Improvement of Myocardial Perfusion and Left Ventricular Function After Coronary Artery Bypass Grafting in Patients with Unstable Angina

ALBERT J. KOLIBASH, M.D., JOHN S. GOODENOW, M.D., CHARLES A. BUSH, M.D., MARC R. TETALMAN, M.D., AND RICHARD P. LEWIS, M.D.

SUMMARY Changes in myocardial perfusion and left ventricular function were evaluated pre- and postoperatively (3–6 months) in 14 patients with unstable angina who underwent coronary artery bypass surgery. Perfusion was studied with intracoronary and intragraft injections of radiolabeled macroaggregated albumin particles. Of 20 abnormal perfusion areas identified preoperatively, 13 demonstrated improved perfusion postoperatively. Segmental analysis of the left ventriculogram demonstrated improved wall motion in 29 abnormally contracting segments; 18 normalized. Areas which showed improvement of left ventricular perfusion were invariably associated with improvement of left ventricular wall motion. Five patients showed improvement in perfusion and contraction in areas of apparent old myocardial infarction.

Thirteen of the 14 patients had significantly less angina whether or not there was evidence of improved perfusion. However, only those patients who demonstrated improved perfusion had a significant improvement in their treadmill exercise tolerance postoperatively. Thus, patients with unstable angina have perfusion defects which may be reversed as a result of saphenous vein graft surgery. Reversal of these perfusion abnormalities results in improved left ventricular performance and better exercise tolerance postoperatively.

CORONARY ARTERY BYPASS SURGERY has an important and widespread role in the treatment of coronary artery disease.1–3 Of particular significance to many investigators is the preoperative detection of ischemic, but viable, myocardium and the ultimate effects of revascularization upon improvement of left ventricular perfusion and function.4–6 Perhaps this problem is best exemplified in those patients with unstable angina who have poorly contracting areas of the ventricle which are being supplied by vessels with highly stenotic lesions. The recent reports of resting perfusion abnormalities in this group of patients6, 7 further raises the question whether these areas represent viable (reversibly damaged) myocardium or nonviable (irreversibly damaged) myocardium.

Various techniques using intervention ventriculography have been used to identify potentially viable myocardium.8–20 Another method used to differentiate between viable and nonviable myocardial tissue is the use of direct intracoronary instillation of radiolabeled, macroaggregated albumin particles.7, 21–26 Since these particles lodge in the coronary microcirculation, the presence of radioactivity throughout the myocardium has been equated with tissue viability.21–23, 25 Although previous studies have suggested that perfusion defects in patients with chronic stable angina represent fibrosis,7, 21 the significance of perfusion abnormalities in patients with unstable angina is not well understood. Particularly, the relationship of perfusion defects to segmental left ventricular contraction abnormalities and their potential for reversibility after coronary artery bypass surgery has not been demonstrated.

If such perfusion defects do not always imply infarction, myocardial revascularization would be expected to correct the ischemia by improving blood flow, resulting in concomitant relief of angina and improvement of left ventricular performance. There have been relatively few studies examining changes in preoperative and postoperative perfusion as provided by individual coronary artery bypass grafts.27, 28 Furthermore, even though numerous studies have shown that bypass grafts will provide significant relief of angina in 75–80% of patients who undergo surgery,5–9, 20 the effectiveness of this procedure in improving left ventricular function remains controversial.29–42

In this study we investigated the significance of preoperative perfusion defects and left ventricular contraction abnormalities in patients with unstable angina by measuring the effects of coronary artery bypass surgery upon reversing such perfusion and contraction abnormalities and relating the results of surgery to the clinical response.

**Methods**

We studied 14 patients with unstable angina (New York Heart Association functional class III and IV) who were admitted to Ohio State University Hospital and who underwent coronary artery bypass surgery. Most patients had resting or nocturnal pain despite treatment with nitrates and propranolol. None had evidence of acute myocardial infarction at the time of coronary angiography or before surgery. ECG criteria for old infarction were based upon significant Q waves defined as Q wave duration \( \geq 0.04 \) seconds and Q wave depth \( \geq 25\% \) of the R wave amplitude. An inferior infarction was diagnosed when significant Q...
waves appeared in leads II, III, and aVF and anteroseptal infarction when similar Q waves were seen in leads V1-3. In the three patients who also had vectorcardiograms (VCG), the VCG diagnosis of infarction was made according to the criteria of Benchimol.43 Treadmill exercise testing using the Bruce protocol was performed pre- and postoperatively in patients in whom the test was not contraindicated. Left ventriculography, selective coronary angiography, and resting myocardial perfusion scanning were performed in all patients preoperatively and 3–6 months postoperatively.

Left ventriculography was performed in the 30° right anterior oblique (RAO) and left lateral (LAT) views. Left ventricular function was evaluated by calculating the ejection fraction, left ventricular end-diastolic pressure (LVEDP), left ventricular end-diastolic volume (LVEDV), stroke volume (SV) and segmental analysis. Segmental analysis was performed according to the method of Leighton et al.44 This method involves a quantitative analysis of left ventricular wall motion along the anterior and inferior heart borders as seen in the 30° RAO left ventriculogram. Systolic motion for each of seven segments is expressed as percentage shortening of each hemi axis. Segments 1–4 represent contraction along the anterior left ventricular wall and segments 5–7 are located along the inferior heart border. A segment was considered abnormal if the percent shortening was less than two standard deviations of the previously reported normal values.44 After left ventriculography, we performed selective coronary angiography using the Judkins technique.

Myocardial perfusion distribution was studied by intracoronary instillation of 250–300 thousand radio-labeled MAA particles 10–40 μ in size. The particles were tagged with technetium-99m (99mTc) and either Indium-111 or 113m (In). The particles were instilled slowly in the resting state, 3–5 minutes after the final coronary arteriogram. Preoperatively, 1 mCi of In MAA was instilled into the left coronary artery and 0.4 mCi of 99mTc MAA was instilled into the right coronary artery. Postoperatively, approximately 0.4 mCi of the isotope was instilled into each graft and native vessel. Because only two radionuclides were used in each patient, we could not always image the perfusion distribution of all grafts and both native vessels in the same patient. Grafts to the left anterior descending artery (LAD) and right coronary artery (RCA) were always injected, if patent, since these vessels were most commonly the recipients of grafts. If these grafts were occluded and the radionuclide was available, grafts to the circumflex (CX) and patent native vessels were studied. There were no complaints of angina, no ECG changes, and no intracoronary pressure changes during the instillation of isotopes nor at ventriculography.

Myocardial scintigraphy was completed within 30–60 minutes after angiography. Patients were imaged in the 45° left anterior oblique (LAO), anteroposterior, and LAT projections using a scintillation camera with a medium energy or ultrafine collimator. An information density (1000 counts/cm²) was collected for all cardiac images in every case; this reduced statistical variation in total counts in each image and among patients. Separate and dual images were recorded on either x-ray or polaroid film. Because of the fixed information density, similar exposure was made on all patients regardless of the type of film used. The scintigrams were qualitatively and blindly analyzed by three independent observers. A normal scintigram was considered to be a homogenous distribution of radioactivity, while perfusion defects were defined as areas of minimal or no activity. Postoperative improvement in perfusion was felt to be present only when there was a definite increase in area of radioactivity compared with the preoperative scan. Variations in density of activity in similar areas before and after surgery were not considered to represent a change in perfusion. Interobserver agreement was 95%. In those cases where interobserver variation did occur, the interpretation of two of three observers was recorded.

Myocardial perfusion was evaluated by defining three perfusion areas in each patient as visualized on the scintigram. These areas correspond to the areas of myocardium supplied by the LAD, CX and RCA.44 The relationship between perfusion and left ventricular function was then studied by matching perfusion areas of the LAD and RCA to their corresponding segments of ventricular contraction as seen on the 30° RAO ventriculogram, i.e., the perfusion distribution of the LAD to the anterior left ventricular wall and the RCA to the inferior heart border. Changes in postoperative perfusion were then compared with changes in segmental ventricular contraction. Since only the anterior and inferior walls are visualized on the RAO angiogram, perfusion areas of the CX artery were not compared to wall motion unless the CX was the dominant vessel supplying the inferior heart border.

In the 14 patients, 33 of 42 perfusion areas received a total of 39 grafts. All vessels grafted had 90% stenosis except two, which had 60% stenosis.

Statistical analysis was accomplished with a Hewlett-Packard 9600-B computer using the t test for paired comparisons and chi square analysis using standard programs.

Results
Pre- and Postoperative Changes in Myocardial Perfusion and Left Ventricular Function

Figure 1 shows the pre- and postoperative changes of perfusion in the 33 areas receiving grafts. Preoperatively, 13 of these areas had normal perfusion and 20 areas had perfusion defects. Preoperatively, nine of the normally perfused areas also demonstrated a normal contractile pattern. Contractility was not studied in the remaining four areas, as these areas were supplied by the left CX artery. Of the 20 preoperative areas which had abnormal perfusion, 16 demonstrated segmental contraction abnormalities, two had normal contraction and two were not studied.
Eight of the 13 areas which had normal perfusion preoperatively continued to show normal perfusion postoperatively. Five of these areas were supplied by six patent grafts. Three areas continued to show normal perfusion postoperatively even though the blood supply was not provided by the grafts. These three areas were supplied by the native vessels and collateralization as identified by either the angiogram or scintigram. One of these three areas received the majority of its perfusion from the native vessel despite the presence of a patent graft. The remaining five areas of normal preoperative perfusion were not studied postoperatively due to limitation of the number of isotopes. However, four of these five areas were associated with five occluded grafts and the fifth was supplied by a patent graft.

Of the 20 areas of abnormal preoperative perfusion, 13 had improved perfusion postoperatively. Since minimal or no activity was seen in these 13 areas preoperatively, the increased activity seen postoperatively strongly suggests that blood flow to these areas had been improved. However, it should be emphasized that the radionuclide method employed in this study was qualitative and, therefore, statements concerning quantitative changes in blood flow cannot be made. Ten of these areas were supplied directly by 13 patent grafts and three areas were supplied by grafts in adjacent areas. Figure 2 is a typical example of improvement in perfusion after vein graft surgery. The remaining seven areas had persistent perfusion defects postoperatively. Six grafts to these areas were occluded. The seventh area did not show improved perfusion with a patent graft.

In table 1 the patients are divided into two groups, based on the presence or absence of postoperative improvement in perfusion. Nine of 14 patients demonstrated at least one area of improved perfusion, while five patients showed no improved perfusion. An increase in left ventricular wall motion was seen in 33 of 63 segments revascularized in the patients with improved perfusion and in only five of 26 segments revascularized in those without improved perfusion ($p < 0.05$). In examining segmental analysis more closely, there were 26 hypokinetic and three akinetic segments (< 5% shortening) of which 15 hypokinetic and the three akinetic segments normalized (12.5% to 35.2%, $p < 0.01$) (fig. 3). All 18 normalized segments were associated with improved perfusion via patent grafts. Of these 18 normalized segments, 13 were located along the inferior heart border and five along the anterior left ventricular wall. Those segments along the inferior wall correspond to improved perfusion in the distribution area of the RCA and those along the anterior wall to the distribution area of the LAD. There were no normalized segments in the five patients without improved perfu-
sion (table 1). Figure 4 is an example of marked improvement in contractility along the inferior heart border after vein graft surgery. Improved perfusion occurred in the area supplied by the RCA, and this area corresponds to those segments demonstrating improved contractility.

In addition to segmental analysis, changes in ejection fraction, LVEDP, LVEDV and SV were examined within each group pre- and postoperatively and between the two groups postoperatively (table 1). Only the ejection fraction significantly improved (p < 0.05) pre- to postoperatively within the group with improved perfusion. There were no significant changes in the LVEDV, LVEDP and SV within each group nor between groups. Although the patients with improved perfusion had a higher graft patency rate, the difference between the two groups was not statistically significant.

### Preoperative Perfusion and Graft Patency

Whether the knowledge of preoperative perfusion was useful in predicting graft patency was examined (fig. 1). Thirteen areas of normal preoperative perfusion received 16 grafts. At the time of the postoperative catheterization, only eight of these grafts remained patent, while eight were occluded. Twenty-three grafts were placed in the 20 areas with abnormal preoperative perfusion. Fourteen of these grafts remained patent and nine were occluded. This difference was not significant and, therefore, the preoperative assessment of perfusion was not beneficial in predicting graft patency in this group of patients.

### Perfusion and Contraction in Areas of Apparent Old Infarction

Changes in myocardial perfusion and left ventricular contraction were also examined in areas of apparent old infarction. Preoperative ECG in these 14 patients showed six with an old myocardial infarction, three with left anterior hemiblock, two with a right intraventricular conduction delay, and three with nonspecific ST and T wave changes. Three patients also had VCGs, which confirmed the presence of an inferoposterior infarction as shown on the ECG in one patient, and disclosed an inferior myocardial infarction in another patient. Thus, a total of seven patients had ECG or VCG criteria for an old myocardial infarction.

In four of these patients perfusion improved in areas of apparent old myocardial infarction. A fifth patient (JM) also showed improved perfusion along the inferior heart border despite developing ECG changes consistent with an inferior infarction in the immediate postoperative period. Data relating to these five patients are summarized in table 2. Three of these five infarctions were inferior, one was infero-posterior and one anteroseptal. One patient (JH) had only VCG evidence of an inferior infarction (fig. 4). In two patients with inferior infarction (LS and JH), the

![Graph showing improvement in contraction](image-url)

**Figure 3.** This graph shows improvement in contraction of 18 hypokinetic and akinetic segments which normalized after revascularization.

<table>
<thead>
<tr>
<th>Areas of improved perfusion</th>
<th>Improved perfusion (patients n = 9)</th>
<th>No improvement in perfusion (patients n = 5)</th>
<th>Significance between the two groups</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>p &lt;0.05</td>
</tr>
<tr>
<td>Segments with improved contractility</td>
<td>13/81</td>
<td>0/26</td>
<td>None</td>
</tr>
<tr>
<td>Segments normalized</td>
<td>18</td>
<td>0</td>
<td>NS</td>
</tr>
<tr>
<td>Grafts patent</td>
<td>18/30</td>
<td>4/9</td>
<td>NS</td>
</tr>
<tr>
<td>Ejection fraction (%)</td>
<td>53→61 (p &lt;0.05)</td>
<td>63→62 (NS)</td>
<td>NS</td>
</tr>
<tr>
<td>Angina</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Completely alleviated</td>
<td>6</td>
<td>3</td>
<td>NS</td>
</tr>
<tr>
<td>Subjectively improved</td>
<td>3</td>
<td>1</td>
<td>NS</td>
</tr>
<tr>
<td>Increase in exercise tolerance</td>
<td>4'23&quot; (=60&quot;)</td>
<td>p &lt;0.01</td>
<td>None</td>
</tr>
</tbody>
</table>

Abbreviation: NS = not significant.
postoperative improvement in perfusion and contraction occurred along the inferior heart border as a result of a patent graft to the RCA. The patient with an old anteroseptal infarction (KS) demonstrated improved perfusion and contraction of the anterior left ventricular wall and had a patent graft to the LAD. The improvement along the inferior left ventricular wall in patient SS was attributed to a patent graft to a large posterolateral artery. This patient had a dominant left coronary artery, accounting for the changes seen inferiorly, an area more commonly supplied by the RCA. The postoperative changes in perfusion and contractility seen in patient SM, who developed Q waves in leads II, III and aVF in the immediate postoperative period, were due to collateral flow from the LAD which had received a patent graft. The RCA in this patient was not grafted and was totally occluded. Postoperatively, the Q waves persisted in all patients except SM, whose postoperative ECG revealed a significant Q wave in lead III only.

In the three remaining patients with old infarction, perfusion was unchanged after revascularization and all patients continued to show abnormal contractility in the areas of infarction. Three additional patients

![Figure 4](http://circ.ahajournals.org/)

**FIGURE 4.** Pre- and postoperative scintigrams and segmental analysis in a patient with patent saphenous vein graft (SVG) to the right coronary (RCA) and posterolateral (PL) arteries. Preoperatively, $^{111}$In was injected into the left coronary artery (LCA) and $^{99m}$Tc into the RCA. The preoperative dual views indicate combined perfusion of both vessels in anterior (ANT) and left anterior oblique (LAO) projections. There is markedly decreased perfusion of the right ventricle, inferior wall and septum. The perfusion abnormality of the inferior wall corresponds to the abnormal contraction pattern of the inferior wall (segments 5, 6 and 7). Postoperatively, injection of the grafts with $^{99m}$Tc demonstrates considerable improvement of perfusion of the inferior wall and lower septum (white arrows) concomitant with normal contractility of the inferior wall (segments 5, 6 and 7).

**TABLE 2. Patients Showing Improved Perfusion and Contractility In Areas of Apparent Infarction**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Graft site</th>
<th>Area of improved perfusion</th>
<th>ECG</th>
<th>Segments improved</th>
<th>Ejection fraction Preop</th>
<th>Ejection fraction Postop</th>
</tr>
</thead>
<tbody>
<tr>
<td>LS</td>
<td>RCA</td>
<td>RCA</td>
<td>Inferior MI</td>
<td>5,6,7</td>
<td>48</td>
<td>71</td>
</tr>
<tr>
<td>KS</td>
<td>LAD</td>
<td>LAD</td>
<td>Antero-septal MI</td>
<td>2,3</td>
<td>48</td>
<td>62</td>
</tr>
<tr>
<td>SS</td>
<td>Posterior Lateral</td>
<td>CIRC</td>
<td>Infero-posterior MI (+ VCG)</td>
<td>6,7</td>
<td>50</td>
<td>59</td>
</tr>
<tr>
<td>JH</td>
<td>RCA</td>
<td>RCA</td>
<td>Inferior MI VCG only</td>
<td>5,6,7</td>
<td>50</td>
<td>60</td>
</tr>
<tr>
<td>SM</td>
<td>LAD</td>
<td>RCA</td>
<td>Inferior MI</td>
<td>5,6,7</td>
<td>56</td>
<td>68</td>
</tr>
</tbody>
</table>

Abbreviation: MI = myocardial infarction.
with ECG evidence of infarction postoperatively also showed impaired perfusion and worsening of contractility in the area of infarction.

**Graft Occlusion, Perfusion and Left Ventricular Function**

In order to assess more accurately the effects of graft occlusion upon postoperative perfusion and left ventricular function, we made the following observations (fig. 1).

Eight grafts were occluded in seven areas of myocardium in which preoperative perfusion was normal. Three of these areas continued to show normal perfusion after surgery despite the occluded grafts. Blood flow to these areas was supplied through the native vessel or from adjacent grafts via collateral vessels. Perfusion in the remaining four areas with five occluded grafts was not evaluated. However, contractility was abnormal in three of these areas, implying impaired perfusion.

Of nine areas of abnormal preoperative perfusion receiving nine occluded grafts, six continued to show abnormal perfusion and contractility postoperatively and three actually showed an improvement in perfusion. This seemingly paradoxical situation of improved blood flow in three areas receiving three non-patent grafts may be explained by the blood supply via collaterals originating from another native vessel which was associated with a patent graft. The origin and extent of perfusion could be detected scintigraphically by selectively injecting the graft with MAA. Figure 5 shows the improvement along the inferior heart border. In this patient, blood supply to the inferior heart border was via a graft to the LAD. Two other grafts and both native vessels were occluded in this patient. Since only the graft to the LAD was injected and since activity is seen in areas of myocardium normally supplied by the RCA, perfusion of the inferior heart border must be attributed to left-to-right collateral vessels. Furthermore, normal contractility is maintained and both native vessels along with two additional grafts are totally occluded.

This analysis indicates that graft occlusion usually results in impaired perfusion and worsening left ventricular function. However, graft occlusion is not uniformly associated with such adverse results and under certain circumstances perfusion and contractility may improve despite a nonfunctioning graft.

**Pre- and Postoperative Changes in Exercise Tolerance and Angina Pectoris**

Thirteen of 14 patients had less angina postoperatively (table 1). In the five patients who demonstrated no improvement in perfusion, three had no angina, one had angina of less severity, and one patient was...
unchanged. Three of these patients had perioperative myocardial infarctions.

Of the nine patients with improved perfusion, six were free of angina and three were clinically improved. One of these patients had a perioperative myocardial infarction. Thus, the majority of patients were improved postoperatively regardless of whether perfusion was improved.

Not all patients had pre- and postoperative treadmill exercise studies, as some had resting chest pain before surgery. Two patients without improved perfusion did have exercise tests pre- and postoperatively. Neither of these patients showed any improvement in exercise tolerance postoperatively. Six patients with improved perfusion increased their exercise tolerance by an average of 4 minutes, 23 seconds (± 60 seconds, p < 0.01). Thus, even though there were no significant differences in improvement of angina, patients with demonstrably improved perfusion significantly improved exercise tolerance postoperatively.

Discussion

Several studies using myocardial scintigraphy have indicated that most perfusion defects represent areas of old infarction and therefore are irreversibly damaged. Similar to the segmental nature of contraction disorders seen in coronary artery disease are well recognized and markedly hypokinetic and akinetic segments are felt to be indicative of severely damaged or nonviable myocardium in the majority of instances. These studies imply that coronary artery bypass grafts to underperfused, poorly functioning myocardium would not be beneficial.

Although these conclusions may be accurate when applied to most patients with coronary artery disease, the results of this study show that perfusion defects and left ventricular contractile abnormalities in patients with unstable angina do not always represent irreversibly damaged myocardium. Since 13 of the underperfused areas demonstrated improved perfusion, preoperatively, these areas probably represented ischemic viable myocardium rather than nonviable or irreversibly damaged tissue. This suggests that in some patients with unstable angina myocardial blood flow is reduced to such minimal levels that it produces perfusion abnormalities on scintigraphy and abnormal segmental contraction. However, this markedly reduced blood flow seems to be sufficient to maintain the tissue in a viable state and, if the area receives additional blood flow such as through a graft, the perfusion abnormalities may improve and result in better left ventricular performance. These findings confirm impressions that perfusion defects in patients with unstable angina may represent transient ischemia. However, improvement of blood flow postoperatively was not a uniform finding. Seven preoperative perfusion defects were unchanged postoperatively; six of these were associated with occluded grafts. Thus, preoperative perfusion defects with unstable angina pectoris may represent either viable or nonviable myocardium.

The improvement in left ventricular performance which accompanied the improvement of perfusion is notable. It has become increasingly recognized that some abnormally contracting segments may have the potential to regain a degree of function. This reversal of contractile abnormalities implies not only tissue viability, but also suggests that a bypass graft can provide sufficient blood flow to correct the underlying ischemia. Although increased myocardial blood flow through grafts has been previously demonstrated with xenon-133, reports of the association between surgical revascularization and improved left ventricular function differ. The discrepancies might be clarified if the patients were clearly separated into those with unstable angina and those with angina of less severity. The concomitant improvement in both perfusion and wall motion in our study indicates that functionally patent grafts can provide adequate blood flow to severely ischemic, but viable, myocardium, resulting in improved left ventricular function in patients with unstable angina. Finally, the preoperative distinction between viable and nonviable tissue could not be made with myocardial scintigraphy.

Improvement of blood supply to areas of apparent infarction was not entirely surprising. Although Q waves on the ECG are generally a good indicator of coronary artery disease and localized ventricular contractile abnormalities, it is apparent that Q waves are not always synonymous with extensive transmural myocardial infarction. The possibility of improved left ventricular performance in areas of apparent infarction has been alluded to by several investigators. Zef et al. reported six patients with reversal of left ventricular dysfunction and restoration of R waves in regions of apparent infarction. Conde showed that abnormal Q waves could disappear after aorticcoronary bypass surgery. Therefore, the combination of Q waves and asynergy does not preclude the possibility of improved perfusion and function after coronary artery bypass surgery, particularly in patients with unstable angina.

The observation that graft occlusion is generally associated with deterioration of left ventricular function was expected and has been previously reported. The patency rate for our study is somewhat lower than reported in the literature and may be explained by the severe degree of disease in our patients. However, several patients demonstrated improved perfusion and function despite occluded grafts, and this is surprising. Conversely, the lack of improvement in perfusion and performance in one patient with a patent graft was unexpected. Although these latter two observations occurred in a minority of instances, they demonstrate the importance of using additional parameters to evaluate the functional significance of bypass grafts.

The high incidence of alleviation of angina in our patients is similar to that previously reported. The decrease in angina in the five patients without improved perfusion suggests mechanisms other than improved myocardial blood flow for pain relief, as proposed by others. The occurrence of a
perioperative myocardial infarction in three of these patients may, in part, explain their decrease in angina. On the other hand, improvement of angina in the remaining nine patients may have been the result of improved blood flow to the myocardium. This is supported by both the improved perfusion in all nine patients and the significantly improved exercise tolerance postoperatively in six of these patients. Increased exercise tolerance after bypass surgery may correlate with graft patency, and Zaret has shown that successful bypass grafts can prevent the development of exercise-induced perfusion defects. Our results indicate that improvement in exercise capacity in postoperative patients is a result of improved myocardial blood supply.

This study indicates that the presence of a perfusion defect as defined by myocardial scintigraphy does not necessarily indicate fibrosis or irreversibly damaged myocardium in patients with unstable angina pectoris. Such areas of myocardium may show improvement in both perfusion and function after coronary artery bypass surgery, even when associated with marked left ventricular contraction abnormalities and ECG or VCG evidence of myocardial infarction. Differentiation between severely ischemic, but viable, myocardium and fibrosis was difficult, since preoperative assessment of myocardial perfusion was not of value in predicting graft patency. The patients in this study all had severe progressive angina refractory to appropriate medical therapy, so the results of this investigation cannot be applied to patients with less severe angina. Our data also indicate that in most instances graft occlusion is associated with absent perfusion and worsening left ventricular function; however, this was not a uniform finding, as a few patients actually had improved perfusion and function when the graft was occluded. This observation is explained by improved blood supply via collaterals from patent grafts in adjacent areas or by persistent patency of the native vessel which received the graft. Virtually all patients were functionally improved with respect to symptoms of angina; however, only those patients with improved perfusion had significantly improved exercise tolerance postoperatively.

References
17. Dumesnil JG, Ritman EL, Davis GD, Gau GT, Rutherford BD, Frye RL: Regional left ventricular wall dynamics before and after sublingual administration of nitroglycerin. Am J Cardiol 36: 419, 1975
27. Gander MP, Jansen C, Wareham E, Huse W, Judkins MP:
Internal mammary to anterior descending coronary anastomosis, evaluated postoperatively with high resolution arteriography and myocardial perfusion scanning. (abstr) Circulation 48 (suppl IV): IV-90, 1974


44. Leighton RF, Wilt S, Lewis RP: Detection of hypokinesis by a quantitative analysis of left ventricular cineangiograms. Circulation 50: 121, 1974


Improvement of myocardial perfusion and left ventricular function after coronary artery bypass grafting in patients with unstable angina.
A J Kolibash, J S Goodenow, C A Bush, M R Tetalman and R P Lewis

Circulation. 1979;59:66-74
doi: 10.1161/01.CIR.59.1.66

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1979 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/59/1/66