

# Influence of coronary artery bypass grafting performed in patients with unstable angina on left ventricular remodelling in medium-term follow-up

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## Abstract

**Introduction:** Left ventricular remodelling is a process of change in size, shape, wall thickness and heart function, initiated by a noxious stimulus such as ischaemia. Methods of pharmacological and surgical inhibition or reversal of remodelling are being sought.

**Aim:** To assess the influence of coronary artery bypass grafting on echocardiographic measures of left ventricular size and shape in medium-term follow-up.

**Methods:** In a group of 30 patients three echocardiographic examinations were performed: before CABG operation, 3 months after and 20 months after the operation. Left ventricular area and volumes as well as indices of sphericity, thinning and expansion were calculated.

**Results:** After the operation, left ventricular areas measured in short axis and in apical four-chamber view increased among patients with a history of myocardial infarction. Improvement in the sphericity index occurred after the operation in patients with a history of myocardial infarction in whom the ejection fraction before the operation was less than 50%.

**Conclusions:** The left ventricular remodelling process progresses after coronary artery bypass grafting in patients with a history of myocardial infarction. Inhibition of remodelling may be expected in patients without myocardial infarction, with preserved left ventricular systolic function.

**Key words:** left ventricular remodelling, ischaemic heart disease, CABG

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## Introduction

Left ventricular remodelling is a process of change in size, shape, wall thickness and function of the heart, caused by a noxious stimulus [1]. Remodelling occurring after MI is best recognized. The first observations of changes in shape and systolic function of the heart muscle following coronary artery ligation were made by Tennant and Wiggers in 1935 [2].

The most pronounced feature of remodelling is the dilatation of the heart chamber occurring in 30-50% of MI cases, which may develop early (within one month)

after infarction or later (within six months) or may progress gradually from the onset of infarction [3-5]. Chamber dilatation is accompanied by a change in shape from ellipsoid to spherical [6] and, as a consequence, according to Laplace's law, wall stress increases [7]. Chamber dilatation has prognostic significance for the development of congestive heart failure [8, 9] and mortality [10]. Gross alterations that are pronounced at the organ level result from processes taking place in the tissue and cells. Ischaemia leading to necrosis causes activation of an inflammatory reaction

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with release of metalloproteinases and proteolytic enzymes from granulocytes and destruction of collagen scaffolding supporting cardiomyocytes. Resulting slippage of cardiomyocytes leads to wall thinning and elongation. This process occurs early, within several days after MI, and is called infarct expansion [11]. Later, scar tissue is formed, and in areas distant from the necrotic area adaptive mechanisms compensating for increased wall stress develop aimed at maintaining cardiac output. Cardiomyocyte hypertrophy is initiated by mechanical stimuli, stretch and by humoral factors: increased activity of norepinephrine, endothelin, the tissue renin-angiotensin system, insulin-like (IGF), fibroblast (FGF) growth factors and cardiotrophin [12, 13]. Activated protooncogenes (c-fos, c-myc and c-jun) produce regulatory proteins which in turn initiate synthesis of contractile fibres characteristic for foetal life:  $\beta$ -myosin heavy chain,  $\alpha$ -actin and  $\beta$ -tropomyosin [14]. When compensation of increased loading conditions by hypertrophy are exhausted, dilatation of heart chambers takes place and overt congestive heart failure develops [15].

There is no doubt that post-MI remodelling may be hampered by medical therapy. It was proven that  $\beta$ -adrenolytic agents and angiotensin converting enzyme inhibitors (ACEI) have a favourable influence on left ventricular volume and shape measured with the spherical index [1]. However, the question remains as to how revascularisation affects heart remodelling. The results of investigation of this issue are equivocal. Reported effects on cardiac remodelling of coronary angioplasty performed in the chronic state after MI differ among authors [16]; even the role of fast opening of an infarct-related artery in remodelling prevention was recently challenged [3]. As regards surgical revascularisation, which is rarely performed in the setting of acute MI, its impact on remodelling may be assessed in patients in whom coronary artery bypass grafting (CABG) is performed later after MI. The results of the CASS study in a subgroup of patients with the left ventricular ejection fraction below 35% proved the survival benefit of coronary artery bypass grafting (CABG) in this group of patients compared to medical treatment [17]. The role of myocardial viability assessment before surgery for the prediction of systolic function improvement is established [18]. To examine how cardiac remodelling is modified by CABG, diastolic left ventricular volume and indices of its geometry should be measured.

The aim of our study was to follow by serial echocardiographic examinations the changes in left ventricular volume and geometry in patients undergoing CABG: before surgery and in the medium-term follow-up period.

## Methods

### Patients

The study group consisted originally of 59 patients who underwent CABG surgery in the Cardiac Surgery Department of our institution between June 2000 and June 2001. They were consecutive patients with symptoms of unstable angina who were operated on urgently [19]. In all the patients creatine kinase (CK) and its isoenzyme CKMB activities were measured to exclude MI. Myocardial infarction was diagnosed when CKMB levels exceeded 5% of total CK [20, 21].

The following data were analysed:

- demographic data (age, gender, body mass index – BMI),
- history of MI and percutaneous coronary angioplasty, presence of arterial hypertension, diabetes, lipid disorders,
- medical treatment,
- localisation and percentage of stenosis in coronary arteries revealed by angiography, number and site of bypass grafts implantation,
- completeness of revascularisation.

### Echocardiography

Echocardiographic examination was performed three times: 1-4 days before surgery, 8-11 weeks after surgery and 18-20 months after surgery. Each time the following parameters were measured or calculated:

- in apical four- and two-chamber views:
  - end-diastolic and end-systolic left ventricular area (AREA D 4C and 2C, AREA S 4C and 2C),
  - end-diastolic and end-systolic left ventricular long axis dimension (L D 4C and 2C, L S 4C and 2C),
  - end-diastolic and end-systolic left ventricular volume (EDV 4C and 2C, ESV 4C and 2C),
  - left ventricular ejection fraction (EF 4C and 2C),
  - ejection fraction calculated by biplane method (EF BP).
- in parasternal long axis at the level of papillary muscles:
  - end-diastolic left ventricular area in short axis (AREA SAX D),
  - length of the anterior segment measured along the endocardial border contained between an anterior and posterior papillary muscle (P),
  - length of the posterior segment measured along the endocardial border contained between a posterior and anterior papillary muscle (T),
  - interventricular septum thickness (A),
  - posterior wall thickness (B).

Parameters of AREA D 4C and 2C, AREA S 4C and 2C, LD 4C and 2C, ESV 4C and 2C, EF 4C and 2C, EF BP and AREA SAX D were measured according to the guidelines of the American Society of

Echocardiography [22]. Anterior (P) and posterior (T) segment length and interventricular septum (A) and posterior wall thickness (B) were measured as described by Jugdutt and Michorowski [23]. In accordance with these authors the expansion index (Eix) as a quotient of P and T and the thinning index (Tix) as a quotient of A and B were calculated. The sphericity index (Six) was calculated in accordance with Gannau et al [24] using the following formula:  $Six = \sqrt{(AREA\ SAX\ D/3.14) / (L\ D\ 4C/2)}$ . Hewlett-Packard Sonos 2500 and Sonos 2000 equipment was used.

### Statistical analysis

Results are presented as mean  $\pm$  standard deviation. The value of  $p$  ( $\alpha$ ) $<0.05$  was considered significant. The Fisher exact test was used to compare the nonparametric variables. Repeated echocardiographic measures were analysed by ANOVA.

### Results

Among the 59 patients originally enrolled into the study, five died during the perioperative period. In one patient perioperative MI was diagnosed. In five the quality of echocardiographic view was poor, and another five had coronary angioplasty in the past. These patients were not included in the analysis. During the whole follow-up period two patients died, one had re-CABG surgery, and four had MI or percutaneous coronary angioplasty. In three patients recurrence of angina occurred, and another three did not attend the follow-up echocardiographic examination.

Finally, we analysed the data of 30 patients, in whom all three echocardiographic examinations were completed. Clinical characteristics of the study group, coronary angiography data and data pertaining to implanted grafts are shown in Table I.

Analysis of the echocardiographic data obtained in the three examinations was performed in two subgroups of patients: with [MI(+)] and without [MI(-)] a history of MI. There were no significant differences between the subgroups in age [66 $\pm$ 6 in MI(+) and 65 $\pm$ 6 in MI(-)], gender [4 females in MI(+) and 8 in MI(-)], number of patients with three-vessel disease [11 in MI(+) and 14 in MI(-)] or completeness of revascularisation [10 in MI(+) and 11 in MI(-)]. Patients with MI history more frequently had hypertension (14 patients, 100%) than patients without MI (9 patients, 64%),  $p=0.007$ , and also more frequently were treated with ACEI [13 (93%) and 5 (31%), respectively,  $p=0.001$ ].

Also a subgroup was identified consisting of patients who had MI (or there was no history of MI but chronic occlusion of a coronary artery was diagnosed on angiography – there were three such patients) and

**Table I.** Coronary angiography data, number and types of implanted grafts in the study group. LAD – left anterior descending artery, RCA – right coronary artery, Cx – circumflex artery, OM – marginal branch of Cx, LM – left main artery, LIMA – left internal mammary artery, VG – venous graft, ASA – acetylsalicylic acid, ACEI – angiotensin enzyme inhibitor, BB – betaadrenolytic agent

Gender	12 females, 18 males
Age	50-76 years (mean 65.8 $\pm$ 6.3)
BMI	19-31 (mean 24.4 $\pm$ 2.5)
History of infarction	14 (46%), in 8 anterior
Hypertension	23 (76%) patients
Diabetes	4 (13%), treated with insulin 4
LDL cholesterol >100 mg%	12 (40%)
Treatment: nitrate, ASA, ACEI, BB, statin	30 (100%), 30, 16 (53%), 26 (86%), 20 (66%)
Lesions >70% of luminal diameter	LAD – 29 (97%) patients, in 4 occlusion RCA – 23 (76%) patients, in 4 occlusion Cx – 21 patients LM (lesion >50%) – 10 (33%) patients
Implanted grafts	LIMA to LAD – 13 grafts VG to LAD – 16 grafts VG to RCA – 21 grafts VG to OM – 15 grafts

divided into patients with depressed baseline BP EF (<50%) – group Efo – and with normal left ventricular systolic function (EF $\geq$ 50%) – group EFp. Groups Efo and EFp did not differ significantly in demographics, risk factors, medical treatment, results of coronary angiography or surgical treatment.

Two-factor analysis of variance ANOVA of the echocardiographic parameters measured at the three time points provided the following results.

Two parameters showed significant fluctuations in time (between examinations). The MI(+) and MI(-) groups differed significantly in the course of time as to the parameters of AREA D 4C and AREA SAX D.

AREA D 4C increased significantly in MI(+) patients from an initial examination through follow-up examination 1 and to follow-up examination 2. In MI(-) group this parameter was stable.

A post hoc analysis was also performed for the parameter AREA SAX to assess the significance of changes of this parameter between examinations. Significant changes and  $p$  values are listed below. AREA SAX D increased gradually in MI(+) group. In MI(-) patients this parameter did not change between the baseline examination and follow-up examination 2, and a significant change occurred between the baseline and follow-up

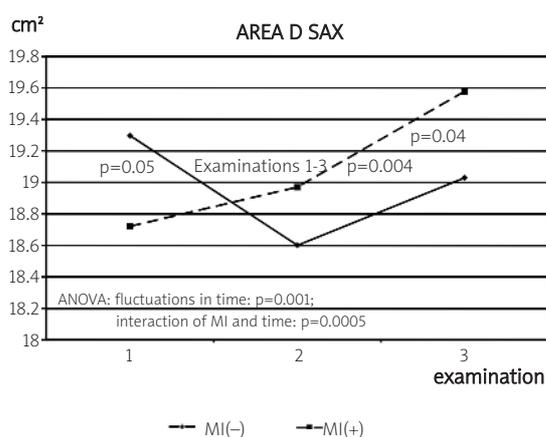
**Table II.** Two-factor analysis of variance with repeated measures: mean values and standard deviations of echocardiographic parameters in the three examinations in patients without a history of MI [MI(-)] (N=16) or in patients with a history of MI [MI(+)] (N=14).

P value is given for significance of fluctuations in time and for differences in time between groups BZ and Z (interaction between analysed factors: factor 1 – presence of MI in history and factor 2 – fluctuation in time)

Parameter	Group	Echocardiographic examination			p value
		baseline	follow-up 1	follow-up 2	
AREA D 4C [cm <sup>2</sup> ]	MI(+)	26.33±4.82	26.04±4.35	25.85±4.73	fluctuations in time: p=0.04 interaction: p=0.0007
	MI(-)	25.83±4.26	26.40±4.27	27.75±4.88	
AREA SAX D [cm <sup>2</sup> ]	MI(+)	19.30±6.04	18.60±5.03	19.03±5.49	fluctuations in time: p=0.001 interaction: p=0.0005
	MI(-)	18.72±3.19	18.97±3.51	19.58±3.20	

**Table III.** Two-factor analysis of variance with repeated measures: mean values and standard deviations of echocardiographic parameters in the three examinations in patients without a history of MI [MI(-)] (N=16) or in patients with a history of MI [MI(+)] (N=14). P value is given for differences in time between groups BZ and Z (interaction between analysed factors: factor 1 – presence of MI in history and factor 2 – fluctuation in time)

Parameter	Group	Echocardiographic examination			p value
		baseline	follow-up 1	follow-up 2	
L D 4C [cm]	MI(+)	6.91±0.57	6.88±0.56	6.95±0.57	p=0.01
	MI(-)	7.19±0.75	7.38±0.71	7.25±0.82	
Elx	MI(+)	1.447±0.569	1.465±0.585	1.454±0.570	p=0.05
	MI(-)	1.368±0.743	1.335±0.657	1.427±0.718	



**Figure 1.** Diastolic area of the left ventricle measured in short axis (AREA SAX D) in three examinations in groups with [MI(+)] and without [MI(-)] history of myocardial infarction

examination 1 (Figure 1). Mean values of the above parameters for the three examinations in the MI(+) and MI(-) groups are summarised in Table II.

Parameters of AREA S 4C, L S 4C, EDV 4C, AREA D 2C, L D 2C, L S 2C, AREA S 2C or thinning index (Tlx) did not change significantly.

Two parameters, L D 4C and Elx, were different between MI(+) and MI(-) groups only in the time course.

L D 4C increased transiently in MI(+) patients in follow-up examination 1 compared to the baseline and again decreased in follow-up examination 2. However, in MI(-) group this parameter transiently decreased in follow-up examination 1 compared to the baseline and in follow-up examination 2 returned to a higher value.

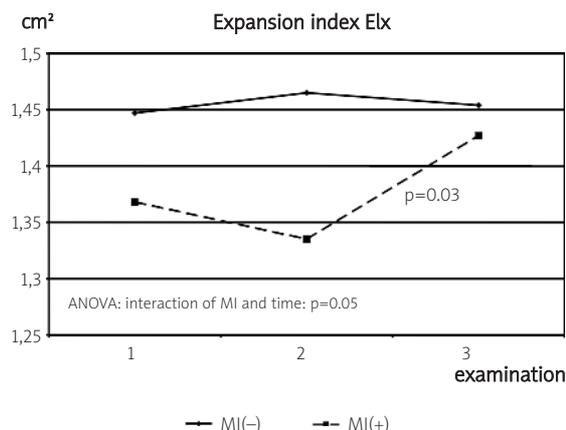
The Elx index decreased transiently in follow-up examination 1 compared to the baseline in MI(+) patients (p=NS) and increased again in follow-up examination 2, p=0.03. In the MI(-) group this index transiently showed an increase in follow-up examination 1 and returned to its initial value in follow-up examination 2 without significant changes between examinations in this group (Figure 2). Mean values of L D 4C and Elx for the three examinations in MI(+) and MI(-) groups are shown in Table III.

Two-factor analysis of variance of the parameters ESV 4C, EF 4C, EDV 2C, ESV 2C, EF 2C, EF BP and sphericity index measured at the three time points showed significant fluctuations of these parameters over time, but without significant differences between MI(+) and MI(-) patients. Mean values of these parameters are shown in Table IV.

The analysis performed among patients with a history of MI, divided into subgroups with decreased baseline EF (<50%) – group Efo – and normal EF (≥50%) – group EFp – revealed that the values of this parameter changed significantly with time. In the group with normal EF, the value of EF did not increase significantly between the initial examination and follow-up examination 2, whereas in group with depressed baseline EF the increase was pronounced. Table V shows the mean values of EF BP for the three examinations.

The sphericity index changed significantly with time and its time course was also different between groups Efo and EFp. In group Efo the sphericity index changed as follows: its value decreased (improvement) from the baseline examination to follow-up examination 1 and there was some increase in follow-up examination 2. In group EFp the sphericity index gradually increased between examinations (Table VI, Figure 3).

No significant changes or differences occurred between subgroups Efo and EFp in the thinning index (Tlx) and the expansion index (Elx).



**Figure 2.** Expansion index (Elx) in three examinations in groups with [MI(+)] and without [MI(-)] history of myocardial infarction

### Discussion

Left ventricular EF increased significantly after CABG surgery in the study group patients. The increase was seen in both four-chamber and two-chamber apical views and, as a consequence, also in biplane assessment. Such improvement was expected considering published reports on this issue [25]. Improvement in systolic function after revascularisation depends on confirmation of myocardial viability [18]. In the study group comprising patients with unstable angina, viability

**Table IV.** Two-factor analysis of variance with repeated measures: mean values and standard deviations of echocardiographic parameters in the three examinations in patients without a history of MI [MI(-)] (N=16) or in patients with a history of MI [MI(+)] (N=14). P value is given for fluctuations in time

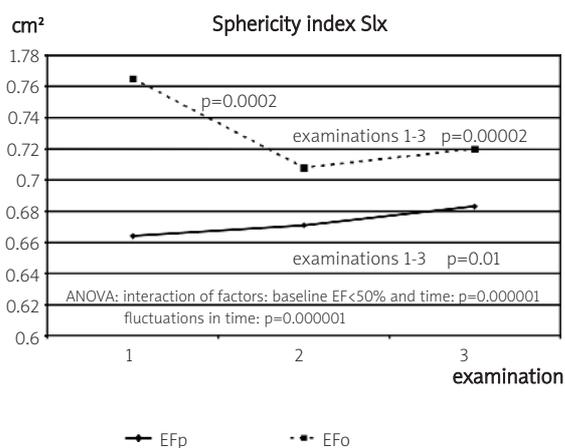
Parameter	Group	Echocardiographic examination			p value
		baseline	follow-up 1	follow-up 2	
ESV 4C [mL]	MI(+)	40.66±16.10	39.86±18.31	35.73±15.79	p=0.00005
	MI(-)	48.12±18.15	51.46±14.47	39.82±15.67	
EF 4C [%]	MI(+)	50.72±7.11	52.71±7.58	56.60±9.06	p=0.002
	MI(-)	44.34±12.79	42.59±11.22	51.72±13.16	
EDV 2C [mL]	MI(+)	87.45±30.21	87.83±30.96	83.44±29.14	p=0.007
	MI(-)	79.90±25.73	83.21±24.46	80.61±26.76	
ESV 2C [mL]	MI(+)	43.85±25.35	43.74±27.46	39.03±24.84	p=0.003
	MI(-)	35.36±15.43	35.93±16.20	34.43±16.38	
EF 2C [%]	MI(+)	51.86±10.79	52.76±11.81	55.74±11.42	p=0.001
	MI(-)	56.53±12.46	58.15±12.06	59.80±13.80	
EF BP [%]	MI(+)	50.76±7.84	52.26±8.39	55.82±8.98	p=0.0002
	MI(-)	52.86±8.45	50.54±6.76	56.71±10.34	
Six	MI(+)	0.715±0.100	0.690±0.084	0.702±0.084	p=0.003
	MI(-)	0.682±0.079	0.669±0.082	0.693±0.080	

**Table V.** Two-factor analysis of variance with repeated measures: mean values and standard deviations of echocardiographic parameter EF BP in three examinations among patients with myocardial infarction with normal (group EFp, N=9) and depressed (group Efo, N=8) left ventricular ejection fraction. P value was given for differences in values between groups Efo and EFp and for significance of fluctuations in time

Parameter	Group	Echocardiographic examination			p value
		baseline	follow-up 1	follow-up 2	
EF BP [%]	EFp	56.40±5.90	57.03±6.77	57.93±8.32	EFp vs Efo: p=0.02 fluctuations in time: p=0.02
	Efo	45.13±4.94	47.49±7.29	53.71±9.67	

**Table VI.** Two-factor analysis of variance with repeated measures: mean values and standard deviations of echocardiographic parameter – sphericity index Six among patients with myocardial infarction with normal (group EFp, N=9) and depressed (group Efo, N=8) left ventricular ejection fraction. P value was given for fluctuations of parameter in time and differences in the course of time between groups EFp and Efo (for interaction between analysed factors: factor 1 – presence of depressed EF at baseline and factor 2 – fluctuation in time)

Parameter	Group	Echocardiographic examination			p value
		baseline	follow-up 1	follow-up 2	
Six	EFp	0.664±0.069	0.671±0.064	0.683±0.060	fluctuations in time: p<0.000001 interaction: p<0.000001
	Efo	0.765±0.105	0.708±0.102	0.720±0.103	



**Figure 3.** Sphericity index (Six) in three examinations in patients with myocardial infarction in subgroups with baseline EF<50% (group Efo) and EF≥50% (group EFp)

studies were not performed; angina was considered as an indication for surgery and also as an indirect sign of myocardial viability [26]. An increase in EF should be attributed to a decrease in end-systolic volume measured in both apical views. End-diastolic volume measured in apical two-chamber view also decreased. Improvement in left ventricular systolic function occurred in the whole study group, without differences between patients with or without MI.

The sphericity index, an important measure of left ventricular remodelling, did not differ either between groups with or without MI. Its values changed with time, but the time course was similar in both groups. Compared to the baseline, the sphericity index increased (deteriorated) at follow-up examination 2 among patients with MI; however, there was no significant difference compared to the group without MI. As the value of L D 4C was stable, changes in the sphericity index should be attributed to alterations in AREA SAX D, which remained unchanged in the subgroup without MI and increased in the subgroup with MI. These changes, although their time course differed between subgroups, did not result in significant differences in AREA SAX D values between subgroups with or without MI.

It should be noted that the two-factor analysis of variance ANOVA did not show significant differences in values of measured echocardiographic parameters between groups with or without MI. The reason for the lack of differences may be attributed to the fact that patients with unstable angina have a high probability of presence of contractile dysfunction (myocardial hibernation) which cannot be differentiated from necrosis before revascularisation without viability testing. However, after revascularisation increased myocardial perfusion achieved in patients with a history of MI might have improved the function and structure of infarcted areas where residual viability was present, as well as of ischaemic, hibernated areas, most probably

present in patients with multivessel coronary artery disease [27].

The differences in measured parameters were significant between the time points (as the increase in EF described above) and in the pattern of time course in subgroups. For the expansion index EIx also there were no differences in values between subgroups with or without MI history; however, its time course differed, being stable in those without MI and increasing in those with MI. An increase of the EIx index indicated remodelling progression and was caused by elongation of the anterior segment localised between the papillary muscles, measured in the short axis of the ventricle. Elongation occurred in patients with anterior wall MI. The posterior segment elongated in one patient with inferior wall MI and in one with anterior wall MI history, but this did not influence its value.

Among patients with a history of MI, EF increased in the follow-up period if it did not exceed much more 50% at baseline, compared to cases of baseline  $EF \geq 50\%$ . The increase should probably be attributed to perfusion improvement in hibernated myocardium, but it cannot be stated unequivocally whether the hibernation comprised infarcted area or myocardium subtended by stenotic arteries. In cases where the systolic function was not compromised in spite of ischaemia (patients with  $EF \geq 50\%$ ), improvement did not occur after revascularisation either. The value of the sphericity index was not different between subgroups EFo and EFp, but it improved significantly in the group with depressed EF at baseline and even deteriorated in the group with baseline  $EF \geq 50\%$ .

The results of our investigation in the aspect of an increase in EF and decrease in end-diastolic volume after CABG are in concordance with published data. Improvement in the systolic function of heart muscle after graft implantation is attributed to better blood supply delivered by a patent graft [27]. Besides influencing heart function, surgical revascularisation may lead to a decrease in chamber volume; in patients with markedly dilated left ventricles operated on at Yale University the left ventricular volume index decreased from 175 ml/m<sup>2</sup> to 144 ml/m<sup>2</sup> after CABG. On the basis of this observation it was postulated that CABG surgery may reverse the remodelling process [28].

Our results suggest that myocardial revascularisation by CABG may modify the process of left ventricular remodelling in a different way in a heart muscle damaged by infarction and in an ischaemic myocardium without necrosis. It is accepted that left ventricular remodelling is a consequence of infarction and necrosis, but remodelling was also observed in the setting of chronic hypoperfusion without concomitant infarction. Chen et al [29] in an experimental model described wall thinning, chamber dilatation and compensatory hypertrophy following chro-

nic stenosis placement in a left anterior descending artery with resting blood flow reduction to 40%. A histological examination did not reveal a transmural infarction, but in some cases small foci of necrosis and fibrosis were seen, that did not exceed 2% of the total area subtended by a stenotic artery, localised in the subendocardial region. The authors suggested that in the state of chronic hypoperfusion leading to myocardial hibernation and depression of contractile function, adaptive mechanisms similar to those that follow infarction are initiated: increased curvature of the akinetic wall increases its stress, which in turn promotes cardiomyocyte hypertrophy, ventricular filling pressure increases and the chamber dilates in order to maintain stroke volume. It may be assumed that this mechanism, dysfunction caused by ischaemia and its reversion through revascularisation, was responsible for changes in parameters describing remodelling seen in our investigation.

In the subgroup of patients without a history of MI, expansion and sphericity indices improved with time compared to the subgroup with a history of MI. Among patients with MI, improvement of the sphericity index with time occurred in cases where baseline EF was below 50%. In these cases also, compared to patients with normal baseline EF, significant improvement in EF occurred. As the study group comprised patients with unstable angina, we assumed that anginal symptoms may indicate the presence of viable myocardium. Our results are similar to those reported by other authors [30] who observed a decrease in left ventricular end-diastolic volume following surgical revascularisation in patients without previous MI and significant improvement in EF in patients with MI history, in whom it was depressed before the procedure.

As controversies exist about the role of infarct-related artery patency in the remodelling process [31], the importance of microcirculation integrity for myocardial viability has been raised [32]. The requirement for viability confirmation before surgical revascularisation to achieve systolic function improvement is doubtless [18, 26]. Also, the results of our investigation suggest that CABG may hamper or reverse ischaemic remodelling when the revascularised myocardium is viable. Besides CABG, cardiac surgery offers different operative techniques for patients with ischaemic heart failure [33]; an ongoing STICH trial [34] of surgical reconstruction of the left ventricle aims to answer the question of which patients will benefit from this procedure alone, and for which it should be combined with CABG.

## Conclusions

1. Coronary artery bypass grafting may improve those indices of left ventricular remodelling which describe chamber shape and size, while indices of wall thickness remain unchanged.

2. In spite of improvement in left ventricular systolic function, in patients with a history of MI left ventricular remodelling progresses after CABG surgery.
3. In certain patient groups – with left ventricular ejection fraction below 50%, without a history of MI – surgery may inhibit the process of ischaemic remodelling.

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## Wpływ operacji pomostowania naczyń wieńcowych wykonywanej u pacjentów z niestabilną dławicą piersiową na występowanie przebudowy lewej komory w obserwacji średnioterminowej

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### Streszczenie

**Wstęp:** Przebudowa lewej komory jest procesem polegającym na zmianie wielkości, kształtu, grubości ścian i funkcji serca, zapoczątkowanym przez działanie czynnika uszkodzającego, np. niedokrwienia. Trwają poszukiwania metod farmakologicznego i zabiegowego zahamowania lub odwrócenia przebudowy serca.

**Cel:** Ocena wpływu operacji pomostowania naczyń wieńcowych na parametry echokardiograficzne opisujące wielkość i kształt lewej komory w obserwacji średnioterminowej.

**Metody:** U 30 chorych wykonano 3 badania echokardiograficzne: bezpośrednio przed operacją CABG, a następnie 3 mies. i 20 mies. po operacji. Dokonywano pomiarów pola powierzchni i objętości lewej komory wg wskaźników sferyczności, ścienienia i ekspansji.

**Wyniki:** W podgrupie pacjentów po przebytych zawale serca po operacji doszło do powiększenia pól powierzchni lewej komory w osi krótkiej i w projekcji koniuszkowej czterojamowej. Poprawę sferyczności obserwowano po operacji wśród chorych po przebytych zawale, u których frakcja wyrzutowa lewej komory przed zabiegiem była niższa niż 50%.

**Wnioski:** Po operacji CABG wykonanej u chorych po przebytych zawale serca postępuje przebudowa lewej komory. Zahamowania przebudowy można spodziewać się u chorych, którzy nie przeżyli zawału, z zachowaną funkcją skurczową lewej komory.

**Słowa kluczowe:** przebudowa lewej komory, choroba niedokrwienna serca, CABG

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