolic (ESV) and end-diastolic (EDV) volumes, ejection fraction (EF) and valve function were assessed. Segmental wall motion was visually evaluated and scored as normal, hypokinetic, akinetic or dyskinetic in a 16-segment model according to the American Society of Echocardiography recommendations by two independent echocardiographists [2].

Mapping procedure

All patients underwent electroanatomical mapping with the nonfluoroscopic CARTO (2001-2003) or CARTO XP system (2004-2005, Biosense Webster Inc.). Under fluoroscopic guidance the reference electrode(s) was (were) introduced to the right heart (apex of the right ventricle – coronary sinus – near the His bundle). Mapping of the LV was performed with a deflectable ablation catheter (Navistar, Biosense Webster Inc.) during sinus rhythm. The catheter was moved throughout the LV endocardium, local unipolar/bipolar voltages were registered at different locations or voltage maps were constructed by interpolation. Anatomic landmarks such as a mitral ring and aortic valve were marked.

The patients were heparinised to avoid thrombus formation.

Segmental analysis

The LV maps were divided into three equal parts (apex, midventricle and base) by two experienced electrophysiologists. These three parts were further divided into four regions: anterior (80°), lateral (80°), inferoposterior (80°) and septal (120°) to obtain a 12-segment model of LV (Figure 1).

According to Langenhove et al. the 16-segment echocardiographic division was reduced to 12 segments to achieve comparable sets of data [3]. The segments were divided into three groups according to regional

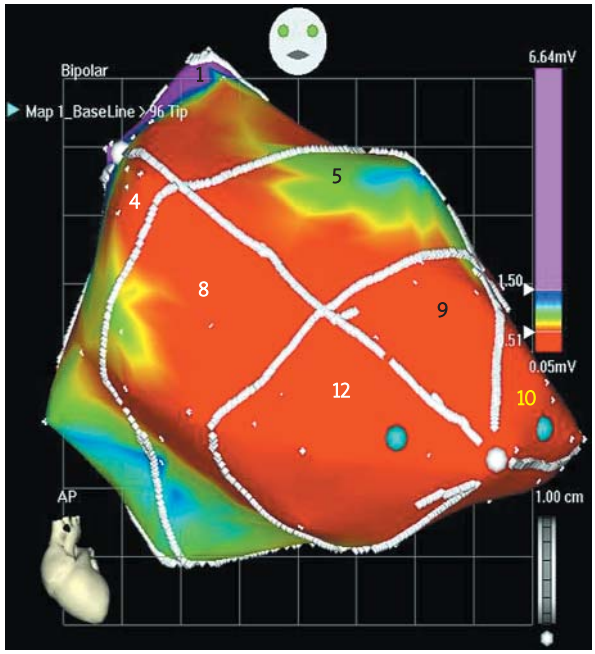

Figure 1. Left ventricular segments in a 12-segment model. Number in: black – anterior wall, yellow – lateral wall, white – interventricular. Bipolar voltage scale: purple – normal heart muscle (BP voltage >1,5 mV), red – scar area (BP voltage <0,5 mV)

Table I. Characteristics of patients

Parameter	Mean ± SD or number [%]
Gender (F/M)	7 (22%)/25 (78%)
Age [years]	64±9
NYHA class III or IV	8 (25%)
CCS class ≥II	2 (6%)
Previous MI: anterior/inferior/ anterior and inferior	4 (44%)/113 (40%)/5 (16%)
Hypertension	10 (31%)
Diabetes mellitus	4 (12%)
VT only/VT+VF	24 (75%)/8 (25%)
Revascularisation: CABG/PCI/CABG+PCI	6 (19%)/12 (37%)/0 (0%)
EF [%]	33±10
WMSI	1,8±0,34
EDD [mm]	64±7
ESD [mm]	49±8
IVS [mm]	10±2
PW [mm]	10±1
EDV [ml]	271±162
ESV [ml]	198±133
LA [mm]	43±5
MVI (grade ≥III)	7 (22%)
TVI (grade ≥III)	1 (3%)

Abbreviations: NYHA – New York Heart Association classification, CCS – Canadian Cardiovascular Society classification, MI – myocardial infarction, VT – ventricular tachycardia, VF – ventricular fibrillation, CABG – coronary artery bypass grafting, PCI – percutaneous coronary intervention, EF – ejection fraction, WMSI – wall motion score index, EDD – end-diastolic diameter, ESD – end-systolic diameter, IVS – interventricular septum, PW – posterior wall, EDV – end-diastolic volume, ESV – end-systolic volume, LA – left atrium, MVI – mitral valve insufficiency, TVI – tricuspid valve insufficiency, SD – standard deviation

function (normo-, hypo- and a- or dyskinetic). Segments with potentials recorded at fewer than three points during mapping were excluded from the analysis.

Statistical analysis

ANOVA and Student's t-test for unpaired parametric samples and Kruskal-Wallis, median test and U Mann-Whitney test for nonparametric samples were used. A p value <0.05 was considered significant. Logistic regression analysis was applied to assess factors influencing voltage.

Results

Clinical and echocardiographic characteristics of the patients are shown in Table I. The majority of them had multivessel coronary artery disease and low EF (median =30%). In 18 (56%) patients percutaneous or surgical revascularisation was performed, but only in 4 cases was it complete.

Thirty-one patients underwent ICD implantation before mapping and in one patient the device was implanted 1 month later. The mean time from MI was 15±8 years (range: 3 months to 27 years).

After initial examination of 512 segments, 65 (13%) segments were excluded due to insufficient quality to evaluate contractility and 52 (10%) segments could not be assessed because fewer than 3 points per segment were registered during the mapping procedure or the quality of the signal was not optimal. Finally, after reduction from a 16-segment to a 12-segment model, 282 comparable segments including 2664 points were available for analysis. A mean of 97 points were sampled per patient map.

Differences between unipolar (UP) and bipolar (BP) voltages among all groups of segments are presented in Table II. Both UP and BP voltages were significantly lower in the segments with advanced systolic dysfunction. Moreover, the UP voltage in hypokinetic segments was lower than in normokinetic segments

Table II. Mean unipolar and bipolar voltages for individual LV segments differing from contraction to each other

Echo scores	Segments [%]	Points [%]	Unipolar voltage [mV]	Bipolar voltage [mV]
			Mean±SD	Mean±SD
1 – Normokinesia	132 (46.5%)	1216 (46%)	7.8±4.2[a), b)]	2.1±1.5 [b)]
2 – Hypokinesia	84 (30%)	850 (32%)	6.5±4.2[a), b)]	1.9±1.9[b)]
3 – Akinesia + dyskinesia	66 (23.5%)	598 (22%)	4.7±2.5 [b), c)]	1.1±1.0[b), c)]
Total	282	2664	6.7±4.0	1.8±1.6

[a)] p <0.01 for normokinetic vs. hypokinetic segments;
[b)] p <0.01 for normokinetic vs. akinetic+dyskinetic segments and hypokinetic vs. akinetic+dyskinetic segments;
[c)] p <0.01 for all groups of segments

(p <0.01). The high standard deviation in a- and dyskinetic segments proves the presence of points with higher unipolar and bipolar voltage. In 56% of those segments bipolar voltage amplitude was ≥0.5 mV and in 19% >1.5 mV. This demonstrates the electrical heterogeneity of the postinfarction scar.

Taking into account specific regions of low electrical activity (such as the fibrous ring of the mitral valve) similar estimation was performed after excluding basal segments. There was no significant difference compared to earlier results.

In normokinetic segments, voltages were analysed against angiographic and echocardiographic parameters, MI location and time from MI (Tables III and IV). The mean unipolar and bipolar voltage was significantly lower in normokinetic segments in patients with dilated LV. Normokinetic segments of patients with EF ≤30% and after both inferior and anterior MI also had lower unipolar voltage. Patients with occluded infarct-related artery and with remote MI had higher voltage in normokinetic regions than patients with open infarct-related artery and more recent infarction. Logistic regression analysis revealed that only low EF was a significant factor for low bipolar voltage (≤0.5 mV) in normokinetic segments.

Discussion

In the present study a very special group of patients with low left ventricular EF and a history of recurrent VT after MI was analysed. Our results show that akinetic and dyskinetic segments had significantly lower values of both unipolar and bipolar voltages than normokinetic and hypokinetic segments. Previous studies have also identified that electroanatomical endocardial mapping could distinguish infarcted myocardium from non-infarcted zones. Kornowski et al. showed that myocardial segments with fixed perfusion defects (assessed with SPECT) had the lowest average voltage potentials (UP: 7.5±3.4 mV) [4]. Low values (UP: 4.6±1.9 mV), almost identical to those in our patients, were reported by Perin et al. in segments assessed as transmural scar by cardiac magnetic resonance imaging [5]. According to Marchlinski et al. a bipolar voltage <0.5 mV is typical for scarred areas [1]. Our study indicates that even in the akinetic and dyskinetic segments there are some points with normal or only moderately diminished voltage. Thus, segments that are akinetic on echocardiography do not necessarily reflect scarred tissue, because viability may be present. Recently, Arenal et al. also demonstrated that the scar tissue is not homogeneous and that there are areas of higher voltage which could form slowly conducting channels inside the scar participating in the VT circuit [6].

In our patients, average voltage potentials in normokinetic segments (UP: 7.8±4.2 mV, BP: 2.1±1.5 mV) were lower than in other studies in patients with normal or mild LV dysfunction [7-9]. In patients with prior MI, Kornowski et al. recorded average voltages of 17.8±4.6 mV (UP) and 4.5±1.1 mV (BP) in healthy zones remote from MI [4]. Perin et al. also demonstrated higher unipolar voltage in normal segments (11.6±4.5 mV) than in the present study [5]. It seems that

Table III. Mean unipolar and bipolar voltage in normokinetic segments against some clinical, echocardiographic and angiographic parameters

Parameter	Mean UP±SD [mV]	Mean BP±SD [mV]	Parameter	Mean UP±SD [mV]	Mean BP±SD [mV]	UP p	BP p
EDD ≤56 mm	14.7±6.2	3.9±2.2	EDD >56 mm	7.3±3.6	2.0±1.4	<0.01	<0.01
EF >30%	9.0±4.2	2.3±1.6	EF ≤30%	6.4±3.8	1.8±1.4	<0.01	NS (0.07)
Open IRA	6.7±3.3	1.5±1.2	Occluded IRA	8.2±4.5	2.3±1.6	NS (0.06)	<0.01
MI ≤5 years	5.5±2.7	1.3±1.1	MI >5 years	8.5±4.4	2.3±1.5	<0.01	<0.01

Abbreviations: UP – unipolar voltage, BP – bipolar voltage, SD – standard deviation, EDD – end-diastolic diameter, EF – ejection fraction, IRA – infarct-related artery, MI – myocardial infarction

Table IV. Mean unipolar and bipolar voltage in normokinetic segments depending on myocardial infarction location

Parameter	Anterior MI	Inferior MI	Anterior and inferior MI
Mean UP±SD [mV]	8.2±4.9[a)]	8.4±3.6[b)]	5.4±2.5[a), b)]
Mean BP±SD [mV]	2.3±1.6	2.1±1.5	1.6±1.3

Abbreviations: UP – unipolar voltage, BP – bipolar voltage, SD – standard deviation, MI – myocardial infarction
a) p <0.05 for anterior MI vs. both anterior and inferior MI;
b) p <0.05 for inferior MI vs. both anterior and inferior MI

these differences are due to advanced dysfunction and remodelling after MI in a substantial number of our patients. Left ventricular remodelling is characterized by progressive dilatation, hypertrophy, diminished contractility and distortion of cavity shape.

In the present study we found significantly diminished voltage potentials in normokinetic segments in patients with either prior anterior and inferior MI, poor LV systolic function or dilatation. Patients with advanced LV dysfunction (EF ≤30%) were characterised by the lowest average values of UP (6.4±3.8 mV; p <0.01) and BP (1.8±1.4 mV) voltages in normokinetic segments. Similar analysis of electroanatomical mapping data was performed in patients with chronic severe ischaemic cardiomyopathy and a mean EF of 31% by Samady et al., who found higher UP voltage in normal segments (10.5±4.7 mV) [9]. It should be stressed that these patients had no history of malignant ventricular arrhythmias and, despite low EF, had a lesser degree of LV dilatation.

In addition, we noted that average voltage potentials were higher when measured in normokinetic segments of patients with occluded infarct-related artery and with remote MI. The impact of myocardial hypertrophy on electrical activity is a possible explanation.

Study limitations

This was a retrospective study. The goal of electroanatomical mapping was successful ablation of VT circuits, and areas closer to the ablation target were mapped more densely than other areas. Thus, as in other studies, segments with fewer than 3 acquired points were excluded from analysis.

Another disadvantage of this assessment is possible misalignment of the corresponding regions leading to findings discordant with the method. Additionally, standard two-dimensional echocardiographic evaluation of regional wall motion is dependent on the reviewer's ability and is characterised by limited accuracy.

We expect that the use of more advanced echocardiographic modalities (e.g. tissue Doppler techniques) and electroanatomical mapping (e.g. CARTO Merge technology) could help to increase our knowledge about the relation between electrical and mechanical function in patients with postinfarction LV dysfunction.

Conclusions

Uni- and bipolar voltages in segments with advanced systolic dysfunction significantly differ from normo- and hypokinetic segments.

Preserved local electrical function indicating viable tissue could be found in a/dyskinetic segments.

Previous both anterior and inferior MI, LV dilatation and low EF were strongly related to lower voltage in normokinetic myocardium. Only low EF was associated with bipolar voltage <0.5 mV in these areas. On the other hand, open infarct-related artery and more recent history of MI were associated with lower voltages in normokinetic segments.

References

1. Marchlinski FE, Callans DJ, Gottlieb CD, et al. Linear ablation lesions for control of unmappable ventricular tachycardia in patients with ischemic and nonischemic cardiomyopathy. *Circulation* 2000; 101: 1288-96.
2. Schiller NB, Shah P, Crawford M, at al. Recommendations for quantitation of the left ventricle by two-dimensional echocardiography. American Society of Echocardiography Committee on Standards, Subcommittee on Quantitation of Two-Dimensional Echocardiograms. *J Am Soc Echocardiogr* 1989; 2: 358-67.
3. Van Langenhove G, Hamburger JN, Roelandt JR, et al. Comparison of mechanical properties of the left ventricle in patients with severe coronary artery disease by nonfluoroscopic mapping versus two-dimensional echocardiograms. *Am J Cardiol* 2000; 86: 1047-50.
4. Kornowski R, Hong MK, Gepstein L, et al. Preliminary animal and clinical experiences using an electromechanical endocardial mapping procedure to distinguish infarcted from healthy myocardium. *Circulation* 1998; 98: 1116-24.
5. Perin EC, Silva GV, Sarmento-Leite R, et al. Assessing myocardial viability and infarct transmurality with left ventricular electromechanical mapping in patients with stable coronary artery disease: validation by delayed-enhancement magnetic resonance imaging. *Circulation* 2002; 106: 957-61.
6. Arenal A, del Castillo S, Gonzalez-Torrecilla E, et al. Tachycardia-related channel in the scar tissue in patients with sustained monomorphic ventricular tachycardias: influence of the voltage scar definition. *Circulation* 2004; 110: 2568-74.
7. Keck A, Hertting K, Schwartz Y, et al. Electromechanical mapping for determination of myocardial contractility and viability. A comparison with echocardiography, myocardial single-photon emission computed tomography, and positron emission tomography. *J Am Coll Cardiol* 2002; 40: 1067-74.
8. Koch KC, vom Dahl J, Wenderdel M, et al. Myocardial viability assessment by endocardial electroanatomic mapping: comparison with metabolic imaging and functional recovery after coronary revascularization. *J Am Coll Cardiol* 2001; 38: 91-8.
9. Samady H, Choi CJ, Ragosta M, et al. Electromechanical mapping identifies improvement in function and retention of contractile reserve after revascularization in ischemic cardiomyopathy. *Circulation* 2004; 110: 2410-6.

Związek pomiędzy właściwościami elektrycznymi i mechanicznymi lewej komory serca u chorych z pozawałowym częstoskurczem komorowym

Wojciech Wróbel, Krystian Wita, Andrzej Hoffmann, Cezary Czerwiński, Anna Maria Wnuk-Wojnar, Anna Rybicka-Musialik, Zbigniew Tabor, Artur Filipecki, Maria Trusz-Gluza

I Klinika Kardiologii, ŚAM, Katowice

Streszczenie

Wstęp: Analiza elektroanatomicznej mapy serca pozwala na odróżnienie żywotnego miokardium od blizny. Echokardiografia jest szeroko stosowana w celu oceny kurczliwości serca. Związki pomiędzy elektrofizjologiczną a echokardiograficzną oceną funkcji lewej komory (LV) nie zostały dotąd należycie zbadane.

Cel: Próba korelacji oceny mechanicznej i elektrycznej funkcji LV u chorych z pozawałowym częstoskurczem komorowym oraz ustalenie klinicznych, echokardiograficznych i angiograficznych parametrów wpływających na lokalną czynność elektryczną.

Metodyka: Grupę badaną stanowiło 32 chorych (25 mężczyzn, średni wiek 64±9 lat), u których wykonano elektroanatomiczną mapę serca (system CARTO), oceniając średnią amplitudę sygnałów unipolarnych i bipolarnych dla 12 segmentów LV, scharakteryzowanych w zależności od stopnia zaburzeń kurczliwości jako segmenty prawidłowe, hipokinetyczne, akinetyczne lub dyskinetyczne. Średnia amplituda sygnałów unipolarnych dla tych grup segmentów wynosiła odpowiednio 7,8±4,2 mV, 6,5±4,2 mV i 4,7±2,5 mV, p <0,01, a dla sygnałów bipolarnych 2,1±1,5 mV, 1,9±1,9 mV oraz 1,1±1,0 mV, p <0,01. Wartość frakcji wyrzutowej LV ≤30%, wymiar końcoworozkurczowy LV >56 mm, przebyty zawał ściany przedniej lub dolnej, zawał w wywiadzie ≤5 lat oraz drożna tętnica dozawałowa były związane z obniżoną amplitudą sygnału w segmentach normokinetycznych.

Wnioski: Segmenty LV z zaawansowaną dysfunkcją skurczową charakteryzują się istotnie niższą amplitudą sygnałów elektrycznych niż segmenty normo- lub hipokinetyczne. Niektóre segmenty akinetyczne lub dyskinetyczne mogą jednak mieć zachowaną lokalną czynność elektryczną. Przebudowa LV, czas od dokonania się zawału serca i jego lokalizacja oraz drożność tętnicy dozawałowej wpływają na wartość amplitudy sygnałów elektrycznych segmentów normokinetycznych.

Słowa kluczowe: częstoskurcz komorowy, echokardiografia, elektroanatomiczna mapa serca

Kardiol Pol 2007; 65: 24-29

Adres do korespondencji:

dr n. med. Wojciech Wróbel, ul. Ligocka 5/16, 40-570 Katowice, tel.: +48 504 935 197, e-mail: wowr @wp.pl

Badanie było wspierane przez grant naukowy KBN.