Surgical Treatment of Mitral Insufficiency by Total Circumferential “Purse-String” Suture of the Mitral Ring

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A new surgical procedure for correction of mitral insufficiency is presented. The clinical and hemodynamic results in 12 cases are discussed.

SUCCESS in the surgical treatment of stenotic valvular disease of the heart has emphasized the urgency for the development of equally satisfactory methods for the correction of valvular insufficiencies. This need applies particularly to the mitral valve.

Although mitral stenosis is quite common, it is relatively less so in the “pure” state. Bland stated that of 730 cases of rheumatic heart disease followed at the Good Samaritan Hospital in Boston, 128 (18 per cent) had mitral stenosis, 115 (16 per cent) had mitral insufficiency, and 398 (55 per cent) had a combination involving both lesions.

The vast majority of patients undergoing surgery for mitral stenosis have some degree of well tolerated coexisting regurgitation. In a significant number of such cases the degree of regurgitation may be aggravated by surgery. In a few, serious regurgitation may be produced by a technically satisfactory commissurotomy. There are also many patients who cannot benefit from surgery for stenosis because of “too much” concomitant regurgitation. In these instances correction or control of the regurgitant factor is essential.

The incidence of mitral insufficiency appears to be quite high. Most writers agree that the initial valvular lesion of rheumatic fever is insufficiency.

Although the mortality of the acute rheumatic attack is relatively low, even in patients with definite carditis, as time goes on the toll is greater. DeGraff and Ligg follow 644 rheumatic patients until their death and noted that 75 per cent succumbed before the age of 40. The average case showed onset of the original attack at age 17, diminished cardiac reserve at 28, failure at 30, and terminated fatally at 33. The factors determining the cause of death in such cases are disputable although many authors agree that cardiac failure in patients with old rheumatic disease is often precipitated by rheumatic reactivation, or that failure does not appear in the absence of myocardial inflammation. On the other hand, the greatest number of deaths from rheumatic heart disease occur in patients in whom valvular lesions are present. Some authorities believe it is the valvular lesions that lead eventually to failure and death. It has been stated that the disturbances in circulatory dynamics, the symptomatology, and the type of heart failure are due to the valvular lesion rather than to the disease that produced it. The presence of active inflammation and the severity and extent of myocardial damage, and other factors as well, are, nevertheless, important in the production of failure by reducing the ability of the heart to tolerate the strain imposed by the valvular dysfunction.

The classic experiments of Wiggers and Feil elucidated the importance of a strong myocardium in mitral insufficiency and led to the conclusion that this lesion cannot be tolerated in the presence of a weak muscle. There is as yet no satisfactory experimental work on the chronic effects of the valvular lesion per se upon the heart. It is, however, well known that mitral insufficiency produced experimentally or in patients is followed by

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dilatation of the left side of the heart. This dilatation subsequently results in hypertrophy in order to maintain "compensation" for the overload added to the normal work requirements. The enlargement of the left chambers results in dilatation of the atrioventricular ring with consequent increase in the magnitude of the regurgitation (relative insufficiency). The progression of these phenomena sets up a vicious cycle that culminates in myocardial exhaustion. Although many have attempted to do so, the chronic effects of mitral insufficiency are yet to be demonstrated under controlled experimental conditions.

In the light of considerable experience in the surgical treatment of mitral stenosis, the mechanical function of the valve appears of major importance. Relief of the obstruction clearly renders great and lasting improvement in cases that not so long ago were thought to show the consequences of myocardial inflammation. Surgery has not altered the course of the latter aspect of the disease but commissurotomy has changed the balance of factors in the patient's favor.

These arguments form the basis for the authors' suggestion that there appear to be at least 2 general indications for the correction of mitral insufficiency. The first is in the patient with mitral stenosis in whom opening of the valve is at present contraindicated because of concomitant insufficiency or in whom commissurotomy has resulted in significant regurgitation. The second is in the patient with "pure" or predominant mitral insufficiency with or without demonstrable myocardial disease. Effective correction or amelioration of the valvular dysfunction, by relieving this source of myocardial overload, may result in significant or even marked improvement for these patients in whom death is so frequently inevitable despite the most assiduous medical management. Indeed, the more severe the myocardial impairment, the greater might be the urgency for the relief of the valvular defect.

To test these ideas it is first necessary to develop a safe technic for correcting the mechanical dysfunction. Many methods have been suggested that, in general, have been based on the principle of repair or replacement of the occluding elements of the valve. Techniques employing prosthetic devices, grafts of pericardium or veins or methods of suturing the leaflets have been tried. Although these procedures have been applied clinically in significant numbers of patients, usually with little or no preliminary experimental evaluation, no objective evidence of their efficacy has been shown. The results have been admittedly unsatisfactory and disappointing or have not been reported.

In previous communications the authors have suggested a new approach to the problem based on the principle of reducing the size of the orifice that the valve must occlude. This approach is attractive because it avoids intracardiac foreign bodies and utilizes fully
the available valvular elements. It does not entail damage to existent valvular structures and does not reduce the capacity of the effective atroventricular orifice. Objective evidence has been shown, for the first time, in experimental and clinical studies\textsuperscript{45, 46} that this method consistently and effectively corrects mitral regurgitation.

Following a preliminary publication of this approach a number of technics have been reported based on this principle.\textsuperscript{24, 40-42, 47} These technics are still too new and untried for critical evaluation of modifications of the method. On anatomic and physiologic grounds, after extensive laboratory experience with many modifications, the authors favor the total circumferential constriction of "mitral purse-string" because its effects are consistent experimentally and clinically. It makes possible accurate reduction of the ring to the optimum degree, and it provides permanent stabilization, thus preventing future dilatation in the event that regurgitation is not totally eliminated.*

The technic consists of placing an encircling suture around the mitral valve ring. The anatomic feasibility of the method has been clearly shown\textsuperscript{46} (fig. 1). The technical success of the operation depends on precision in its execution. An exact knowledge of the pertinent anatomy is required, as well as practice in animals and postmortem specimens. Once the technic is mastered, however, it is quite easy to perform with consistent accuracy. Relatively little intracardiac manipulation and no significant cardiac trauma are involved when the procedure is properly performed. No demonstrable damage to either the coronary circulation, the myocardium, the valve itself, or the cardiac conduction system has been noted when the technic is correctly performed in controlled experiments. Narrowing of the normal mitral annulus to the degree necessary to correct otherwise fatal experimental insufficiency in dogs has not been associated with the creation of stenosis and has not produced significant hemodynamic changes beyond those indicative of correction of the insufficiency.\textsuperscript{44}

**Case Reports**

The first 12 consecutive cases treated by this method are summarized here (tables 1–3) to emphasize (1) the clinical applicability of this method, (2) the immediate correction of regurgitation even in cases where annular dilatation was not the major valvular pathologic factor, (3) the improvement that follows correction of regurgitation even in far advanced stages of the disease or in the presence of probable recurrent rheumatic activity, and (4) the value of objective data in judging the result.

*Case 1*

R. M. (No. 164783). This 28-year-old woman was admitted to Presbyterian Hospital on January 12, 1955, complaining of shortness of breath on exertion. At age 16 she was told that she had a "leaky heart." One year prior to her present admission she was hospitalized elsewhere because of congestive heart failure. Six months prior to her present admission she began to have dyspnea. Symptoms progressed rapidly, with severe dyspnea, orthopnea, moderate ankle edema, inability to work, and almost complete inactivity for 3 months and sudden syncope while at rest 1 week before admission. She then improved considerably upon treatment with digitals and diuretics.

Physical examination revealed a buxom woman in no severe discomfort. The lungs were clear. A marked precordial pulsation was observed. The cardiac rhythm was totally irregular. There was a grade III to IV, rough mitral systolic murmur and a grade II to III mitral diastolic rumble. There was also a harsh, grade II systolic murmur heard over the aortic valve, transmitted to the neck. The mitral first sound and the pulmonic second sound were accentuated 2 plus. The liver was not palpable. There was no dependent edema.

Laboratory results were normal. Roentgenologic examination revealed clear lung fields and a large cardiac silhouette (fig. 2). The electrocardiogram showed atrial fibrillation and right ventricular hypertrophy. A phonocardiogram showed a prominent apical systolic murmur and no diastolic abnormalities. The second heart sound was accentuated. A brachial artery tracing was considered compatible with minimal aortic stenosis.

The patient was operated upon on January 20,
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1955. The lung was moderately turgid but expanded well. A systolic, prominent thrill was palpable over the lateral and posterior aspects of the left atrium. There were no thrills over the left ventricle or the aorta. The aortic valve ring was not abnormal to external examination. Intracardiac pressures were measured directly (table 2).

Digital intracardiac examination revealed a grade III regurgitant jet over the entire mitral valve orifice. The effective orifice admitted 2½ to 3 fingers,

| Table 1.—Pressures Obtained by Cardiac Catheterization |
|---|---|---|---|---|
| No. | Brachial artery | Right atrium | Right ventricle | Pulmonary artery |
| 1 | 125/75 (80)* | 25/14 (18) | 88/10 (40) | 80/47 (60) |
| 2 | 125/65 | 15/7 (11) | 30/0 (10) | 35/5 (24) |
| 3 | 8.5/2 | 75/1 (30) | 56/30 (40) | RPA 85/35 (55) |
| 4+ | 125/62 | 1 5/-3 | 20/-5 | 28/10 |
| 5 | 98/57 (67) | 1/-3 (-2) | 31/-6 (7) | 30/11 (20) |
| 6 | 10/6 | 48/0 | RPA 46/11 | 49/17 |
| 7 | 7/2 | 50/10 (18) | 55/20 (33) | (16) |

All pressures in mm. Hg.

* Figures in parenthesis indicate mean pressures.
+ Catheterization done elsewhere.
† Data not obtained due to technical difficulties.
§ Pulmonary venous or capillary pressure.

| Table 2.—Intracardiac Pressures Obtained During Surgery before and after Circumferential Suture |
|---|---|---|---|---|---|---|
| No. | Before | After | Left atrium | Left ventricle | Aorta | Pulmonary artery | Systemic artery |
| 1 | Before | 30/10 (17) | 108/-10 (25) | 110/55 (80) | — |
| 2 | After | 30/10 (17) | 108/-10 (25) | 110/55 (80) | — |
| 3 | Before | 19/6 (11) | 106/-3 (27) | — | — |
| 4 | After | 19/6 (11) | 106/-3 (27) | — | — |
| 5 | Before | 78/15 (35) | 110/10 (50) | — | 110/65 (84) | 105/95 |
| 6 | After | 78/15 (35) | 110/10 (50) | — | 110/65 (84) | 105/95 |
| 7 | Before | 17/7 (11) | 116/6 (54) | — | 102/58 (75) | 110/100 |
| 8 | After | 17/7 (11) | 116/6 (54) | — | 102/58 (75) | 110/100 |
| 9 | Before | 24/6 (14) | 112/2 (45) | — | — | — |
| 10 | After | 24/6 (14) | 112/2 (45) | — | — | — |
| 11 | Before | 22/11 (15) | 160/-3 (60) | — | — | — |
| 12 | After | 22/11 (15) | 160/-3 (60) | — | — | — |
| 13 | Before | 28/11 (17) | 155/-2 (55) | 130/65 (90) | 54/25 (37) | 130/15 |
| 14 | After | 28/11 (17) | 155/-2 (55) | 130/65 (90) | 54/25 (37) | 130/15 |
| 15 | Before | 18/6 (11) | 150/-2 (55) | 105/62 (80) | 54/26 (37) | 120/110 |
| 16 | After | 18/6 (11) | 150/-2 (55) | 105/62 (80) | 54/26 (37) | 120/110 |
| 17 | Before | 38/5 (19) | 102/-5 (38) | 110/80 (87) | 110/70 (82) | 110/80 |
| 18 | After | 38/5 (19) | 102/-5 (38) | 110/80 (87) | 110/70 (82) | 110/80 |
| 19 | Before | 27/0 | 80/-8 | — | 48/12 | 120/70 |
| 20 | After | 27/0 | 80/-8 | — | 48/12 | 120/70 |
| 21 | Before | 10/0.5 | 96/-4 | — | — | — |
| 22 | After | 10/0.5 | 96/-4 | — | — | — |
| 23 | Before | 20/-3 | 90/-8 | — | 25/12 | 110/90 |
| 24 | After | 20/-3 | 90/-8 | — | 25/12 | 110/90 |
| 25 | Before | 5/-4 | 92/-10 | — | 24/7 | 110/80 |
| 26 | After | 5/-4 | 92/-10 | — | 24/7 | 110/80 |
| 27 | Before | 48/13 | 128/0 | — | 120/60 | 120/80 |
| 28 | After | 48/13 | 128/0 | — | 120/60 | 120/80 |
| 29 | Before | 25/-7 | 112/0 | — | 94/44 | 140/80 |
| 30 | After | 25/-7 | 112/0 | — | 94/44 | 140/80 |
| 31 | Before | 24/14 | 115/0 | — | 33/10 | 90/80 |
| 32 | After | 24/14 | 115/0 | — | 33/10 | 90/80 |
| 33 | Before | 100/60 | 128/? | — | 76/3 | 105/60 |
| 34 | After | 100/60 | 128/? | — | 76/3 | 105/60 |
| 35 | Before | 47/-10 | 100/-8 | — | 30/0 | 90/70 |
| 36 | After | 47/-10 | 100/-8 | — | 30/0 | 90/70 |
| 37 | Before | 0/-10 | 100/-10 | — | 18/0 | 80/70 |
| 38 | After | 0/-10 | 100/-10 | — | 18/0 | 80/70 |
| 39 | Before | 29/7 | 138/-8 | 128/78 | 116/60 | 130/100 |
| 40 | After | 29/7 | 138/-8 | 128/78 | 116/60 | 130/100 |
| 41 | Before | 8/1 | 138/-2 | 132/84 | — | 130/100 |
| 42 | After | 8/1 | 138/-2 | 132/84 | — | 130/100 |
### Table 3—Pertinent Data in Twelve Cases

<table>
<thead>
<tr>
<th>Case</th>
<th>Operation date</th>
<th>Purse-string or purse-string and commissurotomy</th>
<th>Living survival time</th>
<th>Present status</th>
<th>Survival time before death</th>
<th>Cause of death</th>
<th>Pre-operative stage of disease</th>
<th>Rheumatic activity</th>
<th>Immediate hemodynamic result</th>
<th>Immediate clinical result</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1/20/55</td>
<td>P</td>
<td>1 year</td>
<td>Excellent</td>
<td>—</td>
<td>—</td>
<td>Far advanced</td>
<td>Terminal</td>
<td>Good</td>
<td>Excellent</td>
<td>Slight increase in intensity of systolic murmur</td>
</tr>
<tr>
<td>2</td>
<td>2/1/55</td>
<td>P</td>
<td>—</td>
<td>—</td>
<td>2 months</td>
<td>Advanced disease</td>
<td>Terminal</td>
<td>?</td>
<td>Excellent</td>
<td>Good</td>
<td>Died in congestive failure</td>
</tr>
<tr>
<td>3</td>
<td>2/2/55</td>
<td>P + C</td>
<td>1 year</td>
<td>Excellent</td>
<td>—</td>
<td>—</td>
<td>Far advanced</td>
<td>Terminal</td>
<td>?</td>
<td>Excellent</td>
<td>Slight increase in intensity of systolic murmur</td>
</tr>
<tr>
<td>4</td>
<td>3/1/55</td>
<td>P + C</td>
<td>—</td>
<td>—</td>
<td>3 weeks</td>
<td>Advanced multivalvular disease</td>
<td>Terminal</td>
<td>—</td>
<td>Good</td>
<td>Aortic stenosis, tricuspid regurgitation</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>3/3/55</td>
<td>P</td>
<td>—</td>
<td>—</td>
<td>Ventricular fibrillation before purse string</td>
<td>—</td>
<td>Terminal</td>
<td>—</td>
<td>Good</td>
<td>Fair</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>4/19/55</td>
<td>P</td>
<td>—</td>
<td>—</td>
<td>3 months</td>
<td>Advanced disease</td>
<td>Terminal</td>
<td>—</td>
<td>Excellent</td>
<td>Excellent</td>
<td>Sudden unexplained death</td>
</tr>
<tr>
<td>7</td>
<td>4/21/55</td>
<td>P</td>
<td>—</td>
<td>—</td>
<td>5 months</td>
<td>Advanced disease</td>
<td>Terminal</td>
<td>—</td>
<td>Excellent</td>
<td>Good to Excellent</td>
<td>Sudden unexplained death</td>
</tr>
<tr>
<td>8</td>
<td>5/20/55</td>
<td>P + C</td>
<td>—</td>
<td>—</td>
<td>6 weeks</td>
<td>Advanced disease</td>
<td>Far advanced</td>
<td>Terminal</td>
<td>—</td>
<td>Good</td>
<td>Pulmonary edema at autopsy</td>
</tr>
<tr>
<td>9</td>
<td>6/2/55</td>
<td>P</td>
<td>8 months</td>
<td>Fair</td>
<td>—</td>
<td>—</td>
<td>Terminal</td>
<td>—</td>
<td>Fair to Good</td>
<td></td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>6/6/55</td>
<td>P</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>Ventricular fibrillation 65 min. with recovery</td>
<td>Terminal</td>
<td>—</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>9/15/55</td>
<td>P</td>
<td>—</td>
<td>—</td>
<td>3 months</td>
<td>Technical error 1 hr. postop.</td>
<td>Far advanced</td>
<td>—</td>
<td>Excellent</td>
<td>Infected vegetations on suture in left atrium</td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>9/20/55</td>
<td>P</td>
<td>5 months</td>
<td>Excellent</td>
<td>—</td>
<td>—</td>
<td>Far advanced</td>
<td>—</td>
<td>Excellent</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Fig. 2.** Anteroposterior roentgenograms taken preoperatively (left) and 1 year postoperatively (right) in case 1.
the potential orifice was enlarged to a capacity of 4 to 5 fingers. The valve leaflets were only moderately thickened but quite tense, stretching across the atrioventricular orifice like a diaphragm. The aortic, or septal, leaflet was large and very pliable. The mural leaflet was short, rolled, and fixed. The commissures were rounded by a slight degree of fusion or scarring. There was no calcification. The chordae tendineae were thick, short, and tense.

A circumferential suture was placed as described elsewhere.44 When the suture was tied, the regurgitant jet immediately diminished to a "whiff" localized to the region of the posteromedial commissure that was barely detectable by the intracardiac finger. The potential orifice was reduced to a capacity of approximately 3 fingers. The effective orifice remained unchanged. The aortic leaflet of the mitral valve was felt to relax and move almost like a normal leaflet, closing quite efficiently during systole. Direct intracardiac pressure tracings then showed a significant drop in atrial pressure (table 2).

The postoperative course was uneventful. Frequent postoperative electrocardiograms showed an axis shift to the left and episodes of atrial flutter-fibrillation but were otherwise unremarkable. Chest roentgenograms showed a significant decrease in the cardiac silhouette (fig. 2). A systolic murmur was still present, but it was a soft, short, grade I murmurm. The postoperative phonocardiogram substantiated the marked decrease in intensity and duration of the murmur. No diastolic murmur was noted.

The patient was last observed 12 months after surgery. She was able to do all but the heaviest housework shortly after the first month and after 4 months she returned to work as a seamstress. Examinations have shown constant clinical improvement. The chest x-ray, electrocardiogram, and phonocardiogram have not changed significantly since discharge. She gained 30 pounds in weight and has remained completely asymptomatic. She continued to take digitoxin.

Comment. This patient approaches what may come to be considered the ideal case for treatment by this method. Preoperatively there was considerable question as to the
diagnoses of mitral stenosis versus mitral insufficiency. This is the first recorded case in which objective evidence of correction of mitral regurgitation is available.

Case 2

B. F. (No. 164854). This 24-year-old white woman was admitted to the Presbyterian Hospital on January 15, 1955. She had rheumatic fever at age 9 and was confined to bed for 2 years. At 19 rheumatic fever recurred and left her a semi-invalid ever since. In February 1952 an operation disclosed "pure" mitral regurgitation, which was not corrected. Progressive cardiac failure developed with ascites, ankle edema, shortness of breath, orthopnea, venous engorgement of the neck, and a chronic, nonproductive cough that persisted despite absolute bed rest and a rigid medical therapeutic regimen.

Physical examination revealed an emaciated, dyspneic young woman. There were marked engorgement of the neck veins and a strong precordial "heaving." The lungs presented scattered fine rales. The heart rhythm was totally irregular. There were a prominent apical systolic thrill and a grade IV, rough, mitral systolic murmur transmitted to the axilla, back, and left sternal border. The aortic second sound was diminished. The pulmonic second sound was accentuated 2 plus. The mitral first sound was greatly diminished. A grade II, mitral, diastolic rumble was detected by some examiners but not by others. A firm, tender liver was palpable 5 fingers below the right costal margin and pulsed slightly. Two-plus pretilial edema and ascites were present.

Preoperative laboratory studies were not remarkable. Roentgenologic examination revealed left basal pleural thickening and an enormous cardiac shadow (fig. 4). Electrocardiograms showed evidence of combined heart strain and hypertrophy with predominance on the right as well as atrial fibrillation and frequent ectopic ventricular beats. A phonocardiography showed prolonged systolic vibrations of moderate amplitude, but no diastolic murmur (fig. 4). Cardiac catheterization data are summarized in table 1.

Surgery was performed on February 1, 1955. The pleural and pericardial cavities were obliterated by dense adhesions. The heart was enormous; it seemed to fill the chest. There was no left atrial appendage. A strong systolic thrill was palpable over the entire left atrium. Direct intracardiac pressure tracings were made (table 2). The left atrium was entered through an artificial rubber appendage sewn onto its anterolateral surface. There was a strong, wide, grade IV systolic jet. The effective orifice admitted 3 to 4 fingers, the potential orifice 5 fingers. The valve leaflets were shortened, slightly thickened, tense, and diaphragmlike. The mural leaflet was rolled and fixed. There

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* The term "effective orifice" refers to the opening that is bounded by the edges, or "lips," of the mitral valve cusps. This orifice determines the effective atrioventricular diastolic flow. The term "potential orifice" refers to the opening that would result if the valvular obliterating elements were eliminated completely. The size of this "valveless" atrioventricular communication is generally determined by the annulus fibrosus. These terms are fully discussed elsewhere.44
was little, if any, fusion at the commissures, and no calcium was present. The circumferential suture was placed and the potential orifice was reduced to a capacity of 3 to 4 fingerbreadths but the effective orifice remained unchanged in size. The regurgitant jet practically disappeared, the size of the left ventricle diminished visibly and the left atrium, which had been very tense and distended, became flaccid. Intracardiac pressure tracings were repeated (table 2).

Postoperatively the patient was hypotensive and required vasopressor drugs. Positive pressure oxygen was necessary for about 3 hours. In 24 hours, however, the vital signs stabilized. Retained tracheobronchial secretion required aspiration and mucolytic vaporization, but after the fourth postoperative day her course was smooth and she improved progressively except for several episodes of epistaxis, which required cauterization of the nasal membrane. The ascites disappeared in 3 weeks. The liver receded to 1 or 2 fingerbreadths beneath the right costal margin. Daily electrocardiograms during the first postoperative week and periodic tracings after that showed no new abnormality. She developed mild anemia and an elevated sedimentation rate (82 mm. per hour) on the eighteenth day after operation. The antistreptolysin and streptococcal antihyaluronidase titers at this time were 128 and 384 respectively, which are intermediate but compatible with rheumatic activity. After 4 weeks the patient’s condition was improved; she was ambulatory most of the day and was afebrile, the liver was no longer palpable, and she had no shortness of breath or orthopnea. A grade I apical systolic murmur was audible, and the phonocardiogram showed a marked decrease in the systolic murmur vibrations. There was only slight, if any, decrease in the cardiac silhouette on x-ray.

![Fig. 3. Posteroanterior and left lateral roentgenograms, taken preoperatively in case 2, showing intractable congestive failure.](#)

![Fig. 4. Phonocardiogram and direct left atrial and left ventricular pressure pulse tracings before and after surgery in case 2. Note the dramatic pressure changes and also the changes in contour.](#)
The patient was discharged on February 20, 1955, in a much improved state, which continued for a month. However, during the last week in March she developed what appeared to be an upper respiratory infection associated with fever and signs of decompensation. Frank congestive failure with hemoptysis rapidly followed. The downhill course continued and she died at home on April 1, after 4 days of anuria. An autopsy was not performed.

Comment. Correction of the regurgitation, as shown by the hemodynamic studies and phonocardiogram, probably accounts for the rather remarkable and rapid initial improvement. However, without postmortem studies one can only speculate as to the factors determining her ultimate demise. Rheumatic activity is strongly suggested by some of the early postoperative studies as well as by the terminal course.

Case 3

P. F. (No. 165623). This 27-year-old white woman was admitted to the Presbyterian Hospital on February 14, 1955, complaining of exertional dyspnea, orthopnea, fatigability, and periodic ankle edema. She had been told that she had heart disease at the age of 14 after a routine school physical examination. She had pneumonia the following year and again in 1944, at which time she was treated with prolonged bed rest and oxygen and was told that she had a bad heart. She was completely incapacitated for the next 5 years, being unable to engage in any useful activity. Gradually improvement followed until age 22 when she married. She had 1 pregnancy during which she noted moderate exertional dyspnea and ankle edema. Six months before her present admission she was hospitalized for 10 weeks with atrial fibrillation that was treated with digitalis and quinidine. Thereafter she was unable to carry on her usual household activities.

Physical examination revealed a well developed, tense young woman. There were fine rales at both lung bases. The cardiac rhythm was totally irregular and there was a prominent apex impulse. A grade III mitral systolic murmur transmitted to the base and axilla and a grade II systolic murmur over the aortic area were heard. A grade II mitral diastolic rumble followed the opening snap. The aortic second sound was present but split, and in the mitral first sound and the pulmonic second sound were accentuated. The liver was palpable 1 fingerbreadth below the right costal margin but there was no dependent edema.

Laboratory studies were not remarkable. Roentgenologic examination showed an enlarged heart with calcification in the region of the mitral valve (fig. 5). The electrocardiogram showed combined ventricular strain and atrial fibrillation. Phonocardiograms showed vibrations of wide amplitude lasting for about one third of systole following the first sound. There were also diastolic vibrations of long duration but relatively small amplitude. The data of cardiac catheterization are summarized in table 1.

At surgery, on February 2, 1955, the lungs were moderately turgid and the pericardium contained 300 ml. of fluid. There were a grade I systolic thrill over the aortic root, slight dilatation of the ascending aorta, a strong systolic thrill over the entire atrium, and a prominent diastolic thrill over the left ventricle. Externally no calcification of the aortic valve could be detected. Intracardiac pressures were taken (table 2). Intracardiac exploration revealed mitral

Fig. 5. Anteroposterior roentgenogram taken preoperatively (left) and 11 months postoperatively (right) in case 3.
stenosis with an effective orifice that admitted 1 to 1½ fingers and marked calcification in the region of the posterior commissure. However, the leaflets retained moderate pliability even though they were thickened and rolled. The potential orifice was enlarged moderately with a capacity of 3 to 4 fingers. A grade III regurgitant jet was palpable. As the needle for the circumferential suture was manipulated around the medial zone of the ring, ventricular fibrillation appeared. The needle was withdrawn and cardiac massage and resuscitative measures were carried on for 22 minutes until effective ventricular contractions were restored and the heart beat improved progressively. The blood pressure became stabilized in about 15 minutes. The suture was then placed easily without further disturbances of myocardial action. Anterolateral commissurotomy was then performed whereupon the regurgitant jet increased markedly in intensity and size. After the circumferential suture was tied, the jet diminished dramatically. Effective action of the aortic leaflet of the mitral valve was clearly palpable even though the leaflet margin was indurated. The effective orifice there admitted 2 to 2½ fingers and the potential orifice 2½ to 3 fingers. A small, faint jet was still detectable posteriorly. Pressure readings were repeated (table 2).

Postoperatively the patient did very well despite an interval of hypotension and lassitude lasting about 48 hours. There was rapid clinical improvement with no signs of congestive failure at any time postoperatively. Electrocardiograms showed no significant changes. The phonocardiogram showed a marked decrease in the systolic murmur. On auscultation a grade I to II systolic apical murmur and a grade I diastolic apical murmur were heard, but the murmur at the aortic area was unchanged. The x-ray of the chest showed no significant change in cardiac size. The patient left the hospital in excellent condition on March 30, 1955.

She remained asymptomatic until May 23, when she was readmitted to the hospital with a diagnosis of left basilar pneumonitis. Cardiac examination was unchanged. She improved promptly with antibiotics and was discharged in 2 weeks.

She was admitted again on June 28 after another asymptomatic interval because of left-sided chest pain. A left pleural effusion was found that required thoracentesis. Prompt improvement followed and she left the hospital in 11 days.

She was last seen 6 months later, still completely asymptomatic. She has been doing most of her housework for approximately the past 5 months. Electrocardiogram, phonocardiogram, and findings on physical examination were essentially the same as those of her original discharge from the hospital following surgery. An x-ray of the chest 11 months after operation showed reduction in cardiac size (fig. 5).

Comment. This is the first case in which we had an opportunity to test the efficacy of this method for correcting mitral regurgitation in a valve in which a "double lesion" was present. Despite only slight enlargement of the annulus and considerable induration and calcification of the valve the mitral purse string diminished the regurgitation to a marked degree. Whether the degree of regurgitation was more important before surgery than the coexisting stenosis is impossible to know. It is certain, however, that the aggravation of the regurgitant factor following the commissurotomy would have been of very serious consequence had it not been controlled by the suture.

Case 4

B. W. (No. 165890). This 41-year-old white woman was admitted to the Presbyterian Hospital on February 25, 1955. She had rheumatic fever at age 11, and 1 year later she was unable to climb steps, was dyspneic, and had pain in her back and chest. During the next few years she was hospitalized several times and required digitalis and occasional diuretics during these episodes. She improved and did well until age 31, when dyspnea lasting several weeks followed a "strept" infection. She married and had an uneventful pregnancy and a normal delivery at age 34. Until 1954 she was reasonably well but she did not do much work and was under constant, careful medical observation. In the year before admission she had frequent attacks of paroxysmal cough as well as weakness, increased dyspnea on exertion, orthopnea, and ankle edema.

On physical examination she was orthopneic and had frequent dry cough. There was moderate engorgement of the neck veins, dullness over both lung bases, and crepitant basal rales. The cardiac rhythm was totally irregular. The apical impulse was prominent. A grade IV systolic murmur was loudest at the apex and transmitted to the axilla; there were also a grade II mitral diastolic murmur and a grade II aortic systolic murmur. The mitral first sound and the pulmonic second sound were accentuated 2 plus and the aortic second sound was present. The liver was palpable 3 to 4 fingers below the right costal margin, and there were dullness in the flanks and moderate ankle edema.

Laboratory studies were not remarkable. X-ray showed a large pleural effusion on the left and an enlarged heart (fig. 6). The electrocardiogram showed changes considered compatible with rheumatic carditis. Phonocardiograms showed vibrations of wide amplitude throughout systole at the apex as well as over the aortic area. The aortic vibrations were of greater amplitude in mid systole. The pul-
monic second sound was marked by very wide deflections. There was no diastolic abnormality.

Cardiac catheterization performed elsewhere 1 week earlier was compatible with significant aortic and mitral lesions. The pressures are listed in table 1.

Surgery was performed on March 1, 1955. The left pleural cavity contained 1200 ml. of fluid, the lungs were turgid and discolored, and there was a dense adhesive pericarditis. A coarse systolic thrill was palpable over the lateral and posterior surfaces of the left atrium. There were a grade I to II poststenotic dilatation of the aorta and a grade II aortic systolic thrill. Considerable thickening and induration about the aortic valve was found. Direct intracardiac pressure tracings were taken (table 2). Digital exploration of the mitral valve revealed extensive damage consisting of a stenosis of the effective orifice to 1 to 1½ fingers, with dense calcification almost entirely around the orifice. The potential orifice was only slightly, if at all, enlarged, admitting to 3 to 3½ fingers. The leaflets were thick and of the consistency of shoe leather. The aortic leaflet of the mitral valve retained some degree of flexibility of its base and body. The orifice was slit-like and did not open well in diastole. A grade II to III regurgitant jet was present.

Both commissures were opened and the effective orifice was enlarged to 2½ fingers, but the regurgitant jet increased to grade IV. There was significant improvement in the motion of the aortic leaflet. A circumferential suture was placed and the mitral ring was reduced to a capacity of 2½ fingers, but the effective orifice was not reduced. The regurgitant jet was then detectable posteriorly only and was reduced to grade I or less. Intracardiac pressure readings were repeated (table 2).

Aortic commissurotomy was judged inadvisable in view of the condition of the myocardium and of the relatively low aortic-ventricular gradient.

Postoperatively there was mild hypotension for the first 48 hours. During the ensuing 5 days progress was good except for a daily temperature spike to 101 F. The edema, hepatomegaly, and dyspnea diminished notably within the first 10 days. The patient improved further, becoming afebrile on antibiotic and cortisone therapy. In the third postoperative week she became listless and complained of fatigue. Signs of congestive failure reappeared. A mild electrolyte imbalance was corrected but the patient continued to deteriorate. On the twentieth postoperative day she became comatose and cyanotic; severe hepatomegaly and marked edema were present. She died on the twenty-second postoperative day.

An autopsy was performed that disclosed multiple fresh small pulmonary infarcts and areas of bronchopneumonia with evidence of chronic passive congestion, fibrosis, and pulmonary edema. There were also hemosiderosis and anthracosis of the lung and pleural thickening. The liver was cirrhotic, and the spleen showed marked changes of chronic passive congestion. The kidneys showed arteriolar nephrosclerosis. The adrenal glands showed changes of chronic passive congestion.

The heart was very large. There was severe mitral, aortic, and tricuspid valve damage. The mitral and aortic valves were severely calcified. There was significant aortic stenosis. The tricuspid valve leaflets were fused at the commissures but there was a large, rounded, effective tricuspid orifice that admitted 3 fingers; the leaflets did not close. The mitral "purse-string" suture was placed accurately, the potential orifice being as described at surgery. The stiffened aortic leaflet of the mitral valve could occlude the orifice quite well. The effective orifice admitted 2 to 2½ fingers. The myocardium showed chronic rheumatic disease. The coronary arteries were patent.

![Fig. 6. Posteroanterior and left lateral roentgenogram, preoperatively, in case 4. Note left pleural effusion that could not be cleared by medical means.](http://circ.ahajournals.org/doi/abs/10.1161/01.CIR.21.4.670?journalCode=circ)
Comment. Autopsy findings in this patient substantiated the operative impression, supported by intracardiac pressure studies, that the mitral stenosis and regurgitation were significantly improved by surgery. This was demonstrated dynamically on the pulse duplicator,\textsuperscript{46} which permitted direct observation of valvular action. The explanation of the postoperative course and eventual demise of this patient is obscure. It was probably related to the left ventricular insufficiency in the face of aortic stenosis, which was possibly aggravated by the improved ventricular filling. The additional severe tricuspid regurgitation and extensive pulmonary pathology were important contributory factors.

Case 5

L. T. (165936). This 19-year-old white woman was admitted to the Presbyterian Hospital on February 26, 1955. At the age of 8 years she was hospitalized with rheumatic fever, following which she had to be carried upstairs. For 15 years she suffered from continual edema, shortness of breath, dyspnea on exertion, and occasional chest pain. She was unable to do more than a minimal amount of indoor walking, and she spent most of her time confined to her bed or a chair. She received digitalis and diuretics for 5 years. Several episodes of epistaxis occurred that sometimes required nasal packing. She had a chronic cough productive of white sputum with occasional blood streaking. Periodically she had episodes of vomiting, which lasted 2 or 3 days.

Physical examination revealed a small, very thin girl with pale moist skin. There was engorgement of the neck veins. The anterior chest wall was prominent and there was marked precordial heaving. The lungs were clear except for questionable dullness at the left base. The cardiac rhythm was totally irregular. There was a grade IV mitral systolic murmur, which was transmitted to the left axilla, the back, and toward the sternum; no diastolic murmur was heard. The mitral first sound was normal or decreased. The pulmonary second sound was accentuated 2 plus and widely split. The liver was felt 2 or 3 fingers below the right costal margin. There was no significant ankle edema.

Laboratory studies were not remarkable. Roentgenologic examination revealed enormous cardiac enlargement (fig. 7). Electrocardiogram showed atrial fibrillation, digitalis effect, and right heart strain. Phonocardiograms showed prominent, wide vibrations in the first half of systole as well as early and mid-diastolic vibrations of much less amplitude. Cardiac catheterization data are summarized in table 1. The pulmonary "wedge" pressure could not be obtained because the loop of the catheter within the heart was so large (due to cardiac enlargement) that the 100-cm. length of the catheter was not sufficient to allow the tip to wedge.

Operation was performed on March 3, 1955. The lungs were turgid and thickened. The left atrium was enormous, almost twice the size of the rest of the heart. It extended posteriorly and downward to the diaphragm. A prominent systolic thrill was palpable over the entire left atrium. The direct intracardiac pressures are summarized in table 2. Left atrial and left ventricular pressure pulse contours were diagnostic of mitral regurgitation. Intracardiac digital exploration revealed annular dilatation with a potential capacity of 4 to 5 fingers and an effective orifice of 3 to 4 fingers. The aortic leaflet of the mitral valve was of the consistency of a kid glove and very pliable. It was taut and restricted in motion by the chordae tendineae. The mural leaflet was rolled and contracted.

![Fig. 7. Preoperative roentgenograms in case 5 showing the marked aneurysmal dilatation of the left atrium that is almost routinely found in advanced cases of mitral insufficiency.](http://circ.ahajournals.org/doi/10.1161/01.CIR.2.11.671)
Before the circumferential suture was placed, ventricular fibrillation appeared without obvious precipitating factor. For 1 $\frac{1}{2}$ hours resuscitative measures were carried out—massage, repeated electric countershocks, and administration of various drugs—to no avail. In view of the massive regurgitation and the failure to restore cardiac action the circumferential suture was placed during the fibrillation with brief interruptions of the massage. After the suture was tied, the regurgitation decreased markedly. The peripheral pulse, synchronous with cardiac massage was greatly improved. Despite the better output, however, cardiac action could not be restored. Finally the left atrial wall was lacerated and severe hemorrhage resulted. Autopsy revealed hemosiderosis, chronic passive congestion, vascular thickening, and fibrosis of the lungs; chronic passive congestion, periportal fibrosis, and acute and chronic inflammation of the liver; chronic passive congestion of the spleen; and acute and subacute necrotizing glomerulonephritis of the kidneys. The adrenal glands showed marked cortical atrophy with acute congestion and lipoid depletion. The largest chamber of the heart was the left atrium. There was also marked left ventricular dilatation and hypertrophy. There was no aortic, tricuspid, or pulmonic valvular disease. The mitral valve was obviously incompetent. The annulus was easily identifiable and greatly dilated, the chordae tendineae were thick and short, the mitral leaflets were incapable of coaptation. On visual and cinematographic study on the pulse duplicator the magnitude of the regurgitation was quite obvious. It was also shown by this method that the mitral purse string completely corrected the insufficiency by constricting the ring to about 3 to 3.5 cm. in diameter from its original of 5.5 to 6 cm. diameter (fig. 8).

Comment. This patient represents a surgical death that was due to factors not related to the corrective procedure itself, since irreversible ventricular fibrillation ensued prior to the placement of the circumferential suture. Up to this point the patient had undergone the usual preliminary exploratory cardiotomy. As was shown on postmortem examination, complete correction of the regurgitation would probably have been technically possible in this case.

Case 6

R. S. (No. 167057). This 28-year-old Negro woman was admitted to Presbyterian Hospital on April 11, 1955. Sixteen years previously she had "inflammatory rheumatism." Eight years later after a successful pregnancy, she suffered exertional dyspnea, orthopnea, and fatigueability. Despite digitalis and careful medical management she was unable to do any useful work.

Physical examination revealed marked precordial pulsations. The lungs were clear. Cardiac rhythm was totally irregular. The aortic second sound could not be heard. There was a grade IV mitral systolic murmur transmitted to the axilla and back and to
the pulmonic area. The mitral first sound and the pulmonic second sound were both accentuated. A grade II mitral diastolic rumble was present. There was a questionable grade II aortic systolic murmur.

Laboratory studies were not remarkable. Roentgenologic examination revealed fibrosis and pleural thickening at the lung bases and moderate calcification over the mitral area. The heart was tremendously enlarged as in case 5. The electrocardiogram showed myocardial damage, atrial fibrillation, and probable left ventricular hypertrophy. Cardiac catheterization data are summarized in table 1. The phonocardiogram showed systolic vibrations of wide amplitude and medium frequency lasting throughout systole as well as less impressive early diastolic vibrations. The first sound at the apex and the second sound over the pulmonic valve were accentuated.

At operation on April 19, 1955, the lungs were turgid and thick and the heart was enormous. The walls of the aneurysmal left atrium appeared pale and fibrinous, noncontractile, and tense. Left atrial enlargement extended inferiorly to the diaphragm and to the right. There was a coarse, prominent thrill over the entire left atrial surface. Superficial exploration revealed no evidence of other valvular pathology. Intracardiac pressures were recorded (table 2).

Intracardiac digital exploration revealed a potential mitral ring of 4 to 5 fingers' capacity. The effective orifice was not stenotic, admitting 3 to 4 fingers. The aortic leaflet, somewhat contracted and thickened, was quite pliable and moved well although under tension by shortened chordae tendineae. There was a plaque of calcium at the tip of the leaflet. The mural leaflet was rolled and thickened. The circumferential suture was placed and tightened until the potential orifice was reduced to a capacity of 3 to 4 fingers. The effective orifice remained unchanged. The regurgitant jet was barely detectable in the region of the posterior commissure. The atrial thrill disappeared and the tension decreased to such an extent that this chamber became a flaccid sac. The tension in the pulmonary artery also diminished noticeably. The volume of the left ventricle decreased appreciably. Pressure tracings were repeated (table 2). The left atrial and left ventricular pressure pulse contours, which had been typical of mitral insufficiency before the procedure, reverted to tracings that appeared normal except for the lack of atrial contractions.

The postoperative course was uneventful except for fever up to 101 F. on the first day after surgery. Electrocardiograms showed a shift in electric axis to a more upright position and a slight ST elevation suggesting epicardial or pericardial changes that subsided by the seventh day. There was no suggestion of coronary insufficiency or of conduction defect. Antistreptolysin and streptococcal antihyaluronidase titers were intermediate and inconclusive.

The patient was ambulatory on the seventh postoperative day, free of dyspnea and orthopnea, and was discharged in excellent condition on the twenty-fifth postoperative day.

The patient did very well in the following weeks and felt relieved of all symptoms. On July 2, shortly after returning home from a shopping trip, she suddenly complained of feeling faint and fell dead. At autopsy no explanation for the sudden demise was evident. The viscera showed chronic congestive changes. The "purse-string" suture was well placed, accomplishing its purpose very effectively. The mitral valve leaflets came together well and also opened widely. The portion of suture passing on the right side of the interatrial septum was covered by glistening endothelium-like tissue. There was no intracardiac thrombosis or coronary occlusion. The other valves were not significantly deformed. The major pathologic features were marked dilatation of the left atrium, left ventricular dilatation and hypertrophy, and the deformity of the mitral valve that was as noted at surgery.

Comment. The death of this patient, occurring suddenly after a period of uninterrupted improvement, is quite disturbing, the more so because we have no explanation for it.

Case 7

J. L. (No. 160123). This 25-year-old woman was admitted to the Presbyterian Hospital on April 17, 1955. She had rheumatic fever at the age of 5 and did not attend school for the following 2 years. From that time on she has been under continuous medical care. She had been reasonably well until September 1953, when she had the first of several episodes of pneumonia, allegedly accompanied by "pulmonary and cerebral embolism." Other than a transient amnesia there was no sequela. In October 1954 she collapsed and required oxygen therapy for 2 days. On admission she complained of 2-pillow orthopnea, exertional dyspnea, fatigability, and occasional lapse of memory. She was confined to bed except for meals. She was taking digitalis, diuretics, and oral penicillin.

The patient was pale, thin, and weak. The lungs were clear. There were marked precordial pulsations. The cardiac rhythm was regular with occasional extrasystoles. A grade III mitral systolic murmur and a grade II aortic systolic murmur were heard (possibly a transmission of mitral murmur to aortic area?).

Laboratory studies were not remarkable. The electrocardiogram showed normal sinus rhythm with multifocal ectopic beats, and probable left ventricular hypertrophy; an electrocardiogram on a previous admission showed atrial fibrillation. Phonocardiograms showed high frequency vibrations of moderate amplitude throughout systole. Roent-
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genologic studies revealed calcific pulmonary scars, generalized emphysema, and a very large heart. The patient had been catheterized previously and the findings are summarized in table 1. Although the pulmonary wedge pressure was not greatly elevated the pressure contour was compatible with mitral insufficiency. The brachial artery tracing did not confirm the clinical impression of aortic stenosis.

Surgery was performed on April 21, 1955. There was a strong systolic thrill over the entire left atrial surface. No thrill was palpable over the left ventricle. External exploration of the aorta and aortic ring gave no suggestion of aortic valve disease. Direct pressures taken at this time are summarized in table 2. The mitral anulus was dilated to 5 fingers' capacity and the effective orifice was easily 4-finger-breadths in capacity. There was a grade IV regurgitant jet. The mural leaflet was markedly thickened and contracted. The aortic leaflet was contracted and under tension, but quite pliable. No calcification was present. A circumferential suture was placed as usual; when it was tied, the anulus was reduced to 3 or 4 fingers. The regurgitant jet was felt to diminish dramatically. The effective orifice remained about 3½ fingers and the valve was felt to close well. A faint "whiff" was detectable posteriorly. Pressures were again recorded (table 2).

On the second postoperative day the patient showed mild respiratory distress, which after x-ray examination was attributed to retained secretions and atelectasis. She was also hypotensive at this time. Norepinephrine infusions and tracheal aspiration produced improvement. A state of depression and lassitude with fever and tachycardia persisted about 1 week, but improved following cortisone therapy. After the second postoperative week improvement continued and the patient required no medication except for digitalis. She was discharged home 1 month postoperatively. She did well at home on an ambulatory status with restricted activity and had no dyspnea, orthopnea, or cough.

September 22, 1955, she died suddenly and unexpectedly. There has been no change in her status and nothing was found to explain her demise. The findings at autopsy consisted of the following: bronchial hypertension, emphysema, chronic passive congestion, bronchothiasis and edema of the lungs; localized parenchymatous atrophy of the liver; renal calcinosis and healed polynephritis. In the heart the purse-string suture was placed correctly and was well tolerated. The anulus was narrowed sufficiently to allow coaptation of the leaflets and was about 3.5 cm. in diameter. The heart except for enlargement of all chambers and adhesive pericarditis was otherwise negative.

Comment. This is a second instance of a satisfactory postoperative course followed by sudden, unexplained death after continuing improvement. The postmortem findings indicated far advanced disease as well as multiple lesions. The immediate postoperative course was suggestive of rheumatic activity. Nevertheless, there is no specific factor that explains the ultimate outcome. Neither in this patient nor in the previous one was there any postoperative electrocardiographic change to suggest conduction disturbances.

Case 8

M. G. (No. 167976). This 33-year-old white woman was admitted to the Presbyterian Hospital on May 16, 1955. She gave a history of severe orthopnea and dyspnea of several months' duration. Some months before at cardiomyopathy elsewhere "pure" mitral insufficiency was discovered. Thereafter she was totally incapacitated and at complete bed rest.

Physical examination revealed a thin, dyspneic, chronically ill woman. The thorax revealed prominent precordial impulses. There were crackling rales over both lung bases. The cardiac rhythm was totally irregular and a grade IV mitral systolic murmur was present. The liver was palpable 5 fingers below the costal margin. The extremities showed mild to moderate ankle edema.

The laboratory findings were not remarkable. Roentgenologic studies showed congested lungs and slight pleural thickening at both bases. No abnormal calcification was noted. The heart was enlarged. The electrocardiogram showed marked right ventricular hypertrophy and atrial fibrillation. Cardiac catheterization was performed and the available data are summarized in table 2.

At operation on May 26, 1955, the left atrium was greatly enlarged and a prolonged systolic thrill was felt over the entire surface. Direct pressures were recorded (table 2). The left atrial pressure contour was diagnostic of mitral insufficiency. The potential orifice of the mitral valve was about 5 fingers in capacity. The valve leaflets were pliable, thickened, and under tension, stretching diaphragm-like across the orifice. The commissures were somewhat fused making the effective orifice about 2 to 3 fingers in size. A strong grade IV regurgitant jet was found and much calcium was present, particularly over the lateral half of the aortic leaflet of the mitral valve and in the region of the commissures. The mural leaflet was larger than usual. The orifice was located postero-medially as a result of more extensive fusion of the anterolateral commissure. A circumferential suture was placed in the established manner. The anterolateral commissure was opened to the myocardium, so that the effective orifice was enlarged to 3 to 4 fingers. The aortic leaflet was then considerably more mobile and the circumferential suture was tied until the potential orifice was reduced to 3 to 4 fingers whereupon the regurgitant jet was barely discernible as a faint localized "whiff." Intracardiac pressures were repeated (table 2).
The patient had a smooth postoperative course with prompt disappearance of edema and recession of the liver edge beneath the costal margin within the first week. She had a temperature elevation up to 101 F., for 4 days. The postoperative electrocardiograms showed occasional premature ventricular contractions and digitalis effect. A lung biopsy showed marked arteriolar thickening. The patient was discharged much improved physically and in excellent spirits.

She died on July 7, 1955. No details of her course are available. At autopsy at the local hospital the following diagnosis was noted: mitral insufficiency due to rheumatic heart disease, pulmonary edema and passive congestion, cardiac cirrhosis of the liver, salmonella infection (parathyroid) of the ileum. The heart showed coronary occlusion or intracardiac thrombosis. The suture was well placed and accomplished annular constriction as desired. A specific cause of death was not stipulated.

Comment. This patient was in a far advanced, practically terminal stage of the disease. The pulmonary edema suggests heart failure as a likely cause of death. It could have been the result of chronic, extensive myocardial insufficiency or due to renewed rheumatic activity.

Case 9

H. S. (No. 168237). This 38-year-old white woman was admitted to the Presbyterian Hospital on May 26, 1955. She had “rheumatism” at the age of 7 years and took digitalis ever since, but had repeated bouts of dyspnea, orthopnea, and ankle edema. For 2 years she had a 2-pillow orthopnea and nocturnal dyspnea. She complained of constricting pain about the lower chest and upper abdomen, abdominal fullness, and ankle edema, but not of cough or hemoptysis. She also complained of occasional vertigo, tinnitus, and near-syncope. She had had 4 pregnancies with normal deliveries. In 1938 she had left pleural effusion that required thoracentesis.

The thorax showed a prominent precordium with marked pulsations. A grade IV mitral systolic murmur was transmitted to the base, left axilla, and the back. M1 was normal or diminished and P2 was accentuated 1 to 2 plus. There was a marked systolic thrill over the precordium, most prominent in the third interspace. There was no diastolic murmur and the lung fields were clear. The liver was slightly tender and extended 2 fingers below the right costal margin. There was no dependent edema.

The laboratory findings were not remarkable. Roentgenologic examination revealed pulmonary congestion, cardiac enlargement, and no calcification. The electrocardiogram indicated atrial fibrillation, digitalis effect, and myocardial damage. Cardiac catheterization was performed elsewhere and the data are summarized in table 1.

Surgery was performed on June 2, 1955. The left atrium was huge, it bulged into the left pleural space, elevated the left main bronchus to almost a right angle, and extended down to the diaphragm. There was a grade IV systolic thrill over its entire left atrial surface. Direct intracardiac pressures were taken (table 2).

The potential orifice of the mitral valve was large enough to admit 4 or 5 fingers; the effective orifice was about 3 fingers. The aortic leaflet was thickened but pliable and the mural leaflet was rolled, contracted, and immobile. No calcium was present. A grade IV jet of regurgitation was palpable. Because of the tremendous size of the heart it was difficult to place the medial zone of the suture in the annulus. After completion of the procedure and without obvious initiating circumstance the ventricles began to fibrillate. Various resuscitative measures were carried out including cardiac massage, multiple electric shocks, and administration of procaine, calcium chloride, and epinephrine. Effective ventricular contraction was resumed after a period of 65 minutes. The circumferential suture was tightened during the period of ventricular fibrillation and no regurgitant jet could be detected immediately after the recovery of effective contraction. Direct intracardiac pressures were not repeated. At the conclusion of the procedure the patient recovered from the anesthesia without apparent ill effects from the prolongation period of circulatory failure.

The postoperative course was complicated by a period of hypotension lasting 2 days that required norepinephrine infusions. On the fifth day bronchoscopy was done for retained tracheobronchial secretions with satisfactory results. On the sixteenth postoperative day the temperature rose to 103 F. and scattered rales were heard over the entire chest. Very slight ankle edema was noted and large amounts of sputum were raised. Marked improvement occurred during the next 2 days. Edema disappeared and the liver edge receded beneath the costal border. During the first 3 postoperative weeks the patient manifested amnesia for recent events but was otherwise rational and reasonably active. Her orthopnea and shortness of breath diminished. The postoperative electrocardiograms showed lowered QRS voltage with an axis shift to the right. There was a return to the preoperative configuration by the third postoperative day. Occasional premature ventricular beats were seen on the twenty-sixth and thirty-third postoperative days. They were attributed to the digitalis; by the thirty-fifth day, after reduction of digitalis, they diminished. The patient was discharged on July 11, the thirty-fifth postoperative day.

Reports from her family physician indicate that her condition remained satisfactory although there has been no decrease in the size of her heart by x-ray.

Comment. In this patient we can have no objective measure of the immediate reduction of
regurgitation because of the lack of postoperative pressure studies. The effectiveness of the operation in this case will have to be judged on the basis of her clinical course and future studies.

Case 10

J. M. (No. 168324). This 33-year-old white man was admitted to the Presbyterian Hospital on May 31, 1955. He gave a history of rheumatic heart disease and dyspnea on exertion in childhood and progressive incapacity from congestive failure. In 1953 a mitral commissurotomy was performed following which his condition deteriorated rapidly. He became severely incapacitated, with hepatomegaly and ascites. At the time of admission he complained of dyspnea, cough, nausea, orthopnea, edema, and abdominal fullness.

Physical examination disclosed a thin, chronically ill man with venous engorgement in the neck. There were pulmonary rales. The cardiac rhythm was totally irregular. There was a prominent pulsation over the precordium and a strong apical systolic thrill. A harsh apical systolic murmur, grade IV, was transmitted throughout the left hemithorax, but there was no diastolic murmur. The mitral first sound was markedly decreased and the pulmonic second sound was accentuated 2 plus. The liver was palpable 3 fingerbreadths below the right costal margin. No ascites or dependent edema was present.

Laboratory studies were not remarkable except for an elevated erythrocyte sedimentation rate (50 mm. per hour). Roentgenologic examination revealed pleural thickening and marked cardiac enlargement. Electrocardiograms indicated atrial fibrillation, a vertical heart with marked clockwise rotation, and probably combined ventricular hypertension and strain. Cardiac catheterization data are summarized in table 1. Phonocardiography showed vibrations of wide amplitude throughout systole.

Surgery was performed on June 6, 1955. Several hypotensive periods during induction of anesthesia necessitated the use of norepinephrine and delay in surgery. The lung was turgid and thickened but expanded readily. There was an obvious, coarse systolic thrill over the entire left atrium transmitted to the pulmonary parenchyma. No evidence of valvular disease other than mitral was present. Direct intracardiac pressure tracings were made (table 2). Intracardiac digital exploration revealed numerous areas of calcification on the atrial walls and a tremendous regurgitant jet. The mitral orifice seemed wide open with a potential orifice of 4 to 5 fingers and an effective orifice of 3 fingers’ capacity. The circumferential suture was placed easily around the medial zone of the ring. As the posterior tail of the suture was stitched laterally into the atrioventricular groove, ventricular fibrillation ensued. The placement of the suture was completed with only brief interruptions in cardiac massage. The size of the potential orifice was reduced to about 3 fingers in capacity. Resuscitative measures were continued for 35 minutes until normal ventricular action was restored. The patient’s condition improved rapidly but it was deemed unwise to re-explore the valve or to repeat the pressure tracings. Continuous monitoring of the electrocardiogram during the operation showed that following the period of ventricular fibrillation there was a return to effective ventricular contraction with QRS complexes that rapidly resumed their previous characteristics. There was no suggestion of either coronary occlusion or conduction defect.

The patient regained consciousness almost immediately after completion of the closure. He was rational and had no evidence of central nervous system damage. He even asked questions and engaged in conversation before returning to his room. Less than 1 hour later the pulse suddenly stopped. The chest was opened promptly and the ventricles were found to be fibrillating. Persistent efforts for 1½ hours to restore heart action failed.

At autopsy cardiac enlargement was noted with enormous dilatation of the left atrium. There was no disease in the aortic, tricuspid, or pulmonic valves. There was marked contraction of the mural mitral leaflet with a drawing up of all the chordae tendineae of both leaflets into 2 columns densely adherent to the lateral ventricular wall at points near the center of the mural leaflet. The free edge of the aortic leaflet of the mitral valve was also drawn toward the central point of the mural leaflet. Thus an eccentric stenosis that could have scarcely admitted a match head had been produced. At the time of commissurotomy it must have been impossible to identify the usual landmarks and the “finger fracture” that was intended in the anterior commissure was actually a laceration through the aortic leaflet extending from its margin centrally to the base of the cusp. The circumferential suture effectively decreased the size of the potential orifice, but the completely unsupported, flail edges of the torn leaflet remained totally incompetent.

Comment. This case must be considered a surgical death since the operation was actually completed. Probable factors include the extremely advanced stage of the disease and inability to correct the lesion by any method. The unchanged dynamics in the face of advanced disease and the burden of anesthesia, thoracotomy, and cardiac manipulation were not compatible with recovery.

Case 11

A. T. (No. 170404). This 38-year-old white man was admitted to the Presbyterian Hospital on Sep-
tember 1, 1955. At the age of 17 a kidney was removed for pyelonephritis. A heart murmur was first noted at age 25 years and symptoms began 10 years ago with palpitation and a “rattle” in the chest on reclining. Digitalis and diuretics were given for a year during which time he had precordial pain on exertion, and progressive dyspnea, cough, and pulmonary congestion. The patient was able to carry on his duties as a clergyman but on a limited basis.

On physical examination the lungs were clear. The heart rhythm was regular with frequent premature ventricular contractions. There were a grade III, harsh mitral systolic murmur transmitted throughout the chest anteriorly and posteriorly and also a grade II rough, mitral diastolic rumble. The liver edge was palpable and slightly tender.

Laboratory studies were not remarkable. Preoperative electrocardiograms showed sinus bradycardia with a rate of 53 per minute. Roentgenologic examination showed pulmonary congestion and cardiac enlargement. Cardiac catheterization was unsuccessful and phonocardiograms were not obtained preoperatively.

The patient underwent surgery on September 15, 1955. The lung was turgid and the heart was enlarged. The left atrium and the pulmonary veins were particularly large. A coarse systolic thrill was felt over the atrium and pulmonary veins. Direct intracardiac pressure tracings were made and the data are summarized in Table 2. Digital intracardiac examination revealed a potential orifice of 4 to 5 fingers’ capacity and an effective orifice of 3 fingers. There was a grade IV regurgitant jet. The aortic leaflet of the mitral valve was mobile and pliable. The mural leaflet was moderately thickened but not completely fixed. No calcification was detected. A circumferential suture was placed and tied, reducing the potential orifice to 3 to 4 fingers in capacity. The effective orifice remained unchanged. The regurgitant jet decreased until it was difficult to detect. Pressure tracings were repeated (Table 2).

The immediate postoperative course was smooth except for temporary urinary retention. ACTH was given. The pulse rate continued slow (55 to 65 per minute) as it had been preoperatively. On the twelfth postoperative day a temperature of 105°F. occurred accompanied by severe chills. A blood culture revealed Staphylococcus aureus. Intravenous penicillin was started in dosage of 20 million units daily. On the fourteenth postoperative day an electrocardiogram showed complete heart block. The temperature again rose to 104°F. By the eighteenth day he had improved considerably, was afebrile, and had recovered from a bout of pulmonary congestion that followed the last temperature spike. Penicillin therapy was continued at the same dosage. The patient did well until the fifty-first postoperative day when the fever again recurred. The heart block persisted with a pulse rate at 60 per minute. A grade III mitral systolic murmur became apparent. The patient became pale and lethargic and appeared toxic. He developed pulmonary rales and dependent edema. DOCA was administered for 2 days. During the ensuing 3 weeks he had several bouts of acute congestive failure. He was treated with continuing antibiotics in massive doses, mercurials, digitalis, and adrenal supportive drugs. On the eighty-sixth postoperative day the temperature rose to 103°F. and continued to spike daily despite massive wide spectrum antibiotic therapy. Obvious weakness and electrolyte imbalance developed. On the one hundred and fourteenth postoperative day he became very lethargic, developed Cheyne-Stokes respirations, and died.

At autopsy the heart was very large with only mitral valvular involvement. The mitral valve was as described at operation. The posterior portion of the suture hung free in the left atrial and atrioventricular lumen and was covered with poorly organizing thrombus and vegetations. The thrombotic mass was not large enough to obstruct the atrioventricular orifice significantly. The suture had entered the left atrial lumen in its passage toward the septum and right atrium. This avoidable technical error caused a chord of suture to lie in the left atrium like a bow string, which in the course of time cut further along the ring from each puncture site.

Comment. This patient was one of the better surgical risks of this series. The lesion in the valve was ideal for correction by the purse-string technic. The procedure was performed without any apparent difficulty. The error lay in the inaccurate introduction of the suture needle and transfixion of the posteromedial zone of the ring. Such an error in placement of the suture should be recognized by careful palpation of the corresponding zone of the ring; if it occurs, the suture must be removed and replaced correctly. This error has been made in dogs and has resulted in the same type of progressive loosening and erosion of the suture.

Case 12

G. S. (No. 170599). This 16-year-old white girl was admitted to the Presbyterian Hospital on September 11, 1955. She had rheumatic fever at the age of 7 and shortness of breath for 5 years. Nine months prior to admission her liver became enlarged, ascites appeared, and dyspnea increased progressively.

Physical examination revealed a thin girl with pulmonary rales. The cardiac rhythm was totally irregular. There was a grade III to IV mitral systolic murmur transmitted throughout the left chest. There was also a grade II mitral diastolic murmur. The
liver was not palpable, and there was no dependent edema.

Laboratory studies were not remarkable. Roentgenologic studies revealed pulmonary congestion and a very large heart. An electrocardiogram showed atrial fibrillation (fig. 9). Phonocardiograms showed prolonged systolic vibrations of moderate amplitude as well as brief diastolic vibrations of small amplitude. Cardiac catheterization data are summarized in table 1.

At operation on September 20, 1955, the lung was turgid with the consistency of latex rubber. The heart was markedly enlarged and a coarse systolic thrill was palpable over the entire left atrium, the pulmonary veins, and the pulmonary parenchyma. Intracardiac exploration revealed a potential orifice of 4 fingers' capacity and an effective orifice of 3. The valve leaflets were thin and pliable, but tense and restricted in motion. There was a grade IV regurgitant jet, but there was no calcification. Intracardiac pressure tracings were made (table 2). A circumferential suture was placed and the annulus was reduced to 3 fingers in capacity. The effective orifice remained unchanged, the jet became almost undetectable, and intracardiac pressure determinations were repeated (table 2).

The postoperative course was completely uneventful and the patient was discharged on the seventeenth postoperative day. She has continued in good condition to date.

**Discussion**

The first 12 cases of an initially proposed 25-case pilot study have been presented. In this study, except in 2 instances, only patients in the terminal phases of valvular disease were selected, although it was known from years of intracardiac surgical experience that little salvage could be expected from cases in such dire distress and apparently irreversible cardiopulmonary-hepatic disease. Nevertheless, after 2 years of experimental study, it was essential to test the efficacy of "mitral purse-string" in cases such as this before applying it properly to the patient early in the course of progressive myocardial decompensation from mitral insufficiency.

It has been shown that this technic is clinically applicable. It must be emphasized, however, that its performance demands accurate anatomic orientation as well as considerable exercise in the experimental laboratory. The one technical error that occurred in this series of cases was definitely avoidable. The available human postmortem specimens as well as all the experimental material have clearly shown the anatomic and technical feasibility of the procedure.

The experimental data that indicated effective correction or even elimination of regurgitation by this method has been corroborated in these clinical cases. Figures 2 and 3 are examples of hemodynamic and phonocardiographic changes. The specimens obtained at autopsy 3 weeks, 6 weeks, 3 months, and 5 months after surgery (cases 4, 8, 6, and 7 respectively) demonstrated that consistent technical accuracy is attainable and that the immediate correction can be expected to persist. Regurgitation was corrected whether it occurred as a "pure" lesion or in combination with stenosis (cases 3, 4, and 8), even in the presence of severe calcification (cases 3, 4, and 6) and even if annular dilatation were not the major pathologic feature of the lesion (cases 3 and 4).

In the 4 postmortem specimens there was no significant damage to the atrioventricular
groove, the septal zone of the ring, the myocardium, the coronary vessels, or the valve elements. The suture was found where it was placed, encased, and fixed there by a thin fibrous sheath that added to its stabilizing function. The portion of suture that passed on the right of the interatrial septum was uniformly covered by endothelium-like tissue.

Of the first 12 consecutive cases that have undergone this operation (table 3), 4 are living in an improved state; 2 (cases 1 and 3) have survived 1 year after surgery. They are the only reported cases that have lived so long after any operation for mitral regurgitation. One is working full time as a seamstress, the other is doing most of her housework. Two other patients have survived 8 months (case 9) and 5 months (case 12) after surgery. In case 9 the operation was complicated by ventricular fibrillation. This patient showed some immediate improvement but did not improve progressively like the other 3 survivors. This less favorable result could be due to the fact that the suture was tied during the ventricular fibrillation when it was impossible to ascertain the optimum degree of constriction of the annulus. This patient was also the worst candidate of the 4, so that advanced disease might account for the less favorable result.

Of the 3 patients with good to excellent results, 2 (cases 3 and 12) may have had rheumatic activity before or after operation, but both, nevertheless, showed marked improvement.

Two patients did not survive surgery. This relatively low operative mortality for cases such as these demonstrates again that even the most damaged heart will tolerate the necessary surgical manipulation if hemodynamic function is improved. One patient (case 5) died on the operating table from ventricular fibrillation that occurred before the “purse-string” procedure was initiated. The other patient (case 10) was the only case in which the lesion was not correctable by any known method. Both were in an extremely advanced stage of the disease. In one patient who died in the late postoperative period (case 11) death was directly attributable to an avoidable technical error. Although all 3 cases must be tabulated as surgical deaths, actually only the third patient died as a result of the specific procedure, and this death was due to its improper execution.

The other 5 patients survived surgery up to 5 months with initial improvement. The improvement was transient in 3 patients (cases 2, 4, and 8) and was lasting in 2 (cases 7 and 8), in whom the late cause of death remains obscure.

To consider the results in the light of the patient’s condition preoperatively it is necessary to review the clinical data. It can be seen from x-ray and hemodynamic studies that the grouping of cases 2, 4–9, and 10 in the category of terminal disease and of cases 1, 3, 11, and 12 in that of far advanced disease seems reasonable. Only 1 patient in the terminal group has survived more than 5 months (case 9). Three of the far advanced group are living and doing well (cases 1, 3, and 12), and only 1 of the far advanced group died (case 11), due to technical error.

This experience is much like that seen in the treatment of aortic stenosis. The more extreme the stage of disease the greater the risk of surgery. In aortic stenosis, however, satisfactory reduction of the hemodynamic abnormality (high aortic-ventricular gradient) is not routinely obtainable in instances of far advanced disease with severe calcification of the valve; in mitral insufficiency improvement of the hemodynamic defect was accomplished in 11 of the 12 cases (in case 10 the integrity of the remaining valve leaflet had been previously destroyed). In mitral regurgitation, as in aortic valve disease, the left ventricle is subjected to the greatest load and undergoes progressive exhaustion. Once the left ventricular myocardium has reached a certain state of compensatory dilatation and hypertrophy, it seems no longer capable of recovery despite relief of the hemodynamic burden. Rheumatic activity notwithstanding (cases 3 and 12), if relief of the valvular dysfunction is attained before this end point of irreversible myocardial insufficiency, recovery and ultimate improvement seem likely.

In view of the excellent mechanical correction of regurgitation obtained in 11 cases
and of the good clinical results in the survivors despite the difficulties encountered in this initial series of intractable cases, it appears mandatory to attempt this corrective procedure in patients who are somewhat better candidates. Reasonable criteria for selection at this time include patients with mitral insufficiency with or without associated stenosis who show progressive symptoms, even to the point of chronic congestive failure, provided the latter is still controllable by medical means.

**SUMMARIO IN INTERLINGUA**

Es presentate un nove manovra chirurgic pro le correction de insufficientia mitral. Le resultatos clinic e hemodynamic obtenite in 12 casos es discutite.

**ADDENDUM**

Since submission of this article for publication 24 additional cases (for advanced terminal stage IV) have been operated upon to complete a pilot study of 36 operated patients. Forty-two per cent are presently improved, representing pure clinical salvage. Five other patients have been operated upon more recently, for the most part earlier cases, with no mortality and more dramatic improvement.

**REFERENCES**

The practice of medicine in its broadest sense includes the whole relationship of the physician with his patient. It is an art, based to an increasing extent on the medical sciences, but comprising much that still remains outside the realm of any science. The art of medicine and the science of medicine are not antagonistic but supplementary to each other. There is no more contradiction between the science of medicine and the art of medicine than between the science of aeronautics and the art of flying.—FRANCIS WELD PEABODY. The Care of the Patient. Harvard University Press, 1927.
Surgical Treatment of Mitral Insufficiency by Total Circumferential "Purse-String" Suture of the Mitral Ring
ROBERT P. GLOVER and JULIO C. DAVILA

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