Does Mitral Stenosis Recur after Commissurotomy?

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The ultimate fate of the incised commissure following surgery for mitral stenosis will remain unknown for years to come. A clinical appraisal of patients living five years after commissurotomy and a review of available autopsy material from patients surviving surgery for periods up to four years indicates that valvular restenosis does not occur within the first half decade provided the commissurotomy has been properly performed.

SINCE the inception of definitive cardiovalvular surgery in 1948 there has been much conjecture about the ultimate fate of the incised valve. One of the first questions asked by the patient contemplating valvular surgery is, “Will the valve close again?” To this query the physician has had no authoritative answer than to suggest that recurrence of the stenotic state seems unlikely, provided the full-blown rheumatic state does not return. While such an answer has satisfied the disturbed patient, it is the duty of the physician, if it is possible, to supply more factual information. To that end, the experience, both medical and surgical, in nearly 600 consecutive commissurotomies, performed over a period of five years, has been exhaustively reviewed.

The protean nature of chronic rheumatic valvular heart disease was recognized from the outset as a major obstacle to definitive prognostic conclusions short of several decades of observation. It is to be understood, therefore, that we are not attempting here to predict the future of these surgically treated patients either from the clinical or valvular standpoint. It is a presentation (as the material available lends itself to such analysis), of what has happened to the incised valve during this initial five-year period of observation. Therefore, rather than to draw specific conclusions as to the state of the valve from clinical material alone, the method of investigation which has been adopted comprises a study of the following factors. First and foremost, the operation of mitral commissurotomy must be clearly defined and the technic for its proper performance understood. Second, the pertinent literature has been reviewed to ascertain the experience of others. Third, all available autopsy material obtained in the early and late postoperative period has been studied with particular reference to the incised valve. Fourth, the clinical course and present subjective and objective findings of the oldest commissurotomized patients have been detailed.

Technical Considerations of Commissurotomy

The rationale and technic of mitral commissurotomy have been published on many previous occasions by the authors and others. Only certain features of this operation will be emphasized here as they have a very real bearing on the problem of recurrence of stenosis. The purpose of commissurotomy is twofold as it concerns the valvular lesion of stenosis: to enlarge the mitral orifice and to restore, insofar as the scarred valve tissue will allow, the maximum degree of valve leaflet mobility. To be most effective, this must be accomplished without the production of significant valvular insufficiency. To this end, the anterolateral commissure is cut or split through the

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Presented at the Annual Meeting of the American Heart Association, Section on Clinical Cardiology, Chicago, Ill., April 3-4, 1954.
Fig. 1.—An adequate commissurotomy necessitates complete anatomic separation of the fused valvular cusps from the stenotic orificial rim out to the annulus (c). This can be achieved best by initiating a cut through the thickened portion of the fused commissure at the rim of the orifice (a) and subsequently by either digital separation of the remainder of the commissure (b) or when necessary by multiple cuts with the hooked guillotine knife.

area of pathologic fusion outward to the myocardium at the atroventricular annulus (fig. 1). Thus, the two mitral leaflets are completely separated from each other at this angle and are capable of motion independent of each other, there now being no scar tissue bridge between them. In addition, the finger must be advanced through the valve opening for subvalvular dissection. Ofttimes, chordae tendineae and papillary muscle, fused to each other or to the myocardial wall, can be gently freed to add considerably to the desired overall improvement in valve function (fig. 2). Such a newly constructed valve orifice should readily admit the introduction of two fingers, placed side by side; this constitutes the ideal valve opening when dealing with the tissues usually encountered in rheumatic mitral stenosis. Under these circumstances, the posteromedial commissure may be left intact, for it is usually short, heavily indurated and ill-defined, due to the fusion and shortening of the chordae tendineae peculiar to this location in the valve ring. Frequently, however, a centimeter or so of commissural separation can be accomplished in this area as well, to further enlarge the orifice and to increase the mobility of the valve leaflets.

Anything less than the above-described commissurotomy is not considered to be ideal, although, to be sure, the distortion of the valve tissues, the imbedded calcium, the shortened, fused and puckered chordae and papillary muscles may prevent the operator from accomplishing this desired ideal in a number of cases. It must be the pathologic changes of the valve tissues that dictates the limitations of the operative procedure and not the surgeon's ignorance of what constitutes an ideal commissurotomy. Within the past two years, intracardiac valvular surgery of this type has suddenly become the ambition of almost every
A surgeon who has ever had occasion to open the thorax, and, as a result, commissurotomy is now being attempted by many operators who have had little or no experience in this field; nor a complete understanding of the fundamental surgical goal. For example, there are many who merely thrust the index finger through the tiny stenotic valve orifice, feel the tissues split to a degree on either side and rapidly withdraw the finger, feeling that a proper commissurotomy has been performed. Under these circumstances, the valve leaflets have not been adequately separated and a considerable bridge of fused tissue remains to prevent adequate restoration of valve motion (fig. 3). Most certainly such an incompletely divided commissure may act as the locus for reagglutination, either because of the proximity of the raw cusp margins, or from the deposition of fibrin and particulate matter leading to thrombosis. Even more experienced operators seem unaware of the fact that the base of the valve cone at the lateral angle of the commissure may lie parallel to the ventricular myocardium and be unrecognized without specific exploration for it. Such pliable commissures might well be opened by finger pressure or with a straight bladed knife or both to the point where the commissural tissue lies against the myocardium, leaving a centimeter or more of commissure unopened, with the operator under the impression that the valve angle had been completely opened (fig. 4). For these reasons and others, the dull hooked knife with a guillotine blade is preferred and used whenever the commissural tissues do not separate readily with a moderate degree of finger pressure. An understanding of these fundamental principles in the technique of mitral commissurotomy is obviously essential for the intelligent evaluation of the valvular status of patients so treated.

**Review of Literature**

A review of the literature reveals very little data on the subject of stenosis recurring after surgical intervention. This is understandable since the follow-up period of observation in the experience of most surgeons has been short. Five specific instances in which a recurrence of stenosis is postulated have come to the attention of the authors. In the cases reported by Jordan and Hellems, and by Donzelot and others, the suspicion that an adequate commissurotomy was not performed is entertained. In the latter instance it is stated in the protocol that due to the precarious condition of the patient the procedure was limited to a single thrust of the finger through the stenotic orifice. The statement, attributed to Wood, that in 5 per cent of the cases followed by his group, there was reactivation of the rheumatic process and recurrence of stenosis needs considerable clarification and more detailed information. One would hardly think that he meant to imply that the two conditions are one and the same. Janton and Soloff, the first to call attention to the occasional recurrence of rheumatic activity following surgery on the rheumatic heart, have not observed what could be considered a return of structural valvular stenosis, and indeed they feel, that such a stipulation could hardly be made on clinical grounds alone. Avery and Priest have noted the reagglutination of an adequately opened posteromedial commissure in a heavily calcified valve. In such a badly damaged valve and with the commissurotomy necessarily localized to

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*Fig. 4.*—In some instances, even though care is taken to attempt a separation of the cusps out to the annulus, the false impression that this is achieved may be the result of "pressing" the relatively pliable edge of the membranous "cone" against the myocardial wall (a and b). This pitfall is indicated as it may occur in the course of finger dissection or even when cutting of the commissure is attempted with a straight bladed instrument. (c) The guillotine knife with the blunt hooked end is particularly valuable in assuring that the membrane be held and effectively cut.
the posteromedial angle (the area of least excursion of motion even in a normal valve), sufficient valve motion could obviously not be restored; under such circumstances one might rarely expect a thrombus to plug the orifice in this manner. In a case early in the experience of Julian, an anterolateral commissurotomy was thought to have been made, enlarging a 5 mm. orifice to 18 mm. The excellent clinical result obtained terminated in 18 months with a recurrence of the patient's original symptoms. At reoperation, "the mitral valve orifice was again found to be 5 mm. in length, but this time, being somewhat more expert with the handling of such fibrous valves, the opening resulting from incisions along both anterolateral and posteromedial commissures was over 3 cm. This patient has again recovered normal activity." Obviously the original commissurotomy was either very small or consisted primarily of orificial dilatation.

Muller has reported the findings in postoperative autopsies on two patients, one at seven months, in whom no evidence of restenosis was found. Brock has stated that in the absence of reactivation of the rheumatic process restenosis is unlikely. He elaborated on the mechanism of possible reocclusion in that situation in which a mass of fused chordae and papillary muscles might produce a subvalvular site for thrombosis. In this regard, it might be permissible to theorize and speculate on the possible mechanism of fusion of incised valve elements whether effected by old or recent rheumatic activity. In mitral stenosis, at the stage in which commissurotomy is performed, one is dealing with a densely scarred structure which has admittedly no blood supply within its densely fibrous or calcified substance. Tissues with these characteristics are notorious for their poor healing qualities. On this basis and in the absence of a reactivation of the rheumatic process which involves the valve elements proper, it seems quite unlikely that fusion can recur. However, due to inadequate pliability of the leaflets, or due to distorted anatomy (foreshortening and fusion of the chordae tendineae and papillary muscles into a dense subvalvular column), when such a commissure is cut, a deep welled crevice is created. Vascular particulate matter and local stasis of blood can predispose to thrombosis or deposition of fibrin leading to subsequent organization and fibrosis. Hence narrowing of the mitral orifice may result. As stated, the prevention of this form of recurrence depends to a great extent upon the adequacy of the technic of commissurotomy.

In just under 600 cases of mitral commissurotomy, there has been a total of 42 deaths in the late postoperative period. The ultimate clinical course and the cause of death in five of these cases is not known. The remaining 37 cases were adequately followed to the time of their demise. Of these the longest survival following an adequate commissurotomy was 36 months. There was not a single instance in this group in which death could be attributed to the recurrence of mitral stenosis. As shown (table 1) the cause of death in the majority was the far advanced stage in which the patient presented himself for surgical treatment. That is, these patients were in the group referred to as a functional stage V, in which irreversible cardiac, pulmonary, hepatic and/or renal changes existed. Such cases do not obtain benefit from commissurotomy. In a significant number of patients, mitral insufficiency or aortic valvular disease was an important or even major factor in the ultimate outcome. In several, existing mitral insufficiency was further aggravated by surgery.

### Table 1.---Causes and Contributing Factors in the Deaths of 42 Patients in the Late Postoperative Period

<table>
<thead>
<tr>
<th>Cause</th>
<th>Cases</th>
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<tbody>
<tr>
<td>1. Stage V cases which showed progressive failure due to irreversible state despite adequate commissurotomy</td>
<td>5*</td>
</tr>
<tr>
<td>2. Stage III to V cases in which multivalvular disease was an added factor</td>
<td>10*</td>
</tr>
<tr>
<td>3. As in 2 but mitral insufficiency was aggravated by surgery</td>
<td>10*</td>
</tr>
<tr>
<td>4. Pure mitral stenosis but in which significant mitral insufficiency was produced surgically</td>
<td>1</td>
</tr>
<tr>
<td>5. Adequate commissurotomy was not technically possible</td>
<td>3</td>
</tr>
<tr>
<td>6. Subacute bacterial endocarditis</td>
<td>1</td>
</tr>
<tr>
<td>7. Unknown</td>
<td>6*</td>
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</tbody>
</table>

* One case in each group had presumed rheumatic activity postoperatively.
There was one patient with "pure stenosis" in whom severe mitral regurgitation was produced surgically. Group 5 in Table 1 is of importance. These three patients can be said to have died of essentially unrelieved mitral stenosis. In none of these was an adequate commissurotomy technically possible and therefore their obstructive lesion was not relieved. In one case the appendage was so tiny (child, age 4) that a finger could not be introduced into the atrium and despite a blind attempt to open the valve by means of a curved Kelly clamp the diastolic murmur persisted without change in quality. This patient died in congestive failure during another attack of rheumatic fever 22 months later. A second case had a densely calcified, thickened, puckered valve, and, although a slight commissural separation might have been accomplished (initial valve orificial size, one finger; opened to one and one half fingers), the course was one of gradual deterioration until his death 42 months postoperatively. The third patient (early in this series) had a completely thrombosed and obliterated appendage and the commissurotomy was attempted through a pulmonary vein (not a recommended technic). Access to the valve was unsatisfactory and although a slight separation was achieved, it was totally inadequate. The patient died 15 months later. In none of the three patients was there any postoperative clinical improvement. In only this last case was autopsy permitted and examination of the valve revealed the original slight degree of separation but no evidence of restenosis.

### TABLE 2—Length of Survival in 31 Autopsied Cases

<table>
<thead>
<tr>
<th>Deaths within 24 days of surgery</th>
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<tbody>
<tr>
<td>In O.R.</td>
<td>6 cases</td>
</tr>
<tr>
<td>Within 24 hrs.</td>
<td>5 cases</td>
</tr>
<tr>
<td>24 to 72 hrs.</td>
<td>4 cases</td>
</tr>
<tr>
<td>3 to 24 days</td>
<td>5 cases</td>
</tr>
</tbody>
</table>

Late Deaths

| 4 to 5 wks.                       | 3 cases |
| 8 to 13 wks.                      | 3 cases |
| 7 to 10 months                    | 2 cases |
| 15 to 18 months                   | 3 cases |


### Valvular Findings in 31 Autopsies

Thirty-one autopsies upon patients dying after mitral commissurotomy are reviewed. Table 2 lists the survival periods of these cases. In the postmortem protocols of 20 patients who died within the first 24 postoperative days, there was no evidence that restenosis had occurred nor was there any thrombotic material or granulation tissue in or about the incised commissures. In all, the separation of the incised commissures was clearly evident. Figure 5 shows the valve in one of these cases in which a typical commissurotomy is seen.

The findings in 11 autopsies performed upon patients who died in one to 18 months postoperatively are available. One of these patients who survived surgery for 13 weeks was presumed to have suffered a flare-up of rheumatic activity postoperatively. There was no indication of acute valvulitis in this case. In none of the 11 was there any evidence to suggest endothelialization of the cut commissures nor was there evidence of restenosis by any mechanism. Material currently available on five of these cases is presented and pictures of these five valves are reproduced in figure 5. Microscopic sections from the anterolateral commissure in four cases are illustrated in figure 6. It is of interest to note that even in the one case autopsied of the three noted in Table 1 (E) who had had an inadequate commissurotomy, the partial separation has persisted (not illustrated).

### Clinical Analysis

Although even a meticulously accurate clinical appraisal of the operated patient cannot be used as specific evidence to either prove or disprove the state of the incised valve, such an analysis lends considerable weight when it tends to corroborate the observed pathologic findings. Rather than indulge in a detailed discussion of the total number of cases in this entire series, it seemed wise to concentrate upon those patients, treated consecutively, who have lived the longest period of time and who, therefore, represent the best available span of observation. Ten patients in various clinical stages of rheumatic heart disease with mitral
FIG. 5.—Photographs of six valves at various intervals after commissurotomy. The anterolateral commissure, which has been cut in all these specimens, is shown towards the top of the illustrations. Note that in all cases the surgically produced separation has remained. Furthermore, there is no gross evidence of new cicatrization, fibrin deposition, granulation tissue reaction, nor thrombosis. Note that these valves are all badly scarred and calcified. The five-week old specimen is interesting because this case died of a purulent pericarditis and bacterial endocarditis. A zone of septic vegetations is present, located in the region of the annulus and atrial wall. There were no such vegetations on the margins of the valve cusps nor on the cut edges of the commissure. The 18-month specimen is from a patient who had an excellent clinical result and who died in a traffic accident (commissurotomy done elsewhere).

FIG. 6.—Sections from four valves taken at various intervals after commissurotomy: (a) three days, (b) four weeks, (c) five weeks, (d) seven months. The magnification is 10X. The figure shows the cut commissures separated and the surfaces of the cut of the previously fused cusps. (e, f and g) High power microphotographs of the cut edge which is marked by circles in a, b and d respectively. (h) A section from valve (d) from a zone upon which no surgical manipulation was carried out. The magnification of the last four illustrations is 400X. No evidence of healing or reagglutination can be detected in these specimens.
stenosis were subjected to commissurotomy in 1949. Three of these died in the immediate postoperative period. The other seven are living today, four and one-half to five and one-half years later. As such they are among the oldest living postcommissurotomy patients. So that the reader may form his own opinion as to their present status, individual summaries of the case histories have been prepared.

In the opinion of the authors, the referring physicians and the patients themselves, five of these patients (cases 1, 3, 5, 6 and 7) have obtained an excellent functional result. They are asymptomatic, require no cardiac medication and are living a normally active life. One (case 4) is improved but has not as yet reached her ultimate status since a tricuspid commissurotomy was necessary just eight months ago. One (case 2) is unimproved; in fact her condition understandably is slowly deteriorating after having obtained moderate improvement for three years. In view of her postoperative course it would seem likely that her myocardium is failing in the face of a valvular stenosis inadequately opened due to the type of pathology encountered.

For the past two years we have been impressed by the fact that the obvious functional improvement obtained in 75 per cent of our patients is not routinely reflected in the objective cardiac findings. For example, in these seven patients, five have retained some element of their original diastolic murmur although there is some diminution of the pulmonic second sound. Two have mitral systolic murmurs of the same intensity as heard before surgery.

Fluoroscopically and by complete roentgenographic studies, two patients (cases 1 and 3) show reduction in their cardiac size to practically normal limits, three show no appreciable change (cases 2, 5 and 7) and in two there is some increase in cardiac size (case 4 and 6). In general those who have shown a decrease in overall size (most patients will show a diminution or even a concavity in the pulmonary conus-pulmonary artery segment of the left border silhouette probably from obliteration of the left auricular appendage) were the smaller hearts initially. One must be careful to recognize in the larger hearts that postoperative reduction in size may merely mean that the patient was in subclinical failure just prior to or at the time of surgery. In such instances a more rigorous dehydration regime might have further reduced the size of the cardiac silhouette preoperatively comparable with that noted postoperatively, and which fallaciously has been attributed to the surgical intervention. These factors may greatly confuse much of the roentgen data forthcoming in the future.

That there was little or no significant electrocardiographic change observed was somewhat surprising. The four cases in normal sinus rhythm and the three in auricular fibrillation prior to surgery remain the same to date. Minor changes in the voltage and direction of the various complexes, while varying from time to time, have remained essentially unchanged in five (cases 1, 2, 3, 4 and 6). In cases 5 and 7, there was a slight decrease in the magnitude of the right axis deviation accompanied by an increase in the voltage of R waves in leads V5 and V6.

Physiologic studies are playing an ever increasing role in evaluation of patients undergoing cardiac surgery. In many instances, too much reliance has been placed on the cardiac catheterization findings originally regarded as almost "factual without question." Most observers now agree that such studies when performed on patients with acquired heart lesions must not be taken "out of context" but are of value only when subject to correlation with all other findings including clinical appraisal, fluoroscopic and electrocardiographic observations.

Each of these seven patients has been subjected to pre- and postoperative catheterization studies although these studies are by no means complete. The original examinations in 1949 were carried out during the embryonic phase in the development of our laboratory and consist of pressure recordings in the pulmonary artery and right heart chambers only. The results are tabulated in table 3. A significant decrease in pressure was obtained in four instances (cases 1, 3, 5 and 6) all of whom have remained clinically excellent to date. In case 7, also considered to be an excellent result four
and one-half years later, the early postoperative catheterization was technically unsuccessful. Additional catheterization data at the present time would, of course, be highly desirable and have been suggested to these individuals. Each patient, however, has declined with regret after having already been subjected to two, and some to three such studies. This attitude is understandable if the physician changes places with the patient. Case 4, the patient, who underwent mitral commissurotomy originally and tricuspid commissurotomy four years later, remains an enigma from the catheterization standpoint. Reference to her case history will clarify this statement.

**Case Reports**

**Case 1.** J. B., a 32 year old, white, male, miner and truck driver, was admitted to Hahnemann Hospital on Jan. 18, 1949, complaining of chest pain, cough, hemoptysis and shortness of breath. He gave no definite history of rheumatic fever. He had pneumonia in 1942, recovered and was taken into military service but discharged after five and one half months. Shortly thereafter, he noted the onset of periodic bouts of right chest pain accompanied by severe hemoptysis. These attacks occurred about four times a year and lasted one or two days. Exertional dyspnea and intermittent palpitations have been noted for several years. He was unable to work for some time prior to his admission to our service.

Cardiac examination revealed a blood pressure of 110/60. The lungs were clear. The heart showed a long apical diastolic murmur, an exaggerated first sound at the apex and the second aortic and pulmonic sounds equal. Regular rhythm was present at this time although he was known to have had transient atrial fibrillation in the past.

At operation on Feb. 2, 1949, a tight mitral steno-

**Table 3.—Catheterization Data**

<table>
<thead>
<tr>
<th></th>
<th>Preoperative</th>
<th>Postoperative</th>
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<tbody>
<tr>
<td></td>
<td>RA</td>
<td>RV</td>
</tr>
<tr>
<td>1. J. B.</td>
<td>90/17 (41)</td>
<td></td>
</tr>
<tr>
<td>2. E. W.</td>
<td>82/2</td>
<td>84/38</td>
</tr>
<tr>
<td>3. V. S.</td>
<td>141/5 (50)</td>
<td>143/56 (85)</td>
</tr>
<tr>
<td>4. R. M.</td>
<td>5/-2</td>
<td>14/4.5</td>
</tr>
<tr>
<td>5. T. S.</td>
<td>43/3 (16)</td>
<td>42/12 (22)</td>
</tr>
<tr>
<td>6. J. K.</td>
<td>60/4</td>
<td>81/31</td>
</tr>
<tr>
<td>7. S. G.</td>
<td>1/0</td>
<td>42/?</td>
</tr>
</tbody>
</table>

* Prior to tricuspid commissurotomy.
† Technically unsuccessful.

sis which would not admit the finger tip was found. The cusp margins about the orifice were rolled and beaded with calcium but the leaflets retained good pliability. The anterolateral commissure was split and cut and the valve opened to a size which would now admit two and one half fingers. The postero-medial commissure was not opened. (See fig. 7, J. B.) Left atrial pressures taken at surgery were 45/30 before commissurotomy and 14/2 after commissurotomy.

The patient made an uneventful postoperative recovery, was discharged on Feb. 15, 1949, and returned to his previous occupation two months later. He has been working full time in the coal mines and as a truck driver ever since. He requires no medication or dietary precautions. He leads a completely normal and full life in all respects. Present cardiac findings show a normal sinus rhythm and persistence of an exaggerated mitral first sound with a short, late presystolic mitral murmur with no systolic component.

The preoperative electrocardiograms showed normal sinus rhythm with right axis deviation and notched and deformed P waves in the limb leads. The postoperative tracings have remained unchanged except for slight decrease in the magnitude of the right axis deviation.

Before operation in the posteroanterior projection, the overall size of the heart was estimated to show 2 plus enlargement. The pulmonary artery and the right ventricular outflow and inflow tracts showed 2 plus enlargement. The left ventricle was of normal size. The pulmonary vascular markings were moderately accentuated. Mitral valvular calcification was not detected. At the present time, five and one-half years after operation, the overall size of the cardiac silhouette was considered to show 1 plus enlargement, as was the right ventricular outflow tract and pulmonary artery. There was a diminution in the convexity of the corresponding segment of the left border, noted in the posteroanterior view. In the left lateral view the left atrium appeared unchanged. There was a decrease in the ac-
centration of the pulmonary vascular markings. These changes are comparable with those seen in case 3 (V. S.), whose radiographs are illustrated in fig. 8. Cardiac catheterization data are shown in table 3.

Case 2. E. W., a 38 year old, white, housewife, was admitted to Hahnemann Hospital on April 6, 1949, complaining of "heart disease." She had rheumatic fever at the age of 12. Since that time she had been subject to exertional dyspnea, cough and progressively increasing hemoptysis. She was given digitalis when she was 16 but only for a short time. At the age of 25 she developed congestive failure and had another short course of digitalis therapy. She had been on digitalis from age 28 until admission. Ankle edema had appeared three years ago but none had been noticed in the past two years. In January 1949 the patient had a "renal embolus" and bronchopneumonia. She had progressively increasing dyspnea, orthopnea, cough, hemoptysis and nocturia despite adequate medication and had been unable to do her housework since January 1949.

Her cardiac examination revealed a blood pressure of 132/82. The lungs were clear. The important cardiac findings were: atrial fibrillation with an apical rate of 96, apical diastolic and systolic murmurs, and a moderately accentuated second pulmonic sound. The liver was tender at the right costal margin but not palpably enlarged. No ankle edema was observed.

At operation on April 20, 1949, the mitral valve was found to be greatly distorted by scarring and calcification. Due to the extent and location of the calcified masses, an adequate commissurotomy could not be done. However, the orifice, which originally would not admit even the finger tip, was opened to an estimated one and one half fingers. This was accomplished by splitting and cutting directly through solidly calcified tissues. No restoration of valve motion could be obtained so that the incised cusps tended to remain in close proximity unless the finger was inserted to separate them. (See fig. 7, E. W.) Although a drop in the left atrial pressure was noted (35/16 before and 7/—1 after the procedure), it was stated in the operative protocol that it was doubtful that this procedure would result in lasting improvement.

The patient recovered and was discharged from the hospital on May 10, 1949. Although she was able to do a little housework a few weeks after her operation and is still able to do so, she has not continued to improve. In November 1952 she had an attack of left chest pain which necessitated rest and inactivity. She has been slowly deteriorating ever since this episode, although there has been no increase in her digitalis requirements. She states that she had defi nitely felt improved postoperatively until November 1952 but has been slowly deteriorating since that time.

Present cardiac examination reveals atrial fibrillation, a loud grade III to IV apical diastolic rumble ending in an accentuated first sound, a rough, loud grade II to III systolic murmur transmitted to the left axilla and a markedly accentuated second pulmonic sound. The liver is 3 fingerbreadths below the right costal margin and is tender. There is 2 plus edema of feet and legs.

The preoperative electrocardiograms showed atrial fibrillation with marked right axis deviation and digitalis effect. The postoperative tracings to date show no significant change.

Cardiac x-ray studies showed a general 3 plus enlargement of the heart shadow in the preoperative posteroanterior view. The pulmonary vascular markings were greatly accentuated. The pulmonary ar-
FIG. 8.—X-ray films taken before surgery and several years after to illustrate the three types of changes noted in the seven cases presented. In case V. S. there was an obvious decrease in the size of the cardiac silhouette. In case T. S. there was no significant change in the size of the heart shadow. In these two cases there is significant decrease in the degree of pulmonary vascular congestive markings. There was apparent increase in the size of the cardiac shadow in case J. K. Note the change of the contour in the region of the left auricular appendage in all of these cases. This is due, in part, to the partial amputation of this structure.

tery and right ventricular outflow tract showed 3 plus enlargement. Enlargement of the right ventricular inflow tract was 2 plus; of the left atrium, 3 plus and of the right atrium, 2 plus. The left ventricle was of normal size. Mitral valvular calcification was marked. There was no significant change in the cardiac size postoperatively. Those findings are comparable to those of case 5 (T. S.), whose radiographs are reproduced in fig. 8. Cardiac catheterization data is given in table 3.

Case 2. V. S., a 29 year old, white, female, office worker, was admitted to Hahnemann Hospital on June 20, 1949, complaining of “rheumatic heart disease” since July of 1944, at which time she first noticed dyspnea and cough. One month later she had her first episode of hemoptysis (about 80 to 100 cc. of blood). This was repeated three times in the next three months during which time she developed frank congestive failure. During the ensuing five years she has been hospitalized five times, on each occasion in fulminating failure. She required constant digitalization, diuretics and sedatives. From 1947 to June 1949 she was unable to work except on very sporadic occasions, could not negotiate one flight of stairs and for the most part was confined to her room.

Cardiac examination revealed a blood pressure of 108/64. Her neck veins were engorged. Her lungs were clear. Her cardiac findings revealed a normal sinus rhythm, a sharp and accentuated mitral first sound and pulmonic second sound. There was a long diastolic murmur with presystolic accentuation at the apex followed by a short systolic murmur. The liver was not enlarged and there was no ankle edema.
She was operated on June 27, 1949. The mitral valve was found to be densely fibrosed but not calcified. The tiny orifice was eccentrically placed lying adjacent to the anterolateral myocardial wall. There was no identifiable anterolateral commissure. The posteromedial commissure was split and further opened with two cuts of the knife, the opening being enlarged from an estimated 5 mm. to about two fingers (fig. 7, V. S.). Very considerable valve motion was restored. Left atrial pressures immediately before and after the procedure were 32/17 and 23/7 respectively.

The patient had an uneventful recovery, was discharged from the hospital on July 11, 1949 and returned to her work one week later, against advice. She has worked regularly ever since. In November of 1949 she had a cholecystectomy and in 1952 an excision of a Bartholin cyst. She states that she has had no symptoms whatsoever since her surgery. She has not taken digitalis or any other cardiac medication since leaving the hospital. She now works five eight-hour days a week as a typist. She also does extra work on weekends as a waitress. She leads a full life, dances, bowls and does all her own housework.

Recent cardiac examination revealed a blood pressure of 104/70, a normal sinus rhythm, a grade II apical diastolic rumble with presystolic accentuation, slight accentuation of the mitral first sound and the pulmonic second sound. There is no evidence of congestive failure.

Preoperative electrocardiograms showed normal sinus rhythm with marked right axis deviation. The P waves were broad and notched in the limb leads. At the present time there is normal sinus rhythm, the P waves are now less notched and are decreased in amplitude. The previously marked axis deviation has reverted to normal.

X-ray films preoperatively showed 2 plus cardiac enlargement. The pulmonary artery and right ventricular outflow and inflow tracts were enlarged 2 plus. The left atrium was enlarged 2 plus and the left ventricle was of normal size. The pulmonary vascular markings were accentuated. There was no detectable valvular calcification. At the present time there is a striking decrease in the overall size of the heart (fig. 8, V. S.). Cardiac catheterization data is shown in table 3.

Case 4. R. M., a 36 year old, white female was admitted to Hahmemann Hospital on October 10, 1949, with a history of chorea at age seven. She had another rheumatic flare-up in 1932 and again in 1935. This last episode was followed by severe decompensation for which she was, rather surprisingly, kept in bed for eight years. She had ankle edema, ascites, orthopnea and dyspnea at times despite bed rest. Her condition improved very slowly after prolonged rest, digitalis and diuretics. Just prior to admission she still had edema, dyspnea, cough and orthopnea as well as "palpitations." Occasional hemoptysis had occurred and she suffered a peripheral embolic accident from which she recovered by treatment with conservative measures.

Her cardiac examination revealed a blood pressure of 102/70. The mitral first sound was sharp and the pulmonic second sound accentuated. There was a long, mitral, diastolic rumble and a grade I systolic murmur. Normal sinus rhythm was present. Her lungs were clear. Ankle edema was present. There were full neck veins with marked pulsations.

Mitral commissurotomy was performed on Oct. 14, 1949. The mitral valve was fused into the typical "cone shaped" membrane, pliable but thickened like the kid skin of a glove. There was a minimal regurgitation. The 2 cm. orifice was opened to 4 cm. by cutting the anterolateral commissure. The posteromedial commissure was not cut. (See figure 7, R. M.).

She recovered without incident and was discharged on Oct. 26, 1949. Her dyspnea and edema disappeared for four months. Soon after this she again developed peripheral edema. In 1952, she noted swelling of the face and breasts. Although her breathing had been easier, from that time on she noted progressive exertional dyspnea and fatigue. In May 1953 the diagnosis of tricuspid stenosis was made by catheterization. On July 17, 1952, she underwent tricuspid commissurotomy. The tricuspid orifice was estimated to measure 13 x 2 mm. It was opened by cutting two commissures to about 10 x 30 mm.

She had a smooth postoperative course and was discharged on July 28, 1953, on a strict medical regimen. Her condition has continued to improve to the present time.

Her present cardiac examination reveals a normal sinus rhythm, a faint diastolic blow to the left of the sternum in the fifth intercostal space and a sharp mitral first sound. She has remained on digitalis therapy.

The preoperative electrocardiogram showed normal sinus rhythm with marked right axis deviation and broad notched P waves in all limb leads. Her present tracing is essentially unchanged.

Preoperative chest x-ray films revealed 1 plus cardiac enlargement involving primarily the pulmonary artery, the right ventricular outflow tract and the left atrium. The pulmonary vascular markings were normal. At the present time there is a slight increase in the size of the cardiac silhouette, although, no actual increase in the individual chambers can be detected. The postoperative increase in heart size seen in this case is comparable to that of case 6 (J. K.) illustrated in fig. 8. The cardiac catheterization data is shown in table 3.

Case 5. T. S., a 28 year old, white male, butcher, was admitted to Hahmemann Hospital on Oct. 31, 1949, complaining of shortness of breath. He gave a
history of “growing pains” at age 15. In 1940 he was rejected for military service because of a “murmur.” From 1937 until 1940 he had suffered very frequent “colds, one after another.” In 1946 he suffered a severe, persistent “cold” accompanied by severe cough and hemoptysis. He was hospitalized and placed on digitalis. He was fairly well after this episode until May 1949 when he went back to work as a meat cutter (having given up this work some time previously). At this time, exertional dyspnea and tachycardia became quite severe and continued to progress until admission.

His cardiac examination revealed a blood pressure of 138/78. The lungs were clear. There was atrial fibrillation, a short apical systolic murmur and a soft, rumbling, mid-diastolic murmur at the apex with presystolic crescendo. The pulmonic second sound was accentuated. His liver was not palpable.

At operation on Nov. 7, 1949, the mitral valve was found to be of the consistency of kid glove skin and the cusp margins were studded with scattered beads of calcium. The orifice would not admit the tip of the finger. The anterolateral commissure was cut and digitally split, accomplishing a final opening of two fingers breadth. The posteromedial commissure was not opened (fig. 7, J. K.). No significant degree of regurgitation was noted. Left atrial pressures immediately before and after the procedure were 37/17 and 19/7 respectively.

He made an uneventful recovery, was discharged from the hospital on Nov. 19, 1949 and returned to work six weeks after surgery. He has been working full time as a butcher ever since. He has required no cardiac medication since surgery and remains completely asymptomatic. He is able to swim and play baseball. He has had no “colds” since surgery. In all respects he leads a completely normal and active life.

Recent cardiac examination revealed a blood pressure of 126/70, atrial fibrillation, murmurs, the same as preoperatively. There is less accentuation of the mitral first sound and the pulmonic second sound. There is also a soft diastolic blowing murmur in the pulmonic area. The preoperative electrocardiogram showed atrial fibrillation with normal electrical axis. His recent tracing reveals no significant change.

X-ray films made preoperatively showed a 3 plus overall enlargement of the heart with moderate increase in the pulmonary vascular markings. The pulmonary artery and right ventricular outflow tract as well as the left atrium showed 3 plus enlargement. The left ventricle was of normal size. At the present time these x-ray findings show no significant change. (See fig. 8, T. S.) Cardiac catheterization data is given in table 3.

Case 6. J. K., a 17 year old, white, male, truck driver, was admitted to Hahmemann Hospital on Nov. 14, 1949, complaining of shortness of breath and frequent “colds.” He had had rheumatic fever at ages 6, 9 and 11. He has had progressively increasing dyspnea, orthopnea, cough and hemoptysis. He was unable to work or to participate in sports or recreation with youngsters of his age. He had been on digitalis in recent months.

His cardiac examination revealed a blood pressure of 130/78. His lungs were clear. The cardiac findings revealed atrial fibrillation and a long, mitral diastolic rumble with presystolic accentuation. The mitral first and pulmonic second sounds were accentuated. There was no liver enlargement and no edema of lungs or extremities.

He was operated on Nov. 23, 1949, and the mitral valve was found to be of the consistency of kid glove skin. There was considerable fibrous thickening about the orifice which was too small to admit the tip of the finger. The anterolateral commissure was cut and the valve opened to about one and one-half fingerbreadths (fig. 7, J. K.). Due to the fact that the auricular appendage purse-string suture broke at this point with ensuing hemorrhage no further manipulation or exploration of the valve was accomplished.

The patient recovered without incident and was discharged on Dec. 8, 1949. He did not return to work until a year after surgery when he obtained a job as a truck driver. He worked regularly for two years and was then laid off, remaining unemployed to the present time. He reports that during the past one to two years he has noted some dyspnea on heavy exertion. He remained on digitoxin for more than a year postoperatively but takes no medication now. He is depressed at present due to his inability to find employment.

At last examination in March 1954, by Dr. John Lenox, Myers Clinic, Philippi, West Virginia, he was reported to be much better than before surgery but not quite as well as last year. The lungs were clear. The heart sounds were essentially the same as those noted preoperatively. The pulmonic second sound was less pronounced. Atrial fibrillation was present.

The preoperative electrocardiogram showed atrial fibrillation, marked right axis deviation and digitalis effect. The tracing of March 9, 1954, was essentially unchanged.

The preoperative x-ray films showed a 2 plus overall enlargement of the heart. The pulmonary artery, right ventricular outflow tract and left atrium were enlarged 2 plus. The right atrium and right ventricular inflow tract were enlarged 1 plus. The left ventricle was of normal size. The pulmonary vascular markings were moderately accentuated. At the present time, there is generalized increase in heart size from 2 to 3 plus noted especially in the transverse diameter and the right ventricular outflow tract. The left atrium shows no significant
change. (See fig. 8, J. K.) Cardiac catheterization findings are shown in table 3.

It may be that due to the technical difficulties with the appendage at the time of surgery resulting in considerable hemorrhage a complete commissurotomy (cut out to myocardium) was not accomplished. Possibly he may be reconsidered for reoperation in the future should his course ultimately prove to be not entirely satisfactory.

Case 7. S. G., a 35 year old farmer and truck driver, was admitted to Hahnemann Hospital on Nov. 16, 1949. He had no history of rheumatic fever. He complained of progressive dyspnea since 1946 and orthopnea since 1948. He had been digitalized since 1947. There was obvious progression in his dyspnea and fatigue during the past year.

His cardiac examination revealed a blood pressure of 132/90, a normal sinus rhythm and a sharp mitral first sound. There was a long mitral diastolic rumble with presystolic accentuation followed by a grade II apical systolic murmur. The lungs were clear. The liver was not enlarged and there was no peripheral edema.

On Nov. 28, 1949, he underwent mitral commissurotomy. The valve was found to be tightly stenosed, moderately flexible and moderately calcified. It was opened by splitting and cutting both commissures to two fingerbreadths with restoration of some valve function (fig. 7, S. G.).

Following his discharge from the hospital on Dec. 23, 1949, he returned to work as a truck driver. He has been drinking and working excessively and has failed to follow his doctor's advice to moderate his activities. He had some dyspnea on heavy exertion in the early postoperative months but is now completely asymptomatic and able to carry on a normal active life without cardiac medications.

His present examination reveals a normal sinus rhythm, a slightly exaggerated mitral first sound but no diastolic murmur. There is a soft grade I systolic murmur at the apex.

The preoperative electrocardiogram showed normal sinus rhythm with slight right axis deviation and broad notched P waves. His recent tracing shows no significant change other than a decrease in the degree of right axis deviation.

The preoperative x-ray showed a general cardiac enlargement of 2 plus. The pulmonary artery, right ventricular outflow tract and left atrium were enlarged 2 plus. The left ventricle was of normal size. The pulmonary vascular markings were markedly accentuated. At the present time, there is no significant change in cardiac size. There is some decrease in the pulmonary vascular markings. The findings in this case are comparable to those of case 5 (T. S.) illustrated in figure 8. The cardiac catheterization data is given in table 3.

Summary and Conclusions

1. There is as yet no authoritative answer to the question of whether restenosis of the mitral valve will eventually follow commissurotomy. Undoubtedly it will take many years of careful observation before the proper answer can be unequivocally given.

2. The importance of clearly defining the correct technic of mitral commissurotomy, bearing in mind the objectives of this procedure, is emphasized. Only by having a clear understanding of precisely what was accomplished at surgery will it be possible to assess the course of the operated patient and to determine whether restenosis has occurred or whether the original stenotic state was adequately relieved initially.

3. A review of the literature revealed isolated instances of so-called restenosis after mitral valve operations. The authors feel that the conclusion that restenosis had occurred is highly speculative in view of the strong indications that adequate commissurotomy (either due to technical reasons or to the pathology present) was not achieved.

4. In an attempt to obtain at least initial clarification of the problem now that over five years of experience with the operation is available, our entire series of approximately 600 cases (both living and dead) was reviewed.

5. A total of 42 patients have died in the late postoperative period. The causes for these deaths have been outlined. In no instance could death be attributed to the recurrence of mitral stenosis.

6. The autopsy records and material available in 31 cases (early and late deaths) were reviewed, the longest survival among these 31 cases after a technically adequate commissurotomy being 36 months. Again, in no instance was there evidence that death was due to recurrence of mitral stenosis. Serial sections of the valves showed little or no evidence of true endothelialization of the cut surfaces, no evidence of active rheumatic valvulitis and no evidence that any process which might lead to mechanical occlusion of the mitral orifice had occurred. Indeed, many months after operation the tissues and edges of the cut valves appeared
to be practically the same as those observed only one or two days after commissurotomy.

7. Seven patients followed from four and one-half to five and one-half years, the oldest living commissurotomized patients, show no clinical, objective or laboratory evidence to justify the presumption that restenosis of the mitral valve has occurred. Five of these patients obtained and have maintained an excellent functional result to date and are living an active, normal life without cardiac medication. The sixth patient is improved but her ultimate functional status cannot now be completely assessed due to the additional performance of tricuspid commissurotomy just eight months prior to this report. The seventh patient, although alive and able to assume minor household duties, is essentially no better now than she was prior to surgery. In this instance the nature of her valvular pathology was such (extensive calcification) that a proper commissurotomy could not be performed at the time of surgery.

The fact that the obvious functional improvement obtained in approximately 75 per cent of our entire series of commissurotomized patients is not routinely reflected in the postoperative objective cardiac findings has been pointed out. This is readily understandable when one appreciates the fact that this surgery is performed for the relief of a mechanically strictured mitral valve and not for rheumatic heart disease as a disease complex. Those rheumatic stigmata and changes present in the myocardial and valvular tissues prior to surgery obviously remain after surgery. Should the patient's subclinical rheumatic state continue in the future, and there is no reason to suppose that it may not, unless present day medical therapy (antibiotics and other drugs) is bringing the disease under better control, it is entirely within reason that objective cardiac findings (electrocardiographic changes, heart size and other evidence) will remain essentially unchanged or even, upon occasion, progress. The point is that the patient whose valve has been properly opened with resultant relief of left atrial, pulmonary vascular and right heart hypertension is now in a far better mechanical state to cope with his rheumatic state; therefore emphasis to date has been placed on the patients' improved functional status. It is for this reason that the authors have continually stressed early operative intervention, once the rheumatic victim has demonstrated the pattern of obstructive symptomatic progression, before cardiopulmonary changes have advanced to a stage where they are on the verge of becoming irreversible.

Thus, based upon the data as presented, it may be stated that the mitral valve leaflets stenosed by rheumatic infection when adequately divided by commissurotomy have shown no sign of either partial or complete return to their previous state within a five year period.

**SUMMARIO IN INTERLINGUA**

Le destino final del commissuras incisionate in operationes pro stenosis mitral va continuar incognoscite in le proime futuro. Un evaluation clinic de patientes ancora in vita cinque annos post commissurotomia e un revista del disponibile datos autopic ab patientes qui superviveva le operation pro periodos usque a quatro annos indica que restenosis valvular non occurre durante le prime medie decade providite que le commissurotomia eseva execute correctemente.

**ACKNOWLEDGMENT**

The authors gratefully acknowledge the very considerable help and guidance given by Drs. Philip Custer and James Butcher of the Department of Pathology, Presbyterian Hospital, Philadelphia, Penna., in the preparation of the pathological aspects of this communication.

**REFERENCES**


DOES MITRAL STENOSIS RECUR AFTER COMMISSUROTOMY?

17 Avery, E. E., and Priest, W. S.: Personal communication.
18 Julian, O. C.: Personal communication.
Does Mitral Stenosis Recur after Commissurotomy?
ROBERT P. GLOVER, JULIO C. DAVILA, THOMAS J. E. O'NEILL and O. HENRY JANTON

Circulation. 1955;11:14-28
doi: 10.1161/01.CIR.11.1.14

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on
the World Wide Web at:
http://circ.ahajournals.org/content/11/1/14

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