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# **Diagnostics and Surgical Treatment of Left Ventricular Aneurysm with Ventricular Tachycardia**

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Additional information is available at the end of the chapter

<http://dx.doi.org/10.5772/54126>

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## **1. Introduction**

Left ventricular aneurysm (LVA) in postinfarction period makes for worse prognosis of coronary artery disease (CAD) course due to concomitant complications. With the natural course of postinfarction aneurysms 5 year survival varies from 25 to 60%, according to various authors. Ventricular arrhythmias cause death in 50% of the patients with remodelled left ventricle (LV) after myocardial infarction [1].

Contrast-enhanced magnetic resonance imaging (MRI) is the method of choice for the evaluation of myocardial viability in patients with chronic CAD and with LVA in particular [2,3,4].

Meanwhile, data of contrast-enhanced MRI pictures with condition of electrophysiological activity and topical diagnosis of ventricular tachycardia in patients who had experienced myocardial infarction complicated with LVA have not been compared. At the same time, need for surgical treatment of cardiac aneurysm combined with intraoperative ablation of arrhythmogenic areas of myocardium arises no doubts since it allows for better treatment outcomes in the early postoperative period and in the late follow-up [4,5,6].

Thus, the objective of our study was to enhance efficacy of topical diagnostics and of surgical treatment in patients with postinfarction LV aneurysms complicated with ventricular rhythm disorders through application of contrast-enhanced MRI, electrophysiological study (EPs) of the heart and optimal dissection or ablation of scarred and arrhythmogenic endocardium.

## 2. Materials and methods

The study included 188 patients operated for postinfarction anterior septal and apical LVA. The disease was diagnosed basing on the data of echocardiography (EchoCG), coronary ventriculography (CVG) and contrast-enhanced MRI.

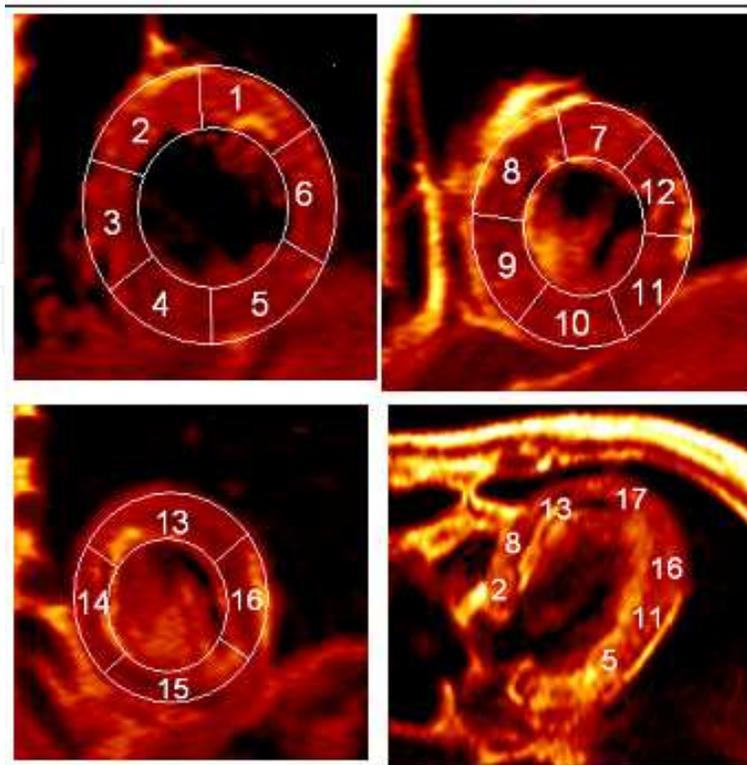
Prior to the surgery the patients mostly had III-IV CC angina class, their condition corresponded to that of New York Heart Association (NYHA) class II-III for chronic heart failure. All the patients demonstrated evidence of postinfarction LV remodeling according to the results of ventriculography and EchoCG.

Cardiac inversion recovery and T-1 spin-echo weighed MRI study with ECG synchronization was performed with a patient lying flat with no additional functional stress. Axial slices on the level of thorax with the complete coverage of heart area were recorded. The field of view was 350-380 mm wide and 7-8mm thick slices were recorded into the matrix of 256x256 voxels. Synchronization of the recordings of MRI pictures with ECG was performed by standard means of an open PRI scanner Magnetom-Open (0,2 T by Siemens Medical) or high field open MRI scanner Vantage Titan (1,5 T, by Toshiba) by R-wave of ECG; end-diastolic images were acquired in all the cases. Parameters of the acquired T1-weighted images in spin-echo mode were as follows: repetition time (TR) 550 – 1040ms, echo time (TE) – 20 ms. MRI included slices with long axis two-chamber and four-chamber views as well as short axis view covering all the myocardial volume of LV. The study was performed in 12-20 minutes after injection of paramagnetic contrast agents with the concentration of paramagnetic agent itself of 0,5M (Omniscan, Magnevist, Optimark, Cyclomang, Viewgam) in dosage of 2ml/10kg of a body weight. The short-axis and long-axis slices in four-chamber view were divided semi-automatically into 17 segments taking into account generally accepted segmentation of LV myocardium (Fig.1) [7].

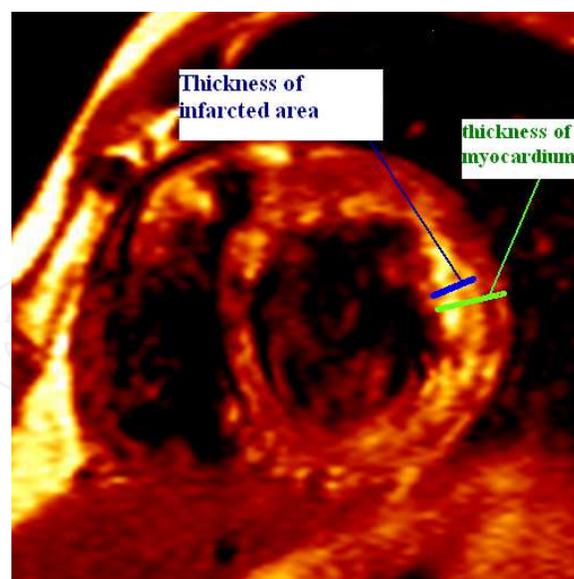
In particular, for each segment  $i$  ( $i = 1 - 17$ ) we calculated the depth of damage by the degree of paramagnetic contrast agent uptake as follows:  $[\text{Index of Transmurality}]_i = (\text{maximum thicknesses of paramagnetic contrast agent uptake})_i / (\text{thickness of myocardium in a particular segment})_i$  (Figure 2). (original data)

To see the association between MR images with the data of electrophysiological condition of heart muscle, and in particular with the location of the areas with lowered voltage of local electrical activity, the patients underwent electrophysiological study of the heart (EPhS) with electroanatomical CARTO reconstruction of LV [20]. Besides, there were identified the areas of delayed conduction, zones of possible re-entry and inducible VT (Figure 3).

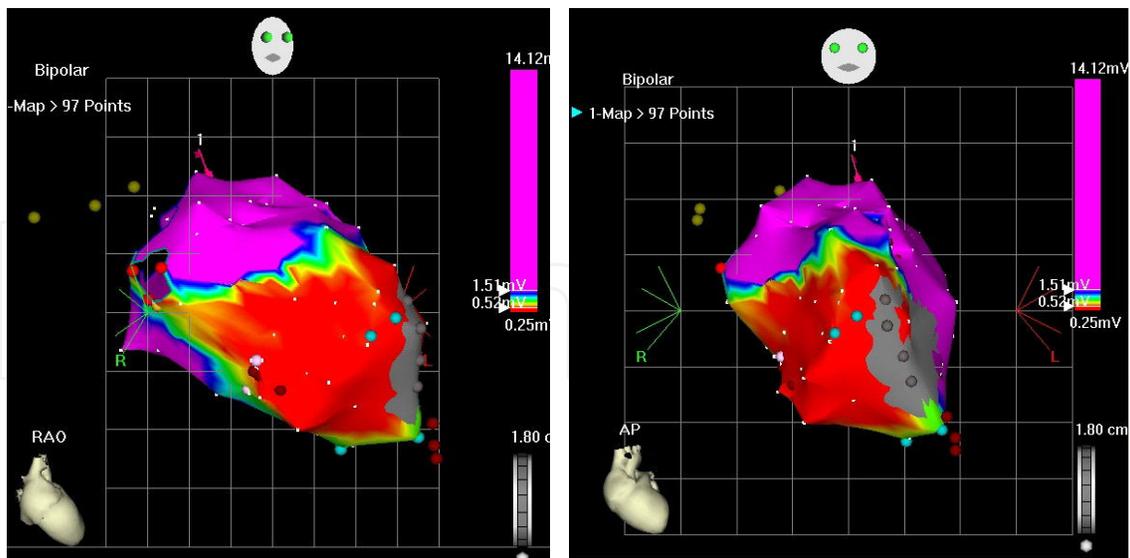
Locations of intracardiac leads and thus of segmental electrical activity corresponded to the locations of left ventricular segments during contrast-enhanced MRI. In accordance with the amplitude of the curve during EPhS for a definite myocardial segment, degree of potential reduction was graded as follows: 0 – for the amplitude of the potential from 1,5 to 8 mV, when the segment was considered to be a zone of normal potential; 1 – for the amplitude of 0,5 – 1,5 mV, a transient zone; 2 – for the amplitude 0,05 – 0,5 mV, low potential zone; 3 – zone of «electrical scar» - lack of electrical activity when the amplitude was 0 – 0,05 mV. This grading was preconditioned by the fact that myocardial areas with the 1<sup>st</sup> or 2<sup>nd</sup> grades of lowered potential were, as a rule, sources of life threatening tachycardias, while for grade 3 “electrical scar” this was less possible and in the zones with normal electrical activity of grade 0 VTs did not occur [9,10].



**Figure 1.** Segmentation of LV myocardium, used in evaluation of a local paramagnetic contrast agent uptake during myocardial MRI



**Figure 2.** The scheme of TI calculation by the data of delayed contrast-enhanced MRI of myocardium. The values of thickness of the infarction zone (accumulation of paramagnetic) and the value of the total myocardial thickness were identified on the LV slices in short axis. Thickness of the contrast-paramagnetic accumulation in myocardium is thought to be the thickness of lesion resulting from acute myocardial infarction. Thus TI is equal to  $\{(thickness\ of\ an\ infarcted\ area) / (thickness\ of\ the\ myocardium)\}$ . (original data)



**Figure 3.** Patient T, 56 year old. Before the surgery. EPhS with LV reconstruction of a patient with LV aneurysm. The area of electrical “silence” (scar) is highlighted with grey color; the low-amplitude ventricular potential area of 0,5 mV – with red; the transient zone of 0,5 - 1,5 mV – with yellow-green; the zone of viable myocardium – with violet; the double potential zone – with blue dots and the zone of delayed potential – with pink dots. Front view, right oblique view. (original data)

Surgical ventricular reconstruction (SVR) was performed by the standard methods by V. Dor and in L. Menicanti modification. [4,6]. After cardiac arrest with a calculated injection of cardioplegia solution (Kustodiol) there was performed grafting of distal coronary anastomoses. Left ventricle was opened with a longitudinal incision in the apical area to be parallel to the anterior descending artery along visually identified scarred tissue. After revision of left ventricular cavity thrombotic mass if any was eliminated. In case of endocardectomy we performed resection of scarred and transient areas of LV. Residual volume of LV cavity was calculated by a physiological norm of 50-60 ml/m<sup>2</sup> of a patient’s body surface, and was limited by a special sizer (Chase Medical Richardson, TX, USA). To close LV cavity we used an endocardial synthetic patch (Gore-tex). When L. Menicanti modification was applied, LV neoapex was formed with one or two u-shaped sutures. LV was closed with a double running suture [11,12].

Endocardectomy was performed in 84 patients who were referred to the study group (LVR +EE); on average there was dissected 44cm<sup>2</sup> of LV endocardium (from 17 to 84 cm<sup>2</sup>) including ventricular septum. The control group consisted of 104 patients in which endocardial resection was not performed (LVR without EE). The patients were allocated into the groups randomly. All the patients signed the informed consent form. The study was approved by the local ethic committee.

Resection of aneurysm and left ventricular reconstruction (LVR) was performed by V. Dor procedure in 130 patients, by L. Menicanti modification - in 58 patients. In 29 patients from both groups mitral valve fibrous ring repair was done. All the patients underwent coronary artery bypass grafting (CABG). The area of an endocardial synthetic patch varied from 5 to 20 cm<sup>2</sup>. Clinical data and the data of instrumental examinations did not show significant differences between the patient groups (Table 1).

Characteristics		LVR with EE n=84	LVR without EE n=104
Age, years old		55	56
Angina class Canadian Cardiovascular Society, (%)	II	10	10
	III	37	40
	IV	34	33
	Unstable angina	19	17
Current NYHA heart failure class, (%)	I	5	6
	II	20	21
	III	70	69
	IV	5	4%
Type of LV aneurysm, (%)	1	56	58
	2	35	34
	3	9	8
Ventricular tachycardia, (%)	Spontaneous	14	13
	Induced	32	30
Ventricular extrasystoly, (%)		44	48
Mitral regurgitation 2+, fibrous ring more than 35 mm, (%)		18	13
Lesions of coronary arteries, (%)	1	30	34
	2	35	36
	3	35	30

**Table 1.** Clinical characteristics of the patients.

SPSS 11.5 for Windows software was used for the analysis. Shapiro-Wilk test was applied to assess normality of distribution law of quantitative values. The parameters conforming with the normal distribution test were described with the use of a mean value (M) and a standard deviation (SD). Qualitative data were described by the rate of occurrence or its percentage. Student's t-test was used to evaluate significance of the differences of quantitative values in the compared groups when distribution law was normal. To see the significance of differences among quantitative values Z criterion (Fisher's exact test) was used. Evaluation of significance of differences in postoperative mortality was carried out by Kaplan-Meier method. With  $p < 0.05$  all the statistical parameters were considered significant.

### 3. Results

#### 3.1. Survival rate

Intraoperative mortality for the patients underwent LVR comprised 5% (9/188). For the patients of the study group (LVR with EE) mortality was 4% (3/84), for the patients of the control group (LVR without EE) – 6% (6/104). One year survival was 92% (77/84) for the patients subjected to LVR with EE and 87% (90/104) for those from the control group. The causes of mortality are shown in Table 2.

Causes of postoperative mortality	LVR with EE (N=7 from 84)	LVR without EE (N = 14 from 104)
Low cardiac output syndrome	1	2
Progressing HF	3	4
Acute myocardial infarction	2	1
Stroke	-	2
Sudden cardiac death	-	4
Non-cardiac reason	1	1

**Table 2.** Surgical outcomes of the patients in 1 year after the intervention.

#### 4. Cardiac function

Thus, in the study group patients left ventricular end-diastolic volume index (LV EDVI) was increased on average up to 118 ml/m<sup>2</sup>, end-systolic volume index (LV ESVI) – up to 74 ml/m<sup>2</sup>, LV ejection fraction (EF) was lowered to 38% and in the control group patients these parameters were: LV EDVI - 114 ml/m<sup>2</sup>, LV ESVI – 69 ml/m<sup>2</sup>, EF - 40%. MRI of diastolic phase in synchronizing mode showed perimeters of affected myocardium; on average they were 52% and 49% of the entire myocardial perimeter in the groups.

EchoCG performed in 2 weeks after the surgical intervention showed statistically significant ( $p < 0,01$ ) change of the values in comparison with preoperative data: increased EF up to 49% и 52%, decreased EDVI down to 79 and 77 ml/m<sup>2</sup>, ESVI to 49 and 48 ml/m<sup>2</sup> in the patients of the study group and control group correspondingly. There were no statistically significant differences found between the groups as for preoperative and postoperative hemodynamic values.

## 5. EPhS with LV electroanatomical mapping

Analyzing the results we decided to allocate the patients who underwent EPhS with electroanatomical LVR before and after the surgery into separate groups. Forty patients from the study group were included into group 1 and 38 from the control group into group 2. In the early postoperative period in the patients of group 1 the values of EPhS improved: “electrical scar” zones were found on endoventricular patch only, areas of lowered potential disappeared completely, transient zones (from 0,5 to 1,5 mV) took a limited area without possibility of re-entry and VT induction (Figure 2).

In 2<sup>nd</sup> group patients spontaneous VT spells were registered by Holter monitoring in 6 cases; in 8 cases VT was induced during EPhS which made in total 37% of the patients. In 12 patients cardioverters-defibrillators were implanted for the secondary prevention of a sudden cardiac death.

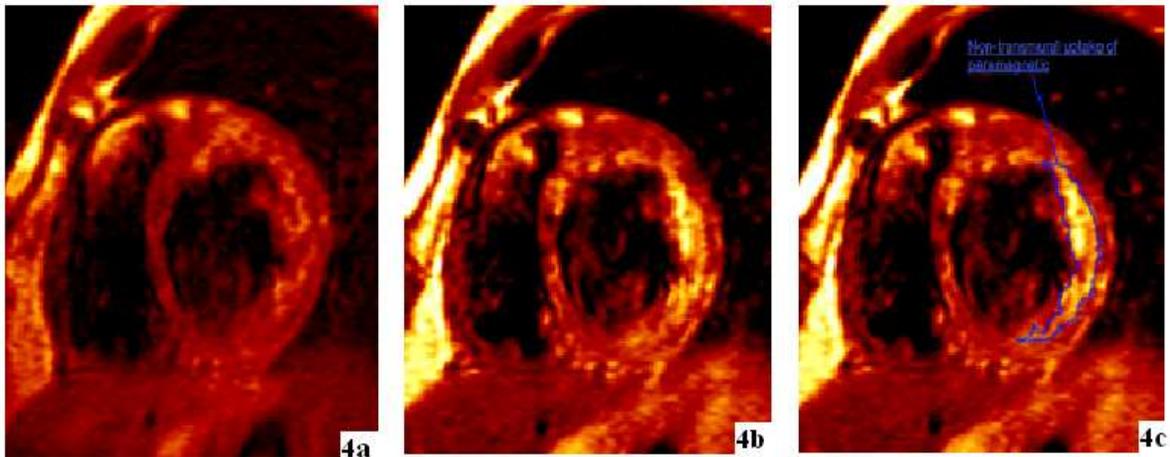
## 6. Contrast-enhanced MRI

Analyzing the obtained MRI values characterizing local morphological condition of the myocardium with values of local electrical myocardial potential we found a significant difference as for the thickness of viable myocardium (i.e. myocardium which does not accumulate contrast paramagnetic agent) in comparable segments. Thus in the zones with normal potential (0 decrease) the thickness of viable myocardium was more than 7 mm - on average 9,8mm; in transient zone (lowered potential 1) it was 6,2mm; in low potential zone (lowered potential 2) – 5,3mm and in “electrical scar” zone (lowered potential 3) – 2,8 mm. In the latter case viable myocardium was thinner than 3,5mm in all the segments. Figure 4 shows an example of a typical MR image in a patient with a previous acute myocardial infarction and affected lateral LV wall.

In the segments 10,11, 12 the uptake of contrast with the index of transmural index ranging from 0,20 to 0,55 is obviously seen. Later on during the electrophysiological study the activity of proarrhythmogenic type 2 was revealed. (original data)

Besides, the value of transmural index (TI) of paramagnetic contrast agent accumulation in myocardium differed significantly between unaffected segments with 0 degree potential lowering and segments with the 1<sup>st</sup> and 2<sup>nd</sup> degrees of potential lowering –the most arrhythmogenic degrees (Figure 4). In electrically normal myocardial segments, in particular, TI value was  $0,072 \pm 0,020$ . In the group of segments in transient zone TI was  $0,46 \pm 0,046$ , and in the low potential zone -  $0,32 \pm 0,052$ . Finally, the most affected myocardium with TI of  $0,32 \pm 0,052$  was found in the area of an “electrical scar” with no electrical potential.

By the data of ROC analysis and discriminative analysis the most appropriate breaking value allowing to differentiate segments with abnormal electrical activity became TI value of 0,27. In other words, when  $TI \geq 0,27$  one should consider probable arrhythmogenic activity in such a segment and pay closer attention to such areas during EPhS.



**Figure 4.** Patient K, has had an acute MI in the circulation of a left circumferential artery with a long area of subendocardial lesion of a lateral wall. Fig.4a – T1- weighed spin-echo ECG-gated MRI study before injection of paramagnetic contrast; Fig. 4b – T1-weighted spin-echo ECG-gated MRI 15 min after injection of paramagnetics, as 2ml of 0,5M solution per 10 kg of BW. Fig. 4c – the same as 4b, after semi-automatic bordering of subendocardial contrast uptake.

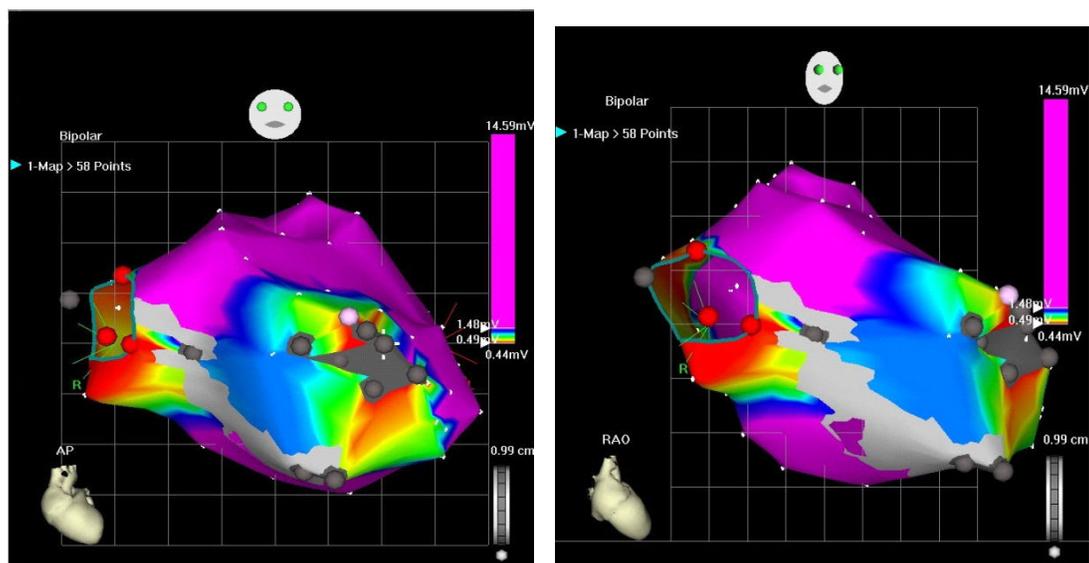
## 7. Clinical case

Patient T, 56 year old was admitted to the department of cardiovascular surgery at Tomsk Institute of Cardiology in 4 months after transmural anterior-septal myocardial infarction with complaints on occasional angina pangs and dyspnea. The patient was examined routinely. Holter monitoring showed ventricular extrasystoly (grade III by Lown). By EchoCG ejection fraction was 25% lower than normal (in B mode), LV was dilated with LV EDVI as high as 154 ml/m<sup>2</sup> and LV ESVI of 116 ml/m<sup>2</sup>; local LV contractility was disturbed, there was found akinesia of apical, medial septal and anterior segments as well as hypokinesia of lateral and posterior-lateral segments. EchoCG also showed the 2<sup>nd</sup> type aneurysm.

By MRI there were found postinfarction cicatricial changes in all apical and, ventricular septal and anterior segments; perimeter of the affected LV endocardium was 43%. In the apical and septal segments TI varied from 0,35 to 0,56. Data of coronaroveniculography showed LV deformation due to the aneurysm on the plane of anterior-lateral and apical segments and due to atherosclerosis of coronary arteries which included occlusion of the LAD artery in its proximal third and 75% stenosis of the right coronary artery. After mapping and electroanatomical LV reconstruction (Figure 3) there were identified the areas of an “electrical scar” on the apex, ventricular septum and anterior LV wall, zones of delayed conduction (pink dots in the picture) and those of double potential (blue dots) in transient zone, around the scar on ventricular septum and partially on the lateral LV wall. On the border of affected areas and viable myocardium radiofrequent (RF) dotted tags were applied (maroon dots in the picture) by an ablation lead.

After careful examination the decision was made to perform surgical myocardial revascularization and LV endoventriculoplasty with endocardectomy of the affected area. During the

surgery we performed epicardial EPhS with overdriving stimulation of 200 impulses a minute; VT was induced. In conditions of CP bypass and cardioplegia mammary-coronary artery bypass grafting of the LAD artery, LV aneurysm dissection, endocardectomy of the apex, ventricular septum, anterior and lateral LV walls along RF tags were performed as well as SVR including endoventricular circular repair with a synthetic patch by the method of V.Dor. Postoperatively the patient received routine care. Postoperative period was uneventful. By EchoG done in 3 weeks after the surgery one could notice better contractile cardiac function – LV EF grew up to 40% (B-made), LV sizes became smaller – EDVI was 70ml, ESVI – 48ml. The data of 24-hour ECG monitoring did not reveal any signs of ventricular rhythm disturbances. Postoperative mapping (Figure 5) showed significantly smaller transient zone, lack of re-entry and VT.



**Figure 5.** Patient T, 56 year old. EPhS with LV reconstruction of the patient after LV aneurysmectomy (LVR) : electrical scar in the area of the patch. Low-potential areas with the potential from 0,5 mV and transient zones (from 0,5 to1,5 mV) take a limited area with no possibility of re-entry and VT induction. Front view, right oblique view. (original data)

The patient was discharged from the hospital in satisfactory condition.

## 8. Discussion

In 1956 Couch O.A. performed LV aneurysm resection in a patient with VT thus beginning an era of surgical treatment of ventricular rhythm disorders [13].

It has been more than 50 years since; nevertheless the issue of complications and approaches of surgical treatment associated with the appearance of VT in patients with remodelled LV after previous MI is still quite challenging [14]. It was at that time already when specialists were aware of the fact that LV myocardium affected by infarction was a source of fatal ventricular rhythm disorders. Initially there were offered methods of indirect surgical interven-

tion such as thoracic sympathectomy, CABG, resection of a cardiac wall for the treatment of recurrent ventricular arrhythmias associated with CAD [15,16,17]. Since these methods appeared to be inefficient, over the course of time there were implemented direct endocardial methods performed under control of intraoperative electrophysiological mapping. The first endocardial procedure developed for the treatment of VT combined with CAD was a circular endocardial resection performed by Guiraudon in 1978 [18]. This procedure involves endocardial incision made on the borderline between endocardial fibrosis and viable myocardium and continued around the whole base of aneurysm or infarction area. In 1982 to enhance efficiency of a circular endocardial resection J. Moran modified this procedure by resecting all the fibrous endocardium connected with LV aneurysm or infarction and called it an expanded endocardial resection [19]. Supporting development of the ideas referred to endocardial resection V. Dor offered resection of fibrous endocardium from the side of inter-ventricular septum during surgical LV reconstruction [11].

This kind of intervention appeared to be efficient for the treatment of «refractory» ischemic VTs but did not make any effect on VTs coming from papillary muscles' base or from areas adjacent to a ring of an aortic or mitral valves.

In 1981 Leo Bokeria one of the first in the world began resection of LV aneurysm and cardio-destruction in the areas of early activity after intraoperative epicardial EPhS [20]. Developing cryosurgical methods of intervention in 1985 J. Cox performed endocardial cardiodestruction but the procedure resulted in lethal outcome in 27% of the cases and was ineffective in 17% of the cases [15]. In 1980<sup>th</sup> M. Mirovsky (the USA) offered an alternative method of VT treatment – implantation of cardioverter-defibrillator [21].

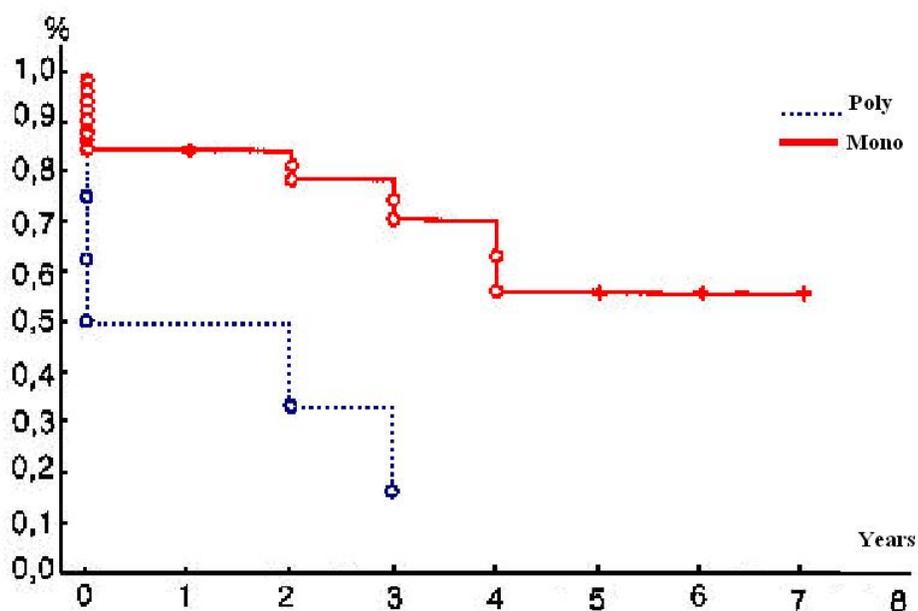
As a result, for the treatment of postinfarction LV aneurysms and associated ventricular tachyarrhythmias there have been used different methods, either alone or in combination. Significant clinical experience have been acquired.

Thus, Bokeria L.A. in his study including 59 patients demonstrated a clear dependence of actuarial survival rate from the type of tachycardia and from the presence of VT relapse in the early postoperative period [20]; the worst prognosis was noticed with the presence of polymorphic ventricular extrasystoly (Figures 6, 7).

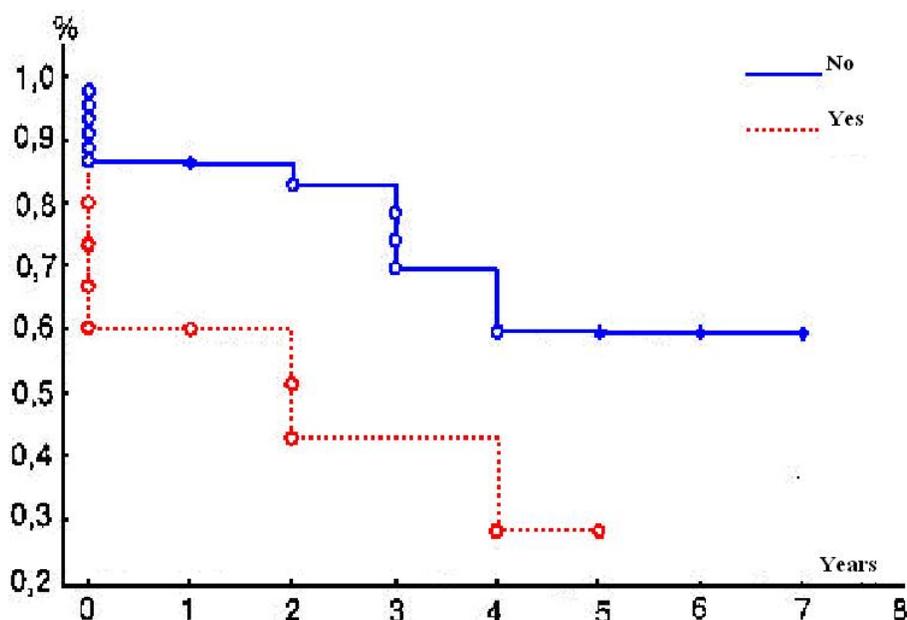
Interesting data were presented by the group of authors headed by M. Di Donato [22]; they analyzed data of 382 patients proving that spontaneous VTs after surgical treatment of LV aneurysms and VT significantly worsen prognosis for late postoperative period if compare with induced VTs of cases without arrhythmias (Figure 8).

After careful study of immediate ablation results in 71 patients with LV aneurysm and VT J. Pirk showed that epicardial cryoablation alone was successful in 63,3% of the cases and aneurysmectomy and endocardial cryoablation and/or subendocardial resection were successful in 73,2% of the cases [23].

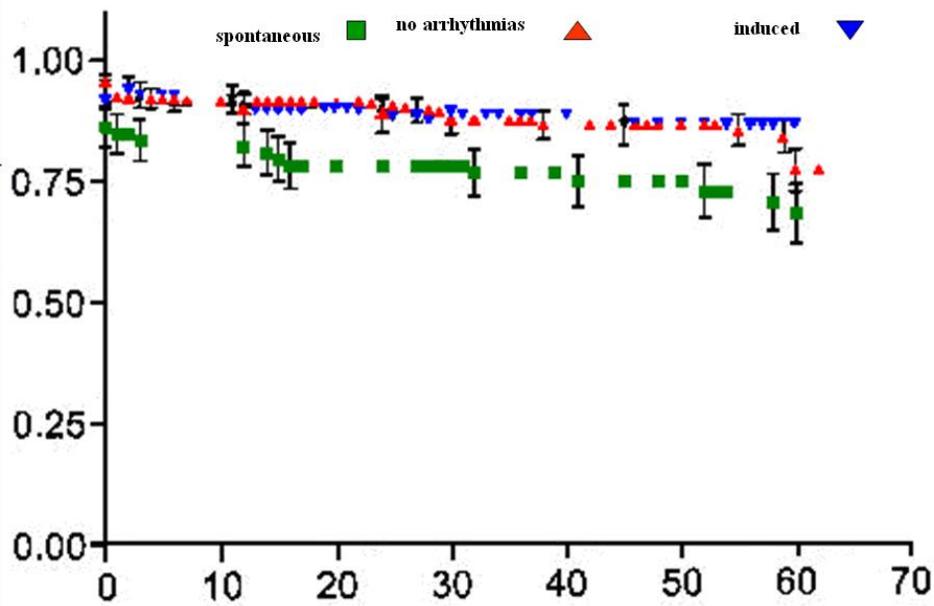
Sartipy U. studying combination of V. Dor procedure and surgery for VT in 53 patients came to the conclusion that combination of these procedures keeps survival rate high in the postoperative period (Figure 9) and that majority of the patients did not need implantation of an automatic implantable cardioverter-defibrillator (AICD) [24].



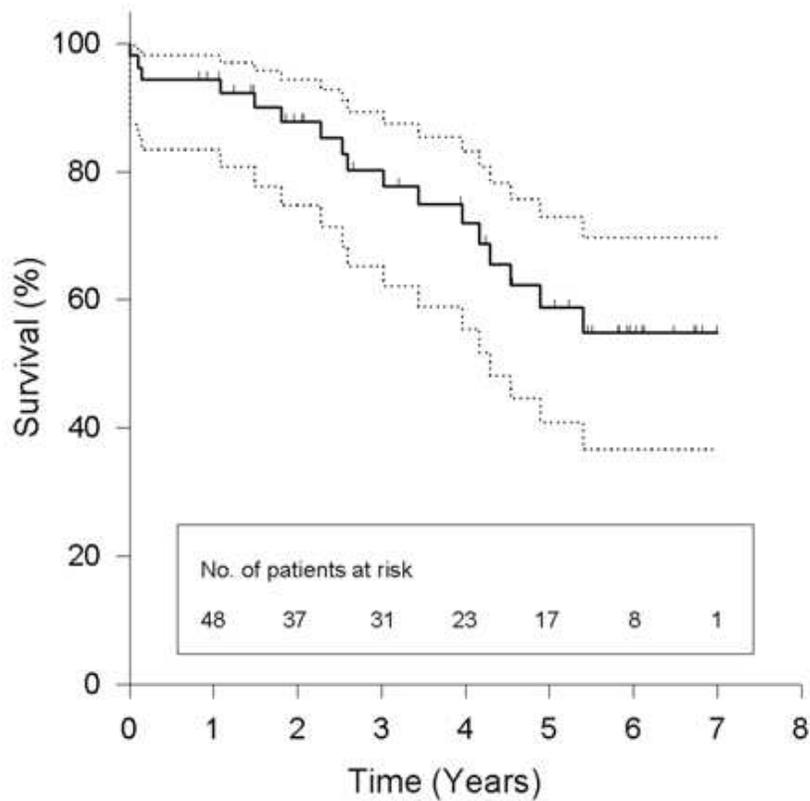
**Figure 6.** Actuarial survival curve depending on a type of VT (Kaplan-Meier);  $p=0,00739$  (Bokeria L.A et al.// Journal of Thoracic and Cardiovascular Surgery –1999.– №6.).



**Figure 7.** Actuarial survival curve depending on VT relapse (Kaplan-Meier);  $p=0,012$  (Bokeria L.A et al.// Journal of Thoracic and Cardiovascular Surgery –1999.– №6.).



**Figure 8.** Kaplan-Meier survival curves by the groups with VT in postoperative period (months) after surgical treatment of LV aneurysm and VT. (Di Donato et al. *Seminars in Thorac and Cardiovasc Surg.* Vol.13;4:480-485).



**Figure 9.** Overall actuarial survival after the Dor procedure including ventricular tachycardia surgery. Dotted curves are upper and lower 95% confidence intervals (Sartipy U. et al.; *Ann Thorac Surg* 2006;81:65-71).

Contemporary therapeutic methods are not able to solve this problem also. By the data of a multicenter trial MADIT II implantation of AICD in patients with ventricular rhythm disturbances lowers the risk of a sudden cardiac death for 31 % which is more efficient than antiarrhythmic therapy but still is not 100% saving [21]. In a year after endovascular treatment of VT the rate of relapses comprises 20% [25,26]. Nevertheless, antiarrhythmic therapy, implantation of AICD, catheter isolation of ectopic focuses do not touch an issue of coronary arteries lesion.

According to the data of a multicenter STICH trial there were no significant differences found between the patients with ICMP and postinfarction LV aneurysm subjected to CABG only (group 1) and those subjected to CABG with LV reconstruction (group 2) during 5 year follow-up. Nevertheless, postoperatively AICD was implanted into 20% of the patients from group 1 and into 17% from group 2 [27]. The study did not suppose to perform extended endocardectomy during LV reconstruction. Taking into account the aforesaid, one may claim that almost every 5<sup>th</sup> patient is destined for AICD implantation after surgical remodeling of LV. Although, by the data of multiple authors endocardial resection either with intraoperative mapping or without it prevents VT paroxysms in 90% of the cases and more [19, 24].

Thus, we saw clearly that at that time to treat patients with postinfarction LV aneurysm complicated with ventricular rhythm disorders is was necessary to perform reconstruction of LV cavity with endocardial resection and CABG; to use contemporary antiarrhythmics and AICD implantation in postoperative period if necessary.

Though, at that point there were unclear issues connected with topical diagnostics of potential re-entry zones which was important for adequate resection of affected endocardium. In our study we tried to enhance efficacy of topical diagnostics and surgical treatment of the patients with postinfarction LV aneurysm complicated with VT, due to combined application of contrast-enhanced MRI, EPhS and advanced surgical treatment (SVR and EE). It is well-known that MRI is a golden standard in diagnostics of LV aneurysm [4, 28], but MRI data may provide only indirect evidences about the presence of arrhythmogenic zones.

Prognostic role of contrast-enhanced MRI in evaluation of myocardial viability were reported in literature in as far as 1986 [29]. In particular, it was supposed, that with the presence of irreversible ischemic lesion of myocardium MRI made at rest demonstrated significant decrease of end-diastolic thickness of myocardium (EDTM) and simultaneously – contractility index. At the same time it was assumed that secure thickness of myocardium evaluated by the value EDTM meant also a secure viability of myocardium in that location.

Comparison of myocardial MRI made at rest with the results of PET with 18F-FDG and SPECT with repeated injection of tallim-201 in patients with chronic coronary disease and pronounced LV dysfunction was made in a number of studies [30]. It was found, that as a rule MRI visualized secure thickness of myocardium and the value of EDTM more than 5,5-6,0 mm in the affected areas in LV segments classified as viable by PET and SPECT. Later, Baer et al [31] making a direct comparison of MRI at rest and PET data with 18F-FDG found that with EDTM  $\leq$  5,5mm there were no signs of viability on myocardial tomography

slices during radionuclide study. As for prognosis for restoration of myocardial viability and contractility after CABG in such patients, their criterion {  $EDTM \leq 5,5 \text{ mm}$  } had high sensitivity up to 92-95%, but low specificity – just about 56-60%.

As a rule, for contrast-enhanced MRI visualization of affected myocardium contrasting agents – paramagnetics are used, usually they are complexes of Gd or Mn with derivatives or analogues of diaethylenetriaminopentacetic acid (DTPA). Their intravenous bolus injection makes possible qualitative evaluation of myocardial perfusion by the degree of changing brightness of myocardial image during the first few seconds after injection. Later on, in 12-20 minutes after injection one can evaluate the picture of myocardial lesion by accumulation of contrasting agent in affected areas.

There exist an established and commonly accepted opinion that transmural accumulation of paramagnetics in myocardium during contrast-enhanced MRI means irreversible lesion, and lack of accumulation vice versa evidences viability of myocardium and makes for favourable prognosis [32]. Nevertheless, relationship of contrast-enhanced MRI picture with the possibility of arrhythmogenesis in this or that myocardial area is still of a great interest.

Electrophysiological mechanism of the observed interrelationship between results of cardiac contrast-enhanced MRI and decrease of electrical potential in a definite LV segment is nothing but a particular case of a well-studied pathogenesis of arrhythmias appearance in the area of ischemic myocardial lesion [33].

It is in the area of thickened and partially replaced by subendocardial scarred tissue of myocardium where one can notice lowered electrical potential proportionally to the lowering mass of viable myocardium. This fact, in its turn, is favorable for the functioning of local re-entry circuits which are electrophysiological basis for ventricular tachycardias [33, 34].

That is why during contrast-enhanced MRI it makes sense to calculate TI index value in all the cases keeping in mind further electrophysiological study and evaluation of risks for ventricular tachycardias. Epicardial mapping provides information about the presence of excitement zones in LV and approximate anatomy of their localization for a further surgical treatment [4, 35]. Preoperative endocardial EPhS with electroanatomical LV reconstruction is able to demonstrate vividly disturbances in cardiac conduction system. Examining the results of endocardial EPhS we found consistency of myocardial lesion and its electrophysiological properties. In patients suffered from extensive myocardial infarction complicated with aneurysm one can identify zones of low-amplitude ventricular potential less than 0,5mV which is a scarred zone more often anatomically involving an apex of LV with a part of anterior wall and ventricular septum. Viable myocardium has potential amplitude higher than 1,5 mV. A subject of a special interest is a transient zone from 0,5 to 1,5 mV situated between the scar and viable myocardium where they register double potential and/or delayed conduction able to cause re-entry and ventricular tachycardia; a surgeon is just to perform dissection of affected endocardium. EPhS and MRI allow to identify borders for endocardial dissection.

Postoperative EPhS worth electroanatomical LV reconstruction performed in patients without endocardectomy showed that re-entry and VT sources revealed preoperatively were still

there and made for a high risk for the patients' lives. Contrast-enhanced MRI gives additional prognostic information about arrhythmogenicity of particular areas and segments of LV after myocardial infarction. More often arrhythmogenic areas are located in the areas of a pronounced non-transmural lesion of LV myocardium with TI higher than 0,27.

## 9. Conclusion

Thus, data of contrast-enhanced MRI not only have diagnostic significance concerning a degree of a cardiac muscle lesion but also identify arrhythmogenicity of this or that myocardial area. In surgical treatment of postinfarction aneurysm endocardectomy of scarred and transient LV zones' endocardium is an inseparable stage to prevent VT spells. MRI and endocardial EPhS with electroanatomical LV reconstruction allow to find potential areas where re-entry may occur.

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