Evaluation of the Surgical Correction of Mitral Regurgitation

By Earle B. Kay, M.D., David Mendelsohn, M.D., and Henry A. Zimmerman, M.D.

Usually the first questions to be asked by patients, their relatives, and their physicians, are: "What are the dangers of operation, and will it be a cure?" Obviously, any patient with rheumatic valvular disease will not be cured but may be given varying degrees of benefit dependent upon the degree of pathology, the degree of myocardial reserve, and the degree of effectiveness of the operative technic. The best results are obtained in good-risk patients who have less than severe cardiomegaly, reversible pulmonary vascular sclerosis, and a valvular mechanism amenable to correction. The myocardial component of valvular dysfunction cannot be overemphasized. No operative technic will be highly successful when employed in the presence of a failing heart. Similarly, the persistence of pulmonary vascular sclerosis may negate benefit gained by valvular correction.

Only too often in the past have physicians lost sight of the fact that cardiac disease is compensated at the expense of cardiac reserve. Only too often a patient with rheumatic valvular disease is followed medically until rapidly progressive deterioration becomes obvious, before surgical intervention is sought in the hopes of stopping this otherwise hopeless course.

In the past this policy has been justified in that surgical technics were not adequate to provide reasonable safety or assurance of benefit. The recent progress in surgical correction has now reversed this situation, so that safety and benefit are not so dependent upon the technic employed as they are on the time or state of the disease when surgery is performed.

Obviously, all patients with rheumatic valvular disease and specifically all patients with evidence of mitral regurgitation do not require operation. Many compensate for their disease and remain relatively stable for years. It is mandatory, however, for their physicians to recommend periodic examinations, and to determine from objective evidence of cardiac size and function whether the disease is progressive or stable.

Pathology

Twenty-three per cent of the regurgitant valves were of the pure variety in which the regurgitation was largely due to a disproportion between valvular tissue and annulus size so that the leaflets failed to approximate (fig. 1). A fourth of these patients had either disruption or stretching of the chordae tendineae with herniation of the leaflets into the atrium. The majority of these valves were repaired by variations of annular plication and resuturing or replacement of the chordae tendineae. These hearts had the greatest degree of myocardial hypertrophy. Several of the larger hearts of this group later stretched the valvular correction with recurrence of a regurgitant murmur.

Thirty-four per cent had combined, but predominantly regurgitant valves with an element of stenosis (fig. 2). Approximately 25 per cent of these had varying degrees of calcification. The heart size in patients with combined mitral stenosis and regurgitation was smaller than those with pure mitral regurgitation. There was considerable variation in the pathologic process causing the regurgitation, which necessitated individual adaptation of the operative technic. The majority were corrected with relative ease,

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whereas others were corrected only partially and with difficulty.

In 29 per cent of the patients, the element of stenosis was greater than the regurgitation (fig. 3). These hearts were smaller than those in the first two groups. A large majority of the valves in this group were satisfactorily corrected.

The last group, with destroyed valves, comprised 14 per cent of the patients operated upon (fig. 4). In 11, the valvular destruction was due to extensive calcification detected in 10 instances preoperatively by image amplification or by over-penetrated roentgen examinations. In three, the valvular destruction was due to absorption and replacement of the valvular components by scar. These patients were by far the most seriously ill of all, and had persistent myocardial failure. Though we recognized preoperatively the critical risk involved, we elected to proceed in the hopes that some benefit could be afforded them. In nine, no worthwhile surgical correction short of valvular replacement could be obtained. All died postoperatively from myocardial failure.

Since the development of an artificial mitral valve, five have had valvular replacement. Though there have been no successful long-term survivors, the artificial valve appeared to function properly. The cause of death in two patients was from associated coronary occlusion with myocardial infarction; one was due to progressive myocardial failure in a patient operated upon 5 years previously for mitral regurgitation by one of the closed technics; one died 7 days postoperatively from renal failure while being perfused on the artificial kidney; and one died 14 days postoperatively from poor pulmonary function resulting from asthmatic bronchitis, emphysema, pulmonary vascular sclerosis, a refractory electrolyte imbalance, and myocardial failure. This patient also had been operated on 7 years previously. The gross appearance of the artificial valve in this patient is noted in figure 5. There were no complications relating to the valve.

The majority of the patients with valvular destruction (10 out of 14) were known preoperatively to be so involved and could have been denied a chance for help. It is our opinion that certain centers should continue their efforts to benefit this unfortunate group of patients, but that the poor results initially obtained should not unduly overshadow the good results in other patients.

Results in Relation to Valve Pathology

The results of surgical correction in relation to valve pathology are tabulated in Table 1.

**Pure Regurgitation**

There was an over-all operative mortality during this 4-year period of 9 per cent. This was due mainly to air embolism occurring during the developmental stage of left-sided cardiotomies. This complication has been entirely corrected. One patient subsequently died suddenly 16 months postoperatively from acute rupture of the chordae tendineae.
SURGICAL CORRECTION OF MITRAL REGURGITATION

Examples of variations in valvular pathology in patients with combined but predominantly regurgitant lesions indicating valvular addition in the form of plastic material (left) and selective annular plication at the posteromedial commissure (right).

Table 1
Results in Relation to Valvular Pathology

<table>
<thead>
<tr>
<th>Type</th>
<th>No.</th>
<th>Operative mortality No.</th>
<th>%</th>
<th>Status 6 mo.—3–1/2 yr.</th>
<th>Dead</th>
<th>Fair</th>
<th>Good</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pure regurgitation</td>
<td>23</td>
<td>2</td>
<td>9</td>
<td>1</td>
<td>4</td>
<td>16</td>
<td>80%</td>
</tr>
<tr>
<td>Predominant regurgitation</td>
<td>34</td>
<td>4</td>
<td>12</td>
<td>1</td>
<td>6</td>
<td>23</td>
<td>80%</td>
</tr>
<tr>
<td>Combined (predominant stenosis)</td>
<td>29</td>
<td>3</td>
<td>10</td>
<td>1</td>
<td>6</td>
<td>26</td>
<td>100%</td>
</tr>
<tr>
<td>Destroyed</td>
<td>14</td>
<td>14</td>
<td>100</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
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</table>

Table 2
One-Year Analysis of Patients Referred for Surgery

<table>
<thead>
<tr>
<th>No.</th>
<th>Av. age</th>
<th>Failure</th>
<th>Size</th>
<th>Av. Press. mm. Hg</th>
<th>Pathology</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>M</td>
<td>S</td>
<td>PA</td>
<td>PC</td>
</tr>
<tr>
<td>6</td>
<td>47</td>
<td>100%</td>
<td>17</td>
<td>83</td>
<td>74</td>
<td>28</td>
</tr>
<tr>
<td>8</td>
<td>43</td>
<td>100%</td>
<td>12</td>
<td>88</td>
<td>76</td>
<td>37</td>
</tr>
<tr>
<td>4</td>
<td>34</td>
<td>100%</td>
<td>25</td>
<td>75</td>
<td>40</td>
<td>28</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>23</td>
<td>39</td>
<td>34%</td>
<td>78</td>
<td>22</td>
<td>56</td>
<td>24</td>
</tr>
</tbody>
</table>

*Autopsies not obtained on two patients. Two valves replaced with artificial valves.
†M, moderate; S, severe; D, destroyed; C, corrected.

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Until then he had been doing well. Of the surviving 20 patients, 20 per cent have had fairly good results. All had grade-IV cardiomegaly. In three, subsequent stretching of the surgical correction by hypertrophied myocardium caused the regurgitation to recur. Subsequent modifications of the operative technic have largely solved this complication. Earlier intervention in the future before grade-IV cardiomegaly develops, may also eliminate this problem. A possible endocarditis in a fourth patient may have compromised the surgical correction. Eighty per cent of the surviving patients have continued to do well from 6 months to 3½ years following surgery.

**Predominant Regurgitation**

There was an operative mortality of 12 per cent, due to early technical complications, incomplete surgical correction, and myocardial failure. One patient died 6 months postoperatively from myocardial failure. Of the 29 surviving patients, 20 per cent had fair response. Failure of greater benefit was due to poor myocardial reserve or to incomplete surgical correction. Greater benefit may be forthcoming in the future if valvular replacement proves effective. Eighty per cent of the patients received considerable benefit.

**Predominant Stenosis**

There was an operative mortality of 10 per cent. There were no intervening deaths during this 3½-year period. All surviving patients continued to do well during this period of evaluation.

A better understanding of the results of surgery in the treatment of mitral regurgitation can be obtained by an analysis of 41 consecutive patients being referred for operation over a 12-month period. These were patients who had been evaluated 2 or 3 months previously and who were considered at that time to be suitable candidates for surgery. They can be classified into four groups: Group I, six patients admitted to the hospital in the hope of surgical relief...
who deteriorated and died prior to surgery; Group II, eight patients who underwent surgery but failed to survive, largely due to insufficient myocardial reserve; Group III, four patients who obtained only partial benefit from surgery; and Group IV, 23 patients who were significantly improved following surgery (table 2).

It is readily apparent from table 2 that the patients who died immediately prior to and following surgery were the ones most severely disabled from their disease and who had greater degrees of cardiac failure, cardiomegaly, altered hemodynamics, and pulmonary vascular sclerosis. They were also in an older age group and had a higher incidence of destroyed valves with coexistent artery disease.

During this 12-month period the preoperative mortality was almost as high as the postoperative mortality. The operative mortality would have been lower had we not chosen to proceed with operation in four of the five patients with destroyed valves who were known preoperatively to have extensive calcification of their mitral valves. The higher operative mortality in obviously high-risk patients should not deter physicians from recommending operation in good-risk patients who would have a much lower operative risk.

Results Based on Clinical Evaluation

Evaluation of results in patients surviving operation as based on clinical appraisal 6 months to 3½ years postoperatively is presented in table 3. The benefit obtained in the majority of patients corrected by means available through the open approach has continued to warrant our enthusiasm. Two patients (3 per cent) have died during this 4-year period following surgery as noted previously. This small mortality is considerably less than that seen in unoperated patients. In the eleven (15 per cent) doing fair to fairly well, four had valvular mechanisms for which maximal correction could not be obtained. The other seven patients had large hearts with lessened degrees of myocardial reserve. Though the degree of improvement is certainly less than desired, the majority are out of failure and are able to perform most of their duties.

Eighty-two per cent of the patients are continuing to do well from 6 months to 3½ years postoperatively. The degree of improvement was proportionate to the degree of cardiopulmonary damage existing at the time of the operative procedure. Modifica-
Figure 6

A. Preoperative and postoperative phonocardiograms illustrating disappearance of the systolic murmur characteristic of mitral regurgitation following surgical correction in a patient with pure mitral regurgitation. B. Preoperative and postoperative phonocardiograms illustrating disappearance of murmurs following correction in a patient with combined but predominant stenosis.

The electrocardiographic pattern closely parallels the roentgen findings; the greater the abnormality preoperatively, the greater the findings are postoperatively. Though 70 per cent of the patients with atrial fibrillation have converted to normal, there has been some reduction in heart size postoperatively, due largely to reduction of the cardiac dilatation. It is anticipated that the mass of myocardial hypertrophy existing over a number of years will decrease slowly with the lessened burden following correction.
been little change in the pattern of left ventricular overload.

**Murmurs**

The best method to correlate change before and after operation or improvement in the character of a murmur is by phonocardiographic examinations (fig. 6). The majority of patients with pure mitral regurgitation had either no systolic murmur or only a slightly detectable one in the immediate postoperative period. In 3 or 4 months a systolic murmur was evident in five patients with large hearts. Over two thirds of the patients had a definite change in the character of the murmur that suggested improvement.

<table>
<thead>
<tr>
<th>Type</th>
<th>No.</th>
<th>Hemodynamic</th>
<th>Clinical</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>No change</td>
<td>Fair</td>
</tr>
<tr>
<td>Pure regurgitation</td>
<td>10</td>
<td>4</td>
<td>1</td>
</tr>
<tr>
<td>Predominant regurgitation</td>
<td>9</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td></td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>Combined (predominant stenosis)</td>
<td>12</td>
<td>3</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td></td>
<td>6</td>
<td>6</td>
</tr>
</tbody>
</table>

*Figure 7*

Demonstrates good roentgen and hemodynamic improvement in a patient with pure mitral regurgitation.
Demonstrates excellent roentgen and hemodynamic improvement in a patient with pure mitral regurgitation.

In the other third the murmur was only questionably detectable. The presence and character of the murmur are largely correlated with the degree of cardiomegaly. Murmurs are more likely to be absent or markedly diminished in those patients with smaller hearts.

Approximately 10 per cent of the patients had a febrile episode postoperatively. Though the etiology could not be determined with certainty, bacterial endocarditis was suspected. The recurrence of murmurs occasionally followed this complication. The majority of patients with combined but predominant mitral regurgitation had persistence of the murmur, though markedly changed in character. Less change in the character of the murmur was noted in patients with valvular calcification. In contrast, the majority of the patients with combined regurgitation but predominant stenosis had marked improvement in the character of the murmur, and in many instances the murmurs were hardly detectable.

Hemodynamic Findings

Thirty-one patients were studied by catheterization after operation (table 4). Their findings have fallen into three groups: (1) those with no significant hemodynamic improvement, (2) those with significant improvement, and (3) those that returned to essentially normal hemodynamic levels.

Pure Regurgitation

There were four patients in the first group, all of whom had severe cardiomegaly. In two instances the murmur indicative of mitral regurgitation after 1 to 2 years was still absent but catheterization studies showed no reduction in either pulmonary artery or
wedge pressures. One of these patients had only fair clinical improvement. The other three had considerable improvement over preoperative status.

One patient had both clinical and hemodynamic improvement. As demonstrated in figure 7 the resting pulmonary artery pressures, as well as the pulmonary wedge pressures, returned to normal but the pulmonary artery pressure with exercise was still elevated, which suggested that significant pulmonary vascular sclerosis was still present 16 months postoperatively.

Five patients had returned to normal pressures (fig. 8). These patients had smaller hearts than those in the above two categories.

**Predominant Regurgitation**

Nine patients had postoperative catheterization studies. In three there was no significant reduction in hemodynamic abnormalities, although clinically they have done well. Six patients had significant reduction in catheterization abnormalities. It may be that these studies performed on the average of about 1 year following surgical correction were performed too soon to allow pathologic changes to be reversed. This was suggested by one patient having studies performed 6 months and again 1 year following correction. The preoperative pulmonary artery pressures were 80/35 mm. of Hg, with a wedge pressure of 56/27 mm. of Hg. Six months postoperatively the pulmonary artery pressures had reduced to 67/30 mm. of Hg, and the wedge pressure to 40/20 mm. of Hg. At the end of a year the pulmonary artery pressure had fallen to 55/15 mm. of Hg.

**Predominant Stenosis**

All of the patients with predominant stenosis but with some element of regurgita-
tion had both clinical and hemodynamic improvement (fig. 9).

Discussion

It is still too soon to present a valid opinion as to the lasting benefit of the original correction of mitral regurgitation. The benefit derived, however, in the majority of patients during this 3½ year period lends enthusiasm to the surgical approach to this otherwise progressively deteriorating disease. Further improvement in surgical technic will undoubtedly add to the effectiveness of the procedure. An analysis of the patients and their response to surgical therapy suggests that benefit derived is largely related to the status of the myocardium at the time of the surgical intervention and that greater help could have been provided if the operative correction had been performed when it became obvious that the pathologic process was progressive, rather than delaying until it was quite apparent that surgical help was mandatory.

On Permanent Patency of the Mouth of the Aorta, or Inadequacy of the Aortic Valves

By DOMINIC JOHN CORRIGAN, M.D.

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... In the perfect state of the valvular apparatus at the mouth of the aorta, the valves support by intervals the column of blood in the aorta, and the heart with its ordinary complement of fibre and of muscular strength, is with this assistance competent to the office it has to perform. But when, in consequence of a deficiency in the valvular apparatus, the heart does not receive its due share of assistance from these valves, and is obliged to perform not only its own function of propelling the blood, but has in addition to support after each contraction a portion of that weight of blood which should then be wholly supported by the valves, it is no longer in its ordinary state equal to the task imposed upon it. In such circumstances, nature, to enable the heart to perform the additional labour thrown on it, increases its strength by an addition of muscular fibre, and the heart thus becomes hypertrophied, in accordance with the general law, that muscular fibres become thickened and strengthened when there is additional power required from it. Is this hypertrophy disease, or is it a wise provision of nature, by which the organ is thus made equal to the increased labour it has to perform? On the answer depends the treatment to be adopted; and on this there is no room for hesitation. A heart of ordinary strength could not, under the circumstances, carry on the circulation; and nature then wisely endows the heart with the requisite degree of strength. It is at once obvious that to interfere with this wise provision of nature, to diminish the strength of the heart, or, if we choose other words, to direct, according to the advice of Lænnee, Bertin, etc. our measures against the hypertrophy of the organ, is to deprive the system of the only power which enables the heart to carry on the circulation.
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