Influence of Direct Myocardial Revascularization on Left Ventricular Asynergy and Function in Patients with Coronary Heart Disease

With and without Previous Myocardial Infarction

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SUMMARY

The influence of successful aorto coronary artery bypass surgery on left ventricular asynergy and dysfunction was studied by hemodynamic and angiographic methods in 29 patients with coronary artery disease. Eight patients had the preinfarction syndrome, 10 patients had chronic ischemia without previous infarction, and 11 patients had chronic ischemia with previous infarction. LV asynergy was present preoperatively in 12 of the 18 patients in the first two groups. Marked improvement occurred in all and a normal wall motion was restored in the majority following surgery. More pronounced improvement was noted in the preinfarction syndrome as compared to the group with chronic ischemia and no previous infarct. The ejection fraction was reduced in 12 of the 18 patients in these two groups and significant improvement was observed postoperatively ([0.45 ± 0.03 (SEM) to 0.74 ± 0.03]. Increase in ejection fraction was primarily due to a decrease in the end-systolic volume (71 ± 12 to 23 ± 4 ml/m²). The end-diastolic volume was only slightly reduced (114 ± 12 to 97 ± 9 ml/m²). Left ventricular end-diastolic pressure fell from 15 ± 1 to 10 ± 1 mm Hg.

In nine of 11 patients who had previous myocardial infarction, abnormal wall motion was present preoperatively. Following surgery, some abnormalities of wall motion persisted in the areas of known infarction, although significant improvement of wall motion occurred in the noninfarcted segments. The ejection fraction was reduced in seven of these 11 patients and improved postoperatively (0.44 ± 0.05 to 0.59 ± 0.05). The end-systolic volume decreased from 57 ± 5 to 41 ± 6 ml/m², and the end-diastolic volume was unchanged (106 ± 5 to 108 ml/m²). Left ventricular end-diastolic pressure fell from a mean value of 17 ± 3 to a mean value of 10 ± 2 mmHg following successful surgery. These findings are consistent with improved pump function and were associated with improvement in indices of contractile state. The observations indicate that significant improvement in ventricular wall motion and pump function occurs in patients with obstructive coronary disease following successful aorto coronary artery bypass surgery even in the presence of old myocardial infarction. Since the patients of the present study all had normal initial end-diastolic volumes, however, similar beneficial results might not occur in patients with cardiomegaly and more severe heart failure.

Additional Indexing Words:

Systolic wall motion Ischemia Ejection fraction Compliance
Contractile state Ventricular aneurysm

THAT left ventricular asynergy occurs in patients with coronary artery disease is well documented.1-3 It has been postulated that the presence of scar tissue resulting from infarction may be primarily responsible for ventricular asynergy in such patients.1,2 However, significant abnormalities of left ventricular segmental wall motion may also occur in patients without infarction,4,5 presumably

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Supported in part by Contract No. NIH-PH-43-68-1333 under Myocardial Infarction Program, National Heart and Lung Institute, NIH Department of HEW, United States Public Health Service No. 5-S01 RR05468.

Address for reprints: Department of Cardiology, Cedars of Lebanon Hospital, 4833 Fountain Ave., Los Angeles, California 90029.

Received June 16, 1972; revision accepted for publication October 23, 1972.

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due to regional myocardial ischemia due to obstructive coronary artery disease. That regional myocardial ischemia may play a significant role in the genesis of left ventricular asynergy and dysfunction is further supported by the observation that induced hypoxia by atrial pacing may precipitate or exaggerate left ventricular asynergy. Experimentally, myocardial ischemia produced by temporary coronary artery occlusion is consistently associated with akinesis or hypokinesis of the affected wall segments. However, recovery of normal motion of the hypoxic myocardium occurs if the occlusion is promptly relieved. In patients with coronary artery disease, partial or complete recovery of the normal motion might be expected to follow relief of ischemia by direct revascularization by aortocoronary artery bypass surgery. The purpose of this study was, therefore, to analyze by hemodynamic and angiographic means the influence of direct revascularization on left ventricular asynergy in patients with coronary artery disease, both with and without previous infarct. It shows that a striking improvement can occur in left ventricular function following successful relief of acute or chronic myocardial ischemia.

Patients

Twenty-nine patients with ischemic heart disease were studied both before and within 2 weeks after successful aortocoronary artery-saphenous vein bypass surgery. The latter study was designed to evaluate graft patency primarily, and ventricular function secondarily. There were six females and 23 males, age ranging from 37 to 68 years. According to the clinical presentation and the presence or absence of historic and electrocardiographic evidence of previous infarction, the patients were divided into three groups: (1) eight patients with the preinfarction syndrome, (2) 10 patients with chronic ischemia, but without previous infarction, and (3) 11 patients with chronic ischemia and previous infarction. Of 11 patients with previous infarction, four had old anterior, six old inferior, and one recent inferior infarction. The patient with recent inferior infarction also had clinical features of cardiogenic shock at the time of surgery. In 28 of the 29 patients with coronary artery disease in this study, there was no clinical or radiologic evidence of heart failure or cardiomegaly immediately prior to surgery. Only the patient with cardiogenic shock had evidence of heart failure before surgery. Preoperative coronary arteriographic lesions with the indications and types of bypass surgery and postoperative clinical results are summarized in table 1. All patients had patent vein bypass grafts 2 weeks following surgery. This study includes the data of six patients previously reported.

Methods

Premedication prior to each catheterization consisted of seconal 100 mg orally. Left heart catheterization, left ventriculography, and coronary arteriography were performed percutaneously by the Seldinger technic via the right femoral artery. No patient was receiving a

<table>
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<th>Table 1</th>
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<tbody>
<tr>
<td><strong>Coronary Angiographic Data, Indications for Bypass Surgery, and Postoperative Clinical Results in 29 Patients with Coronary Artery Disease</strong></td>
</tr>
<tr>
<td>Data</td>
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<tr>
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</tr>
<tr>
<td>Number</td>
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<tr>
<td>Site of previous infarct</td>
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<tr>
<td>Coronary angiography:</td>
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<tr>
<td>RCA disease</td>
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<tr>
<td>LCA disease</td>
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<td>RCA and LCA diseases</td>
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<tr>
<td>Indications for surgery:</td>
</tr>
<tr>
<td>Uncontrolled chronic angina</td>
</tr>
<tr>
<td>Preinfarction angina</td>
</tr>
<tr>
<td>Shock and heart failure</td>
</tr>
<tr>
<td>Bypass surgery:</td>
</tr>
<tr>
<td>Single-vein graft</td>
</tr>
<tr>
<td>Aorto-RCA</td>
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<tr>
<td>Aorto-LCA</td>
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<tr>
<td>Double-vein graft</td>
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<tr>
<td>Aorto-RCA, LCA</td>
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<tr>
<td>Postoperative clinical results:</td>
</tr>
<tr>
<td>Angina</td>
</tr>
<tr>
<td>Heart failure</td>
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</tbody>
</table>

Abbreviations: RCA = right coronary artery; LCA = left coronary artery; A = anterior; I = inferior.
myocardial depressant drug such as propranolol, and none experienced pain during the preoperative catheterization. LV pressures were recorded through a fluid-filled Teflon loop-end catheter (no. 7F) with a zero reference level 5 cm posterior to the sternal angle. The same catheter-manometer systems were used for both pre- and postoperative evaluations. The rate of rise of LV pressure (dP/dt) was derived electronically with an R-C differentiating circuit.

Overall mean contractile element velocity (VCE) was calculated from the simultaneously recorded LV pressure pulse and its first derivative (dP/dt), using the formula VCE = dP/dt/KP where P is the developed isometric pressure and K is the series elastic constant. This calculation is based on the three-component Maxwell model of muscle mechanics. A value of 32/muscle length was used for the series elastic constant K, appropriate to previous studies in isolated heart muscle. Since the above formula for VCE approaches infinity as developed pressure approaches zero, the value of VCE at 5 mmHg developed pressure was used as an approximation of Vmax. VCE at 5 mmHg was calculated from three consecutive beats and averaged.

A quantitative assessment of LV asynergy and abnormalities of systolic wall motion (SWM) was determined from single-plane (RAO) cineangiography. Left ventricular cineangiograms were obtained using a film speed of 60 frames/sec as 30–40 cc of 75% hypaque was injected into the LV. From the ventriculograms, multiple hemiaxes were drawn and superimposed on both end-diastolic and end-systolic frames. The long axis (midpoint of the aortic root to the apex) was divided into two hemiaxes by drawing a perpendicular at its midpoint. Two more perpendiculars were drawn at the midpoint of these two hemiaxes. Four more hemiaxes were then drawn bisecting the angles formed by the long axis and perpendicular at its midpoint. After correcting for image magnification, percentage changes in these hemiaxes at end-systole were determined from the end-diastolic and end-systolic frames. The changes in the five hemiaxes of the anterior left ventricular wall, and five of the inferior walls were averaged to give a value for relative anterior wall motion (AWM) and inferior wall motion (IWM). The changes in apical motion (APM) were calculated from the percentage change in the base-to-apex axis. These values express the inward motion of the ventricular wall during systole. LV volumes were calculated from single plane (RAO) cineangiograms, assuming the ventricle to approximate an ellipsoid of revolution. The long axis was measured directly and the minor axis was determined by the area-length method of Dodge. The magnification factor was derived from the ratio of the projected width of the catheter to its actual width. The average preoperative magnification factor was 1.40 ± 0.03 (mean ± SEM); and the postoperative magnification factor was 1.40 ± 0.03, suggesting good reproducibility. Angiographically determined volumes were corrected for body surface.

Calculated LV volumes from single-plane cineangiograms in the RAO projection overestimates the actual volumes and correction has been made for this overestimation using the published regression equation of Herman et al., to allow for comparison with other data. However, as postoperative data is compared with preoperative data for each patient, our conclusions are not affected by the use of this correction. Left ventricular cineangiography was always performed prior to coronary arteriography. For calculation of segmental wall motion and ejection fraction, ectopic and postectopic cycles were always eliminated. No patient in this series had clinical or angiographic evidence of mitral regurgitation. Furthermore, in the postoperative studies, all vein-bypass grafts were angiographically patent. Values similarly obtained in six subjects without any detectable cardiac disease served as normal controls.

LV stroke volume (SV) was obtained by subtracting the angiographically determined end-systolic volume (ESV) from end-diastolic volume (EDV). The ejection fraction (EF) was obtained from the ratio of SV/EDV. Stroke-work index (SWI) was calculated from the formula:

\[
\text{SWI} \left( \text{g} \cdot \text{m}^2 \right) = \frac{\text{SVI} \times \text{MAP} - \text{LVEDP} \times 13.6}{100}
\]

where SVI = stroke-volume index (ml/beat/m²), MAP = mean systolic arterial pressure (mm Hg), and LVEDP = left ventricular end-diastolic pressure (mm Hg).

**Results**

**Segmental Wall Motion**

In six normal control subjects there was uniform systolic motion of the anterior (mean 52 ± 12%), apical (mean 33 ± 6%) and inferior walls (mean 41 ± 10%), similar to those reported by others. Seven of the eight patients with the preinfarction syndrome had reduced wall motion preoperatively involving one or more segments which corresponded in general to the angiographically determined coronary artery lesions. Figure 1 (left) shows normalization of severely reduced AWM and APM following LCA bypass surgery in a patient with the preinfarction syndrome. The postoperative changes in segmental wall motion in all patients with the preinfarction syndrome are summarized in figure 2. In all six patients with reduced AWM and in six of the seven patients with reduced APM preoperatively, marked improvement occurred following left CA-bypass surgery. IWM was found to be reduced preoperatively in three of these eight patients and improved postoperatively. Normal segmental wall motion preoperatively remained normal postoperatively.

Five of the 10 patients with chronic ischemia and no previous infarct had reduced wall motion preoperatively involving one or more segments. Figure 3 summarizes the postoperative changes in
LEFT VENTRICULAR WALL MOTION
BEFORE & AFTER AORTO-CORONARY BYPASS

Figure 1

Left ventricular wall motion before and after successful aortocoronary artery bypass surgery in three representative patients. The end-diastolic (solid line) and end-systolic (dashed line) frames together with the multiple hemiaxes (dotted lines) are superimposed for visual comparison. (Left to right) A patient with the preinfarction syndrome and a high-grade LAD lesion, a patient with a previous anterior infarction, and a patient with a previous inferior infarction and anterior ischemia.

Figure 2

Summary of postoperative changes in segmental wall motion in eight patients with the preinfarction syndrome. The normal range (mean ± SD) is indicated by the vertical bar to the left of each panel. Each patient is plotted individually (●).

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wall motion in this group of patients. The three patients with reduced AWM preoperatively had normalization of AWM postoperatively. Overall, seven of the 10 patients had some improvement in AWM postoperatively. Of the four patients with reduced APM preoperatively, three returned to the normal range postoperatively. Similarly three of four patients with reduced IWM preoperatively returned to the normal range postoperatively.

In patients with previous infarction, some abnormalities of wall motion persisted in the area of infarction as determined by preoperative electrocardiogram, although improvement of systolic motion in all other areas was observed frequently. In figure 1 (middle) changes in segmental wall motion in a patient with previous anterior infarction who had both RCA and LCA bypass are shown. Postoperatively, some improvement in inferior and anterior wall motion occurred although there was some remaining abnormality in anterior wall motion. In a similar manner a patient with previous inferior wall infarction showed significant improvement in anterior and apical motion which occurred following LCA bypass without any change in inferior wall motion (fig. 1, right). In general, individuals with anterior infarction demonstrated improvement in IWM and APM, whereas patients with inferior infarction showed improved AWM and APM following appropriate bypass surgery (fig. 4). Individuals with normal SWM preoperatively, as in patients without previous infarction, demonstrated no significant changes postoperatively.

Relation between Segmental Wall Motion and Ejection Fraction

With the improvement in segmental wall motion abnormalities, ejection fraction also increased postoperatively (fig. 5). Before surgery 10 patients-eight with normal segmental wall motion and two with wall motion abnormalities involving one segment-had normal ejection fraction (> 0.60) and 19 patients-18 with wall motion abnormalities involving two or three segments—had significantly reduced ejection fraction. Postoperatively, 25 of 29 patients had both normal segmental wall motion and a normal ejection fraction. In only four patients with persistent wall motion abnormalities was the ejection fraction reduced postoperatively.

After appropriate bypass surgery, improvement in ejection fraction (EF) was observed in all three groups of patients (fig. 6). Thus, of the six patients with the preinfarction syndrome who had a reduced EF, all showed a significant increase postoperatively. Similarly, of the six patients with chronic ischemia and no previous infarct, who had a reduced EF preoperatively, all had a significant increase following operation.

Seven patients with previous infarction had reduced EF preoperatively and in six a significant increase in EF occurred following surgery although the magnitude of postoperative increase in EF was less than in patients without previous infarction. The single patient who did not show any change in
EF postoperatively had only RCA bypass, although he had severe disease of both RCA and LCA preoperatively. In patients who had normal EF preoperatively with or without previous infarction, no significant change was observed following surgery.

Improvement in EF in these patients was mainly due to reduction in ESV (table 2, fig. 7). All patients in this study had normal or near normal EDV before surgery, but in many there was an increase in ESV and a decrease in EF. Postoperatively, there was a slight reduction in EDV in patients without previous infarction (table 2), but a much greater reduction in ESV resulting in an increase in SV and EF. In table 2 patients with the preinfarction syndrome and chronic ischemia without previous infarct have been grouped together because of the small number of patients in each group and the similar results. In patients with previous infarction, there was no change in EDV and the increase in SV and EF was solely due to reduction in ESV following surgery. Although no significant change in EDV occurred postoperatively, LVEDP fell significantly, suggesting increased compliance in those patients (fig. 7) who demonstrated increased EF postoperatively.

Concomitant with the improvement in EF, there was generally a significant improvement in hemodynamic parameters and derived indices of contractility (table 2). Thus, patients who demonstrated improved EF following surgery also had a reduction in LVEDP and an increase in dP/dt, VCE0, and SWI. Arterial pressure did not change significantly, but increased heart rate was observed in the majority of patients irrespective of the postoperative status of LV function.

Figure 8 illustrates typical pre- and postoperative pressure-velocity relations in patients with and without previous infarction. The shift of the curves to the right and upward postoperatively indicate increased contractile element velocity at all levels of developed pressure, suggesting improved contractile state. In patients with normal ejection fraction...
preoperatively, however, there was no significant change in VCE following surgery.

Discussion
Abnormalities of systolic motion of left ventricular walls commonly occur in patients with obstructive coronary artery disease.\textsuperscript{1-3} While reversal of asynery due to scar tissue is not to be expected, the function of ischemic but viable myocardium could be restored following relief of ischemia as in experimental preparations.\textsuperscript{7-8} Some abnormalities of segmental wall motion were detected in 12 of our 18 patients (66\%) with obstructive coronary artery disease and without previous myocardial infarction. In almost all patients significant improvement occurred following direct myocardial revascularization and in the majority normalization of segmental wall motion occurred. The similarity of results between those patients with the preinfarction syndrome and those with chronic angina suggests that ischemia not only produces alterations in ventricular function, but is potentially reversible even after prolonged periods of time. Furthermore, it should be noted that no patient had chest pain during the preoperative catheterization, so that the depression of function observed was not due to acute changes associated with angina pectoris.

The return of normal segmental wall motion in previously akinetic or hypokinetic segments of left ventricular walls (fig. 1) indicates that, as we have previously reported,\textsuperscript{9} in impending infarction such asynery is primarily the consequence of ischemia alone. Hence, even when large areas of akinesia or hypokinesis involving the left ventricle walls are observed, this does not automatically indicate the presence of scar tissue. Therefore this finding does not necessarily mean that surgical excision is indicated since function can be significantly improved or even normalized by direct myocardial revascularization.

On the other hand, in patients with previous myocardial infarction, abnormalities of ventricular wall motion tend to persist in the areas of previous infarction. This lack of improvement in asynery in the areas of old infarction almost certainly indicates the presence of significant scar tissue. However, in almost all patients, significant improvement in wall motion in noninfarcted segments was observed.

With the improvement in segmental wall motion postoperatively, ejection fraction, as expected, also increased. Thus, in 18 of the 19 patients with reduced EF–12 without and seven with previous myocardial infarction–EF significantly increased.
Table 2
Pre- and Postoperative Hemodynamic Data and Derived Parameters of LV Function

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Patients without previous infarct (N = 18)</th>
<th>Patients with previous infarct (N = 11)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Preop reduced EF</td>
<td>Postop</td>
</tr>
<tr>
<td>No.</td>
<td>12</td>
<td>12</td>
</tr>
<tr>
<td>Ejection fraction</td>
<td>0.45 ± 0.03</td>
<td>0.74 ± 0.03*</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>76 ± 3</td>
<td>90 ± 3*</td>
</tr>
<tr>
<td>Mean art pressure (mm Hg)</td>
<td>93 ± 3</td>
<td>87 ± 4</td>
</tr>
<tr>
<td>LVEDP (mm Hg)</td>
<td>15 ± 1</td>
<td>10 ± 1*</td>
</tr>
<tr>
<td>LVESP (mm Hg)</td>
<td>6.4 ± 0.8</td>
<td>2.6 ± 0.6</td>
</tr>
<tr>
<td>LV dP/dt (mm Hg/sec)</td>
<td>1271 ± 79</td>
<td>1602 ± 91*</td>
</tr>
<tr>
<td>SWI (g-m/m³)</td>
<td>49 ± 12</td>
<td>78 ± 6*</td>
</tr>
<tr>
<td>EDV (ml/m²)</td>
<td>114 ± 12</td>
<td>97 ± 9*</td>
</tr>
<tr>
<td>ESV (ml/m²)</td>
<td>71 ± 12</td>
<td>23 ± 4*</td>
</tr>
<tr>
<td>SV (ml/m²)</td>
<td>43 ± 3</td>
<td>73 ± 8*</td>
</tr>
<tr>
<td>VCE at 5 mm Hg (ml/sec)</td>
<td>1.3 ± 0.1</td>
<td>1.8 ± 0.1*</td>
</tr>
</tbody>
</table>

Abbreviations: EF = ejection fraction; EDV = end-diastolic volume; ESV = end-systolic volume; SV = stroke volume; LVESP = left ventricular end-systolic pressure; LVEDP = left ventricular end-diastolic pressure; SWI = stroke work index; LV dP/dt = left ventricular peak dP/dt; VCE = contractile element velocity at 5 mm Hg.

*P < 0.05.
Figure 7

*Left ventricular end-diastolic and end-systolic volumes and pressures before and after bypass surgery in patients with preoperatively reduced (left) and normal (right) ejection fractions. End-systolic and end-diastolic pressure refers to the LV pressures at the beginning and end of diastole. As seen in the upper left panel, the postoperative increase in ejection fraction was primarily due to a reduction in end-systolic volume. Significant reduction in end-diastolic pressure (lower left) with little or no change in end-diastolic volume (upper left) also suggests increased compliance postoperatively.*

Rates of similar magnitude were found postoperatively in patients who had normal EF both before and after surgery and who did not show any significant change in derived indices of contractility.

Improved left ventricular compliance following bypass surgery, as suggested by a fall in LVEDP without change in EDV postoperatively, is also of significant clinical importance. These patients will be less prone to develop pulmonary venous congestion as excessive rise of left ventricular diastolic pressure will not occur due to decreased stiffness of the left ventricle.

Although the present study clearly documents that significant improvement in LV asynergy, contractility, and overall left ventricular function may occur in patients with obstructive coronary
PRE & POST OPERATIVE V_{CE}
FOLLOWING AORTO—CORONARY ARTERY BYPASS SURGERY

![Graph showing pressure velocity relations before (○) and after (●) surgery in two patients, one without (left) and one with (right) previous infarction. The patient on the left had the preinfarction syndrome. Each curve was calculated from three successive contractions. Definite postoperative improvement in myocardial contractility is evident by the shift of the curves upward and to the right. The inset values represent contractile element velocity at 5 mm Hg.]

LV FUNCTION AFTER REVASCULARIZATION

artery disease following appropriate bypass surgery, reports indicating no such postoperative improvement have also appeared in the literature.\(^17, 18\) Kouchoukos et al.\(^17\) reported no improvement in postinfarction patients with large preoperative EDVs following bypass surgery and resection of noncontractile segments. Two patients seen by us with chronic heart failure, cardiomegaly, and large end-diastolic volumes preoperatively also failed to show any significant change in function following bypass surgery and resection of noncontractile segments.

Thus, patients with chronic congestive heart failure and with large end-diastolic volumes preoperatively will probably not show significant improvement in LV function following bypass surgery, and we do not currently accept such patients for surgical treatment.

Improvement in LV function following bypass surgery may be related to the coronary arteries bypassed. Several of the patients reported in the literature with depressed LV function who failed to show any postoperative improvement had right coronary artery bypass only.\(^17\) The only patient in this series who also showed no improvement in ventricular function postoperatively also had only right coronary artery bypass, although he had significant disease of both right and left coronary arteries preoperatively. Isolated disease of the right coronary artery without significant disease of the left coronary artery or its branches usually does not depress LV function significantly and bypass surgery of the right coronary artery is unlikely to cause a significant change in left ventricular function.\(^5, 19\) In contrast, critical lesions of left coronary artery or its branches with or without RCA disease may severely depress LV function and hence, bypass of obstructive lesions of LCA or its main branches is a prerequisite for any improvement of LV function. From the data available in the
literature, it is not known how many patients with previous multiple infarcts had aortocoronary artery bypass surgery. Nevertheless, possible inclusion of such patients in broad series without specific identification may account for some of the disappointing general results reported. The one patient in cardiogenic shock who survived following operation is of some interest because of the special situation involved. Thus the inferior wall was moving poorly because of old inferior infarction, while the anterior wall was moving poorly due to ischemia. Correction of flow to the LCA by vein-bypass graft relieved the ischemia and shock by improvement in anterior wall motion. Conceptually, this combination of circumstances is important because it produced cardiogenic shock with ischemia only, and prior to myocardial infarction.

In conclusion, the present study indicates that marked improvement and even normalization of LV function can be expected in properly selected patients with the preinfarction syndrome or with chronic ischemia and no previous infarct, following appropriate aortocoronary artery bypass surgery. In patients with previous infarction, however, some abnormalities of systolic wall motion may remain in the areas of infarction. Nevertheless, significant increase in ejection fraction may still occur consequent on improvement in LV asynergy in ischemic segments and improvement in the overall contractile state of the left ventricle.

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Circulation, Volume XLVII, February 1973
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Circulation. 1973;47:276-286
doi: 10.1161/01.CIR.47.2.276

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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