Comissurotomy for Mitral Stenosis

By Robert P. Glover, M.D., Thomas J. E. O'Neill, M.D., and Charles P. Bailey, M.D.

The term "mitral comissurotomy" has been suggested to designate a procedure in which the individual anatomic leaflets of the stenotic mitral valve are surgically separated. By incising the angles or commissures of the mitral slit ("fish-mouth") a considerable degree of valve function can be re-established without the production of additional significant regurgitation. Comissurotomy, as described below, was performed in 8 cases of advanced mitral stenosis. Results in 5 were most satisfactory. There were three deaths early in our experience during the period when our technique was being perfected. (Noted in addendum are 22 cases undergoing the operation since this article was submitted for publication, and bringing the total series to 30.)

It was inevitable that the many recent advances in cardiac diagnosis and surgery would shortly foster invasion of the heart chambers. Of equal certainty was the fact that chronic valvular disease of the heart, the most common intracardial disorder, would be among the first conditions subjected to surgical consideration. That valvular disease is common and found universally is well known. Indeed, White\(^1\) has estimated that 0.5 to 1.0 per cent of the community at large is affected, particularly in those areas where rheumatic fever is endemic, as in northeastern United States and northern Europe. The great majority of persons afflicted with rheumatic heart disease (up to 85 per cent) develop some degree of deforming valvular disease, the mitral valve being the most common site of involvement. Structural stenosis of the mitral valve is the most deforming end-result of rheumatic infection.

The prognosis of an individual case of mitral stenosis depends upon many factors: the age of the patient, the severity of the lesion, the presence or absence of other valve defects, the presence or absence of additional rheumatic activity, and the condition of the myocardium. The ultimate outcome, however, is almost invariably unfavorable once the stenotic change gives rise to a progressive pattern. Thus, when the diagnosis of early mitral stenosis has been established the cardiologist can picture and predict with considerable accuracy, both by repeated physical examinations and observance of the patient's general condition, the structural and symptomatic phases through which a given case will pass. His treatment will at once be directed toward the prevention of further rheumatic insults and the support of a myocardium which is attempting to maintain adequate systemic circulation in the face of an increasing mechanical stricture. Under such circumstances both the cardiologist and the myocardium are fighting a losing battle. It is little wonder, therefore, that in his deliberations Sir Lauder Brunton\(^2\) as early as 1902 concluded that direct surgical incision of the stenotic valve provided the only logical method of interrupting the relentless chain of events attendant upon progressive mitral stenosis. Support of the myocardium by intelligent therapy has offered the only approach to date, but it is the treatment of the effects of disease rather than the alleviation of its mechanical cause. True, the ideal approach would be the elimination of the causative agent, rheumatic fever. Failing this, however, the correction of its disastrous cicatrical end result seems most logical.

Pursuing this line of reasoning there followed years of sporadic research, both experimental and clinical. Early investigation was concerned with an experimental production of mitral stenosis and insufficiency, providing material for the study of physiologic pressure changes so produced.\(^3\)\(^,\)\(^4\) Although even to this day true mitral stenosis simulating the clinical form of the disease has never been duplicated, cicatrical constrictions of the mitral ring have provided a medium for the thorough evaluation of obstructive phenomena upon the pulmonary circulation and right side of the heart.

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By 1929 Cutler and Beck7 were able to collect 10 cases from the literature in which surgical relief of mitral stenosis had been attempted. These efforts, in the main, had been made by the introduction of punchlike instruments through the myocardium of the left ventricle, blindly engaging upon a mitral valve cusp with the removal of portions of tissue, thereby enlarging the valve orifice but converting a lesion primarily stenotic into one primarily regurgitant. That this was poorly tolerated is evidenced by the fact that only one of 10 patients survived; thus, all surgical attempts fell into disrepute until very recent years. Powers,8-10 in 1932, demonstrated, again experimentally, that the abrupt conversion of mitral stenosis to mitral regurgitation with its sudden overwhelming increase in pulmonary congestion was responsible for the fatal outcome in patients so treated. This knowledge has prompted more recent investigators to direct their attentions at relief of stenosis by producing either small amounts (controlled) of regurgitation or, more ideally, no additional regurgitation at all. To this end, methods of grafting body tissues into the cardiac chambers to replace deformed valves have been devised but as yet have not been sufficiently perfected to warrant general usage.11, 12 Thus, since 1929, fourteen additional attempts (not including our own) to relieve mitral stenosis by plastic procedures upon the valve itself have been made.13-16 Nine of the patients survived the operation but a number have since succumbed as a result of induced, though possibly limited, mitral insufficiency. It is obvious, therefore, that future efforts must be directed at the relief of stenosis without the production of significant regurgitation if long-lasting benefits are to be derived from surgical intervention. The purpose of this communication is to present a method whereby the production of regurgitation has been so minimized as to be not significantly greater than that which inherently exists with all mitral stenosis. Therefore, it is incumbent upon the cardiologist to classify and re-evaluate mitral stenosis on a more exacting physiologic basis so that proper selection of cases for surgical intervention may be forthcoming. For this re-evaluation the work of Courand and associates17 and of Bing and his co-workers18 has been epochal and their methods of thorough study must be more generally applied.

Because of the possible promise of recent surgical developments, one is forced to the conclusion that heart disease as a whole and mitral stenosis in particular must shortly be viewed by the cardiologist from an entirely new perspective. Thus the first signs of developing mitral stenosis demand immediate and repeated evaluation by all possible methods. Careful clinical examination and appraisal by an experienced cardiologist is obviously essential. The cardiologist’s armamentarium has long included, in addition to subjective and auscultatory analysis: (1) electrocardiograms and sound recordings; (2) teleoroentgenograms, esophagograms, and fluoroscopy for an estimation of the size of individual heart chambers; (3) exercise tolerance tests; and (4) laboratory data for the detection of rheumatic activity. Such a program has been the standard means of determining the benefit derived from therapeutic measures. To this now must be added procedures for the detection of early physiologic changes. To evaluate cases for surgical intervention, there must be included (5) estimations of cardiac output at rest and with exercise as calculated by ballistocardiography or, more accurately, by utilization of the Fick principle; and (6) determinations of the pulmonary arterial and right ventricular pressures by cardiac catheterization.

Nothing need be said regarding the worth of the first four methods of evaluation. Their importance is unquestioned when carried out by experienced clinicians, even though many minor individual variances may lay stress on one finding rather than another. In many cases, however, the conclusions drawn from this type of evaluation alone may be dependent upon the presence of advanced pathologic changes. Significant pathologic and physiologic changes may well be detected earlier by the recently developed studies (5 and 6 in the preceding paragraph) and point the way toward reconstructive surgery before irreversible changes have occurred.
Certain basic questions must then be answered:

A. Has the primary rheumatic infection completely subsided? What is the probability of recurrence? What effect would recurrence exert upon the valve that has been subjected to operation? While no clear-cut answers can be provided, it seems reasonable that present activity can be detected and that future exacerbations can be prevented in large measure by the established practice of intermittent and long-continued chemotherapy or administration of antibiotics. Certain newer substances such as Compound E recently reported by Hench may prove to be of great value. In spite of precautions, should recurrent activity appear, in all probability further cicatrix would nullify the result of surgical treatment.

B. Is the valvular deformity such that the patient cannot reasonably expect a normal life span with but moderate limitation of activity? Preliminary evaluation of the patient by measuring the pressures within the pulmonary circuit may well be paramount in the making of such a decision. There is a strong suspicion that systolic pressures of 50 mm. Hg within the pulmonary artery may represent the critical level above which the patient must shortly expect serious consequences.

C. Finally, to what point may stenosis be permitted to develop before all reasonable chance of surgical relief is lost? Results obtained to date strongly suggest that in far-advanced cases in patients with fixed, calcified valves, who have or have repeatedly had heart failure, the opportunity for surgical relief has been lost. We believe that both the maintenance of a low surgical mortality and the degree of improvement desired require that the patient be a fair surgical risk, that his lesion be primarily early and predominantly stenotic, and that his myocardium, although strained by overwork, be capable of restoring a normal cardiac output once obstruction of the pulmonary outflow has been relieved. One may then ask: Why operate upon a person who is doing well? Surgery is not indicated, obviously, if a patient is doing well and preliminary and repeated evaluation indicates non-progression. On the other hand, many patients with established stenosis, although appearing to be reasonably fit, will demonstrate relatively early the progressive nature of their disease. Operative intervention at this earlier date will be repaid by a far better result and a lower mortality rate. Such reasoning may be likened to the urgency surrounding operative intervention in many other diseases, as in tuberculosis or carcinoma, before irreversible and widespread changes have occurred. As in all surgery for chronic progressive disease, what must be done should be done early.

ANATOMIC, PATHOLOGIC, AND PHYSIOLOGIC CONSIDERATIONS

The interplay of anatomic, pathologic, and physiologic factors, with reference to the mitral valve, when reduced to essential components, is readily understandable.

The normal mitral valve may be likened to a truncated cone of thin, flexible membrane. Its base is attached at the left atrioventricular ring, and its apex extends into the left ventricle. The apex and the outer surface of the apical half of this cone are suspended by numerous “guy wires” (chordae tendineae) attached to papillary muscles arising from the ventricular wall near the ventricular apex. The chordae tendineae are grouped more heavily in two areas of the mitral apex, at either side. These areas represent the anatomic fusion of the two components of the valve (an anteromedial and a posterolateral leaf), and represent the corners of the apex upon which the leaves fold themselves. The apical portion of the valve cone is somewhat folded or flattened upon these points. When modified by rheumatic disease, these points of folding become the “commissures.” The plane of flattening is oblique (with the patient in the supine position), running from the left anterolateral portion of the valve posteromedially. The anterior or anteromedial valve cusp is larger than the posterolateral, and is continuous with the posterior portion of the intracardiac aortic wall. During auricular systole the posterior or posterolateral cusp lies well away from the ventricular wall. During ventricular systole the ventricular wall may approximate and support it. Thus, mitral regurgitation is primarily a defect or dysfunction
of the anteromedial valve cusp. A defect of the posterolateral cusp produces only "limited regurgitation," rather than an uncontrolled reflux of blood, as seen when a defect in the continuity of the anteromedial cusp prevents normal deflection of the ventricular output into the intracardiac aorta. The papillary muscles contract synchronously with the ventricle, drawing the chordae tendineae taut, thus preventing the valve leaflets from becoming inverted or displaced backward into the auricle.

![Diagram](image)

**Fig. 1.—Pathologic changes seen in late mitral stenosis. Note the thin, pliable base of the valve (cross section).**

In rheumatic disease the mitral valve develops numerous minute cauliflower-like vegetations (1 to 2 mm. in diameter) in a row along the line of closure of the valve. Healing leads to the formation of scar tissue. With repeated infection and healing, there is gradual development of fibrosis, thickening, and narrowing and shortening of the apical portion of the valve cone. This scarring and narrowing may be very limited in extent to resemble merely a purse-string puckering of the valve orifice. In other instances, the disease involves one-fourth to three-fourths of the cone, leaving a flexible margin along its base (fig. 1). In far-advanced disease the whole valve becomes a rigid, completely inflexible, often calcified structure resembling a hard, ovoid plaque surrounding a small fish-mouth slit. Most physicians, and even many pathologists, think of such extreme deformity as classical and the rule in mitral stenosis. We have found this condition in only one of our 16 cases.

Usually mitral rheumatic disease hardens or fixes the apex of the mitral cone in the infolded or flattened position so that the commissures become an anatomic reality. Thus stenosis is produced, offering marked resistance to the passage of blood from the left auricle into the left ventricle. Some degree of regurgitation through the mitral slit during ventricular contraction is common. Not infrequently mitral rheumatic disease hardens and shortens the valve cone to a point where the leaves cannot be approximated. Such cases of predominant mitral regurgitation are less common than those predominantly stenotic, and at the present time are not amenable to surgical correction.

Mitral stenosis of any degree interferes with filling of the left ventricle, and thus with the maintenance of normal systemic cardiac output. Many such hearts are unable to increase their output over and above resting bodily requirements. Thus any appreciable amount of work will quickly cause patients with such hearts to become fatigued, dizzy, and even momentarily to lose consciousness. Concurrently, since the egress of blood from the left auricle is impaired, increased pressure within and great dilatation of this chamber results. The high intra-auricular pressure is transmitted to the entire pulmonary vascular system and thence to the right ventricle. A chronic pulmonary hypertension ensues, with nocturnal or exertional pulmonary edema (dyspnea), rupture of pulmonary capillaries (hemoptysis), and failure of the right side of the heart (enlarged liver, ascites, and peripheral edema).

**Surgical Considerations**

The possible surgical approaches to the problem of mitral stenosis seem to take one of the following three courses: (1) Methods of bypassing the stenotic mitral valve. (2) Methods of relieving the associated pulmonary hypertension. (3) Methods of direct surgical attack upon the stenotic valve.
By-Passing the Stenotic Mitral Valve. To our knowledge very little has been attempted or accomplished along the lines of by-passing the mitral valve. In 1913 Jeger suggested that a valved vein might be grafted to serve as an anastomosis between the pulmonary vein and left ventricle, and thereby adequately sidetrack the stenotic mitral valve. Litwak working in our laboratory, has been able to produce such a by-pass by anastomosing a pulmonary vein directly to the left ventricle, utilizing a free graft of azygos, hemiazygos, or femoral vein. This was accomplished in five dogs with function observed for several days, but thrombosis occurred within two weeks to one month in all the dogs.

Satinsky, also in our laboratory, attempted quite a different type of by-pass. He divided the subclavian artery in dogs and anastomosed the distal end of this vessel to a pulmonary vein. The operation was deleterious to normal dogs, causing death, but conceivably might be of some value were the conditions of clinical mitral stenosis present.

Relieving the Associated Pulmonary Hypertension. Various methods have been devised to relieve the associated pulmonary hypertension which accompanies mitral stenosis:

1. Harken and his co-workers have suggested that a measure of improvement may be afforded by removal of the cardiac accelerator and afferent nerves to the heart. Relief so obtained is through the production of a slower heart rate, thereby increasing ventricular filling time or by the interruption of pain fibers. He has observed symptomatic relief in a patient so treated, but suggests that such an approach can only be one of palliation. We have had no experience with this form of treatment.

2. Attempts have been made to destroy the function of the tricuspid valve with the production of tricuspid regurgitation to prevent easy access of blood into the right ventricle and lower its pulmonary output. In our opinion such an approach has little to recommend it.

3. The production of a communicating shunt between the pulmonary and systemic venous systems, thus affording a measure of relief to the hypertension within the left auricle, has some merit. An anastomosis between the azygos and pulmonary veins has been accomplished by Sweet with subjective relief of distressing pulmonary symptoms.

4. Both Harken and our group have produced interauricular septal defects to relieve the pulmonary hypertension of mitral stenosis. Such a venous shunt will reduce the hypertension and strain within the left auricle, and secondarily, the associated hypertension throughout the pulmonary vascular bed and right ventricle. We have obtained pressure readings within the pulmonary artery in certain cases of mitral stenosis which were higher than those within the aorta (150 + mm. Hg). Theoretically, a venous shunt should relieve strain on the entire lesser circulation, and one would anticipate relief of such symptoms as hemoptysis, acute attacks of pulmonary edema, and right-sided heart failure. It is probable that such an effect is accomplished to a degree.

Unfortunately, reduction in the left auricular pressure without enlargement of the mitral orifice results in a destruction of the compensatory mechanism which nature has set up to force blood through the narrowed mitral valve. With the production of such a shunt, the left ventricular output falls. The amount of fall is related to the relative sizes of the shunt orifice and the mitral orifice. Obviously, no patient can withstand a shunt if the cardiac output during exercise does not materially increase over the resting requirements. By the same token, it is essential even in those who can so increase their output, that this should not be cut below the level of resting requirements. Since in most clinical cases the patient can do no more than double his resting output, it follows that one should not produce an opening even as large as the stenotic mitral orifice unless it is planned to render the patient totally bed-fast.

It has been repeatedly stated that nature itself has produced a similar combination of defects in the form of Lutembacher’s syndrome. Indeed, the syndrome does embrace both a mitral stenosis and a large auricular septal defect. In this instance it must be remembered that compensation has been established over many years by gradual changes in the heart.
and by a great increase in the total blood volume. These patients are said to do well, or at least somewhat better than those with "pure" mitral stenosis. Taussig, with reference to this syndrome, states that, "The blood so shunted" (through the auricular septal defect) "passes into the right ventricle and thence is pumped out through the pulmonary artery to the lungs and is again returned by the pulmonary veins to the left auricle. Thus, an excessive amount of blood is pumped around and around the lesser circulation; whereas the left ventricle, aorta, and systemic circulation receive less than their normal quota of blood. The right auricle and ventricle are enlarged. The pulmonary artery is usually twice the size of the aorta. The strain on the left auricle is relieved by the defect in the auricular septum. Therefore, the left auricle is not enlarged. The left ventricle is small." For a time, therefore, the strain on the left auricle and pulmonary vascular bed may be somewhat relieved, but only at the expense of producing a similar strain on the right ventricle, a condition equally serious. Again quoting Taussig, "The late development of cardiac difficulties" (in Lutembacher's syndrome) "occasionally occurs after a relatively minor illness. For example, a patient who has never been known to have any cardiac abnormality, after some slight illness may suddenly develop symptoms which lead to progressive heart failure." It would seem that any condition which causes such great pulmonary arterial and right ventricular enlargement can scarcely be considered desirable. Actually, on an average this defect terminates fatally when the subject is 40 years of age: slight, if any, improvement over longevity in uncomplicated mitral stenosis.

Indeed, the production of a venous shunt for mitral stenosis seems somewhat comparable to the production of an arterial shunt (systemic artery to pulmonary artery) for the treatment of congenital pulmonary stenosis. The production of an artificial ductus arteriosus is life-saving in cases of severe pulmonary stenosis. Too large a shunt is promptly fatal, however, and any arterial shunt increases the load on the myocardium, leading to eventual cardiac enlargement. Helpful as these shunts have been, if a method of direct and successful attack upon the pulmonary stenosis were to become available, we have no doubt that all concerned would promptly embrace the more direct procedure. It would then be necessary, although perhaps not practicable, to recall these patients for the performance of direct valvular surgery and subsequent division of the artificially produced "patent ductus arteriosus." This would also be the case with the venous shunt. If such shunts are created to save life in the face of demonstrably superior direct valvular attack, we will soon be faced with the problem of repairing them.

Direct Attack Upon the Mitral Valve. This undoubtedly has been contemplated for many decades. Brunton, in 1902, suggested that the only proper and logical approach to the problem of mitral stenosis was to "lengthen the slit." How sound his judgment was will become evident shortly.

Since the first section of the mitral ring by Elliott Cutler in 1923, there have been a number of direct attacks upon the stenotic mitral valve. The approach and the methods employed have varied. Thus, the left auricular appendage as a site of entrance has been utilized by Allen and Graham (1922), Souttar (1925), Smithy (1947), and Harken (1947); the left ventricle by Cutler, Levine, and Beck (1924), Pribram (1925), and Smithy (1947); and the left pulmonary vein by Harken (1946). It is our considered opinion that the approach through the auricular appendage is far superior to any other. It leads directly into the wide opening of the mitral funnel and thence to its stenotic orifice. There are no chordae tendineae to interfere with the passage of the instrument or finger. In addition, no serious disturbance of cardiac rhythm or function is produced. The finger or instrument is well tolerated in the roomy left auricle, unless the actual passage of blood into the ventricle is obstructed for more than three beats. In addition, the appendage may be readily and securely ligated at completion of the operation.

On the other hand, the left ventricular approach is obstructed by chordae tendineae and does not insure ready or accurate localization.
of the small opening of the mitral funnel. Such an approach may provoke a serious arrhythmia, does not permit digital insertion or palpation, and may be difficult to close securely.

Methods of dealing surgically with the valve have included: (1) simple incision of a valve cusp, (2) excision of a portion of the mitral ring, (3) digital dilatation of the stenotic orifice, (4) valvuloplasty, and (5) commissurotomy.

1. Simple Incision of a Valve Cusp: Simple incision of a mitral valve cusp has a very deleterious effect upon the experimental animal. If the anteromedial or "aortic" cusp of the mitral valve is completely divided, death is prompt and almost immediate. If the posterior cusp is completely divided, death is usual within twenty-four hours. While Cutler's first patient lived four and one-half years after simple incision, it appears that the cusp was not completely divided. Dogs, too, will tolerate lesser degrees of valve section. One of the objections to simple incision of the ring is the possibility of healing at the site of the incision. It seems improbable that such healing would occur if the valve were widely incised to its base, since the edges would then gape widely during most of the cardiac cycle. On the other hand, if the incision only extended partially through the scar tissue, it could not gape and very probably healing and further cieaatrit would occur. We have never observed healing in an adequately incised heart valve in dogs, followed for periods up to twelve months, or in one patient followed three months postoperatively. Of three such patients reported by Cutler, Levine, and Beck, two died shortly after surgery.

2. Excision of a Portion of the Mitral Ring: Simple excision of the valve ring was practiced by Cutler and Levine (1923),24 by Cutler, Levine and Beck; also by Pribram, and later by Smithy. Since it was felt that the only hope in mitral stenosis was to replace it by a regurgitant type of lesion, partial excision of the valve cusp seemed to be a logical procedure. We are unequivocally opposed to this concept, since experimentally and clinically it has been demonstrated that a suddenly produced mitral regurgitation is poorly tolerated and is as serious a lesion as the original stenosis. Results following this form of treatment have been discouraging in the past. Thus, four patients so treated by Cutler, Levine, and Beck, and one by Pribram, all died within six days after operation. In June, 1948, Smithy reported five living patients out of seven so treated. His better results may be attributed partially to advances in anesthesia and surgery. More pertinent, however, is the fact that by design he excised relatively small pieces of valve tissue. Time will supply the answer as to how well his patients will carry on with their increased, though limited, regurgitation.

3. Digital Dilatation of the Stenotic Orifice: Digital dilatation of the stenotic valve was first practiced by Souttar in 1925 with success in one case. We have since performed three such dilatations with one success.25 26 The first of these was attempted on June 12, 1946. In a practically moribund woman a very hard, calcified valve slit was dilated with marked temporary improvement. Death within three days was disclosed at autopsy to have been the result of clotting at the torn commissures. It was evident that the valve had torn at the line of the commissures sufficiently to establish some temporary valve function. Thrombosis had quickly re-established or increased the stenosis and caused death. At postmortem examination the idea of cutting the commissures well into normal valvular tissue under direct digital guidance or "vision" was born. Since that time we have had to resort to simple digital dilatation in two additional cases, with one success.23 We are well aware from studies upon stenotic mitral valves incidentally found at autopsy that simple digital dilatation does not always result in tearing the fused fibrotic commissures. The ring tears at its weakest point, which may well be across a cusp. Even when the tearing does occur at the commissures, one finger is seldom large enough to force the tearing to extend beyond the fibrotic tissue into normal valve structure. However, this early case of dilatation did result in what we then considered to be an entirely new concept of treatment.

4. Valvuloplasty: Harken has used the term "valvuloplasty" to describe his procedure of resection of portions of the valve ring at the commissures. He rightly recognizes that re-
section will best be tolerated if performed at the commissures, assuming that resection with limited or selective regurgitation is the desired result. Two such operations have been performed. In one the pressure in the left auricle was 450 mm. water, but rose above the readings of a 500-mm. manometer after valvuloplasty of this type. This was attributed to a coincident tachycardia. The patient died in pulmonary edema twenty-four hours later. In the other patient, left auricular pressure dropped from 450 mm. water to 400 mm. during the procedure. The patient was improved subjectively. Localization of the commissures was accomplished by palpating the valve with the valvulotome inserted through the auricular appendage.

5. Commissurotomy: Commissurotomy is a term suggested to us by Dr. Thomas Durant of Philadelphia to designate the procedure which we have employed to re-establish a marked degree of normal mitral valve function. Our present operative technic is simplified and direct. It has been described in detail in a previous publication. Very briefly, it consists of an operative approach through the left anterior chest wall with the patient in the dorsal recumbent position. The pericardium is incised longitudinally one-half inch anterior to the phrenic nerve as it courses downward over the left lateral aspect of the heart. The huge distended auricular appendage now protrudes from the pericardial sac. A purse-string suture of heavy braided silk is passed about the appendage at its base. A Satinsky clamp is closed over the base of the appendage and a generous portion of its tip is amputated. Two gloves are worn on the right hand. An opening is made in the outer one on the palmar surface at the base of the index finger, and another at the tip. The blade of the commissurotomy knife is inserted between the gloves on the index finger (fig. 2), and the finger is inserted into the left auricle as the clamp is released and as the purse-string suture is pulled taut. The finger is well tolerated by the auricle and causes no disturbance to the circulating blood. The valve is quickly and easily located. Its structure can readily be appreciated; the size of the opening and the location of the commissures are determined. The knife is now protruded through the orifice and the hook is engaged upon the anterolateral commissure (fig. 3). A backward stroke usually divides the commissure adequately the first time. The finger again palpatates the opening and gently dilates it. If the cut in the commissure does

![Figure 2](http://circ.ahajournals.org/)

**Fig. 2.**—Commissurotomy knife inserted between two gloves on the palmar surface of the right index finger.

![Figure 3](http://circ.ahajournals.org/)

**Fig. 3.**—Right index finger and commissurotomy knife in left auricle. The stenotic valve is explored; the knife is protruded through the orifice and engaged upon the anterolateral commissure.
not extend well into normal tissue the backward stroke is repeated. In patients who have a rather soft valve orifice markedly diminished in cross-sectional area, simple opening of the lateral commissure is in our opinion sufficient. On the other hand, with a rigid, fixed, and sometimes calcified valve, incision of the medial commissure as well may be necessary.

The finger and knife are now deftly withdrawn from the auricle as the previously placed purse-string suture is drawn tight, preventing more than a few cubic centimeters of blood loss. The suture is tied and the cut edge of the appendage is oversewn. There should be no dis-

This is accomplished by pronating the hand, engaging the medial commissure, and repeating the cutting maneuver as described above.

In order to handle very hard or calcified valves, we have prepared a special narrow-bladed backward-cutting punch (fig. 4). This has a long bite so that it, too, will cut completely through the diseased tissue and into the flexible membranous valve. Thus, we still obtain practically the same effect as by simple incision of the commissures but without the advantage of simultaneous digital guidance.

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FIG. 4.—Narrow-bladed commissurotomy punch with trocar and cannula

### Table 1.—Cardiac Catheterization Studies in One Patient (J. B., Case 6)

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</table>
function. It has the great advantage of direct digital guidance. It should not, and has not in any of our five successful cases, produced any detectable amount of mitral regurgitation. Blood loss is minimal and no apparent disturbance of heart function results. That it effectively accomplishes the desired result is shown not only by the marked clinical improvement in all living patients, but also by objective physiologic studies.

The following representative table (table 1) reveals the pressure changes observed in one of our patients studied before and after commissurotomy. All pressures were recorded with a Lilly electronic manometer. The pressures in the left auricle were taken directly with the heart exposed immediately before and im-

mediately after cutting the valve. Two other patients studied in this manner showed similar changes which have been published elsewhere. In none of these patients was there any appreciable blood loss, significant change of rate or rhythm, or disturbance of systemic circulation. We believe that these changes in the left auricular pressure indicate two things: first, the mitral stenosis was at least partially relieved or pressure would not have fallen; second, no appreciable increase in pre-existing regurgitation was produced or the pressure would have risen.

Long-term follow-up alone can supply the ultimate answer to the effectiveness of commissurotomy. Nevertheless, since years must elapse for such information to become available, we feel justified in proceeding in those patients who meet our present rigid indications and

in whom it might be lifesaving. In this our thinking follows the line already established by Blalock and Taussig in their work on pulmonary stenosis. The day will undoubtedly come when an extracorporeal circulation will permit exclusion of the heart and lungs from active duty during intracardiac surgery. At that time it will be possible to open the heart widely and perform plastic procedures upon the valves and septa under direct vision. Such operations as commissurotomy will then become antiquated.

**RESULTS OF COMMISSUROTOMY**

Our early results were discouraging, but with improved selection of cases they have begun to show promise. A summary of our experience

<table>
<thead>
<tr>
<th>Patient</th>
<th>Date of Operation</th>
<th>Type of Operation</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. W. W.</td>
<td>3/22/48</td>
<td>Commissurotomy</td>
<td>Death in 6 days; technical difficulty</td>
</tr>
<tr>
<td>2. C. W.</td>
<td>6/10/48</td>
<td>Commissurotomy</td>
<td>Living; excellent result</td>
</tr>
<tr>
<td>3. S. S.</td>
<td>6/27/48</td>
<td>Commissurotomy</td>
<td>Living; excellent result</td>
</tr>
<tr>
<td>4. F. G.</td>
<td>7/13/48</td>
<td>Commissurotomy</td>
<td>Death in 8 days; sudden cerebral embolus</td>
</tr>
<tr>
<td>5. A. W.</td>
<td>9/2/48</td>
<td>Commissurotomy</td>
<td>Death in 24 hours; hemorrhage</td>
</tr>
<tr>
<td>6. J. B.</td>
<td>2/2/49</td>
<td>Commissurotomy</td>
<td>Living; excellent result</td>
</tr>
<tr>
<td>7. S. C.</td>
<td>3/23/49</td>
<td>Commissurotomy</td>
<td>Living; excellent result</td>
</tr>
<tr>
<td>8. E. W.</td>
<td>4/20/49</td>
<td>Commissurotomy and dilatation</td>
<td>Living; improved</td>
</tr>
</tbody>
</table>

with commissurotomy to date is appended in table 2. An abstract of each case has appeared in a previous publication. Commissurotomy by the technic described was performed in 8 cases with three fatalities. In the first of these (Case 1) a modified scalpel was employed, as our present commissurotomy knife had not been perfected. Attempts to incise the commissures were attended by repeated disengagement of the blade as it rode over the fibrotic valve, resulting in an inadequate incision extending only a few millimeters into the scar tissue. Pre- and postoperative pressure determinations in the left auricle were unchanged (320 mm. of water). The patient died on the sixth postoperative day in pulmonary edema from unrelieved mitral stenosis. The second patient (Case 4) was operated upon accurately and well, and appeared to be well
on the way to a most satisfactory result, but suffered a cerebral embolus and died suddenly on the seventh postoperative day. Autopsy disclosed an adequate and satisfactory commissurotomy. Thrombus formation had occurred in the left auricular appendage, from which the embolus apparently originated. We had observed such thrombus formation many times in animal appendages after valve surgery, but no adverse complications resulted. We had simply oversewn the incision in the appendage at the conclusion of the procedure. In the

Fig. 5.—Anteroposterior and right anterior oblique preoperative roentgenograms showing size of heart and left auricle (Case 1).

Fig. 6.—Anteroposterior and right anterior oblique roentgenograms made six months postoperatively showing reduced size of heart and left auricle and disappearance of pulmonary vascular congestion (Case 1).
4 succeeding cases (and in one previous one C. W., without purposeful design) we have ligated the auricular appendage at its base to prevent such disaster. The appendage of the third patient operated upon shortly thereafter was ligated at its base without suture of the cut tip. Autopsy indicated that hemorrhage had occurred from this site (Case 5). This experience has led to both ligation at the base and an oversewing suture at the cut tip on all subsequent cases.

The remaining 5 patients represent most satisfactory results. Four patients are clinically well. Two who had been digitalized for some months prior to surgery no longer need such medication. There is definite diminution in the size of the heart as shown roentgenographically (figs. 5 and 6). Their tolerance for exercise is remarkably increased, permitting them to return to normal activity. Careful examination reveals some slight residual evidence of mitral stenosis in 2 of the patients (Cases 2 and 6) and somewhat more in a third (Case 8) in whom conditions were not satisfactory for an ideal commissurotomy. The others have no residual signs of the disease, although admittedly the follow-ups are of short term. Electrocardiographic follow-up shows no significant change from the preoperative state. There has been no evidence of increased mitral regurgitation in any of the 5 patients.

INDICATIONS AND CONTRAINDICATIONS FOR SURGERY (AT PRESENT)

The indications and contraindications for commissurotomy must be considered together. Both must change as experience dictates. Common sense and our present experience have led to certain conclusions to date:

1. Most favorable group:
   A. Excessive fatigability.
      Increasing exertional dyspnea.
   B. No rheumatic activity.
      Normal sinus rhythm.
      Lesion predominantly stenosis.
      Evidence of significantly increased pulmonary hypertension.

2. Less favorable group: The above plus:
   A. Recurrent bouts of hemoptysis.
   B. Arterial embolic phenomena.
   C. Auricular fibrillation without failure.

Hemoptysis in more than amounts necessary to stain the sputum is of grave import. Wolf and Levine\(^{32}\) point out that in their series of cases the average duration of life following the onset of severe hemoptysis is 35.5 months. Levine\(^{33}\) stated that the average duration of life following the initial attack of congestive failure is 4.6 years. The development of auricular fibrillation is usually permanent and irreversible. In this state thrombus formation not infrequently occurs along the endocardium of the dilated and relatively immobile auricular walls. Some 75 per cent of these occur within the lumen of the auricular appendage (left), a common site for the origin of arterial embolization.

It follows, then, that our contraindications would be: (1) Active rheumatic infection. (2) Presence of superimposed subacute bacterial endocarditis. (3) Cardiac failure uncontrollable by medical means. (4) Presence of marked associated mitral regurgitation or other valve (aortic) deformities.

SUMMARY

Our entire experience with commissurotomy for mitral stenosis (8 cases) has been reviewed. There have been four very satisfactory results, one fair result, and three deaths. Had our present knowledge been applied to all cases, the mortality might have been appreciably lower.

Commissurotomy is a simple, direct, effective, and safe procedure. Its exact surgical technic has been outlined.

Commissurotomy accomplishes relief of mitral stenosis by restoring considerable valve function without the production of additional mitral regurgitation.

Digital guidance in the performance of accurate valve surgery is essential until a method of direct vision becomes established.

The left auricular appendage is the most satisfactory avenue of approach to the mitral valve.

The auricular appendage must be ligated at the conclusion of the procedure to prevent arterial embolization. This may prove to be the proper approach to the management of arterial embolization in many cases of auricular fibrillation unassociated with mitral stenosis.
An appreciably enlarged left ventricle in a case of supposed "pure mitral stenosis" is indicative of some additional significant valve lesion (aortic stenosis or regurgitation, or mitral regurgitation) and is by our present criteria contraindicative to commissurotomy.

Study methods of value in addition to clinical evaluation embrace cardiac catheterization for determination of pulmonary vascular pressure, ballistocardiography, and other physiologic studies for the determination of cardiac output.

Venous shunts for the treatment of mitral stenosis are of some value, although their eventual effects upon cardiac output, the right ventricle, and the lesser circulation must be kept clearly in mind.

**Addendum**

Twenty-two additional cases of advanced mitral stenosis have undergone commissurotomy since this paper was submitted for publication, bringing the total series to 30. Six patients died. In 21 the results have been satisfactory to date, both subjectively and by objective improvement similar to that noted in table 1. In 3, improvement has been considerable subjectively, but less striking objectively.

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COMMISSUROTOMY FOR MITRAL STENOSIS


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