Deterioration of Myocardial Function Following Aorto-Coronary Bypass Operation


SUMMARY
Twenty-two patients underwent cardiac catheterization before and an average of five months after aorto-coronary bypass operation (ACBO). Two groups were examined: 10 patients with all grafts patent, and 12 patients with one or more grafts occluded. All patients improved symptomatically, regardless of graft patency. However, in the occluded group, left ventricular end-diastolic pressure (LVEDP) increased (4.4 ± 2.2 mm Hg, \( P < 0.05 \)), stroke volume index fell (9.8 ± 3.1 ml/m², \( P < 0.05 \)), ejection fraction decreased (10 ± 4%, \( P < 0.05 \)), and left ventricular stroke work index fell (12 ± 3 g/m², \( P < 0.01 \)).

Qualitative analysis of segmental left ventricular contractility was performed. Of 28 segments supplied by patent grafts, six improved and nine deteriorated. Of 22 segments supplied by occluded grafts, none improved and eight deteriorated. Frequently no angiographically demonstrable basis for the segmental deterioration was evident.

We conclude that while ACBO may appreciably benefit severely symptomatic patients, our results do not substantiate the claim that ACBO should be recommended when the primary surgical goal is preservation or enhancement of myocardial function.

Additional Indexing Words:
Segmental contractility Ejection fraction Saphenous vein graft Ventricular volumes Myocardial revascularization

AORTO-CORONARY BYPASS OPERATIONS (ACBO) are currently being performed with relatively low operative mortality in patients with stable angina pectoris and no clinical evidence of left ventricular failure.\(^1\)\(^-\)\(^6\) The goals of the operation are the following: improved symptomatic status of the patient, preservation of myocardial function, and enhanced longevity. The first goal has been realized, insofar as it has been the experience of several centers that symptomatic improvement occurs in the large majority of patients after operation.\(^1\)\(^-\)\(^4\)\(^-\)\(^10\) Thus, it would appear reasonable to recommend the operation to patients who are severely limited by angina pectoris. However, patients who have coronary artery disease with minimal symptoms, or with symptoms that are medically well controlled, present a more difficult therapeutic decision. The dilemma arises because it is not known whether ACBO preserves viable myocardium or whether it reduces mortality from coronary artery disease. Despite this lack of basic information, the assumption that these latter two goals will be realized provides the rationale for recommending operation to patients with minimal or mild symptoms attributable to coronary artery disease.\(^11\)

Because of the difficulties inherent in carrying out an extensive randomized study to assess the results of an operation that has generated such enthusiastic support, the critical question of whether or not the operation influences longevity will be extremely difficult to answer. However, some insight regarding the question of whether or not operation preserves myocardial function can be derived from

From the Cardiology Branch and the Clinic of Surgery, National Heart and Lung Institute, Bethesda, Maryland.
Address for reprints: Dr. Stephen E. Epstein, Chief, Cardiology Branch, National Heart and Lung Institute, Building 10, 7B-15, Bethesda, Maryland 20014.
Received July 15, 1973; revision accepted for publication October 8, 1973.

Circulation, Volume XLIX, March 1974 467
more modest studies than those required to answer the longevity question. To achieve this end, we have studied the relationship between graft patency and left ventricular performance before and after ACBO in 22 patients with coronary artery disease and chronic disabling angina pectoris.

Materials and Methods

The results of operation on 39 consecutive patients operated on between September 1970 and February 1972 were reviewed. The operative techniques utilized in all patients were moderate hypothermia (30°C); DC fibrillating electrodes, which were promptly removed once ventricular fibrillation was established; and left ventricular apical vents. The aorta was never cross clamped and blood flow on cardiopulmonary bypass was maintained at a minimum of 2 L/min. The total time on cardiopulmonary bypass averaged 30 min/graft. No perioperative deaths occurred. All of these patients had preoperative catheterization studies, and were catheterized postoperatively regardless of the presence or absence of symptoms, or of the severity of symptoms. Of these 39 patients, 17 were excluded from the study because of the following reasons: eleven because preoperative ventricular cineangiograms were performed nonquantitatively at other institutions; three because of frequent premature contractions and one because of camera malfunction during critical portions of either the pre- or postoperative catheterization studies; two because of coexistent valvular surgery. Otherwise, the patients were unselected and the final study group comprised 22 patients who had left ventricular cineangiograms of sufficient technical quality to measure left ventricular volumes.

All 22 patients had chronic disabling angina pectoris due to coronary artery disease ranging from two months to 16 years (avg 3.7 years). Eleven patients were diagnosed as having a previous myocardial infarction and four additional patients as having a possible previous infarction. The American Heart Association classification of the patients before operation is summarized in figure 1. Eleven patients had type IV and two had type II hyperlipoproteinemia; two others had mild elevation of serum cholesterol which was not further classified. Four patients had mild hypertension and four had diabetes mellitus.

The patients were studied before and one week to nine months (avg 5 months) after ACBO. The postoperative study was obtained within one month on two, within the fourth to sixth month on 13, and during the seventh to ninth month on seven. Studies were performed in the postabsorptive state after premedication with intramuscular pentobarbital. Coronary angiography and cineangiograms of the grafts were performed by either the Sones or Judkins techniques in the left anterior oblique, right anterior oblique, and lateral projections. Cardiac output was measured by left ventricular indicator dilution curves and left ventricular cineangiograms were performed prior to coronary angiography in the right anterior oblique projection using 60-80 ml of 76% meglumine diatrizoate.

The calibration factor for estimation of ventricular volumes was obtained by measuring the distance from the anterior-posterior X-ray tube to the left ventricle by centering the lateral tube of the biplane cine on the tip of the angiography catheter. A one-centimeter cross-hatched grid was then filmed at the same distance from the antero-posterior tube with the image intensifier in the same position as during the left ventriculogram. End-systolic and end-diastolic frames from each of two cardiac cycles of the left ventricular cineangiogram were projected on a Picker Vertex projector and the silhouettes were traced and planimetered by hand. Premature or post premature beats were not employed. In most cases, two of the first five beats after opacification of the ventricle were selected. Ventricular volumes were computed by the area-length formula using the correction factor for the right anterior oblique projection reported by Kennedy.12 Stroke volume was determined angiographically by subtracting the end-systolic volume from the end-diastolic volume. All volume data are the average of the two cycles and are corrected for body surface area (expressed in indices).

Left ventricular stroke work index (LVSWI) was computed by the formula:

\[ \text{LVSWI} (\text{g}-\text{m}/\text{m}^2) = (\text{SAP} - \text{LVEDP}) \times \text{SVI} \times 0.0136 \]

where SAP is the systemic arterial mean pressure (mm Hg), LVEDP is the left ventricular end-diastolic pressure (mm Hg), and SVI is the stroke volume index (ml/m²).

Patients were divided into two groups according to the patency of the aorto-coronary bypass grafts (tables 1 and 2). The patent group consisted of 10 patients with 15 grafts, all of which were patent. The occluded group consisted of 12 patients each having one or more occluded grafts, with or without coexistent patent grafts (22 grafts, 15 occluded). Ten patients had a single vein graft implanted, nine patients had double vein.
### Table 1

**Patent Group Hemodynamic Data**

<table>
<thead>
<tr>
<th>Pt</th>
<th>Age, Sex</th>
<th>Graffe</th>
<th>CI Pre</th>
<th>Post</th>
<th>HR Pre</th>
<th>Post</th>
<th>LVEDP Pre</th>
<th>Post</th>
<th>EDVI Pre</th>
<th>Post</th>
<th>ESVI Pre</th>
<th>Post</th>
<th>SVI Pre</th>
<th>Post</th>
<th>EF Pre</th>
<th>Post</th>
<th>LVSWI Pre</th>
<th>Post</th>
</tr>
</thead>
<tbody>
<tr>
<td>DK</td>
<td>42 M</td>
<td>+</td>
<td>88</td>
<td>72</td>
<td>10</td>
<td>14</td>
<td>68</td>
<td>71</td>
<td>24</td>
<td>31</td>
<td>44</td>
<td>40</td>
<td>63</td>
<td>57</td>
<td>28</td>
<td>45</td>
<td></td>
<td></td>
</tr>
<tr>
<td>OC</td>
<td>53 M</td>
<td>+</td>
<td>63</td>
<td>88</td>
<td>10</td>
<td>11</td>
<td>90</td>
<td>81</td>
<td>42</td>
<td>38</td>
<td>48</td>
<td>44</td>
<td>54</td>
<td>58</td>
<td>49</td>
<td>38</td>
<td></td>
<td></td>
</tr>
<tr>
<td>OV</td>
<td>62 M</td>
<td>+ +</td>
<td>2.5</td>
<td>2.3</td>
<td>16</td>
<td>23</td>
<td>80</td>
<td>101</td>
<td>29</td>
<td>57</td>
<td>50</td>
<td>44</td>
<td>63</td>
<td>44</td>
<td>47</td>
<td>39</td>
<td></td>
<td></td>
</tr>
<tr>
<td>MG</td>
<td>56 M</td>
<td>+</td>
<td>3.4</td>
<td>2.7</td>
<td>14</td>
<td>12</td>
<td>69</td>
<td>53</td>
<td>32</td>
<td>31</td>
<td>37</td>
<td>23</td>
<td>53</td>
<td>42</td>
<td>43</td>
<td>25</td>
<td></td>
<td></td>
</tr>
<tr>
<td>DN</td>
<td>47 M</td>
<td>+</td>
<td>3.0</td>
<td>2.5</td>
<td>15</td>
<td>13</td>
<td>77</td>
<td>57</td>
<td>24</td>
<td>21</td>
<td>53</td>
<td>36</td>
<td>68</td>
<td>63</td>
<td>73</td>
<td>41</td>
<td></td>
<td></td>
</tr>
<tr>
<td>RE</td>
<td>55 M</td>
<td>+ +</td>
<td>3.9</td>
<td>3.0</td>
<td>13</td>
<td>10</td>
<td>62</td>
<td>74</td>
<td>27</td>
<td>21</td>
<td>35</td>
<td>53</td>
<td>57</td>
<td>72</td>
<td>38</td>
<td>59</td>
<td></td>
<td></td>
</tr>
<tr>
<td>LH</td>
<td>43 M</td>
<td>+</td>
<td>2.7</td>
<td>2.9</td>
<td>12</td>
<td>12</td>
<td>68</td>
<td>49</td>
<td>25</td>
<td>18</td>
<td>43</td>
<td>31</td>
<td>63</td>
<td>63</td>
<td>42</td>
<td>34</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CL</td>
<td>45 M</td>
<td>+</td>
<td>3.0</td>
<td>2.7</td>
<td>13</td>
<td>14</td>
<td>76</td>
<td>69</td>
<td>33</td>
<td>33</td>
<td>42</td>
<td>36</td>
<td>54</td>
<td>53</td>
<td>43</td>
<td>36</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Group Mean**: 3.0, 2.7, 77, 85, 13, 14, 76, 69, 33, 33, 42, 36, 54, 53, 43, 36

Abbreviations: Pre = preoperative; Post = postoperative; RCA = right coronary artery; LAD = left anterior descending; LCCA = left circumflex coronary artery; CI = cardiac index; HR = heart rate; LVEDP = left ventricular end-diastolic pressure; EDVI = end-diastolic volume index; ESVI = end systolic volume index; SVI = stroke volume index; EF = ejection fraction; LVSWI = left ventricular stroke work index; M = male; + = graft patent.

### Table 2

**Occluded Group Hemodynamic Data**

<table>
<thead>
<tr>
<th>Pt</th>
<th>Age, Sex</th>
<th>Graffe</th>
<th>CI Pre</th>
<th>Post</th>
<th>HR Pre</th>
<th>Post</th>
<th>LVEDP Pre</th>
<th>Post</th>
<th>EDVI Pre</th>
<th>Post</th>
<th>ESVI Pre</th>
<th>Post</th>
<th>SVI Pre</th>
<th>Post</th>
<th>EF Pre</th>
<th>Post</th>
<th>LVSWI Pre</th>
<th>Post</th>
</tr>
</thead>
<tbody>
<tr>
<td>PH</td>
<td>45 M</td>
<td>0</td>
<td>3.3</td>
<td>3.6</td>
<td>75</td>
<td>90</td>
<td>14</td>
<td>18</td>
<td>64</td>
<td>54</td>
<td>20</td>
<td>17</td>
<td>44</td>
<td>37</td>
<td>69</td>
<td>69</td>
<td>43</td>
<td>34</td>
</tr>
<tr>
<td>SC</td>
<td>45 M</td>
<td>0</td>
<td>2.6</td>
<td>3.3</td>
<td>66</td>
<td>65</td>
<td>11</td>
<td>18</td>
<td>79</td>
<td>60</td>
<td>27</td>
<td>21</td>
<td>51</td>
<td>34</td>
<td>65</td>
<td>65</td>
<td>53</td>
<td>39</td>
</tr>
<tr>
<td>WS*</td>
<td>51 M</td>
<td>0</td>
<td>2.4</td>
<td>3.1</td>
<td>74</td>
<td>103</td>
<td>11</td>
<td>14</td>
<td>61</td>
<td>86</td>
<td>12</td>
<td>48</td>
<td>49</td>
<td>38</td>
<td>81</td>
<td>44</td>
<td>46</td>
<td>41</td>
</tr>
<tr>
<td>JW*</td>
<td>35 M</td>
<td>0</td>
<td>3.4</td>
<td>4.9</td>
<td>75</td>
<td>92</td>
<td>13</td>
<td>3</td>
<td>76</td>
<td>80</td>
<td>35</td>
<td>40</td>
<td>41</td>
<td>39</td>
<td>54</td>
<td>49</td>
<td>36</td>
<td>33</td>
</tr>
<tr>
<td>DL</td>
<td>59 M</td>
<td>0</td>
<td>3.4</td>
<td>1.9</td>
<td>79</td>
<td>102</td>
<td>22</td>
<td>25</td>
<td>84</td>
<td>92</td>
<td>51</td>
<td>63</td>
<td>34</td>
<td>29</td>
<td>40</td>
<td>32</td>
<td>38</td>
<td>31</td>
</tr>
<tr>
<td>TN†</td>
<td>45 M</td>
<td>+</td>
<td>2.8</td>
<td>2.1</td>
<td>87</td>
<td>107</td>
<td>17</td>
<td>35</td>
<td>100</td>
<td>149</td>
<td>53</td>
<td>137</td>
<td>46</td>
<td>12</td>
<td>47</td>
<td>8</td>
<td>40</td>
<td>7</td>
</tr>
<tr>
<td>FK</td>
<td>44 M</td>
<td>+</td>
<td>3.9</td>
<td>3.4</td>
<td>108</td>
<td>97</td>
<td>8</td>
<td>15</td>
<td>78</td>
<td>69</td>
<td>32</td>
<td>37</td>
<td>47</td>
<td>33</td>
<td>60</td>
<td>43</td>
<td>32</td>
<td>23</td>
</tr>
</tbody>
</table>

**Group Mean**: 3.3, 3.1, 81, 92, 12, 16, 74, 77, 32, 43, 42, 33, 59, 49, 45, 33

Abbreviations: Pre = preoperative; Post = postoperative; RCA = right coronary artery; LAD = left anterior descending; LCCA = left circumflex coronary artery; CI = cardiac index; HR = heart rate; LVEDP = left ventricular end-diastolic pressure; EDVI = end-diastolic volume index; ESVI = end systolic volume index; SVI = stroke volume index; EF = ejection fraction; LVSWI = left ventricular stroke work index; M = male; + = graft patent; 0 = graft occluded.

* = perioperative infarction; † = possible perioperative infarction; OM = obtuse marginal; PC = posterior circumflex.
grafts, and three had triple vein grafts. The right coronary artery was bypassed in 10 patients (three grafts were occluded); the left anterior descending artery was bypassed in 14 patients (five grafts were occluded); and the left circumflex artery was bypassed in 12 patients (13 grafts—seven were occluded). The data were analyzed statistically using a two-sided paired t-test.

For purposes of qualitative assessment, the left ventricle was divided into the three following areas: anterior wall, inferior wall and apex. The preoperative and postoperative cineangiograms for each patient were reviewed by at least two of the authors to determine whether or not each segment of the left ventricle had improved, deteriorated or remained unchanged. Wall segments were classified into one of four possible categories: normal, hypokinetic, akinetic, or dyskinetic. 1) Normal—the segment had synchronous and inward movement of good amplitude throughout. 2) Hypokinesis—a portion or all of a segment had inward movement judged qualitatively to be diminished in amplitude. 3) Akinetic—a portion or all of the segment had complete absence of wall motion. 4) Dyskinetic—a portion or all of a segment had paradoxical systolic outward motion. Discrete new areas of impaired apical contractility, which could be attributed to the surgical vent site, were excluded from consideration in the analysis of apical segmental wall function. Improvement was defined as an increase in the amplitude of inward wall motion and/or a decrease in the area involved by any given abnormality. Deterioration was defined as a decrease in the amplitude of inward wall motion and/or an increase in the area involved by any given abnormality.

Based on this segmental analysis, another qualitative judgment was made as to whether or not the left ventricle as a unit had improved, deteriorated, or remained unchanged. After these qualitative changes were assessed, they were correlated with the location and patency of the aorto-coronary grafts. Whenever segmental deterioration was noted, preoperative and postoperative coronary arteriograms were compared to determine whether or not progression of the underlying coronary artery disease accompanied this change.

Results

Clinical Aspects

At the time of postoperative evaluation, all 22 patients were clinically improved although two remained in the same functional class (fig. 1). Of the 37 individual saphenous vein grafts, 22 were patent yielding a patency rate of 60%. Five of the 10 patients in the patent group and eight of the 12 patients in the occluded group still had angina pectoris. Seven patients in the patent group and five patients in the occluded group had preoperative exertional dyspnea; three of the seven in the patent group and two of the five patients in the occluded group continued to have this symptom postoperatively. Two additional patients in the occluded group experienced exertional dyspnea for the first time after operation. Of the seven patients with no patent grafts, two were angina-free and five reported a decrease in angina. Five of these seven patients exhibited new areas of impaired segmental contractility on the postoperative cineangiogram. Two of the seven sustained clinically evident perioperative acute myocardial infarctions; one of these two patients was subsequently free of angina.

Two other patients, each of whom had one occluded and at least one patent graft, probably sustained a perioperative acute myocardial infarction. One was subsequently free of angina. Thus, the incidence of perioperative infarction, probable or documented, was 18% (4/22 patients).

Hemodynamics

The hemodynamic data for both groups are listed in tables 1 and 2. The group mean differences between the pre- and postoperative studies are summarized in table 3. A negative sign indicates that a decrease occurred from the preoperative value.

End-diastolic volume (fig. 2) did not change consistently in either group and none of the patients had mitral regurgitation either preoperatively or postoperatively. However, considering a change of greater than 20% in either direction as significant,

![Figure 2](http://circ.ahajournals.org/lookup/doi/10.1161/01.CIR.49.3.470)
then the data can be broken down as follows: end-diastolic volume in the ten patients with patent grafts increased significantly in one patient and decreased in four; in the eleven patients with occluded grafts it increased significantly in two patients and decreased in two patients. Similarly, end-systolic volume in the patent group decreased significantly in four of 10 patients and increased significantly in two patients; in the occluded group it decreased significantly in one of 11 and increased in three. Ejection fraction (fig. 3) decreased an average of 10% in the occluded group ($P < 0.05$). If 20% is considered to be a significant change in ejection fraction, it was found that no patient in the occluded group improved and four deteriorated; in the patent group, only two of 10 patients showed improvement in ejection fraction while two showed deterioration. Stroke volume index tended to fall in the patent group and fell significantly in the occluded group. Heart rate increased in the patent group, and increased significantly in the occluded group. Cardiac index did not change significantly in either group. Left ventricular end-diastolic pressure (LVEDP) increased significantly in the occluded group, with three patients exhibiting a rise of 10 mm Hg or more.

The change in stroke volume index was related to the change in left ventricular end-diastolic pressure (fig. 4). In the patent group, one patient appeared improved, five were unchanged and four were worse. In the occluded group, none of the patients improved, three were unchanged, eight were worse, and one did not have stroke volume measured. The change in left ventricular stroke work index was related to the change in left ventricular end-diastolic pressure (fig. 5). In the patent group, one patient improved, five were unchanged, and four were worse. In the occluded group, none of the patients improved, four were unchanged, seven were worse and one did not have left ventricular stroke work index measured.

It should be pointed out that there was a good correlation in most patients between stroke volume determined by angiography and by green dye. Occasionally, however, wide discrepancies were noted. As a result, similar hemodynamic analyses to

**Table 3**

*Group Mean Hemodynamic Data*

<table>
<thead>
<tr>
<th></th>
<th>Patent group</th>
<th></th>
<th>Occluded group</th>
<th></th>
<th>Entire group</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Preop</td>
<td>Postop</td>
<td>Difference</td>
<td>Preop</td>
<td>Postop</td>
<td>Difference</td>
</tr>
<tr>
<td>CI</td>
<td>3.0</td>
<td>2.7</td>
<td>-0.3</td>
<td>3.3</td>
<td>3.1</td>
<td>-0.2</td>
</tr>
<tr>
<td>HR</td>
<td>77</td>
<td>85</td>
<td>8</td>
<td>81</td>
<td>92</td>
<td>11*</td>
</tr>
<tr>
<td>LVEDP</td>
<td>13</td>
<td>14</td>
<td>1</td>
<td>12</td>
<td>16</td>
<td>4*</td>
</tr>
<tr>
<td>EDVI</td>
<td>76</td>
<td>69</td>
<td>-7</td>
<td>74</td>
<td>77</td>
<td>3</td>
</tr>
<tr>
<td>ESVI</td>
<td>35</td>
<td>33</td>
<td>-2</td>
<td>32</td>
<td>43</td>
<td>12</td>
</tr>
<tr>
<td>SVI</td>
<td>42</td>
<td>36</td>
<td>-6</td>
<td>43</td>
<td>33</td>
<td>-10*</td>
</tr>
<tr>
<td>EF</td>
<td>54</td>
<td>53</td>
<td>-1</td>
<td>59</td>
<td>49</td>
<td>-10*</td>
</tr>
<tr>
<td>LVSWI</td>
<td>43</td>
<td>36</td>
<td>-7</td>
<td>45</td>
<td>33</td>
<td>-12†</td>
</tr>
</tbody>
</table>

**Abbreviations:** Preop = preoperative; Postop = postoperative; CI = cardiac index; HR = heart rate; LVEDP = left ventricular end-diastolic pressure; EDVI = end-diastolic volume index; ESVI = end systolic volume index; SVI = stroke volume index; EF = ejection fraction; LVSWI = left ventricular stroke work index.

* $P < .05$  † $P < .01$  ‡ $P < .005$
against pre- to postoperative change in left ventricular contractility, whereas movement into the lower right quadrant reflects impaired contractility. Lower left and upper right quadrants reflect no change in contractility. Only one patient, in the patent group, clearly improved. Deterioration was noted in both groups.

**Figure 4**

Pre- to postoperative change in stroke volume index plotted against pre- to postoperative change in left ventricular end-diastolic pressure. Movement into the upper left quadrant reflects improved contractility, whereas movement into the lower right quadrant reflects impaired contractility. Lower left and upper right quadrants reflect no change in contractility. Only one patient, in the patent group, clearly improved. Deterioration was noted in both groups.

**Figure 5**

The pre- to postoperative change in stroke work index plotted against the pre- to postoperative changes in left ventricular end-diastolic pressure. Only one patient in the patent group clearly improved. Deterioration was noted in both groups.

**Figure 6**

Summary of changes in left ventricular segmental function in segments supplied by patent grafts. Segmental deterioration was not uncommon despite graft patency. No co-existent occluded grafts were present.

_Circulation, Volume XLIX, March 1974_
Figure 7 depicts the changes noted in the 22 segments situated in the potential region of supply of an occluded vein graft. Coexistent patent grafts were not present at any of these sites. None of the segments improved, 14 remained unchanged, and eight showed deterioration. Progression of the underlying coronary artery disease proximal to the site of vein graft anastomosis was probably responsible for the deterioration of five of these eight segments. In three, however, no anatomic basis for the deterioration could be found.

Figure 8 depicts the changes noted in segments supplied by both patent and occluded grafts. None of the segments improved, four remained unchanged, and six deteriorated. Deterioration of both the anterior and apical segments in one patient (F.K.) was probably secondary to severe angulation of a coronary arterial branch at its site of anastomosis with an occluded vein graft. No anatomic basis could be found for the functional impairment in the remaining four segments that deteriorated.

Six segments were completely remote from any vein graft site. No change in contractility was noted in any of these segments.

Additional analyses were performed to determine whether or not electrocardiographic diagnosis of infarction is predictive of subsequent failure of segmental wall function to improve following successful bypass operation. An infarct was considered to be present on the electrocardiogram using conventional Q wave criteria. Thirteen segments were unchanged postoperatively despite patent grafts. Of these, seven exhibited abnormal function. Only four of the abnormal segments that did not change after bypass surgery were in areas judged, on the basis of the electrocardiogram, to be in the site of old infarction. Thus, the presence or absence of evidence of old infarction on the electrocardiogram did not predict lack of improvement of segmental function. Six segments supplied by patent grafts improved postoperatively. Two of these segments were judged by an electrocardiogram to be in the site of old infarction. Thus, Q wave evidence in the electrocardiogram of old infarction does not necessarily imply that a given myocardial segment will not improve following a successful bypass operation.

Results of the qualitative analysis of over-all left ventricular contractility are summarized in Table 4. In the ten patients with all patent grafts, three showed over-all improvement, three were unchanged and four had deteriorated. In the twelve patients with at least one occluded graft none were improved, three were unchanged, and nine had deteriorated.

**Figure 7**

*Summary of changes in left ventricular segmental function in segments located in the potential region of supply of occluded grafts. No coexistent patent grafts were present.*

**Figure 8**

*Summary of changes in left ventricular segmental function in segments in the region of supply of both patent and occluded grafts.*
Table 4

Qualitative Assessment of Overall Left Ventricular Contractility

<table>
<thead>
<tr>
<th></th>
<th>Patent group</th>
<th>Occluded group</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Improved</td>
<td>3</td>
<td>0</td>
<td>3</td>
</tr>
<tr>
<td>No change</td>
<td>3</td>
<td>3</td>
<td>6</td>
</tr>
<tr>
<td>Worse</td>
<td>4</td>
<td>9</td>
<td>13</td>
</tr>
<tr>
<td>Total</td>
<td>10</td>
<td>12</td>
<td>22</td>
</tr>
</tbody>
</table>

Discussion

The results of the present investigation indicate that an ACBO is extremely effective in improving or abolishing angina pectoris. However, it was disconcerting to find that improvement and even total abolition of angina occurred whether or not the bypass grafts were patent. Myocardial function deteriorated frequently, even in the group of patients with all patent grafts. It is likely that many patients with improved symptoms and patent grafts experienced symptomatic relief because of restoration of more normal myocardial flow. However, the possibility cannot be excluded that some of these patients improved because the potentially ischemic myocardium was infarcted during the course of operation or shortly thereafter. Alternatively, a placebo effect of surgery might have contributed to the amelioration of symptoms. One or both of these latter two mechanisms may have been responsible for the symptomatic improvement in the patients with occluded grafts.

Our results demonstrating occasional hemodynamic improvement, but not uncommon hemodynamic deterioration, differ from those of Chatterjee and co-authors who demonstrated that ejection fraction increased postoperatively in each of six patients with preinfarction angina studied two weeks after operation. However, it is possible that if ejection fraction is transiently diminished in such patients because of acute myocardial ischemia, normal function may be readily restored once ischemia is relieved either operatively or during the course of medical therapy. While our results, obtained in patients with stable angina pectoris, sometimes demonstrated improvement in segmental wall motion and ventricular function when venous grafts were patent with good run-off, different conclusions emerged when the over-all results were assessed. Thus, segmental wall motion and ventricular function frequently deteriorated in patients with occluded grafts, results similar to those reported recently from other institutions.

Moreover, we found that deterioration of segmental wall motion was not uncommon even in the presence of a patent graft with good run-off.

Deterioration of segmental wall function appeared to be due to graft occlusion and progression of disease in the proximal coronary artery in several of our patients, to severe stenosis of the coronary artery at the site of the graft anastomosis in one, and to severe angulation of the coronary artery at its site of anastomosis with an occluded graft in another. Although an underlying anatomic explanation for deterioration of segmental wall function could be found in some of our patients, no anatomic mechanism could be discerned for nearly two-thirds (14/23) of the segments that exhibited new or increased impairment. These data are similar to those of Bourassa et al. Therefore, it would appear that the most likely cause of the myocardial deterioration in those patients in whom progression of the underlying coronary artery disease could not be documented was intra- or perioperative myocardial infarction. Disruption of the important apical collaterals by the left ventricular apical vent may have been a contributing factor in some patients’ conditions. Furthermore, there was a significant increase postoperatively in mean heart rate. Such a change could have influenced the final outcome by resulting in a chronotropically mediated increase in myocardial contractility. Alternately, the increased rate could have caused increased ischemia and thereby caused a decrease in myocardial function. On the basis of our data it is not possible to determine whether or not the increase in heart rate contributed to the observed results.

One of the problems that often arises when the results of ACBO are assessed accrues from sampling bias. For example, many patients who are asymptomatic postoperatively are often unwilling to be recatheterized. Thus, the results of many studies are necessarily weighted toward the patient with a poor operative result. In our own institution, postoperative studies are carried out routinely, regardless of symptoms, with successful follow-up in over 95% of patients. Therefore, the present investigation comprises a consecutive series of patients. The only patients excluded were those who did not have quantitative ventriculography (patients studied preoperatively at other institutions), and those who did not have adequate studies due to technical problems. Thus, the incidence of impaired myocardial function, observed postoperatively, cannot be attributed to a biased selection of a group with poor symptomatic results.
In summary, it appears that improvement of angina pectoris after ACBO is not related necessarily to patency of the bypass graft, and is often associated with impaired left ventricular contractile function. Hopefully, improvement in surgical techniques, or more aggressive attempts to provide the most complete revascularization possible, will reduce the incidence of deterioration in left ventricular function following operation. However, the likelihood of attaining this goal is uncertain. Meanwhile, the claim that operation should be performed when the primary surgical goal is preservation or enhancement of myocardial function would not appear to be substantiated by the available data.

Acknowledgment

The authors wish to express their thanks to Mrs. Mimi Winterhalter for her expert technical assistance and to Mr. Morton Raff, Dr. William Friedewald, and Mr. Joseph Verter of the Biometrics Research Branch of the National Heart and Lung Institute for their help in the statistical analysis of the data.

References

11. American College of Cardiology, Twenty-second Annual Scientific Session: Symposium, Advances in the Surgical Treatment of Coronary Heart Disease, Feb 17, 1973
Deterioration of Myocardial Function Following Aorto-Coronary Bypass Operation
RICHARD L. SHEPHERD, SAMUEL B. ITSCOITZ, D. LUKE GLANCY, EDWARD B. STINSON, ROBERT L. REIS, GORDON N. OLINGER, CHESTER E. CLARK and STEPHEN E. EPSTEIN

Circulation. 1974;49:467-475
doi: 10.1161/01.CIR.49.3.467

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1974 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/49/3/467

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org/subscriptions/