Results of the Surgical Treatment for Mitral Stenosis

Analysis of One Hundred Consecutive Cases

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An analysis of the first 100 consecutive cases of mitral stenosis treated by commissurotomy is presented. Frank hemoptysis was noted in 25 individuals and systemic arterial emboli in 22 others; no recurrence of these complications was found one to three and one-half years after surgery. "Pure" mitral stenosis was present in 64, and an associated insufficiency in 27; in nine others there was an associated aortic insufficiency. A sinus rhythm changed to auricular fibrillation after surgery in 15; in nine the change was permanent. In one instance auricular fibrillation changed to sinus rhythm postoperatively. A rough correlation was found between the extremes of pulmonary artery systolic pressure and the clinical, roentgen and electrocardiographic findings. Calcium was found in the mitral valves in 52 patients and did not, per se, predicate a poor functional result. No instance of rheumatic fever occurred postoperatively, even though 14 of 78 biopsied auricular appendages indicated rheumatic infection. Functional improvement occurred in 78 patients, and there were nine unimproved. There were 11 deaths due to surgery and two who died during this three-year follow-up from intercurrent infection.

FOR CENTURIES the therapeutic problems of heart disease both valvular and myocardial have been entirely in the province of the internist. With the recent development of intracardiac surgical procedures a major adjunct in the treatment of mitral stenosis has become available. Least one be tempted to suspect that the surgical approach to the problem of mitral stenosis has been entirely the concept of the surgeon one has only to recall that an internist, Brunton,1 in 1902, was the first to propose direct surgical reconstruction of the valve. Forty-six years elapsed before the proper and successful application of Brunton's principle was consummated in 1948.2-3 The purpose of this publication is to present complete data pertaining to our first 100 consecutive commissurotomies for mitral stenosis. Previous communications4-7 have reviewed the painstaking and at times fruitless efforts of many investigators.8-15 These reports have also detailed the pathologic and physiologic considerations of the valvular deformity, and the technical aspects of its surgical correction have been outlined before this association.4-7 It is, therefore, sufficient to state here that mitral commissurotomy is a procedure in which the individual anatomic leaflets of the stenotic mitral valve are surgically separated (fig. 1). By incising the angles or commissures of the mitral slit a considerable degree of valve function can be re-established without the production of additional significant mitral insufficiency (fig. 2). It is to be noted that no valve tissue is removed, thus allowing the liberated although thickened and deformed valve leaflets

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RESULTS OF SURGICAL TREATMENT FOR MITRAL STENOSIS

FIG. 1. Diagrammatic representation of the operative approach and technic of commissurotomy for mitral stenosis.

FIG. 2. Diagrammatic representation of the stenotic mitral cone as viewed from the left ventricle. (a) Line of incision in the anterolateral and posteromedial commissures. (b) The open valve cone during ventricular diastole. (c) Approximation of valve leaflets during ventricular systole minimizing regurgitation.

to open during ventricular diastole and approximate during ventricular systole.

ANALYSIS OF CASES

One hundred cases of mitral stenosis were treated by commissurotomy from March 22, 1948 to November 14, 1950. Of these, 64 were female and 36 were male. The youngest was age 17 and the oldest age 53. Most of these patients were far advanced in the course of their disease and represented the late or terminal stages of mitral stenosis. They have been grouped according to our previously published classification as follows:16

<table>
<thead>
<tr>
<th>Stages of Mitral Stenosis</th>
<th>Cases</th>
<th>Deaths</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Asymptomatic</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>2. Statically incapacitating</td>
<td>6</td>
<td>0</td>
</tr>
<tr>
<td>3. Progressively incapacitating</td>
<td>63</td>
<td>7 (11%)</td>
</tr>
<tr>
<td>4. Terminally incapacitating</td>
<td>29</td>
<td>4 (14%)</td>
</tr>
<tr>
<td>5. Irreversible</td>
<td>2</td>
<td>2 (nonsurgical late deaths)</td>
</tr>
</tbody>
</table>

This classification has been prepared in an effort to combine a functional and therapeutic yardstick for evaluation of the patient for surgery. Stage one includes those with the auscultatory findings of mitral stenosis but who as yet have no symptoms. Cases in stage two have progressed to the point where symptoms under physical activity have developed, but the patient living within his own limitations remains on an even plateau. Stage three, the largest group, and one encompassing many variables includes those who despite the best medical therapy are losing ground. Stage four, terminally incapacitating, includes those patients in whom there is constant evidence of congestive failure even with limited physical activity. Most of these can be rendered relatively free of their accumulating tissue fluid only by the strictest of medical regime. A certain small percentage of those in this group will ultimately prove after surgery to have been in stage five and to have had irreversible changes. As yet, it has been impossible to routinely separate patients in these two stages by clini-
cal and physiologic methods, hence, we reserve stage five to classify those who, despite a techni-
cally adequate commissurotomy, receive no improvement.

Status Prior to Admission

Progressive dyspnea and fatigue were present in varying degree in each of the 100 cases. In addition to this evidence of cardiopulmonary disability, 32 had already progressed to the stage of right heart (congestive) failure as evidenced by one or more episodes of hepatomegaly, ascites and peripheral edema. Twenty-five had had one or more attacks of frank hemoptysis, in several instances to an almost exsanguinating degree. This does not include the many who had expectorated pinkish frothy material during bouts of pulmonary edema.

In 22, cerebral, visceral or peripheral emboli had occurred on one or more occasions. Of these, 19 had auricular fibrillation, three had sinus rhythm, and all had a history of one or more attacks of congestive failure. Interestingly, 10 of these 22 cases had gross thrombi in the left auricular appendage at operation. It is not unusual for the pathologist to report the presence of a small thrombus deep in the trabeculae of the excised appendage which was not noted during the commissurotomy. In one instance embolic episodes were appearing with such frequency that commissurotomy with ligation of the left auricular appendage was urgent.

Autopsy reports of the incidence of thrombi confined solely to the left auricular appendage vary. Graef and co-workers found 47.4 per cent of 19 cases had thrombi limited to the left auricular appendage. Soderstrom found left atrial mural thrombi in 50 cases of rheumatic heart disease. Mural thrombi were found in the left auricular appendage in 29 instances and on the main left atrial wall in 25 instances. No statement was made as to the number of cases in which thrombi in these two sites coexisted. Jordan and co-workers, in a clinical and pathologic analysis of 51 cases of mitral stenosis, found 20 (47.6 per cent) of 42 cases with the thrombi limited to the left auricular appendage. In 22 (54.6 per cent) of these 42 cases, the thrombi in the left side of the heart were situated on the main atrial wall, in the auricular appendage and on the main atrial wall together, or in the left ventricle.

There have been no recurrent embolic episodes in any of these 22 cases (longest three years). It has been previously emphasized that ligation of the base of the left auricular appendage was responsible for this result. However, relief of stasis in the left atrium may be a major factor in preventing these recurrences.

Status Upon Admission (table 1)

In the initial phase of this surgical program every attempt was made to select cases of relatively “pure” mitral stenosis to insure a proper base line for evaluation of the surgically

<table>
<thead>
<tr>
<th>Table 1.—Admission Findings</th>
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</thead>
<tbody>
<tr>
<td>1. “Pure” mitral stenosis</td>
</tr>
<tr>
<td>2. Mitral stenosis with insufficiency</td>
</tr>
<tr>
<td>3. Mitral stenosis with insufficiency and aortic insufficiency</td>
</tr>
<tr>
<td>4. Mitral stenosis and aortic insufficiency</td>
</tr>
<tr>
<td>5. Left ventricular enlargement (x-ray). (Minimal to moderate)</td>
</tr>
<tr>
<td>6. Arrhythmias</td>
</tr>
<tr>
<td>7. Congestive Heart Failure</td>
</tr>
<tr>
<td>8. Electrocardiogram: RAD</td>
</tr>
<tr>
<td>NAD</td>
</tr>
<tr>
<td>LAD</td>
</tr>
</tbody>
</table>

treated stenotic valve alone. It has since become apparent that the auscultatory findings of a minimal systolic mitral and/or aortic systolic or diastolic murmurs in addition to the classic mitral diastolic murmur may be of little consequence provided mitral stenosis is the predominant factor in the production of the patient’s disabling symptoms. For this reason a number of cases were originally deferred for surgery until the eventual picture became clear. Sixty-four cases were classified as “pure” mitral stenosis in that there were no murmurs other than a diastolic rumble at the apex. This diagnosis was corroborated at the time of surgery when no regurgitant jet could be felt by the exploring finger. In 27 cases, there was an associated minimal (grade II systolic murmur) mitral insufficiency, and in two others there was aortic insufficiency of minimal degree. In seven individuals a mitral
diastolic rumble and an aortic diastolic murmur were the only murmurs heard.

In selection of cases for surgery, it is obvious that the careful auscultatory examination by an experienced cardiologist is paramount. The recognition of a typical uncomplicated mitral stenosis by stethoscopic methods usually presents no problem. The difficulty does not lie with this type of individual but rather with the patient who presents evidence of coexisting mitral stenosis and insufficiency, or multivalvular disease. Here, one must decide whether the mitral stenosis is the predominant lesion. Expert fluoroscopic and radiologic evaluation, with emphasis on individual chamber enlargement can be of inestimable value in this respect.

Eighty-eight of the cases presented a normal left ventricle, and in 12 there was minimal to moderate enlargement. In these 12, mitral insufficiency or associated aortic valvular disease was dynamically expressing its presence by enlargement of the left ventricle, a finding which we have come to regard as evidence tending to contraindicate surgery. Slight enlargement of the left ventricle due to mitral insufficiency is not an absolute contraindication but suggests the possibility of a more equivocal surgical result. If left ventricular enlargement is due to an aortic valvular lesion, commissurotomy would seem to be inadvisable at this time. The recognition of minor degrees of left ventricular enlargement in the presence of a markedly enlarged right ventricle may well be perplexing. Enlargement of all chambers of the heart is an absolute contraindication.

Auricular fibrillation, per se, is not a contraindication to surgery unless the ventricular rate is entirely uncontrollable. Fifty-seven patients had auricular fibrillation, and the results in these compared favorably with the results in those who had sinus rhythm.

Eighteen patients presented themselves in various stages of congestive heart failure although others were known to have been in this state prior to admission. Failure was controlled before surgery in all but four in whom failure had existed for many years. With the exception of minimal evidences these too eventually responded. The period of medical preparation of patients in this status was more prolonged, and in several instances six weeks or more were required. Some of the more dramatic results were achieved in this group after a prolonged period of convalescence.

The importance of hypertension of the pulmonary circulation in the natural history of mitral stenosis has been appreciated for many years. Such classic symptoms as dyspnea, orthopnea and hemoptysis have been thought to be indications of increased pressure in the pulmonary vascular bed. The roentgenologist and pathologist have supported the clinician's opinion of the importance of pulmonary hypertension by describing hypertrophy of the right ventricle, marked enlargement of the left atrium and pulmonary artery, and sclerotic changes in the large and small pulmonary arteries.

In patients with mitral stenosis, right heart catheterizations have recorded high systolic pressures in the right ventricle. Borden and co-workers found a rough correlation between the degree of pulmonary artery hypertension and the clinical manifestations. Comparable findings and correlations were observed in 72 of our 100 patients, in whom catheterization of the right heart was done. The upper limits of the pulmonary artery pressure found in this laboratory in normal subjects is 30/10 mm. Hg. As might be expected the higher the pressure in the pulmonary artery the closer the correlation with the clinical and roentgen findings. Usually a systolic pulmonary artery pressure of 50 mm. Hg or greater was associated with a history of prolonged functional disability, episodes of acute pulmonary congestion, hemoptysis or congestive failure. Such pressures, also, were commonly associated with fluoroscopic and x-ray evidence of marked left atrial and right ventricular enlargement and dilated pulmonary arteries. Electrocardiograms in about 75 per cent of this group showed right axis deviation (plus 90 degrees or more), thus reflecting somewhat the degree of pulmonary hypertension.

Chart 1 presents the results of commissurotomy upon the pulmonary arterial pressure. Seventy-two patients were catheterized before
and a number at 1, 6, 12 and 23 months after surgery.

Nineteen had pressures varying from 100 systolic, 35 diastolic to 148 systolic, 61 diastolic, the mean pressure ranging from 57 to 90; the average figures were 117 systolic and 50 diastolic, with a mean of 77. In all instances there was a drop in pressure after surgery, the average drop being 43 mm. Hg systolic, and 21 diastolic and 33 mean.

**Chart 1.**—The Results of Commissurotomy on Pulmonary Arterial Pressure 1 to 23 Months Postoperative

<table>
<thead>
<tr>
<th>Pressure (mm. Hg)</th>
<th>Cases</th>
<th>Systolic (mm. Hg)</th>
<th>Diastolic (mm. Hg)</th>
<th>Mean (mm. Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>100—150</td>
<td>19</td>
<td>117 (110—148)</td>
<td>74 (36—101)</td>
<td>43—F (8—112)</td>
</tr>
<tr>
<td>80—100</td>
<td>9</td>
<td>88 (81—96)</td>
<td>56 (48—76)</td>
<td>32—F (9—48)</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>81</td>
<td>121</td>
<td>40—R</td>
</tr>
<tr>
<td>60—80</td>
<td>8</td>
<td>60 (60—78)</td>
<td>51 (35—75)</td>
<td>18—F (3—31)</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>64 (60—68)</td>
<td>78 (66—89)</td>
<td>14—R (6—21)</td>
</tr>
<tr>
<td>40—60</td>
<td>17</td>
<td>51 (40—55)</td>
<td>40 (28—50)</td>
<td>11—F (2—17)</td>
</tr>
<tr>
<td></td>
<td>5</td>
<td>49 (40—54)</td>
<td>64 (38—70)</td>
<td>15—R (3—30)</td>
</tr>
<tr>
<td>20—40</td>
<td>11</td>
<td>32 (22—38)</td>
<td>28 (18—42)</td>
<td>plus or minus</td>
</tr>
<tr>
<td>Total...</td>
<td>72</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

There were 10 instances of systolic pressure between 80 to 100 mm. Hg. In nine of these the average drop in pressure after surgery was 32 systolic, 12 diastolic with a range of 9 to 48 systolic and 0 to 21 diastolic, the drop in the average mean pressure was 28. In one there was an increase in the pulmonary systolic pressure of 40 mm. Hg which represented an increase from 81 to 121 mm. Hg and was secondary to a severe mitral insufficiency produced at surgery. Interestingly, this marked diastolic in eight. Two of these 10 had an increase in the average systolic pressure from 64 to 78 mm. Hg. However, the clinical course, in both of these two patients, was considered to be markedly improved; thus, the increase in pressure did not indicate a poor clinical result.

Twenty-two had pressures varying between 40 to 55 systolic and 13 to 36 diastolic. Seventeen of these had a drop in the average systolic pulmonary artery pressure of 11 points, and five had an increase in pressure of 15. The
latter five showed improvement clinically. Again, as noted above, the clinical status was not closely correlated with the pulmonary pressure.

In 11 individuals, pressures varying between 22 systolic, 6 diastolic and 38 systolic, 12 diastolic were recorded. There was no significant rise or fall in the pressures postoperatively. In this group we found minimal symptoms and no instance of hemoptysis.

There is a wide range of pressures, probably between 40 to 80 mm. Hg systolic which cannot be closely correlated with the clinical state. These statements are speculative, but a review of our results indicates that only the extreme ranges can be correlated with the functional state. In other words, people with pressures of 80 mm. Hg or above usually have definite evidence of functional incapacity. In this range one expects orthopnea, nocturnal dyspnea, hemoptysis and congestive heart failure. Whereas, those individuals with pressures of 40 mm. Hg systolic or less usually have minimal symptoms, although not infrequently the complications associated with mitral stenosis such as auricular fibrillation and embolic phenomena are found.

It must be borne in mind that most of the above postoperative pressure readings were obtained within a month or two after surgery at a time when maximum changes could hardly be expected. Five patients have now returned for recatheterization after the lapse of over a year. In one patient a pressure of 57 systolic, 24 diastolic fell to 40 systolic, 19 diastolic, within one year. A dramatic change was noted within 15 months in one instance by a change of pressure from 148 systolic, 56 diastolic, to 36 systolic, 15 diastolic, a drop of 112 mm. Hg systolic. Initial pressures in one patient of 90 systolic, 40 diastolic dropped to 60 systolic, 35 diastolic within 21 months. Pulmonary arterial pressures of 48 systolic, 16 diastolic were found to be 27 systolic, 10 diastolic, 23 months later. In another individual right ventricular pressures only were obtained. The initial systolic pressure was 68 mm. Hg; six months later it had risen to 89 mm. Hg. The last pressures obtained were 16 months after the initial ones, when a systolic of 53 mm. Hg was recorded.

Correlation of Clinical Findings with Pulmonary Pressures

(a) Hemoptysis. Twenty-five patients had frank hemoptysis; 12 of these patients had pressures ranging between 100 to 148 mm. Hg systolic; six between 80 to 100 mm. Hg; four between 60 to 80 mm. Hg.; and three occurred between 40 to 60 mm. Hg.

(b) Congestive Heart Failure. There were 18 instances of congestive heart failure. Nine had pressures greater than 100 mm. Hg systolic; four varied from 80 to 100 mm. Hg; three fell between 60 to 80 mm. Hg; one between 40 to 60 mm. Hg; and one had a pressure of 34 mm. Hg systolic. This latter case was one of far advanced failure in a 47 year old white female with aneurysmal dilatation of the left atrium and a tight mitral stenosis, who had been at complete bed rest for one year. We have noted an occasional relatively normal pulmonary artery pressure in these individuals with congestive failure who have been at bed rest for a long period even though the mitral orifice is found to be the size of the head of a paper match at operation.

(c) Graham Steel Murmur. There were six individuals in whom a soft diastolic murmur was localized in the second left intercostal space. There was no change in the quality or intensity with change in position or respiration, and no other murmur could be heard at the base. It was thought that we were probably dealing with a Graham Steel murmur. At operation the pulmonary arteries were enormously dilated (three times the normal), and tense. These findings and the subsequent disappearance of the murmur after surgery seemed to confirm the original diagnosis.

(d) Arrhythmias. There was no correlation between the presence of auricular fibrillation or supraventricular tachycardia and pulmonary artery pressures.

(e) Roentgenologic and Electrocardiographic Correlations. A mild hypertension usually was associated with slight enlargement of the left atrium and straightening of the left border of
the heart. However, no definitive correlation between the size of the left atrium or right ventricle and the degree of pulmonary hypertension could be made.

Right axis deviation (plus 90 degrees or more) was observed in 77 patients, most of whom had pressures of 50 mm. Hg systolic or greater. However, no exact correlation between the pulmonary arterial pressure and the degree of the axis shift could be made.

The prediction of the x-ray and electrocardiographic findings can usually be made more precisely from the physical examination and a history indicative of sustained pulmonary hypertension, than from the pulmonary artery pressure per se.

Table 2.—Operative Findings

<table>
<thead>
<tr>
<th>Category</th>
<th>Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Size of valve orifice (estimated)</td>
<td></td>
</tr>
<tr>
<td>(a) 0.5 cm. (no regurgitant jet)</td>
<td>72</td>
</tr>
<tr>
<td>(b) 0.5 cm. up to 1.5 cm. (regurgitant jet in</td>
<td></td>
</tr>
<tr>
<td>14)</td>
<td>28</td>
</tr>
<tr>
<td>(c) Orifice after commissurotomy</td>
<td></td>
</tr>
<tr>
<td>2.5–3.5 cm.</td>
<td>86</td>
</tr>
<tr>
<td>1.5–2.5 cm.</td>
<td>5</td>
</tr>
<tr>
<td>1.5 cm.</td>
<td>9</td>
</tr>
<tr>
<td>2. Calcification of valve</td>
<td>52</td>
</tr>
<tr>
<td>3. Thrombi</td>
<td>15</td>
</tr>
</tbody>
</table>

The electrocardiogram frequently aids in determining the candidacy of these patients, especially when there is more than one valve defect. Evidence of right ventricular enlargement suggests that the predominant lesion is one of mitral stenosis. It is to be noted that a right axis deviation (plus 90 degrees or more) was present in 77 cases, a normal axis in 21, but more particularly that in only two instances was there evidence of a left axis shift. These latter two occurred in patients with multivalvular lesions and at operation both had major mitral regurgitation. Finding a left axis shift predicates that other factors are at play and a careful re-evaluation must follow.

Operative Findings (table 2)

Seldom in the past has there been the opportunity to examine at first hand the nature of living valvular pathology in situ. It has been a source of wonder to the authors to find the average stenotic valve in relatively good condition when contrasted to the incapacitation of the patient.

1. The size of the valve orifice as estimated by the tip of the exploring index finger in 72 of the cases was approximately 0.5 cm. in diameter or, in more readily appreciated terms, the size of the head of a paper match stick (fig. 3). The cusp margins were thickened, and fibrotic with fixation of the periorificial ring. Nevertheless, just enough pliability remained so that no regurgitant jet of blood could be detected with ventricular systole. In the remaining 28, the orifice was somewhat larger, being 0.5 cm. to 1.5 cm. (one to three match heads) and in 14 of these a distinct regurgitant jet could be appreciated.

In contrast to the firmer cusp margins the thickened valveleaflets proper are considerably more flexible. Thus, the “mitral megaphone” (fig. 2a) with its ventricular mouth-piece rides back and forth as a complete cone in response to changes in pressure on its two sides, always maintaining its construction and unable to open or close at its orifice.

2. Calcification was present in some measure in 52 of the valves. The frequent site for such infiltration was on the anterior valve leaflet over its medial aspect—that portion nearest
the outflow tract of the left ventricle. Many of the valves were lightly flecked with tiny calcific beads along the cusp margins. In others the calcium extended back into the leaflets for varying distances at one or more points of the circumference. Even in these situations considerable mobility could be frequently restored by commissurotomy. In 10 cases calcium was present in larger quantity practically encircling the mitral orifice and appreciably limiting the reconstructive effect obtainable by incising the commissures. Three of these were essentially inoperable. Fortunately the anterolateral commissure is less frequently involved by calcium and separation at this point will improve the patient as a rule, regardless of the status of the remaining valve cone. It is to be emphasized, therefore, that the presence of calcium, per se, need not necessarily mitigate against a good functional result. Occasionally, without detectable calcium, the circumferential contraction of the atrioventricular ring together with extreme thickening of the valve cone and foreshortening of the chordae tendineae result in such fixation that commissurotomy is unavailing.

3. Gross evidence of thrombosis was encountered in the left auricular appendage of 15 patients. Arterial embolization was associated in 10 of the 15 patients and auricular fibrillation in 14. Five patients with thrombosis and auricular fibrillation had no embolic episodes. Interestingly, there was pathologic evidence of rheumatic activity in the excised appendages of four (27 per cent) of the 15 cases. The role such activity plays in thrombus formation in mitral stenosis may be significant.

Immediate Postoperative Status (table 3)

1. Chest Pain. (a) The usual thoracotomy pain, intercostal in type, is to be expected and was experienced by all patients. Its duration is usually for 10 days and is relieved by sedatives. (b) Pleuropericardial pain may be a striking sequel to this surgery and is much more severe and persistent than that experienced by the average thoracic surgical patient. It occurs in about 30 per cent of cases and most frequently during the second postoperative week. However, it may occur sporadically and recurrently at any time during the ensuing six months. The pain is typical of that seen in acute pericarditis and similarly is aggravated by swallowing and change of position. The usual location of the pain is the lower substernal area either localized or radiating to the neck or back, or it may begin posteriorly. In approximately 5 per cent of these a small pleural effusion develops.

The importance of this pain lies in the fact of its persistence, severity, unpredictability and its lack of response to salicylates, antibiotics, and ordinary doses of narcotics. In one extreme instance this distress persisted daily for three months and precipitated right heart failure. Although this morbidity was usually long the patient has now been symptom free for two and one-half years and has returned to normal activity. An attempt has been made to anticipate the probable onset of this complication by investigating the patient's rheumatic state prior to surgery. Unfortunately we are unable to predict with any certainty such a complication, either from the history, physical examination or laboratory studies.

2. Pericardial Effusion. There were seven instances in which pericardial aspiration seemed desirable. This type of effusion is not associated with the usual signs and symptoms of cardiac tamponade. The exhibition of undue anxiety associated with a feeling of strangling and oppression should arouse suspicion. The apical rate may be normal or slightly elevated; slight orthopnea and hypopnea may develop with profuse perspiration and as a rule the systolic blood pressure is lowered. The jugular veins infrequently are distended. Although the physical findings are not typical for effusion, pericardiocentesis will yield 3 to 6 ounces of serosanguineous fluid often containing clotted blood. Even as the
fluid is withdrawn the patient experiences subjective relief. Repeated aspiration has not been necessary.

3. Arrhythmias. Auricular fibrillation developed postoperatively within the first four days in 15 individuals who had been in sinus rhythm prior to surgery. Six were transient and returned to a sinus mechanism without the use of quinidine, and nine became permanent.

One instance of auricular fibrillation reverted to sinus rhythm after surgery and remained so for seven months, when the patient died of streptococcal pneumonia with septicemia.

4. Postoperative Hemiplegia. Three patients awoke from anesthesia with an hemiplegia. In two of these no evidence of gross thrombosis had been observed in the left auricular appendage so that the origin of the embolus is unknown. Thrombotic material was present in the appendage of the third and presumably this was the embolic source. Immediate therapeutic stellate ganglion novacain blocks were done and repeated as indicated. Each made an uneventful recovery except for a minor motor weakness of one extremity.

5. Reactivation of Rheumatic Infection. There was no instance of an exacerbation of acute rheumatic fever following surgery. This was unexpected in view of the histologic evidence of the rheumatic state in 14 of 88 amputated appendages as disclosed by one of us (J. E. G.) (fig. 4). A higher incidence of rheumatic stigmata might be expected if the myocardium proper also had been examined. The previously described postoperative pleuropéricardial episodes have been considered by the authors to be possible manifestations of smoldering rheumatic activity but direct evidence is lacking. Further observation will be necessary before the role of rheumatic infection as influenced by surgery can be evaluated.

6. Auscultatory changes. A loud pericardial friction rub was almost invariably heard immediately after surgery, disappearing slowly within 10 days.

The mitral diastolic rumble was usually greatly diminished in intensity and during the first 7 to 10 days may be inaudible. Less frequently, one may note an increase in the intensity of the diastolic rumble, probably due to increase in the rate of blood flow after commissurotomy.

In almost all of the cases a mitral systolic murmur was produced. Its usual intensity may be graded as I to II and this murmur usually persists. It is felt that as a rule this murmur does not represent a significant degree of insufficiency.

The sharp and accentuated mitral first sound usually remains unchanged in quality even though the diastolic rumble may disappear.

This sound appears to be the least affected by mitral valve surgery. The pulmonic second sound frequently remains accentuated, even though the pressure in the pulmonary artery has been reduced.

Thus, to summarize, the murmurs may change materially but the individual heart sounds may not.

**Present Functional Status (table 4)**

There is no more difficult a task in the compilation of clinical data than to reach agreement on suitable and self explanatory criteria for the evaluation of surgical results. This is especially true when the pathologic and physiologic patterns of a disease entity, such as mitral
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stenosis, are so varied. The common ground for comparison would seem to be the functional

Table 4.--Present Functional Status (one to three and one-half year follow-up)

<table>
<thead>
<tr>
<th>Status</th>
<th>Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>Excellent</td>
<td>32</td>
</tr>
<tr>
<td>Improved</td>
<td>46</td>
</tr>
<tr>
<td>Unimproved</td>
<td>9 (2 have recently died)</td>
</tr>
<tr>
<td>Mortality</td>
<td>11</td>
</tr>
<tr>
<td>(a) Deaths not due to surgery</td>
<td>2</td>
</tr>
</tbody>
</table>

status of the patient observed one or more years after surgery.

Table 4 summarizes the results as obtained in this series. Thirty-two patients are classified as being excellent in that they have been restored to a normal productive life, enjoying all normal activities, without obvious limitation, while no longer requiring cardiac medication. Forty-six patients are considered to be objectively (fig. 5) and subjectively improved as evidenced by their return to almost normal life and activity within their own particular limitations determined on a trial and error basis. Some of this group still require medication because of the demands of their more greatly enlarged sphere of activity. A few so incapacitated as to be placed in class IV, according to the criteria of the New York Heart Association, have now become so amenable to therapy that reasonable activity requires digitalization only. The range of improvement in this group varies widely with the condition of the valve found at surgery and the degree to which valve function could

be restored. Their progressive downhill course has been abruptly terminated or reversed, some to regain a high level of efficiency, and others to remain on an improved plateau.

Nine have been essentially unimproved and two factors were responsible for this. There were three with inoperable valves and six in whom significant mitral regurgitation was produced (fig. 6).

There were 11 deaths directly attributable to surgical technic. The first of these was the first case in this series. Postmortem examina-
tion revealed that the lateral commissure had not been opened more than a few millimeters so that death was occasioned by unrelieved mitral stenosis with the superimposed trauma of surgery. The second death occurred early in the series before the appendage was being ligated at its base. Thrombosis occurred in the appendageal lumen providing the source for subsequent fatal cerebral embolus occurring on the seventh postoperative day in a patient who had had a good technical commissurotomy.

In two, hemorrhage occurred from the appendage—one 24 hours after surgery and the other during an ill-advised attempt to remove an adherent thrombus. One case remained in shock with peripheral vascular collapse for four days, cause unknown. In the remaining six cases surgically produced severe mitral insufficiency caused death, which occurred either in the operating room or within the first postoperative month. Most of these were occasioned by the use of finger dilatation, the line of valvular separation occurring either in the wrong place or extending too far. For this reason we have always preferred the use of a specially devised guillotine knife (fig. 1) with which accurate and definitive incision in the commissures can more readily be controlled. A glance at figure 7 will reveal that our greatest mortality came at a time when the surgical technic was being developed. Certain of these deaths might have been obviated with our present experience.

There were four additional deaths for which surgery cannot be indicted. In two the selection of the cases was obviously poor since there was a marked degree of associated mitral

![Fig. 6. Preoperative (left) and postoperative (right) oblique films showing the rapid increase in size of the left ventricle by the inadvertent surgical production of significant mitral insufficiency.](http://circ.ahajournals.org/)

![Fig. 7. Mortality—First 100 consecutive cases of commissurotomy for mitral stenosis.](http://circ.ahajournals.org/)
insufficiency. Both died within seven months after surgery from unabated progression of their disease. A fulminating streptococcal pneumonia with septicemia was responsible for another death. The fourth died of an acute hemorrhagic pancreatitis.

**DISCUSSION AND CONCLUSION**

In the light of the foregoing presentation, the selection of patients for surgical intervention must necessarily be considered under seven major categories. These are outlined as follows:

1. History: (a) Early progressive cardiopulmonary dysfunction—ideal, (b) Marked dyspnea, hemoptyis, reversible congestive failure—acceptable.
2. Age—elastic range—physiologic rather than chronologic.
3. Valvular Defect: (a) “Pure” mitral stenosis—ideal, (b) Associated mitral insufficiency and/or aortic valve lesion in presence of normal left ventricle—acceptable.
4. X-ray: (a) Left atrium and right ventricle minimally enlarged—ideal, (b) Minimal left ventricular enlargement—acceptable.
5. Electrocardiogram: (a) Normal electrical axis or right ventricular strain—acceptable, (b) Left axis shift—never acceptable.
6. Functional Capacity: (a) Stage 2—statically incapacitating—ideal, (b) Stage 3—progressively incapacitating—acceptable, (c) Stages 4 and 5—debatable.
7. Complicating factors: (a) Arterial embolic episodes, and (b) Recurrent hemoptyis—both are acceptable, and may be urgent.

In conclusion, surgical reconstruction of the stenotic mitral valve is but a major adjunct in the continuing care of these rheumatic victims. Although such patients can and have been restored to a high level of efficiency and, as such, enjoy a more normal life, nevertheless they must always be considered as cardiac patients under the watchful care of their attending physicians just as the surgically treated tuberculous patient will require frequent observation, advice and management. With a greater understanding of the inherent nature of progressive mitral stenosis and the demonstration of a technically successful operation, both as to a lowering mortality rate and the satisfactory results obtained, the selection of patients at an early stage is of utmost importance.

**ADDENDUM**

The personal series of the authors has been extended to 274 cases as of May 1952. The operative mortality has been 5.8 per cent. Marked and sustained improvement has been present in 76 per cent of the patients.

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