Effects of Surgery on Angina (Pre- and Postinfarction) and Myocardial Function (Failure)

By Jack C. Manley, M.D., and W. Dudley Johnson, M.D.

SUMMARY
With the advent of direct bypass surgery, the dramatic clinical responses in some patients has been followed by enormous enthusiasm in many centers. Surgical technics have advanced to such a point that nearly all patients with obstructive coronary disease could have the obstructions bypassed to one or many areas. Criteria for evaluating surgery include mortality, operative infarction rate, patency of grafts, clinical response, and stress testing. Many reports fail to correlate results with angiographic studies of vein function and with completeness of revascularization. When cine studies are performed, a good correlation exists between patent veins and relief of angina and improved stress response. Different patterns of response to stress, sometimes independent of relief of angina, are obtained depending on the completeness of revascularization. Predictable relief of angina is found if revascularization is complete. At times dramatic, but much less predictable, relief of failure (improved ventricular function) follows revascularization. Angina is a valuable aid in selecting some patients for surgery. Stress testing (bicycle ergometry) can now define general groups of patients who are likely, and who are not likely, to show improved myocardial response to stress after surgery. These studies also demonstrate the need for the surgeon to provide complete revascularization whenever possible. The criteria for selection for surgery of patients with symptoms of gross heart failure remain unclear. While revascularization technics could be technically applied to nearly all coronary patients, present methods are unable accurately to define who really needs the surgery and, equally important, which hearts will respond once revascularization is completed.

Additional Indexing Words:
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Vein bypass
Coronary artery disease
Stress testing
Revascularization surgery
Exercise testing
Atrial pacing

During the past 4 years surgical therapy for coronary disease has reached unprecedented enthusiasm. This enthusiasm is based almost entirely on the advances in surgery that have made possible immediate reconstruction of flow to distal coronary arteries. The distal end-to-side vein bypass technic under anoxic arrest is by far the most popular procedure, but other methods of direct reconstruction of flow are at times useful and perhaps preferable to the vein jump graft. These latter methods include endarterectomy with manual or gas technics, mammary artery-to-coronary bypass, and rarely patch graft angioplasty. A large volume of information is accumulating in regard to this surgery, but a number of questions remain unanswered and many areas are unexplored. The following discussion summarizes the surgical achievements, limitations, and the clinical and physiologic responses to coronary surgery.

Early attempts at direct coronary surgery were sporadic, involved the use of patch graft technics, and for the most part were confined to the right coronary artery.1, 2 Patch grafting of the left coronary artery was associated with

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excessive mortality and was rapidly stopped. Under any circumstance, the patch graft was limited to a short segment of disease in the proximal part of a large dominant artery and the high late failure rate of the patch technic was not encouraging.

In experimental studies, methods to apply aortocoronary grafts were investigated and reported at least as early as 1960. Clinical application of aortic-coronary vein grafts began in 1964, but despite a good clinical result further study or use of the technic remained surprisingly dormant for several years. Favaloro, in 1967, began using veins for reconstruction of the proximal right coronary artery, with the method being primarily an interposition technic in which a vein was attached to the proximal and distal coronary in an end-to-end manner. It was the first method used in any significant number of patients and demonstrated the feasibility of the use of vein as a coronary substitute, but it had major drawbacks in that it was limited to the dominant right coronary artery which was large and had a short segment of disease proximal to the marginal branch.

The major factor inhibiting the development of coronary surgery was the assumption that the heart, and especially the heart with coronary artery disease, would not well tolerate anoxic arrest. This fear of anoxic arrest has persisted until relatively recently, even though many have confirmed the early experimental studies which demonstrated its value in performing aortic-coronary bypass. The clinical recognition that the coronary heart does tolerate anoxic arrest well was the major factor allowing vein grafts to be attached to smaller distal coronary arteries in all areas of the heart with end-to-side anastomosis making the left coronary artery equally as accessible as the right for direct revascularization. Time limits for anoxic arrest are not defined, and probably never will be, but certainly periods of 30-40 min at normothermia are tolerated without difficulty. Since it is technically easier, performing the distal anastomoses in a dry, quiet field probably increases the patency rate, but many factors fall into the category of individual preference of the surgeon and are probably not related to surgical results. These factors include normo- or hypothermic bypass, continuous or interrupted suture, variable degrees of hemodilution, and whether aortic or coronary anastomosis should be done first.

Not all patients have distal arteries suitable for direct bypass. When the right coronary artery is diffusely diseased out into the distal branches, endarterectomy, often in association with a vein bypass, can be useful. Most surgeons who do gas endarterectomy would prefer to do vein bypass grafts if possible. The early patency rate with endarterectomy is less than with simple bypass, and late diffuse fibrosis and/or closure is being seen frequently.

Patients with such diffuse coronary disease that not even one bypass can be inserted are rare and make up less than 1% of the coronary population. Much more frequent are patients with multiple diffuse lesions that cannot all be corrected with vein grafts. Some arteries are too small to bypass with consistent success. Particularly common in this regard is the circumflex that divides into three or four small marginal arteries instead of forming one or two large marginal branches. It is in this situation that the Vineberg procedure can be useful. While rarely used alone, it does offer potential to supply blood to areas of the heart not accessible to vein grafts. While not comparable to direct bypass, when implants work, they appear to improve year by year, a condition clearly not occurring in vein grafts. Physiologic studies leave little doubt that artery implants into the myocardium can contribute to myocardial function in some instances.

The patch graft technic is now rarely used and is probably useful only in patients without veins. In patients with two areas of disease in one artery, a vein can often be used as a patch graft over the second lesion while bypassing the first block, thus preserving flow above and below the secondary area of disease. A distal
end-to-side anastomosis, followed by a side-to-side anastomosis more proximally to a separately blocked area of the coronary system, is another variation of technic that increases the versatility of the vein bypass method.

The direct mammary artery-to-coronary bypass, described by Kolessov and popularized by Green et al., is an excellent method to revascularize the anterior descending coronary artery and is particularly useful in patients without veins. The patency rate appears to be higher, with Green reporting 70 of 72 (97%) patent at 1 year (Green GE: Personal communication). If these results persist, it will clearly become the method of choice to revascularize the anterior descending artery and is now our preferred method for anterior descending bypass.

Diffuse coronary disease is often a limiting factor in surgery but rarely a factor contraindicating surgery. Even though adequate distal arteries are not seen angiographically, with adequate surgical exploration satisfactory distal arteries are often found. When arteries are not adequate, endarterectomy, often combined with a vein graft, may open a distal field and allow for increased coronary flow.

The major limitation for the experienced coronary surgeon is not the diffuseness of the coronary disease, but rather the function of the ventricle. No amount of blood will affect scarred and fibrotic muscle. The surgical mortality relates closely to the degree of ventricular damage and not to the number of grafts that can be inserted. Comparisons of surgical mortality in the literature are meaningless. A surgical mortality of 2% from one center in "carefully selected patients" cannot be compared to a series from another center when 70% of patients have abnormal ventricular function. A categorization of ventricular function in coronary patients has been suggested by the Inter-Society Commission for Heart Disease Resources (table 1).

Subsequent reports on surgical results could be more adequately compared if patients were classified by this method. The percentage of patients with coronary disease who are felt to be too far advanced to undergo surgery varies with the ability and experience of the surgeon, and with the degree of risk the surgeon (and cardiologist) is willing to accept for salvage cases. Of patients with significant disease, 85-90% have been accepted for surgery by the authors and their associates during the past 2 years.

The selection of patients for surgery who have distinct ventricular malfunction is often difficult as ischemic but viable myocardium often cannot be differentiated from scar and fibrosis. Ventricular malfunction, plus angina, are distinctly more favorable surgically than malfunction alone as angina arises from ischemic muscle and, once revascularized, this muscle usually works better. The patient who had angina but has now lost it and has a diffusely poor ventricle, is a particularly poor and unacceptable risk. As a generalization, the worse the angina the better the surgical risk.

**Table 1**

**Classification of Patients**

<table>
<thead>
<tr>
<th>Left ventricular function</th>
<th>A. Angiographic appearance:</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Contracts well in all areas</td>
<td>2. Abnormal contraction in one area</td>
</tr>
<tr>
<td>3. Abnormal contraction in two areas</td>
<td>4. Quivering ventricle</td>
</tr>
<tr>
<td>B. Left ventricular EDP (mm Hg):</td>
<td></td>
</tr>
<tr>
<td>1. &lt;15</td>
<td>2. 15-20</td>
</tr>
<tr>
<td>3. &gt;20</td>
<td></td>
</tr>
<tr>
<td>C. Cardiac index (liters/min/m²):</td>
<td></td>
</tr>
<tr>
<td>1. 2.5-4</td>
<td>2. &lt;2.5</td>
</tr>
<tr>
<td>3. &lt;2.0</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Example</th>
<th>Dysfunction</th>
<th>A</th>
<th>B</th>
<th>C</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>1</td>
<td>&lt;15</td>
<td></td>
<td>2.5-4</td>
</tr>
<tr>
<td>Moderate</td>
<td>2</td>
<td>15-20</td>
<td></td>
<td>&lt;2.5</td>
</tr>
<tr>
<td>Severe</td>
<td>3</td>
<td>&gt;20</td>
<td></td>
<td>&lt;2.0</td>
</tr>
<tr>
<td>Gross</td>
<td>4</td>
<td>&gt;20</td>
<td></td>
<td>&lt;2.0</td>
</tr>
</tbody>
</table>

*Surgical risk relates more to ventricular function than to any other factor. To help establish some uniformity in reporting on surgery from different centers, this classification of patients has been recommended by the Inter-Society Commission for Heart Disease Resources.
Unfortunately, many patients have no angina, yet have life-threatening coronary disease and/or grossly poor ventricular function with the decision for or against surgery in this situation being difficult. Much better methods are obviously needed to differentiate, preoperatively, viable but ischemic and poorly functioning muscle from fibrotic and scarred myocardium.

Many criteria need to be considered when assessing the results of bypass surgery for coronary disease (table 2). Besides the obvious survival factor, relief of angina and improvement of ventricular function are major areas of concern. There is very little information in some categories; however, data are beginning to accumulate in several others. In reviewing published reports including abstracts from the November, 1971, American Heart Association meetings it is also apparent that methods of reporting differ from center to center which makes comparison of results difficult. Classification of symptoms, degree of coronary disease, and operations performed varied considerably, and undoubtedly this accounts for the variability of results. Most groups attempt to distinguish between stable angina, preinfarction angina, and acute myocardial infarction but neglect to mention whether revascularization was complete, i.e., all disease bypassed or not, and even whether any or all grafts were patent. Coexisting defects such as a ventricular septal defect and valvular abnormalities are often placed in separate groups, but not ventricular aneurysms, thus influencing results and mortality.

It is difficult to include all the factors that might influence risk and the results. Among these are included the experience of the surgeon, first or second chest operation, and co-existing malfunction of major organ systems. Perhaps even more important in evaluating risk and survival is the assessment of left ventricular function (performance) as judged by left ventricular end-diastolic pressure at rest or during stress. A classification of ventricular function is difficult to agree upon, but one used by our group during the early years is illustrated by table 3. An assessment of surgical mortality during the first half of 1971 is illustrated by table 4. Similar results are evident in the literature to support these concepts.7, 9, 23-27 The early 1971 mortality figures classified according to three of the ventricular function categories is represented in table 5. The early survival experience was evaluated by Dr. D. McRaven demonstrating

Table 2
Criteria for Assessments of Results

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Survival</td>
</tr>
<tr>
<td>2.</td>
<td>Incidence of postoperative myocardial infarction</td>
</tr>
<tr>
<td>3.</td>
<td>Postoperative graft patency</td>
</tr>
<tr>
<td>4.</td>
<td>Relief of angina pectoris</td>
</tr>
<tr>
<td></td>
<td>a. By history</td>
</tr>
<tr>
<td></td>
<td>b. During controlled testing (exercise or tachycardia induced by atrial pacing)</td>
</tr>
<tr>
<td></td>
<td>c. Improvement in left ventricular performance during stress</td>
</tr>
<tr>
<td>5.</td>
<td>Improvement in exercise tolerance</td>
</tr>
<tr>
<td></td>
<td>a. By history (angina or failure)</td>
</tr>
<tr>
<td></td>
<td>b. Controlled testing (exercise)</td>
</tr>
<tr>
<td>6.</td>
<td>Relief of left ventricular failure at rest</td>
</tr>
<tr>
<td>7.</td>
<td>Improvement of ventricular contraction</td>
</tr>
<tr>
<td></td>
<td>a. By angiographic methods (ejection fraction)</td>
</tr>
<tr>
<td></td>
<td>b. By analysis of muscle mechanics (V\text{max})</td>
</tr>
<tr>
<td>8.</td>
<td>Improvement in coronary blood flow</td>
</tr>
<tr>
<td>9.</td>
<td>Improvement in myocardial metabolism</td>
</tr>
</tbody>
</table>

Table 3
Early Method of Classifying Ventricular Function

<table>
<thead>
<tr>
<th>Ventricle function</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Normal</td>
</tr>
<tr>
<td>A. LV by angiography, contracts well in all areas</td>
</tr>
<tr>
<td>B. LVEDP &lt;12 mm Hg</td>
</tr>
<tr>
<td>C. CI normal (2.5-4 liters/min/m²)</td>
</tr>
<tr>
<td>2. Moderate dysfunction</td>
</tr>
<tr>
<td>A. LV by angiography, abnormal contraction in one area (includes discrete ventricular aneurysms)</td>
</tr>
<tr>
<td>B. LVEDP 13-20 mm Hg</td>
</tr>
<tr>
<td>C. CI low normal (&lt;2.5 liters/min/m²)</td>
</tr>
<tr>
<td>3. Severe dysfunction</td>
</tr>
<tr>
<td>A. LV by angiography, abnormal contraction in two or more areas</td>
</tr>
<tr>
<td>B. LVEDP &gt;20 mm Hg</td>
</tr>
<tr>
<td>C. CI low (&lt;2.0 liters/min/m²)</td>
</tr>
<tr>
<td>4. Gross dysfunction</td>
</tr>
<tr>
<td>A. LV by angiography, abnormal contraction in all areas (&quot;quivering ventricle&quot;)</td>
</tr>
<tr>
<td>B. LVEDP &gt;20 mm Hg</td>
</tr>
<tr>
<td>C. CI low (&lt;2.0 liters/min/m²)</td>
</tr>
</tbody>
</table>
the relation between degree of ventricular impairment and survival. Gorlin reviewed and presented a summary of the Cleveland and Milwaukee survival experience, in an address at the American Heart Association Meeting, November, 1971. At present, we are classifying ventricular contraction according to the outline in table 1. Longer survival studies are necessary to assess the true final results, but these data are most encouraging.

It has been known for many years that anesthesia and surgery performed on a group of patients with known coronary artery disease will be associated with a certain percentage of myocardial infarction during the operative and postoperative period. It is not surprising then that myocardial infarction should also occur during the course of coronary bypass surgery. The percentage of infarctions occurring under this setting is difficult to assess because of the variable criteria utilized in diagnosing this complication, and accordingly reports range from 3 to 17%.28-34 In a retrospective study of 200 patients who underwent coronary bypass surgery at St. Luke’s Hospital in Milwaukee during 1970-71 utilizing rather strict criteria (i.e., clinical episode, enzyme rise, Q wave by electrocardiogram, and angiographic documentation of ventricular impairment or change in vascular status), the incidence of infarction was 5%. Infarction was related to graft closure and adequacy of revascularization, as expected. A prospective study is now in progress to further evaluate this important problem, which at present appears unresolved.

Basic to any evaluation of bypass results is the determination of vein graft patency, both early and late. Grondin and Walker and their associates evaluated patency results.35-38 Noteworthy is the early (2–8 weeks) patency rate of 89%, with subsequent late closure rate of 14%, and resultant overall late patency rate of 75%.38 Since most patients receive multiple grafts, a considerably higher percentage of patients have at least one graft open. These late results are biased toward a poor result, as patients having difficulty were more likely to request restudy, whereas those patients completely asymptomatic were among the small group refusing restudy. A more recent larger series (317 patients, 675 grafts) revealed an overall patency rate of 81%, and this corresponds to the majority of reports from other hospitals.15, 27, 39, 40 Causes of graft failure can be multiple, but the majority of early closures are believed related to technical factors at

### Table 4

*Mortality after Bypass Surgery According to Class of Ventricular Function (Milwaukee Group, First Half of 1971)*

<table>
<thead>
<tr>
<th>Ventricular function</th>
<th>No. pt</th>
<th>No. died</th>
<th>Mortality (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Class 1 (normal)</td>
<td>152</td>
<td>3</td>
<td>2</td>
</tr>
<tr>
<td>Class 2 (one wall)</td>
<td>124</td>
<td>7</td>
<td>5</td>
</tr>
<tr>
<td>Class 3 (two walls)</td>
<td>92</td>
<td>10</td>
<td>11</td>
</tr>
<tr>
<td>Total</td>
<td>368</td>
<td>20</td>
<td>6</td>
</tr>
</tbody>
</table>

### Table 5

*Early and Late Mortality after Vein Bypass Surgery (St. Luke’s Hospital, Milwaukee, March ’68–June ’70)*

<table>
<thead>
<tr>
<th>LV angiogram</th>
<th>No. pt</th>
<th>1-mo mortality (%)</th>
<th>6–12-mo mortality (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Contractility normal</td>
<td>146</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>2. Hypokinetic, mild, generalized</td>
<td>18</td>
<td>11</td>
<td>11</td>
</tr>
<tr>
<td>3. Hypokinetic or akinetic (anterior wall)</td>
<td>35</td>
<td>9</td>
<td>11</td>
</tr>
<tr>
<td>4. Hypokinetic or akinetic (inferior wall)</td>
<td>28</td>
<td>14</td>
<td>14</td>
</tr>
<tr>
<td>5. Hypokinetic or akinetic (both walls)</td>
<td>33</td>
<td>30</td>
<td>30</td>
</tr>
<tr>
<td>major one wall, other involved</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>6. Aneurysm one wall (paradox), other involved</td>
<td>27</td>
<td>44</td>
<td>44</td>
</tr>
<tr>
<td>7. Hypokinetic, severe, generalized</td>
<td>23</td>
<td>26</td>
<td>26</td>
</tr>
<tr>
<td>Total</td>
<td>310</td>
<td>18 (3/1/68–5/31/69)</td>
<td>11 (6/1/69–6/30/70)</td>
</tr>
</tbody>
</table>

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surgery, selection of recipient vessels, and evaluation of vascular runoff. It is well established that vein graft flow measured at the time of surgery most closely correlates with patency.35-38

A major limitation of this surgery may be the late closures which appear to be related to a form of subintimal hyperplasia involving the vein graft and occasional thromboses or fibrous constriction of the graft.43-45 This may be diffuse, localized, involve one graft only, or may involve proximal or distal portions. The exact cause of the subintimal hyperplasia remains uncertain, but possibilities include preexisting abnormalities of the vein, effects of systemic pressure on the vein with secondary reaction, and the effects of trauma inflicted prior to or at the time of vein insertion.

Proven late closure can only be ascertained in patients undergoing two or more postoperative studies. Among 92 patients with two or more postoperative studies, Flemma reports a 7% early failure and 13% late failure rate (total 20%) (Flemma RJ, et al.: Unpublished data). Most failures occur within 1 year, and failure after 18 months has not yet been observed. While late failure is important, equally important would be data relating to the failure rate of the stenotic coronaries in a similar group of unoperated patients. Unfortunately no data are available concerning the failure rate of coronary arteries with variable degrees of obstruction. It is our clinical impression that both early and late closure rates are less during 1971, and we are presently evaluating another series of patients for patency during the later period. If the clinical impression is correct that vein fibrosis may be related to poor surgical technic, then improvement in this area may be reflected in a decreased late failure rate. Major areas of importance, technically, are the elimination of testing the vein with saline and substituting blood or a normal pH solution, avoidance of excess pressure in testing the vein, and avoidance of excessive delay in using the vein once it has been removed. It has already been shown by Green et al. that internal mammary bypass has a much higher early and late patency rate, and our initial evaluation of this in patients would tend to confirm this.

Relief of angina pectoris is the primary reason why patients seek this operative procedure. Unreliable as this may be in any objective evaluation, it is at least of interest. The mean value from the significant reports for operative survivors who are improved or asymptomatic ± 12 months after surgery is 90%.5, 15, 22, 27, 40 The mean value of those totally asymptomatic is approximately 70%.

As previously mentioned, the relation of angina to vein graft patency or adequacy of revascularization is not readily apparent from most reports, but in Milwaukee it is definitely related to graft patency and ability to perform satisfactory revascularization. Return of angina pectoris that had been relieved after surgery was definitely related to graft closure or progression of coronary vascular disease. Thirty percent of all repeat coronary bypass graft surgery has been related to progressive coronary disease rather than to graft failure. Angiographic diagnosis is essential in any postoperative patient with recurrent symptoms.

More satisfactory and objective with regard to evaluation of relief of angina following bypass surgery is an assessment using stress. The production of angina is usually (but not always) related to ventricular performance, and the interrelationship between the two has never been easy to explain. Relief of angina and improved hemodynamic performance during stress must be related to improved oxygen supply to the heart or reduced oxygen demand. Major determinants of myocardial oxygen demand have been shown to include intramyocardial tension (interventricular pressure, ventricular volume, myocardial mass), heart rate, and the contractile state.44 Hemodynamic evaluation while stressing the heart has been an objective method of evaluating the results of various medical or surgical interventions, with exercise and atrial pacing being the most common types of stress evaluation in coronary disease.45-66 Various indices derived from hemodynamic measurements have been used to estimate myocardial demand. Arterial
or left ventricular pressure is taken as an indication of wall tension, and the tension-time index (the integral of the ventricular pressure curve) has been widely used. Recent experimental clinical studies suggest that the less complicated blood pressure-heart rate product is a more accurate index than the tension-time index, and several clinical studies support its validity. Use of a triple product of heart rate, systolic blood pressure, and left ventricular ejection time is said to improve accuracy of predicted myocardial oxygen demand. Constructing ventricular function or performance curves according to the Frank-Starling principle has also been clinically useful, especially when the patient serves as his own control for subsequent evaluations. Here an increase in ventricular end-diastolic volume causes an increase in systolic tension and stroke volume, thus maintaining an increasing cardiac output. End-diastolic volume is estimated indirectly by measurement of end-diastolic pressure or directly by angiographic methods. This is viewing the heart as a pump. A more recent addition to the armamentarium is the evaluation of myocardial contractility. These measurements take into account the fact that the heart as a muscle has certain definable mechanical characteristics. An improvement in the contractile state increases the velocity and force of contraction from any ventricular end-diastolic muscle length. Isolated muscle studies have shown that the maximal velocity of shortening at zero load \( V_{\text{max}} \) is characteristic of the contractile state of the muscle which is independent of initial muscle length. These measurements can be obtained by complex methods that combine pressure measurement with quantitative angiography and more simple methods that utilize pressure measurements alone. These measurements of contractility have only recently been applied to the evaluation of coronary disease with certain basic problems remaining to be resolved prior to its final acceptance.

Atrial pacing before and after bypass surgery has been shown to be a reliable means of evaluating relief of angina if angina could be produced in this manner prior to surgery. The technics are well established in many reports. The mean value for relief of angina as evaluated by stress of atrial pacing is 59%. Tristani and associates in Milwaukee reported one such group with significant coronary artery disease evaluated by atrial pacing. In this group, vein graft patency was 80%. Clinically 15 of 17 patients with grafts patent were asymptomatic, whereas five of six patients with one or more grafts occluded still had angina (table 6).

Similar results during controlled testing by exercise have been reported. There is a higher incidence of angina produced by supine ergometer exercise than during atrial pacing (table 7). Thirty-eight patients were evaluated by one of us (J.C.M.) using stress testing. These were consecutive patients with angina and severe coronary artery obstruction, studied preoperatively, 4–6 weeks postoperatively, and 1 year after bypass surgery (Unpublished data). Medications were similar during each study. Hemodynamic evaluation and ventricular performance curves were obtained with each study (table 8), and attempts were made to subject all patients to higher stress after surgery.

Table 6

Results of Atrial Pacing in 23 Patients with Severe Coronary Obstructive Disease

<table>
<thead>
<tr>
<th></th>
<th>Preoperative</th>
<th>Postoperative</th>
</tr>
</thead>
<tbody>
<tr>
<td>No angina (10)</td>
<td>No angina (10)</td>
<td>(9) All grafts open</td>
</tr>
<tr>
<td>Angina (13)</td>
<td></td>
<td>(1) One of two grafts occluded</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(1) Single graft occluded</td>
</tr>
<tr>
<td>Angina (2)</td>
<td></td>
<td>All grafts occluded</td>
</tr>
</tbody>
</table>

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Table 7
Presence of Angina Pectoris before and after Surgery as Evaluated by History and Exercise
(Supine, Ergometer)

<table>
<thead>
<tr>
<th>Factor</th>
<th>Preop No.</th>
<th>Preop %</th>
<th>Postop (1) No.</th>
<th>Postop (1) %</th>
<th>Postop (2) No.</th>
<th>Postop (2) %</th>
</tr>
</thead>
<tbody>
<tr>
<td>History</td>
<td>38/38</td>
<td>100</td>
<td>6/38</td>
<td>16</td>
<td>8/38</td>
<td>21</td>
</tr>
<tr>
<td>Stress</td>
<td>33/38</td>
<td>87</td>
<td>2/38</td>
<td>5</td>
<td>*5/22</td>
<td>23</td>
</tr>
<tr>
<td>Grafts</td>
<td>—</td>
<td></td>
<td>90</td>
<td>*78</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Only 22 restudied (all alive).

Important conclusions were drawn by evaluating the entire group of patients. Even though the group of patients was small (table 8) and expression of coronary disease variable, it was found possible to subdivide the group on the basis of ventricular contraction as evaluated by angiography. Statistical analysis of many hemodynamic measurements disclosed a significant decrease in left ventricular end-diastolic pressure and rise in left ventricular stroke-work index (and stroke index) after successful bypass surgery in the group with good contraction as compared to the group with patent grafts but poor contraction. Ventricular performance curves were also strikingly improved in the former group as compared to the latter. Twenty-four of the 38 patients have been studied both early and late, and in addition another 75 patients have been studied in similar fashion 1 year after surgery. It is evident that the early results are similar to the late results if grafts remain patent. It is also evident that graft closure can be associated with the return of angina and that ventricular performance that was initially improved can deteriorate with or without further recognized ventricular impairment by infarction.

There is insufficient information available to assess improvement in exercise tolerance by patient history, but several reports indicate increased exercise tolerance to standard upright exercise testing. In patients not classified as to graft patency, the mean value for increased exercise tolerance was 75%. In patients with patent grafts, the mean value for increased exercise tolerance was 91% whereas the mean value fell to 32% with nonpatency of at least one graft. Some were worse than preoperatively.

An evaluation of influences of bypass surgery on congestive failure is always difficult because of the variability of criteria employed in definition. The clinical symptom of dyspnea of apparent cardiac origin when supported by left ventricular end-diastolic pressure over 25 mm Hg may be one way of definition, although lesser degrees of “failure” may not be appreciated especially when cardiac output is low.

While dramatic improvement in failure symptoms following surgery is occasionally seen, it should be emphasized that the predictability of this type of response preoperatively is uncertain. It has been noted that in patients with abnormal ventriculograms the improvement in ergometry studies after surgery is much less impressive than in patients with normal ventriculograms. While hemodynamic studies fail to identify these

Table 8
Evaluation of Hemodynamic Measurements during Exercise in Two Groups of Patients as Compared to a Group of Normal Subjects

<table>
<thead>
<tr>
<th>No. pt</th>
<th>Statistical analysis</th>
<th>Performance curves</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group I (13) LV contraction* poor</td>
<td>LVEDP NS</td>
<td>Mild improvement (or none)</td>
</tr>
<tr>
<td>Group II (25) LV contraction† good</td>
<td>$P &lt; 0.01$</td>
<td>Striking improvement</td>
</tr>
</tbody>
</table>

*Poor = One wall or greater area poor contraction (angio).
†Good = no more than mild impairment (angio).
‡Graft closure associated with deterioration.

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patients who will respond, the clinical presence of severe angina along with the failure is often a favorable predictive factor.

In the literature, data used to support relief of ventricular failure following surgery include increased resting cardiac index, stroke index, and stroke work, with decreased end-diastolic pressure at rest, increased ejection fraction, and increased dp/dt. Many patients have normal values at rest but demonstrate gross abnormalities during stress. Certainly this represents failure of the heart to meet its demands under the conditions imposed. Failure signs under stress (i.e. EDP of 35 mm Hg) are clearly expressive of abnormal ventricular function (performance), and often show a severe limitation of reserve of the heart. The degree of abnormal stress response (limited reserve) may eventually be useful in selecting patients for surgery. A normal resting but abnormal stress response appears to be more closely associated with a good surgical result than the more advanced degrees of failure represented by abnormal resting and stress responses.

The development of heart failure as an important clinical manifestation of coronary artery obstructive disease may be related to myocardial fibrosis resulting from previous myocardial cell loss or related to myocardial ischemia, or to both. Fibrosis may present itself as a focal (e.g. aneurysm) or as a diffuse process. With aneurysms, left ventricular function may be reduced because of systolic paradox and associated left ventricular dilatation. Myocardial fibrosis has been shown to be associated with decreased ventricular contractility in the areas of involvement.80

Experimentally, chronic ischemia without actual cell loss has also been shown to decrease myocardial contractility by causing a decrease in myocardial length-tension curves and the first derivative of tension in the ischemic areas. Elevations of resting tension in the ischemic areas have also been noted, produced according to the Frank-Starling principle.89, 90 There are only a few clinical observations that suggest correlation with the experimental work.91–93 During a period of coronary insufficiency, the ischemic myocardium functions on a depressed length-tension curve with more tension developed to achieve the same active tension. This results in a rise in the left ventricular end-diastolic pressure. Even without scar and fibrosis, chronic ischemia could thus lead to ventricular dilatation and rise in end-diastolic pressure with the clinical expression of heart failure.

The rationale behind the surgical (bypass) treatment of heart failure is related to the potential reversibility of chronically ischemic myocardium. There is experimental evidence to support such a concept,90–94 It is obvious that revascularization cannot improve myocardial contractility in areas of diffuse scar or fibrosis. Excision of significant areas may result in functional and clinical improvement, but evaluation of these areas and their significance is often difficult. As has been noted, it is difficult to determine preoperatively whether failure is due to chronic ischemia, and here the association of failure and angina is of some predictive value that viable myocardium is present. Other more reliable methods of identifying ischemic myocardium need to be developed.

It is very difficult to assess results in patients with mild failure as compared to moderate or severe failure, and probably the wide variability of reported results is a reflection of this. Improvement in left ventricular failure appears to occur in about 50% of patients. The same status may remain or actually deteriorate (by the above criteria) in 50% whose bypass grafts were patent.84, 95, 96 In patients with left ventricular failure preoperatively where one or more grafts were shown to be closed, more severe failure postoperatively often was present.96–98

There have been several reports of improvement in ventricular contraction as evaluated angiographically (e.g. ejection fraction).99–101 As might be expected, when the ejection fraction is normal preoperatively, it usually remains normal postoperatively. When ejection fraction is abnormal preoperatively and grafts are patent, some improve and some do not. This is as might be expected in a
scarred ventricle. With closure of grafts ejection fraction may worsen, especially with myocardial injury.\textsuperscript{102}

Improvement in ventricular contraction following bypass surgery as evaluated by analysis of velocity measurements, for example, has been limited and has certain inherent difficulties that remain to be overcome.\textsuperscript{103-104}

Postoperative improvement in coronary flow has been reported in patients undergoing bypass surgery as evaluated by the xenon, rubidium, and coincident-counting methods, and by roentgen videodensitometry.\textsuperscript{88, 105, 106} The number of patients reported is small, with all methods requiring further evaluation and confirmation.

Improvement in myocardial metabolism has been demonstrated in small numbers of patients.\textsuperscript{107, 108} One such group was studied by Brachfeld and Carlson during stress-induced (pacing) hypoxia with measurement of coronary sinus lactate.

**Comments**

There have been many surgical procedures proposed and performed as treatment of coronary artery obstruction. None has been as exciting and promising as the bypass graft procedure. There can be little question that within limits the surgery can be performed with an acceptable risk, that angina is relieved if grafts remain patent, and that ventricular performance (function) improves if preoperative contraction is not badly impaired and nearly complete revascularization is achieved. Supportive data are accumulating that coronary flow and myocardial metabolism are also favorably affected. It is also evident that some patients with heart failure improve as a result of bypass revascularization, but many appear unimproved and some are worse. Those who are worse usually have demonstrated graft closure.

At the present time, patients with significant angina associated with mild heart failure and mildly decreased contraction that is not so severe as to greatly raise operative mortality represent the best potential candidates; but even this remains to be substantiated.

Long-term studies are necessary to evaluate survival of the surgical group as compared to the medically treated group, the quality of life as afforded by both methods of treatment, late patency, rates of vein graft failure and causes of failure, and the results of bypass grafts as treatment of diffuse disease and heart failure.

The technical aspects of surgery have advanced to the point that some degree of revascularization could be achieved in nearly all patients. Much further work is needed to identify the patients who need this surgery, and also to identify the patients whose disease is too advanced to respond to the surgery.

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