

**Right ventricular visualization at SPECT perfusion imaging before  
and after revascularization in patients with postinfarction  
cardiosclerosis**

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**Abstract**

**Aim.** *To evaluate the intersystemic (between the myocardium of the left coronary artery system and the right coronary artery system redistribution mechanisms of perfusion in the myocardium after revascularization in patients with coronary artery disease with focal cardiosclerosis using gated single photon emission computed tomography. Cardiosclerosis foci were initially identified by magnetic resonance imaging.*

**Material and methods.** *The study included 17 patients with coronary artery disease with multivessel coronary disease and large-focal cardiosclerosis according to the results of magnetic resonance imaging with contrast; the diagnosis of left ventricular aneurysm was established*

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*in 14 patients, the focal subendocardial cardiosclerosis was diagnosed in 3 patients. For various reasons, all patients underwent myocardial revascularization without the left ventricle reconstruction (coronary artery bypass grafting in 10 patients, percutaneous coronary intervention in 7 patients). Magnetic resonance imaging was used as the gold standard for focal cardiosclerosis before revascularization. All patients before and after revascularization underwent gated single photon emission computed tomography with MIBI scan. During the initial analysis of peaks on the profile slices of coronal and transversal midsections passing along the lateral walls of the left and right ventricles, we did not notice a clear visualization of in 8 patients (group 1), while an increased MIBI scan accumulation in the right ventricle myocardium was clearly visualized in 9 patients (group 2). Based on the peaks height of profile curves, we compared changes in the maxima of radiopharmaceutical accumulation before and after revascularization in the lateral walls of the left ventricle and right ventricle. All studies were performed using the original Cardiac Functional Imaging medical program in order to obtain quantitative information about the myocardial function of both the left ventricle, and also the right ventricle. This program made it possible to highlight the right ventricle area even in the case of its weak visualization through the initial formation of parametric images, where the right ventricle area was visualized.*

**Results.** *When comparing the revascularization results of the two groups, we noted that the left ventricle ejection fraction increased significantly only in patients without initial visualization of the right ventricular myocardium. Left ventricle ejection fraction did not change after revascularization in patients with initially increased accumulation of the radiopharmaceutical in the right ventricle. Globally, only an improvement in the diastolic function of the left and right ventricles was*

*noted in the latter group of patients. In addition, an increase in the right ventricular uptake level was noted for patients with focal cardiosclerosis and the initially increased uptake in the right ventricle after the maximum possible complete myocardial revascularization, which may indicate a redistribution of perfusion in favor of a more intact right ventricular myocardium.*

**Conclusions.** *1. In patients without signs of increased visualization of the right ventricle (group 1) after revascularization, we revealed a statistically significant increase in the left ventricle ejection fraction (p-value=0.024), a decrease in the end-systolic volume (p-value=0.024), an increase in the motion in segments corresponding to the peri-infarct scar zone (p-value=0.016), and a change in systolic thickening in the segment of the basal parts of the anterolateral wall (p-value=0.046). 2. Initially increased visualization of the right ventricle in patients with extensive focal cardiosclerosis in the myocardium of the left ventricle suggests the absence of the left ventricle ejection fraction increase after myocardial revascularization. 3. An increase in the visualization of the right ventricle after complete myocardial revascularization indicates an intersystemic redistribution of perfusion in favor of the preserved myocardium of this part of the heart.*

**Keywords:** visualization of the right ventricular myocardium, intersystemic mechanisms of perfusion redistribution

**Conflict of interests** Authors declare no conflict of interest

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BP, blood pressure  
CABG, coronary artery bypass grafting  
CAG, coronary angiography  
CARFI, Cardiac Functional Imaging  
CB, circumflex branch  
DB, diagonal branch  
ECG, electrocardiography  
EchoCG, echocardiography  
EDV, end diastolic volume  
EF, ejection fraction  
ESV, end systolic volume  
IHD, ischemic heart disease  
IVS, interventricular septum  
LCA, left coronary artery  
LV, left ventricle  
LW, lateral wall  
MRI, magnetic resonance imaging  
PAP, pulmonary artery pressure  
PCI, percutaneous coronary intervention  
RCA, right coronary artery  
RPH, radiopharmaceutical (radionuclide, radiotracer)  
RV, right ventricle  
RVV, right ventricle visualization  
SPECT, single photon emission computed tomography  
SV, stroke volume

## **Introduction**

Today, no one doubts that revascularization of the myocardium affected with severe focal atherosclerosis is a thankless task, since, as a

rule, complete recovery of function is not observed in such a case. However, there are patients having significant cardiosclerosis foci evidently present in the myocardium who require revascularization. Therefore, many surgeons continue to use myocardial viability testing to make decisions about revascularization in patients with severe left ventricular (LV) dysfunction due to ischemic heart disease (IHD) with the development of heart failure. On the other hand, in patients with acute myocardial infarction, revascularization often precedes a non-invasive assessment of myocardial viability, and therefore the physician in such a case cannot obtain preliminary information about the cardiosclerosis severity during percutaneous coronary intervention (PCI). In both cases, each additional parameter is important for assessing the prognosis of systolic function recovery. We have devoted our small-sample observation study to only one symptom of instrumental findings that occur in such patients

### **Aim**

The aim was to assess intersystem (i.e. between the systems of the left and right coronary arteries) mechanisms of perfusion redistribution in the myocardium after revascularization in patients with multivessel ischemic heart disease with focal cardiosclerosis by using perfusion single photon emission computed tomography in the area of cardiosclerosis foci initially having been detected by means of magnetic resonance imaging.

### **Material and methods**

All investigations were performed using the original CARFI (cardiological functional imaging) program [1] in order to obtain quantitative information about myocardial function of both left, and right

ventricles. This program made it possible to isolate the area of the right ventricle (RV) even in case of poor visualization due to the initial formation of parametric images. To analyze the data of single photon emission computed tomography, nonparametric statistical methods were used. Based on the Wilcoxon signed rank test for two related samples, we identified parameters, for which there were statistically significant changes after revascularization. Also, to assess the reliability of the obtained results, for each parameter, either the initial data on which the calculation was made, or descriptive statistics were presented.

The parameters that changed after revascularization in all patients were presented in a number of tables. An increase in peak LV filling rate was noted (Tables 1 and 2). The null hypothesis that the two samples are equal is rejected in favor of a two-tailed alternative for the parameter of the peak left ventricle filling rate, and the observed difference in related samples (the 1<sup>st</sup> one before and the 2nd one after revascularization) is statistically significant at p-value = 0.05. For the analysis, the Wilcoxon signed rank test (W) was used; hereinafter, for the analyzed parameters, the results of the obtained values of W with  $W_{crit}^1$  and p-value will be presented in tables.

**Table 1. Parameters of the peak left ventricle filling rate in the study patients**

Patient	Parameters of peak filling rate of the left ventricle			
	before	after	$X_2 - X_1$	Signed ranks
V.T.I.	0.21	0.13	-0.08	-10.5
V.O.M.	0.32	0.4	0.08	12
D.V.N.	0.24	0.29	0.05	7
I.Ya.A.	0.34	0.39	0.05	7

<sup>1</sup> It denotes a tabular value that determines the p-value for n non-zero ranks (number of patients for whom the value of the studied parameter (W) changed after revascularization). If W is greater than or equal to  $W_{crit}$ , then the change in the parameter under study is considered statistically significant for a given (taken from the table) p-value

K.M.P.	0.27	0.23	-0.04	-4.5
K.V.A.	0.23	0.36	0.13	14
K.T.V.	0.22	0.3	0.08	10.5
M.G.A.	0.37	0.4	0.03	2.5
M.R.M.	0.28	0.16	-0.12	-13
S.A.I.	0.18	0.25	0.07	9
A.V.S.	0.19	0.5	0.31	16
A.A.A.	0.26	0.31	0.05	7
G.V.N.	0.23	0.24	0.01	1
G.S.N.	0.38	0.38	0	–
G.V.A.	0.24	0.27	0.03	2.5
K.S.I.	0.35	0.54	0.19	15
Sh.P.I.	0.18	0.22	0.04	4.5
Wilcoxon test (n=16, p=0.05)			$W_{crit}=76$	W=80

**Table 2. Descriptive statistics of the peak left ventricle filling rate in the study patients**

All patients	n	Min.	Q <sub>1</sub>	Median	Q <sub>3</sub>	Max.	Mean value	Standard deviation
Before	17	0.18	0.22	0.24	0.32	0.38	0.2641	0.0658
After	17	0.13	0.24	0.3	0.39	0.54	0.3159	0.1114
Wilcoxon test (n=16, p=0.05)					$W_{crit}=76$	W=80		

All patients were characterized by a decrease in the heart rate interval after the intervention, that is, a statistically significant increase in it at p-value = 0.02 (Table 3).

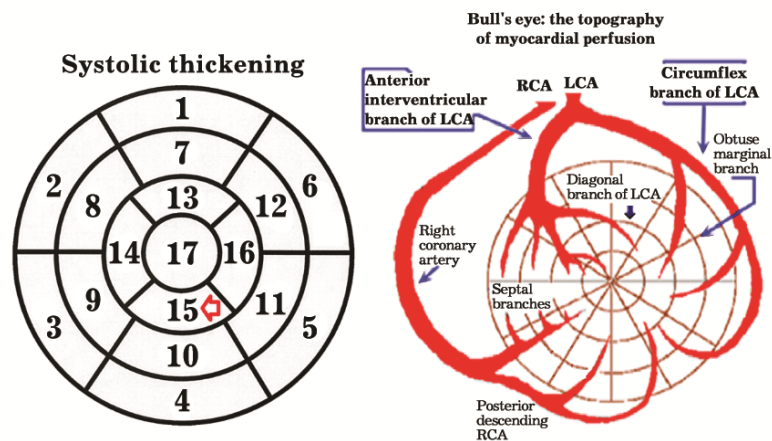
**Table 3. Descriptive statistics of heart rate**

All patients	n	Min.	Q <sub>1</sub>	Median	Q <sub>3</sub>	Max.	Mean value	Standard deviation
Before	17	552	855	1000	1091	1121	955.6471	165.1435
After	17	580	684	815	878	1100	809.4706	152.9269
Wilcoxon test (n=17, p=0.02)					$W_{crit}=97$	W=-119		

An increase in systolic thickening of the myocardium was also noted, corresponding to the apical areas of the diaphragmatic wall (Segment 15) (Table 4, Fig. 1).

**Table 4. Descriptive statistics of the myocardium systolic thickening in the area of the 15<sup>th</sup> segment**

All patients	n	Min.	Q <sub>1</sub>	Median	Q <sub>3</sub>	Max.	Mean value	Standard deviation
Before	17	13	21	30	34	59	29.8824	13.2612
After	17	19	25	36	47	62	36.7059	12.3782
Wilcoxon test (n=17, p=0.05)					W <sub>crit</sub> =83		W=90	



**Fig. 1. The "Bull's eye" plot showing the myocardium segmentation and projection of its perfusion**

Introducing the qualitative parameter of right ventricular visualization (RVV) made it possible to divide patients into two groups according to the presence of this sign and to compare between the 1<sup>st</sup> and 2<sup>nd</sup> groups.



## Observation limitations

1. The small sample size of patients with various diseases of both the myocardium and the coronary circulation was the main limitation of our study.

2. Another important limitation was the convention of the "complete revascularization" concept. A possible example is that among 8 patients of the 1st group, 3 patients did not undergo recanalization of all branches of the circumflex artery, and in 1 patient of the 2<sup>nd</sup> group, occlusion of the right coronary artery persisted. This may explain the decrease in systolic thickening of the myocardium in the 6th segment in patients of the 1<sup>st</sup> group after revascularization.

## Results and discussion

In the 1st group, where there was no increase in myocardial perfusion of the RV free wall, a statistically significant increase in the LV ejection fraction (EF) was revealed (Table 5) after revascularization, and a decrease in the LV cavity size during systole, as indicated by a change in the end-systolic volume of the left ventricle (Table 6). This indicates an improvement in myocardial contractility after revascularization in general in this group of patients.

**Table 5. Descriptive statistics of the left ventricular ejection fraction**

Group 1	n	Min.	Q <sub>1</sub>	Median	Q <sub>3</sub>	Max.	Mean value	Standard deviation
Before revascularization	8	24.64	32.93	36.28	47.555	77.09	42.1713	17.575
After revascularization	8	22.81	47.8025	60.97	69.77	83.76	57.555	19.677
Wilcoxon test (n=8, p=0.024)					W <sub>crit</sub> =32		W=34	

**Table 6. Descriptive statistics of end systolic volume of the left ventricle**

Group 1	n	Min.	Q <sub>1</sub>	Median	Q <sub>3</sub>	Max.	Mean value	Standard deviation
Before revascularization	8	17.34	66.085	106.5	116.9025	157.32	93.386	44.447
After revascularization	8	7.66	30.127	63.37	92.9825	138.62	65.37	45.419
Wilcoxon test (n=8, p=0.024)					W <sub>crit</sub> =32		W=34	

Meanwhile, there was an increased motion in the segments corresponding to the periscar zone (Segments 9 and 13), but at the same time there was a decrease in systolic thickening in the segment of the basal sections of the anterolateral wall (Segment 6) (Tables 7–9).

**Table 7. Descriptive statistics: Motion, Segment 9**

Group 1	n	Min.	Q <sub>1</sub>	Median	Q <sub>3</sub>	Max.	Mean value	Standard deviation
Before revascularization	8	0	2.75	3.5	4.25	8	3.625	2.3261
After revascularization	8	0	4.75	7.5	9.25	17	7.5	5.0427
Wilcoxon test (n=7, p=0.016)					W <sub>crit</sub> =28		W=28	

**Table 8. Descriptive statistics: Motion, Segment 13**

Group 1	n	Min.	Q <sub>1</sub>	Median	Q <sub>3</sub>	Max.	Mean value	Standard deviation
Before revascularization	8	0	5.75	6.5	8.75	12	6.875	3.7201
After revascularization	8	7	8.5	12	16	23	13.25	6.2507
Wilcoxon test (n=7, p=0.016)					W <sub>crit</sub> =28		W=28	

**Table 9. Descriptive statistics: systolic thickening, Segment 6**

Group 1	n	Min.	Q <sub>1</sub>	Median	Q <sub>3</sub>	Max.	Mean value	Standard deviation
Before revascularization	8	0	22.75	30	36.25	46	28	13.7529
After revascularization	8	0	10.5	22.5	26.5	37	18.75	13.3604
Wilcoxon test (n=7, p=0.046)					W <sub>crit</sub> =24		W=-24	

In the 2<sup>nd</sup> group, an increase in myocardial perfusion of the RV free wall was initially observed. After myocardial revascularization, patients did not show an increase in the LV ejection fraction and a decrease in the size of the LV cavity in systole (LV end systolic volume). However, the ratio of radiopharmaceutical (RPH) accumulation peaks in the LV/RV free lateral walls decreased. That is, according to our study data, the perfusion of the RV lateral wall compared with that of the LV lateral wall after revascularization increased. These hypotheses were tested for coronary and transversal sections of the myocardium on scintigrams before and after revascularization, which are presented in Tables 10–13.

**Table 10. The ratio of maximum radiopharmaceutical accumulation in the lateral wall of the left and right ventricle on scintigraphy coronary sections**

Patient	LV/RV ratio on coronal sections			
	before	after	$X_2 - X_1$	Signed ranks
V.O.M.	2.00444765	1.31387072	-0.69057693	-2
I.Ya.A.	2.449912127	2.327447833	-0.122464293	-1
K.M.P.	3.877695878	2.25032754	-1.627368338	-7
M.G.A.	5.870467927	1.952876712	-3.917591215	-8
M.R.M.	3.775	2.495	-1.28	-5
G.V.N.	1.672508215	2.625030201	0.952521986	3
G.S.N.	12.35193697	2.297548045	-10.05438892	-9
K.T.V.	5.314016661	3.958780593	-1.355236068	-6
Sh.P.I.	4.212239285	3.162060973	-1.050178311	-4
Wilcoxon test (n=9, p=0.02)			$W_{crit}=39$	$W=-39$

**Table. 11. Statistical analysis data for the ratio of radiopharmaceutical accumulation in left and right ventricle on scintigraphy coronary sections**

Group 2	n	Min.	Q <sub>1</sub>	Median	Q <sub>3</sub>	Max.	Mean value	Standard deviation
Before	9	1.6725	2.4499	3.8777	5.314	12.3519	4.6142	3.2307
After	9	1.3139	2.2503	2.3274	2.625	3.9588	2.487	0.7433
Wilcoxon test (n=9, p=0.02)					W <sub>crit</sub> =39		W=-39	

**Table 12. The ratio of the radiopharmaceutical accumulation in the projection of the left and right ventricles on scintigraphy transversal sections**

Patient	LV/RV ratio on transversal sections			
	before	after	X <sub>2</sub> -X <sub>1</sub>	Signed ranks
V.O.M.	1.76097888	0.897864036	-0.863114845	-4
I.Ya.A.	2.149936789	2.401802786	0.251865997	2
K.M.P.	4.03	2.44	-1.584436554	-9
M.G.A.	3.378603459	2.046682146	-1.331921313	-7
M.R.M.	3.366	2.487	-0.879	-5
G.V.N.	2.065033584	1.873145185	-0.191888399	-1
G.S.N.	3.718210013	2.646613766	-1.071596247	-6
K.T.V.	5.216933868	3.731832139	-1.485101729	-8
Sh.P.I.	3.155966504	2.865600264	-0.29036624	-3
Wilcoxon test (n=9, p=0.02)			W <sub>crit</sub> =39	W=-41

**Table. 13. Statistical analysis data for the ratio of radiopharmaceutical accumulation in the left and right ventricle on scintigraphy transversal sections**

Group 2	n	Min.	Q <sub>1</sub>	Median	Q <sub>3</sub>	Max.	Mean value	Standard deviation
Before	9	1.761	2.1499	3.366	3.7182	5.2169	3.2046	1.0925
After	9	0.8979	2.0467	2.44	2.6466	3.7318	2.3767	0.7677
Wilcoxon test (n=9, p=0.02)					W <sub>crit</sub> =39		W=-41	

Normally, RPH enters the myocardium in proportion to the blood flow, so most of it accumulates in the myocardium of the left ventricle, which is the reason for its distinct visualization. In the right ventricle, the

RPH accumulation is normally insignificant. Tables 10–13 present a comparison of RPH accumulation in the left and right ventricles of the heart in coronary and transversal sections before and after myocardial revascularization. The analysis of the data given in the tables showed that the accumulation ratio after revascularization in patients of the 2nd group changes in favor of RV. Thus, revascularization resulted in an increased right ventricular perfusion rather than the expected improvement in left ventricular perfusion.

To explain the increase in myocardial perfusion of the RV free wall, it is necessary to assess the relationship of this phenomenon with an increased load on the myocardium. In our study patients, we excluded the most common cause, an increased pulmonary artery pressure [2]. The relationship between perfusion parameters and pulmonary artery pressure (PAP) was assessed by the results of Doppler echocardiography. The analysis was carried out by comparing the ratio of LV/RV RPH accumulation on coronal and transversal sections. Accordingly, Spearman's rank correlation coefficient  $\rho$  was calculated for 9 patients ( $n=9$ ) as a measure of interdependence between rank series (non-parametric method). The following hypotheses were studied:  $H_0$ , hypothesizing that correlation between variables does not differ from 0 (there is no relationship),  $H_1$ , hypothesizing that correlation between variables is statistically significantly different from 0 (there is a relationship).

For the parameters "LV/RV", the  $p$ -value= $-0.0632$  was obtained on coronal sections and PAP, and  $p=-0.438$  on transversal sections. Tables 14–15 present the initial data for calculating the Spearman rank correlation coefficient of the corresponding parameters.

**Table 14. Initial data for calculating Spearman's rank correlation coefficient for the parameters "Ratio of left to right ventricles on the coronary section" and the pulmonary arterial pressure**

Patient	LV/RV on coronary sections (X)	PAP mm Hg, (Y)	Signed ranks X, $d_x$	Signed ranks Y, $d_y$	$(d_x - d_y)^2$
V.O.M.	2.00444765	33	2	7	25
I.Ya.A.	2.449912127	36	3	8	25
K.M.P.	3.877695878	18	5	1	16
M.G.A.	5.870467927	30	8	6	4
M.R.M.	3.775	22	4	4	0
G.V.N.	1.672508215	21	1	2.5	2.25
G.S.N.	12.35193697	21	9	2.5	42.25
K.T.V.	5.314016661	26	7	5	4
Sh.P.I.	4.212239285	28	6	9	9
$\Sigma =$			45	45	127.5

**Table 15. Initial data for calculating Spearman's rank correlation coefficient for the parameters "Ratio of left to right ventricles on the transversal section" and the pulmonary artery pressure**

Patient	LV/RV on transversal sections (X)	PAP mm Hg, (Y)	Signed ranks X, $d_x$	Signed ranks Y, $d_y$	$(d_x - d_y)^2$
V.O.M.	1.76097888	33	1	7	36
I.Ya.A.	2.149936789	36	3	8	25
K.M.P.	4.03	18	8	1	49
M.G.A.	3.378603459	30	6	6	0
M.R.M.	3.366	22	5	4	1
G.V.N.	2.065033584	21	2	2.5	0.25
G.S.N.	3.718210013	21	7	2.5	20.25
K.T.V.	5.216933868	26	9	5	16
Sh.P.I.	3.155966504	28	4	9	25
$\Sigma =$			45	45	172.5

One of the most well-known causes of increased myocardial perfusion of the RV free lateral wall is the presence of a corresponding level of pulmonary hypertension [2]. Therefore, we compared the relationship levels between pulmonary artery pressure and right ventricle visualization (RVV) elevation. As can be seen from the tables (Tables 14,

15), at a significance level of 0.05, the obtained values of  $\rho$  turned out to be less than the critical value of the Spearman rank correlation coefficient equal to 0.68 for  $n=9$ , and therefore, based on the obtained data, we cannot speak of the presence of a relationship between these parameters.

Thus, no relationship was revealed between the level of RV lateral wall perfusion and the pulmonary artery pressure in our patients.

Another cause of the increased RV perfusion level compared to that of the LV is the severity/multiplicity of lesions in the proximal coronary bed [3]. In some cases, the RV is visualized even though LV perfusion is normal. The significance of this fact is suggested by the relationship between RV dysfunction and increased mortality [4, 5]. Finally, myocardial hypertrophy of the RV may be the reason for its increased visualization on SPECT images as an indicator of the risk of death, at least in some patients [6].

Without a doubt, there are many different causes for increased imaging of the RV myocardium. However, the relationship of increased RVV with the cardiosclerosis development in the LV myocardium remains unexplored. In our study, magnetic resonance imaging was used as the gold standard in focal cardiosclerosis, and SPECT with 99 m-Tc-Technetrit was used to assess the redistribution of perfusion in the myocardium. In this case, a reasonable question may arise: “Why is it not enough to perform MRI alone?” But in the interpretation of the MRI results in our patients, none of the experts paid attention to the specific features of the RV images. Moreover, signs of RV myocardial hypertrophy were noted neither in this case, nor in the interpretation of the echocardiography results. It is unclear what awaits patients with initially increased RVV after revascularization. Therefore, the redistribution in the RV myocardium perfusion in case of significant cardiosclerosis of the LV myocardium requires a more thoughtful and

individual analysis of the factors influencing the decision-making process. Such information may be most important for revascularization in the acute phase of myocardial infarction, since patients with recurrent myocardial infarction are at the greatest risk of complications and death.

On the other hand, when evaluating myocardial viability in patients with heart failure, the attention of investigators is usually directed to measuring the focal cardiosclerosis size in the left ventricle [7], while the redistribution of perfusion in the safe myocardium, including the RV myocardium, can provide the most effective intervention. According to the existing point of view, the LV systolic function should improve as a result of myocardial revascularization in ischemic heart failure [8].

In our study, the initially increased perfusion of the RV myocardium increased after revascularization in multivessel disease. This may be the result of blood flow redistribution and an improvement in the function of the “accessible” myocardium; therefore, with a decrease in the cavity size, a corresponding increase in the wall thickness is noted. However, we did not find any significant changes in the RV cavity size in our patients. This means that the RV myocardium was superior to the LV myocardium in its ability to use the perfusion reserve that increased after revascularization. This, in our opinion, can be attributed to the presence of focal cardiosclerosis in the left ventricle, which prevents the improvement of blood flow in it.

The redistribution of perfusion after revascularization in favor of the RV myocardium in patients with focal LV cardiosclerosis, despite the fact that we have not identified its relationship with the level of pulmonary artery pressure, confirms our idea of an independent perfusion redistribution from the left coronary artery bed to the right coronary artery bed.

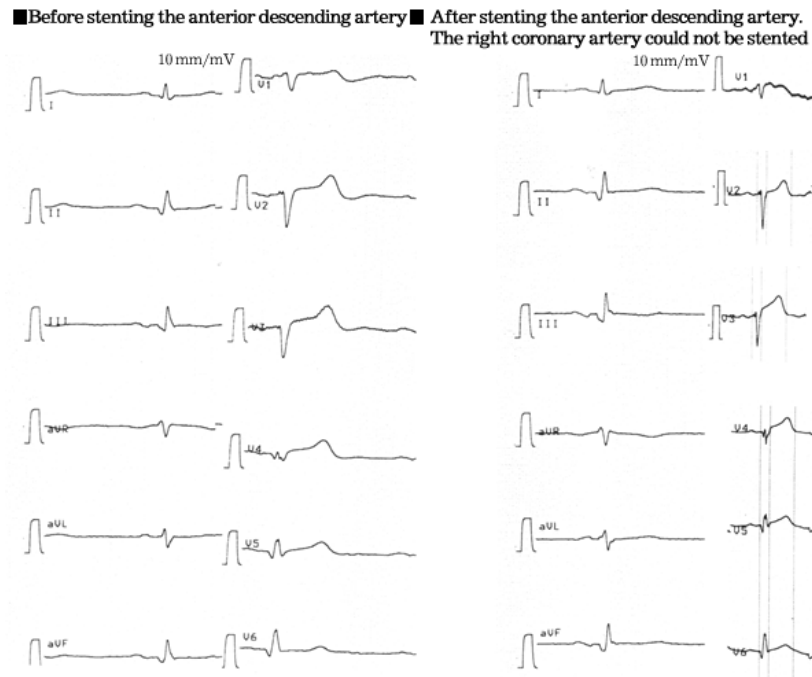


Interestingly, we have previously observed a similar negative predictive role of right ventricular visualization in patients with non-ischemic cardiomyopathy, which were selected for cardiac resynchronization therapy (CRT). Patients not showing an improvement in systolic function in response to CRT underwent heart transplantation. After the explanted heart section, severe diffuse cardiosclerosis of the LV wall was identified at a morphological study with an initially high level of RVV detected at scintigraphy [9].

To illustrate the above, we offer a brief description of the case of incomplete myocardial revascularization in a patient of the 2nd group with multivessel coronary disease and LV aneurysm.

Patient Sh.P.I., aged 56, was admitted to the clinical department with complaints of pressing pain behind the sternum, shortness of breath on exertion and at rest, increased blood pressure (BP) up to 180/100 mm Hg. From medical history it was known that the patient had been suffering from hypertension for a long time with a rise in blood pressure to the indicated figures, which was accompanied by pain behind the sternum. Deterioration of health condition in the form of decreased tolerance to physical activity had been noted over the previous month, while shortness of breath began to bother at night.

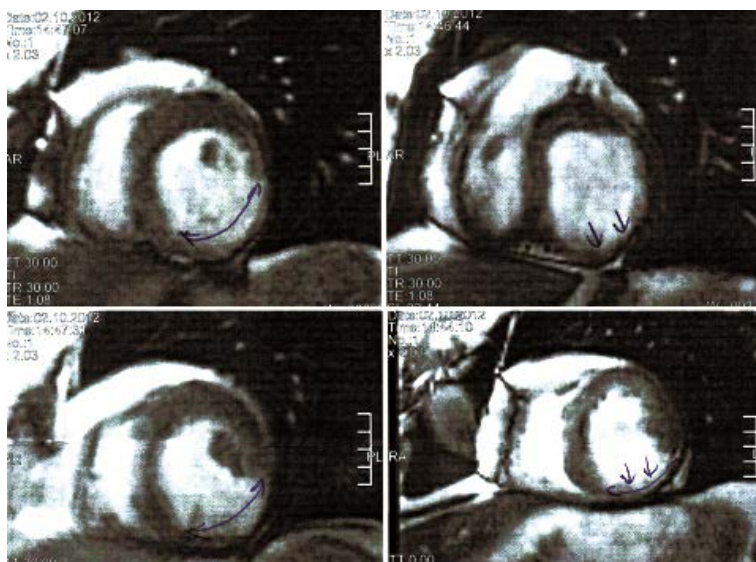
During the examination: on the electrocardiogram *sinus rhythm, heart rate 61 beats/min, PQ 0.14 sec, QRS 0.10 sec, QT 0.44 sec. Small-focal changes in the myocardium along the posterior wall of the left ventricle. Cicatricial changes in the myocardium in the LV anterior septal and apical-lateral regions* (Fig. 2).



**Fig. 2. Electrocardiogram of patient Sh.P.I.**

According to the results of echocardiography (EchoCG), *LV end diastolic volume (EDV) was 232 ml, LV end systolic volume (ESV) was 139 ml, stroke volume (SV) was 93 ml, EF 44%. Hypokinesis was revealed in the areas of the interventricular septum (IVS), lateral segment, lower third of the anteroapical and posterior basal LV segments. The mean calculated pressure in the pulmonary artery was 28 mm Hg.*

Given the history of myocardial infarction, MRI with contrast enhancement was performed for a detailed assessment of the myocardium condition and central hemodynamics (Fig. 3).



**Fig. 3. Magnetic resonance imaging of the heart of patient Sh.P.I.**

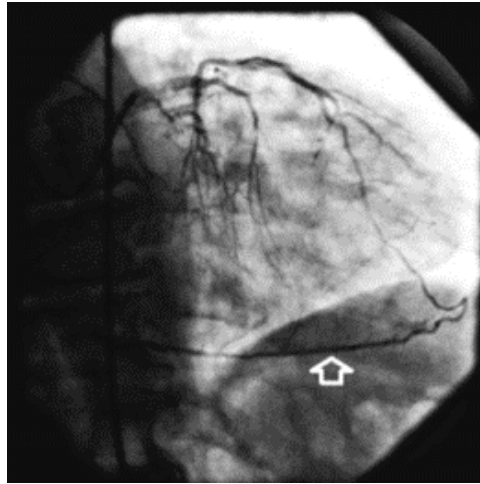
MRI protocol read the following: *LV EDD=7 cm, LV ESR=6.5 cm, IVS=1.4–0.9 cm, LV lateral wall = 0.5–0.6 cm. There were signs of fibrous basal aneurysm of the posterolateral LV wall.*

To assess the perfusion and myocardial function, a perfusion SPECT-ECG of the myocardium was performed, which revealed an increased LV cavity (EDV=260 ml) with a deep decrease in the perfusion of the posterolateral wall, but with preserved function. In the region of the apex, anterior septal wall with the transition to the IVS and the LV posterior wall, hypokinesis was found. LV EF=25%. The sign of RVV was identified.

Taking into account the increasing signs of heart failure and the data of instrumental examinations, it was decided to perform selective coronary angiography.

Selective coronary angiography data were the following: *the right-side type of coronary blood supply. No stenotic changes were found in the region of the LCA trunk. In the anterior interventricular branch, there was Grade 3 stenosis in the proximal third, then occlusion below the origin of the diagonal branch (DB). DB was diffusely affected throughout*

*its length without hemodynamically significant stenoses. There was an occlusion in the proximal third of the circumflex branch. The RCA was occluded from the orifice, communicating with the system of the right interventricular branch (RIVB) via the collateral (indicated by the arrow) (Fig. 4).*

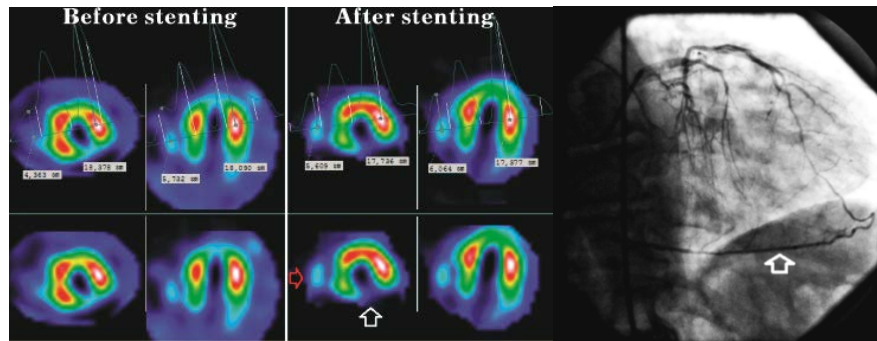


**Fig. 4. Coronarogram of patient Sh.P.I. The collateral between the right coronary artery and the right interventricular branch**

Balloon angioplasty with recanalization and RIVB stenting was performed in two stages (two stents were installed). It was not technically feasible to recanalize the RCA.

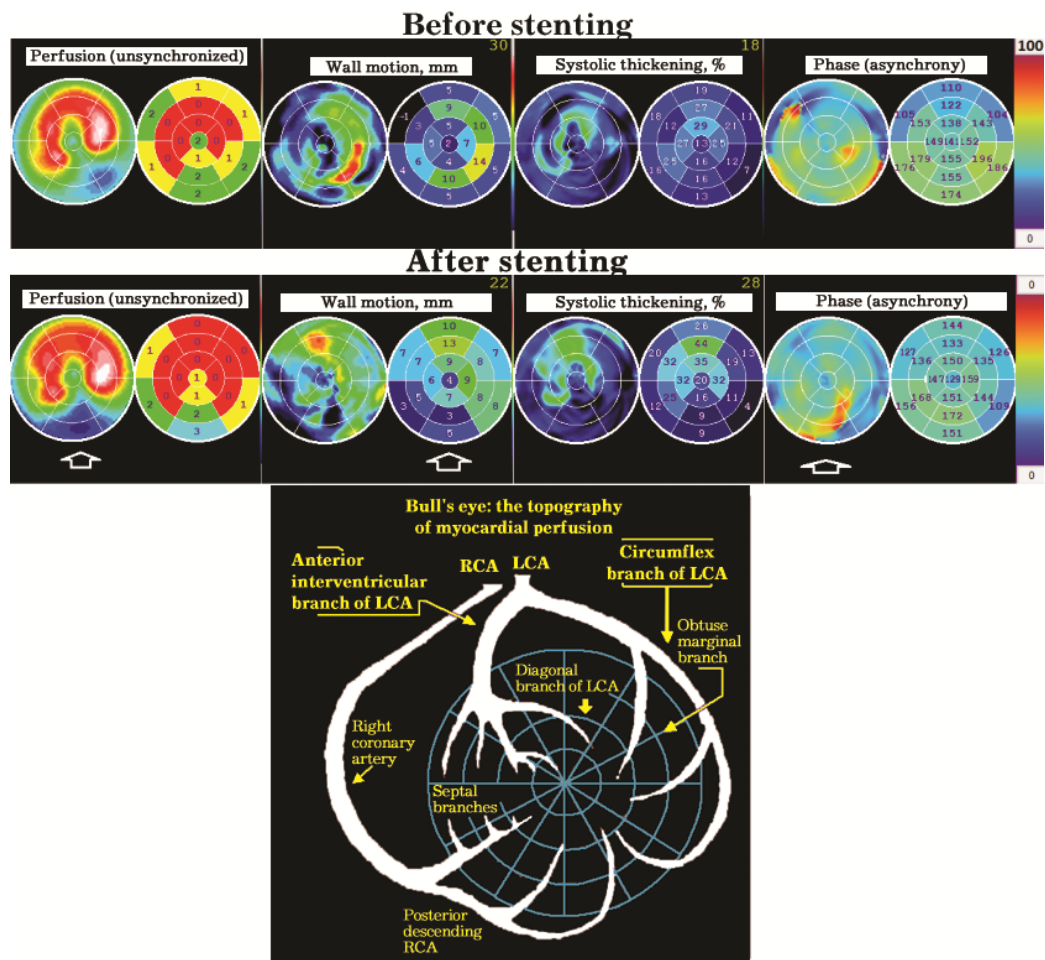
Control echocardiography showed an improvement in the kinetics of the interventricular septum and an increase in LV EF from 25% to 44%.

To assess evaluate the undertaken treatment efficacy, a perfusion myocardial SPECT was repeated. When comparing the image of the myocardium in coronal and transversal sections, shown in Fig. 5, a (relative) increase in perfusion of the RV free wall (RVV) and an improvement in diastolic function (volume/time curve).



**Fig. 5. Coronary and transverse sections of the myocardium before and after revascularization in patient Sh.P.I. Collateral blood flow according to coronary angiography is indicated by an arrow.**

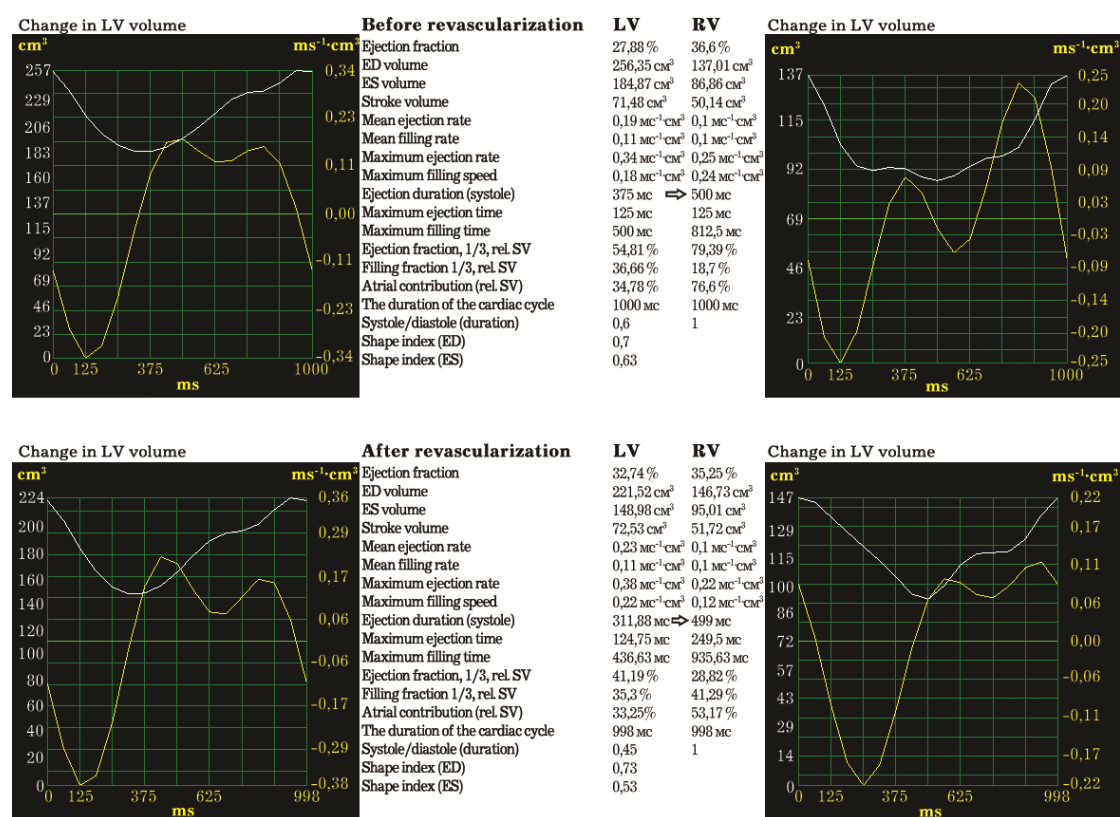
The increase in blood flow in the RV lateral wall (indicated by the red arrow in Fig. 5) is most likely associated with increased collateral blood flow (indicated by the white arrow) from the RIVB to the RCA system. On polar maps, when comparing images after stenting, the zone of reduced perfusion of the posterior wall increased in area (arrowed).



**Fig. 6. Perfusion single photon emission computed tomogram of the left ventricle myocardium in patient Sh.P.I.**

Polar motion maps revealed akinesis of the LV diaphragmatic wall and the appearance of a new focus of pathological asynchrony in the phase image (Fig. 6). In addition, there was an increase in motion and systolic thickening of the myocardium in the segments of the anterior LV wall. The increase in blood flow in the RV lateral wall can be explained by the presence of a collateral from the system of the left coronary artery to the system of the right coronary artery.

The construction of hemodynamic curves made it possible to establish changes in the functions of the LV and RV after revascularization (Fig. 7).



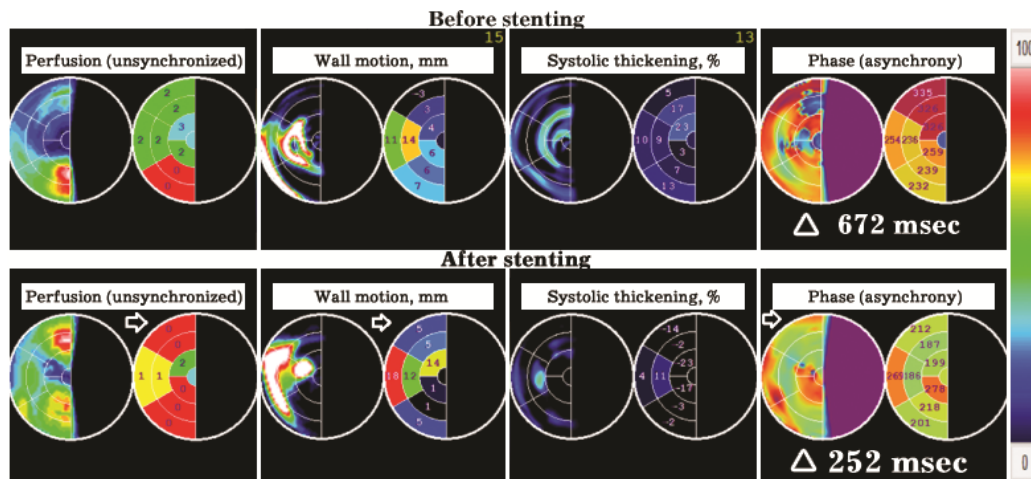
**Fig. 7. Hemodynamic curves of the left and right ventricles as per perfusion single photon emission computed tomography in patient**

These changes were ambiguous. After revascularization, there was an improvement in LV contractile function: an increase in LV EF from 27 to 33% (primarily due to an increase in LV anterior wall perfusion and a decrease in LV cavity). Meanwhile, there was an increased focus of reduced perfusion of the LV diaphragmatic wall.

At the same time, the sections clearly show a relative increase in perfusion of the RV free wall that was accompanied by an improvement in its diastolic function (volume/time curve). In order the reader could see changes in the RV free wall myocardium, we present its images obtained using the CARFI program. Comparison of images of LV polar maps revealed positive dynamics associated with revascularization: an increase in regional perfusion and a decrease in pathological asynchrony from 672



ms to 252 ms in 9 segments, which contributes to the improvement of its function (Fig. 8).



**Fig. 8. Perfusion single photon emission computed tomogram of the right ventricle myocardium in patient Sh.P.I.**

The postoperative course was uneventful, the patient was discharged from hospital in a satisfactory condition, but nevertheless, the question about the cause of deterioration in LV posterolateral wall perfusion and its myocardial function remains open and does not exclude the occurrence of cardiac events.

It is important to note that the relative increase in right ventricular myocardial perfusion and its increased visualization may be associated with right ventricular hypertrophy or an increase in its need for blood supply (for example, in pulmonary hypertension of various origins). To assess the relationship between the ratio of maximum radiopharmaceutical accumulation in the lateral wall of the left and right ventricles on coronal and transversal sections and the pulmonary artery pressure, the Spearman rank correlation coefficient was calculated. In our study, the relationship between the right ventricle increased visualization



and the pulmonary artery pressure was excluded by the results of the correlation analysis (Tables 14 and 15).

On the other hand, using perfusion myocardial scintigrams at rest, contrary to expectations, we detected live cardiomyocytes in the right ventricle, which accumulated the injected radiopharmaceutical. As can be seen, in patients of the 2nd group with initially increased visualization of the right ventricular myocardium, a further increase in the right ventricle visualization may be associated with a significantly greater loss of left ventricular cardiomyocytes than in patients of the 1st group. This statement is supported by an increase in the left ventricle ejection fraction immediately after revascularization, which was observed in the 1st group.

The above confirms that patients with post-infarction focal cardiosclerosis need the perfusion myocardial scintigraphy to determine the prospects for planned revascularization. Further studies will assess the effect of the right ventricular visualization scintigraphic sign on the results of revascularization in various myocardial diseases and coronary circulation disorders.

### **Conclusions**

1. Patients without signs of the right ventricle increased visualization (Group 1) after revascularization showed a statistically significant increase in the left ventricle ejection fraction ( $p\text{-value}=0.024$ ), a decrease in end-systolic volume ( $p\text{-value}=0.024$ ), an increased motion in the segments corresponding to the peri-infarct scar zone ( $p\text{-value}=0.016$ ), and a change in systolic thickening in the segment of the anterolateral wall basal sections ( $p\text{-value}=0.046$ ).

2. Initially increased right ventricle visualization in patients with extensive focal changes in the left ventricular myocardium suggests the

absence no increase in the left ventricular ejection fraction after myocardial revascularization.

3. An increase in the right ventricle visualization after complete myocardial revascularization indicates an intersystem redistribution of perfusion in favor of the preserved myocardium of this cardiac portion.

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