The Responsibility of the Physician in the Selection of Patients with Mitral Stenosis for Surgical Treatment

By Dwight E. Harken, M.D., Laurence B. Ellis, M.D., Lewis Dexter, M.D., Robert E. Farrand, M.D., and James F. Dickson, III, M.D.

The anatomy of the mitral leaflets, a classification of the variations in pathologic morphology of rheumatic stenosis, and the evolution of a practical technic for relief of that stenosis while restoring valve action are considered. The life cycle of patients suffering from mitral stenosis is reviewed and a clinical classification of these patients is presented. Some indications and contraindications for surgery are discussed in the light of strength and weakness of present surgical methods. Results of surgical treatment are reviewed.

Many people badly disabled by mitral stenosis are now being greatly improved by surgical treatment. To those of us who have lived through the troubled era when most of the patients died from attempts at surgical correction, it is gratifying to see properly selected patients subjected to operation with a mortality rate that is not high in relation to the severity of their disease. These results breed enthusiasm in surgeons that may be dangerous. Conversely, the lack of appreciation by physicians of the remarkable advantages that may accrue from the surgical treatment of mitral stenosis is equally dangerous. The possibility of a proper place for surgical intervention in the life cycle of the disease must be investigated carefully and objectively. If in certain phases of the life cycle of the disease surgical treatment is the best treatment, it becomes the responsibility of all participating in the care of the patient to see that the best treatment is given. It is the physician who first sees the patient and therefore it is he who must first bear this major responsibility. Theoretically, in order to assume this responsibility, he should know everything about the life cycle of mitral stenosis and rheumatic carditis; he should also know what medical adjuncts are to be developed in the near future; he should be cognizant of present surgical possibilities and of the surgery that can be expected in the near future. This is, of course, impossible. We can, however, do our best to approximate such a position on the basis of information now available. That is the purpose of this discussion.

The following will be discussed: first, a review of the anatomy of the normal mitral valve; second, the morphologic types of mitral stenosis; third, various indirect and direct forms of surgical treatment and their shortcomings; fourth, the life cycle of mitral stenosis; fifth, a clinical classification of patients in various phases of the life cycle; sixth, which patients should have surgical treatment now and which patients should be deferred in view of present surgical shortcomings.

The Leaflet Pattern in the Normal Mitral Valve

As a background for the study of mitral stenosis and regurgitation it has been necessary to examine the anatomy of the normal mitral valve. Knowledge of the normal assists in making surgical valvuloplasty more nearly
restore the normal state. This has involved detailed gross examination of the mitral ring, its living animal heart and with clinical findings in diseased human hearts.

Fig. 1. This group, "normal," has four leaflets, triangular in shape, with their blunted apexes directed toward the center of the mitral orifice. The radial width of the aortic leaflet is approximately twice that of the ventricular leaflet. The two commissural leaflets may vary considerably in size but their width is usually two thirds that of the ventricular leaflet. This is the arrangement seen in 75 per cent of valves.

leaflets, chordae tendineae and papillary muscles in 35 consecutive normal hearts. The gross anatomic examination of the normal heart at autopsy was correlated with studies of the

The findings revealed that there is considerable variation in the leaflet arrangement. In general, the valve structure consists of a continuous veil inserting around the entire
circumference of the mitral orifice. This continuous veil is divided into leaflets by clefts, none of which reach the attachment. This is at variance with the usual concept of two separate leaves. The most prominent triangular projections of the veil are the aortic and ventricular leaflets, with blunted apexes extending toward the center of the mitral orifice. However, two additional triangular projections are commonly found that might be called the “anterior and posterior commissural leaflets” (fig. 1). Four triangular leaflets, arranged circumferentially, constitute the mitral valve.

While the shape of all four leaflets is essentially similar, their size and distribution of chordae tendineae vary greatly. The aortic leaflet is the largest; its radial length from the mitral ring is approximately twice that of the ventricular leaflet. The two smaller commissural leaflets which arise from the lateral margins of the major (aortic and ventricular) leaflets are but one-half to two-thirds of the length of the ventricular leaflets. Though small and fragile in appearance, their function in maintaining the integrity of the redundant portions of the two larger valve leaflets in systolic closure seems to be considerable. A valve pattern, as seen in 75 per cent of these hearts, will be called the “normal” (fig. 1). In the remaining 25 per cent, variation is principally in the size and shape of the ventricular leaflet in relation to the two commissural leaflets. Instead of being triangular in shape, the ventricular leaflet may be oblong, bifid, trifid or even indistinguishably in continuity with one of the commissural leaflets. On occasion, the radial length of the aortic leaflet may be markedly foreshortened so that the ventricular leaflet may even be the larger. This is of little functional significance in the normal heart due to the redundancy of the aortic and ventricular leaflets. However, when the redundant portions are destroyed, fibrosed or calcified from rheumatic valvulitis, these variations may assume great importance. This could well be a substantial factor in the production of mitral insufficiency, when a fish-mouth stenosis in such a valve is closer to and facing into the outflow tract to the aorta.

Morphologic Classification of Mitral Stenosis

As previously described,¹ there are two basic morphologic patterns of mitral stenosis: Type I is primarily a rigid, fibrous contraction of the leaflets to a stenotic opening with little thickening or fusion of the chordae tendineae. If present, it is likely to occur around the rigid orifice and to extend anteriorly and posteriorly.

![Fig. 2. Type I mitral stenosis: A common pattern of mitral stenosis with (a) marginal leaflet fusion and calcification, (b) leaflet flexibility remaining, (c) only minor thickening in fusion of the chordae tendineae, and (d) right ventricular and pulmonary artery enlargement.](image)

More than 85 per cent of patients are dominantly of this type (fig. 2). Type II consists of an elastic funnel with marked fusion of the chordae tendineae. These fused chordae tendineae may even constitute secondary stenosis (fig. 3). Calcification is common in type I, rare in type II. Less than 15 per cent of patients with marked stenosis have dominant type II processes. There is a whole spectrum of variations of individual patterns of mitral stenosis from I to II. Furthermore, either of these types can have a valve orifice of given
size and yet may or may not be associated with regurgitation. If the mouth of the funnel is turned toward the myocardium, the ventricular wall may close the orifice in systole and prevent regurgitation. This is called group A (fig. 4). If the stenotic orifice rigidly held open by the pathologic process faces out into the cardiac outflow tract, there may be extensive regurgitation. This is called group B (fig. 4). Again, A represents one end of a spectrum of variations and B the opposite pole; any individual lesion may fall between. The use of such a classification facilitates descriptions and also focuses attention on a type of process that responds well to valvuloplasty (type I) and less well (type II).

Indirect Surgical Methods for Treating Mitral Stenosis

Formerly the difficulties attending direct intracardiac surgery prompted the exploration of indirect operations in patients suffering from mitral stenosis. In general these have been attempts at palliation. These are reviewed to indicate their place in the evolution of present therapy and their relation to the current therapeutic armamentarium.

Cardiac Denervation

This has been carried out through low cervical incisions similar to those used in phrenic interruptions. The operation itself is not technically difficult. It consists of bilateral cervicodorsal sympathectomy with the removal of ganglions C-7, D-1, D-2, D-3, and sometimes D-4 and D-5. This has been carried out in an effort to reduce the mean heart rate. It has already been emphasized in previous publications that tachycardia is poorly borne by the patient with mitral stenosis. In this case, it should be to the patient’s advantage to have episodes of tachycardia reduced in number and severity. At least theoretically this might be done by removing some of the cardiac accelerator pathways.

This operation has been performed twice in this series for patients with mitral stenosis. One patient who had had recurrent pulmonary edema every four to seven weeks for two years had no attacks for 12 months following
the operation. However, the patient was re-admitted at the end of a year and died in pulmonary edema. The other patient had some control of her pulmonary edema for four months. She was then readmitted to the hospital with what was probably a pulmonary infarction and associated pulmonary edema. She did not recover. It is interesting, however, that this patient had objective evidence of reduced tendency to ventricular extrasystole following this denervation.

It was recognized that this was but a palliative procedure. Some of the objectives were attained, but the results make it apparent that this therapy is not adequate.

Interauricular Septal Defects

It has long been felt that patients with Lutembacher’s syndrome tolerated mitral stenosis far better than patients with mitral stenosis alone. If this be true, and there is some statistical evidence to support this contention, it might be well to create an interauricular septal defect. It is rational that such patients should tolerate a mild degree of mitral stenosis better than those without the interauricular septal defect because transient periods of pulmonary hypertension might be decompressed into the systemic circuit. Admittedly, such a therapeutic maneuver would be palliative, for people with Lutembacher’s syndrome die at an average age that is far from acceptable as a therapeutic goal.

A possible place for the creation of interauricular septal defects has been mentioned.2 This operation was performed on two patients. There was some brief beneficial effect. At the end of two to four months these patients ceased to enjoy continued palliation, and both have now had relief through finger-fracture valvuloplasty.

It must be assumed that this technic of creating interauricular septal defects is not so satisfactory as that described by Blalock.4 However, the creation of Lutembacher’s syndrome does not seem justified now that the results of finger-fracture valvuloplasty are so satisfactory.

The Bland-Sweet Anastomosis of Inferior Pulmonary Vein to the Azygos Vein5

This ingenious operation has as its objective the creation of a physiologic interauricular septal defect. Bland6 comments on the 14 patients operated as follows: “The later course of this group has been of special interest. Fully half have had striking and continued relief from pulmonary edema, several were much benefited for one or two years but are now beginning to have milder recurrences of their previous trouble, a few had only questionable benefit, and three succumbed within a few days of the procedure.

“As was emphasized originally by the authors, the procedure is a compromise and not a cure. It was designed only for those with relatively strong small hearts as a protection to the lungs. These purely palliative procedures of the past three years have now been overshadowed and quite properly replaced by the more promising direct attack upon the mitral valve. The remarkable improvement in technique and consequent lessening in the risk of the intracardiac approach have rendered other methods less attractive and somewhat obsolete, but nevertheless useful, perhaps, under special circumstances where complicating aortic valve disease (regurgitation) places an added strain upon the left ventricle.”

The Reservoir-Shunt Operation

In the period of consideration of indirect methods for palliating the symptoms of patients suffering from mitral stenosis, we were intrigued by the apparent improvement of some of the patients described by Bland and Sweet. They have created the physiologic interauricular septal defect described in the previous paragraph through the anastomosis of the superior division of the right inferior pulmonary vein to the proximal end of the divided azygos vein. From this procedure we developed our “reservoir-shunt” operation, and this operation was performed twice. The rationale for this procedure was based on two assumptions. First, that the patients were improved by the operation of Bland and Sweet; and secondly, that the anastomosis usually closed. The assumption of improvement
was taken from the observations of the proponents of the operation. The second assumption, that the anastomosis closed, was derived from three facts: (a) 3 mm. and 4 mm. Blake-more tube anastomoses such as were used often close even when used in a site of favorable pressure gradients such as the femoral artery, (b) autopsy examination after two of these operations indicated that the anastomoses were closed, and finally, (c) superior vena caval catheterization failed to demonstrate patency of the anastomosis.

This apparent paradox of improvement in spite of closure of the anastomoses can be explained theoretically as follows: A closed anastomosis is in effect ligation of the azygos vein and ligation of the superior division of the right inferior pulmonary vein. This might have salutary effect in a patient with mitral stenosis in one or all of three ways. First, ligation of the azygos vein might produce trapping of the systemic blood in the “mediastinal venous swamp” during periods of increased blood flow. This would amount to a peripheral venesection. Second, ligation of the superior division of the inferior pulmonary vein might be followed by trapping or reservoir action in the lesser circuit during periods of increased flow similar to a pulmonic venesection. Third, after ligation of the pulmonary vein the regional bronchial veins would enlarge to carry off the blood from the interrupted portions of the pulmonary venous circuit. These bronchial veins drain into the azygos system, and therefore, in effect, the shunt that was initially intended by the pulmonary to azygos vein anastomosis is actually accomplished in another way. In this way, it seemed possible that simple ligation of these two veins might exert a salutary effect in patients suffering from the effects of periodic increase in the pulmonary hypertension of mitral stenosis. Two such “reservoir-shunt” operations were performed; that is, simple ligation of the superior division of the right inferior pulmonary vein and the azygos vein. Both patients seemed to enjoy transient improvement. The improvement was not sufficient to control the symptoms of the disease and no improved circulatory dynamics could be demonstrated by cardiac catheterization. Both patients were offered finger-fracture valvuloplasty. One patient succumbed in pulmonary edema before she came to the direct operation; the other has since had relief of her stenosis through valvuloplasty.

The “reservoir-shunt” procedures represented an interesting physiologic experiment. They were expected to be palliative at best. The procedures seemed reasonable and far simpler than the azygos to pulmonary vein anastomosis of Bland and Sweet. However, there is no evidence that they offered any substantial relief as contrasted with the direct operation of finger-fracture valvuloplasty.

PRESENT VALVULOPLASTY AND ITS DEFICIENCIES

The technic of finger-fracture valvuloplasty has been described in detail elsewhere. It consists of blunt dissection of the fusion bridges with the finger (fig. 5) and is possible in the vast majority of the patients. In some type II forms of mitral stenosis (approximately 10 per cent of all patients), it is necessary to use instruments for incision somewhat similar to Bailey’s valvulotomes. Usually, however, we use a blade that takes advantage of the finger-fracture blunt dissection technic (fig. 6).

The finger-fracture valvuloplasty technic is preferred to valvulotomy or commissurotomy for four reasons. First, delicate digital exploration of the stenotic orifice and of the leaflets is difficult when encumbered by an instrument. Second, incision through large calcific vegetations is both difficult and dangerous because of the possibility of dislodging emboli from the vegetations or because of thrombosis incident to crushing. Conversely, blunt finger dissection around such vegetations is simpler and safer. Third, hook knives and hook guillotines pick up chordae tendineae on the under side of the leaflets and more regurgitation may be produced. Therefore, when a necessity for incision arises in our work, we use a valvulotome that combines a cutting edge with finger-fracture (fig. 6). If a more powerful instrument is required, a modified punch is also available (fig. 7). Fourth, the hazards and complications
of insertion of the finger into the auricle are greater in the presence of thrombosed and contracted auricular appendages or of laminated intra-auricular clots. Such hazards and difficulties as exist with the finger insertion alone are greatly augmented by addition of an encumbering instrument. When there is no auricular appendage and the surgeon needs to enter the heart by way of the superior pul-

Fig. 7. Guillotine valvulotome: A guarded reverse-action valvulotome for initiating fusion bridge fracture that is extended to the annulus by blunt finger dissection.

monary vein, it may be very difficult to thread a rigid instrument around such a curved course safely. These unfavorable situations tend to occur more frequently in the sicker and older patients.

At times, the result of finger-fracture valvuloplasty feels to the surgeon like a nearly perfect restoration. Although this is unusual in advanced disease, nevertheless clinical and laboratory evidence often indicates that marked improvement has occurred. If one considers the

Fig. 6. Double-edged valvulotome. The blade is recessed into the soft tissues of the index finger for introduction into the left auricle. Insert upper left: Blade rotated 90° anteriorly for anterior fusion bridge incision and fracture. Insert upper right: Blade rotated 90° posteriorly for posterior fusion bridge incision and fracture. After the fracture is started with the valvulotome, it is further tailored to the annulus by blunt dissection with the finger, exercising care to avoid damage to the chordae tendineae.

Fig. 5. Above: The operative index finger, introduced through the left auricle, in position to fracture the anterior and posterior fusion bridges. Below: Type I mitral stenosis before and after finger-fracture valvuloplasty of the anterior and posterior fusion bridges.
closing mechanism of the normal leaflets and compares it to that of a moderately to markedly diseased stenotic orifice that has had valvuloplasty correction, he realizes that quite a different type of closing mechanism has been brought about. The normal leaflets close like parachutes that are placed in juxtaposition. The closure is secured by a broad surface of leaflet contact and overlap. The papillary muscle and chordae tendineae anchor the margins, then tighten the closure. Valvuloplasty in advanced disease brings about a simple flutter-valve mechanism. This simpler valve is generally competent and entirely adequate for normal intraventricular pressures. It is less likely to stand high intraventricular pressures such as occur with aortic stenosis, insufficiency or even hypertensive heart disease. This shortcoming of a flutter-valve is, of course, exaggerated when the chordae are markedly shortened and thickened. It is particularly difficult to produce a good flutter-valve when the chordae of the major and the minor leaflets are fused together in continuity with the elastic, flexible fish-mouth, such as is occasionally seen in type II pathology (fig. 3). When this situation is encountered, it is necessary at times to resort to the compromise of producing selective insufficiency, that is, incision or fracture in the ventricular leaflet. This compromise relief of stenosis seems most successful if the incision or fracture is well forward and lateral to the anterior group of chordae tendineae. Unfortunately, there is no method at the present time for anticipating or differentiating type I from type II stenosis before operation. Fortunately, the incidence of type II is something under 15 per cent. Those valves in which the stenosis is not significantly improved by the current surgical approach constitute less than 5 per cent. Progress is being made and this situation will not exist indefinitely.

Valvuloplasty is highly successful in dealing with the anterior fusion bridge in the type I process but it is not always as satisfactory in dealing with the posterior rolled edge or shelf. We may find that multiple incisions of the shelf and even the division of some chordae will be our eventual solution to this problem. At present, the results are good in this group but they may be improved. In type II stenosis considerable improvement in present operative technic is needed.

The place for prosthetic valves in the surgery of mitral stenosis and insufficiency has not been defined. Several interesting possibilities are being explored. Progress is being made experimentally in reducing the diameter of the annulus by excision of an anterior section of the ring. It may be possible to reduce the annulus with sutures. Perhaps eventually a combination of plastic valve and surgery in the annulus and deformed leaflets will prove useful.

Ancillary Problems in the Operating Room

The problems of anesthesia are very great in some patients with high pulmonary vascular pressures and in those with liver and myocardial damage. The respiratory assistor developed by Derrick, Maloney and Whittenberger has been of great value in dealing with people in delicate cardiorespiratory balance. Even so, anesthesia with its concomitant muscular relaxation and peripheral vasodilatation in the patient who has a fixed or falling cardiac output renders hypotension a grave problem. Various means of avoiding this hypotension are being investigated. Drugs such as norepinephrine and Neosynephrine, mechanical adjuncts and intra-arterial transfusion (fig. 8)* are helpful.

The problem of preventing or dealing with pulmonary edema is inextricably tied up with the problem of avoiding tachycardia. The dangers of tachycardia and the mechanisms by which they contribute to pulmonary edema have been stressed elsewhere. The control of excessive ventricular irritability and prevention of fibrillation are improving. In the operating room Prostigmin, 0.25 mg. given intravenously, has been used effectively in reducing rapid rates of various types.

The place for the extracorporeal pump and its field of usefulness will not be elaborated here.

* We are indebted to Dr. Carl Walter for the development of a simple method for the rapid transfusion of blood intra-arterially.
Gibbon, Dennis, Bjork, Clowes, Weleh, Hapgood and others are making noteworthy progress. Contributions from this direction may become a reality within the near future.

The danger of embolic phenomena remains great. Early in our experience (first 30 patients), four patients died from emboli. Recently, there have been no deaths, although two minor accidents have occurred. In short, the problem has not been eliminated but it seems to be far less menacing than it was originally. Three changes in technic are unquestionably associated with this improvement. First, when clot is encountered in the appendage and

auricle, no effort is made to shell out large and extensive cast thrombi. Rather, the finger is insinuated gently along the side of the clot toward the superior vein and thence into the chamber of the auricle without dislodging the clot. Second, during the period of greatest danger of embolization, that is, when the finger is in the auricle, the anesthesiologist presses on the patient’s carotid arteries. This reduces the stream of blood up the carotids and reduces the flow down to the aortic take-off; thus the likelihood of cerebral accident is reduced. Third, patients in whom there is extensive intra-auricular clot at operation are heparinized. This is started eight hours after surgery and continued for five days.

The Life Cycle of Patients with Mitral Stenosis

It is well known that patients with mitral stenosis have a clear-cut history of a previous rheumatic infection in only about one-half of the cases. Looking at it from a somewhat different point of view, if patients are followed from the time they first have their active rheumatic carditis, it has been shown by Walsh, Bland and Jones to take at least 5 to 15 years before fully developed clinical signs of mitral stenosis are present. It is not known what circumstances determine the evolution of the stenosing process, whether it is the repeated insults of a continuing, active, rheumatic process or whether it is due chiefly to the contracture of the scarred valve margins, or both. This of course, has a bearing on the surgical results of operated cases. Because of this time interval, comparatively few patients in the first two or even the first half of the third decade of life, present themselves with clear indications for surgical intervention. Furthermore, the possibility of active rheumatic carditis is greatest in this younger age group.

Although in many patients mitral stenosis runs a comparatively benign course, most patients ultimately die from the effects of their disease. In the same study it was found that heart failure of one type or another accounted for 44 per cent of the deaths; in an additional 33 per cent, acute events in the peripheral circulation (mainly embolic) produced death. Pulmonary infarctions resulted in 11 per cent of the deaths. It can be seen, therefore, that the circulatory breakdown in patients with mitral stenosis occurs in differing fashions. In a good many patients the valvular obstruction apparently does not interfere significantly with effective circulation and this seems to be true even in the presence of a fairly high degree of stenosis in many patients. These patients run a course into the later age groups and die of congestive failure or peripheral vascular accidents, though some patients succumb to associated hypertension or degenerative vascular disease.

In another and younger group, active rheumatic infection dominates the picture. In our
series of pathologic cases studied post mortem, active rheumatic infection was found to be a significant factor in determining death in only one of 100 patients. Many patients develop and still die of the consequences of bacterial endocarditis. During the years 1945-1949, after the advent of penicillin, the incidence of death occurring in patients who had or had recently had subacute bacterial endocarditis was exactly the same (8 per cent) as in a group of patients studied 20 years before at the same hospital by Davis and Weiss.10

There is a final group, and this is a group with which we are particularly concerned in the surgery of mitral stenosis. These are patients who usually have increasing pulmonary difficulty that can be explained largely on a mechanical basis due to the elevated pressure in the pulmonary capillaries leading to edema. This disability may be chronic dyspnea or it may be punctuated by acute attacks of violent dyspnea or pulmonary edema, hemoptysis, cough, wheezing, and frequently by a sense of fatigue and a tendency to lose weight. The deterioration of these patients may be steady and slow, but it frequently increases rapidly. Although slight edema may be present in these patients and many of them do get relief of dyspnea from sodium depleting regimens, true right-sided failure has not yet developed. They may or may not fibrillate but, particularly if they are in auricular fibrillation, the possibility of peripheral emboli occurring at any time is very real. Their disability is severe and increasing, the prognosis under medical management is hazardous, and since their symptoms stem back directly to the obstruction of the mitral valve, the prospect of relief from surgery in these cases is excellent.

The final phase in this group of patients with mitral stenosis is when pulmonary vascular changes become severe. It is then that the right ventricle fails, massive edema occurs with chronically enlarged livers, and elevated venous pressure is present. Pulmonary symptoms may or may not be somewhat alleviated at this point. There frequently is an apparent diminution in pulmonary symptoms, due in part to lessened activity on the part of the patient and in part to the protection of the pulmonary capillaries by the high resistance to blood flow through the arterioles and small arteries. Pulmonary infarctions, however, are common. Most of the patients are in auricular fibrillation. Functional tricuspid insufficiency may develop. These patients are chronic invalids. Since the disease has progressed so far, the prospect of significant improvement by operation is not so good as in the former group; the risk of operation is very much greater.

There is, unfortunately, no simple way of selecting patients who are suitable candidates for operation because of the progression of their disease other than by a complete evaluation of the clinical picture. Objective studies such as by cardiac catheterization are helpful, and in obscure cases occasionally necessary. When pulmonary vascular changes are advanced, the diagnosis can usually be made roentgenographically by a demonstration of large right ventricle and pulmonary artery.

A word is in order in regard to pulmonary vascular changes. It is well known, as demonstrated by Parker and Weiss11 and by Larrabee, Parker and Edwards,12 that organic changes of the nature of pulmonary arterial and arteriolar sclerosis, as well as intra-alveolar fibrosis and edema, may occur in patients with high-grade mitral stenosis. In our clinicopathologic study we were not able to demonstrate, however, a close correlation between the degree of mitral stenosis and the degree of such changes other than the fact that such organic changes are unlikely to be present without well-marked mitral stenosis. However, the reverse is not true; severe mitral stenosis may exist with little or no pulmonary vascular change. From a clinical, physiologic and pathologic study that has also been made by one of us,13 it is evident that some patients tolerate high-grade pulmonary changes with comparatively few symptoms. Since pulmonary changes occur with such frequency in patients with chronic high-grade mitral stenosis with symptoms, it is almost certain that many of the patients who have been successfully operated upon have had such changes. The almost uniform marked relief of symptoms in these patients suggests that such organic changes either do not produce the symptoms or are to some extent
reversible. It has been demonstrated in a small group of patients that high degrees of pulmonary resistance regress to or nearly to normal six months to a year after surgery. At the present time, therefore, the fear that such organic changes may persist in a given patient seems doubtful and their presence is no contraindication to operation.

**Clinical Classification of Patients Suffering from Mitral Stenosis**

It is very difficult to present, for such a protean disease, an accurate and sound clinical classification suitable for use in selecting patients for operation. A working classification useful in the light of our present knowledge is as follows:

Group I comprises patients whose present course is benign. They have auscultatory signs of mitral stenosis but few, if any, symptoms and minimal evidence of increase in pulmonary vascular pressure. Patients in this group may continue to run a benign course or they may develop an acceleration of their illness which shifts them to one of the other groups.

Group II includes patients somewhat handicapped by a static degree of moderate dyspnea on effort or by rare attacks of acute dyspnea or other pulmonary symptoms provoked by an extrinsic cause such as unusual exertion, fatigue or by severe infection. Rarely they may have some peripheral edema but do not have evidence of frank right ventricular failure.

Group III includes patients whose disability is progressive rather than static. There may be increasing dyspnea on effort or easily provoked attacks of hemoptysis, chest pain and pulmonary edema. They may suffer from palpitation, tachycardia, and distress over the liver on exertion. At any time they may slip into group IV or may die in an acute attack of pulmonary edema or from peripheral or pulmonary infarction. Their life expectancy under medical therapy is hazardous.

Group IV is a terminal group. They are completely incapacitated, usually with right ventricular failure manifested by chronically elevated venous pressure, considerably enlarged liver, and a marked tendency to congestion. Their pulmonary disability may or may not be greater than those in group III. They often have poor liver function, even ascites, evidence of decreased peripheral blood flow, and many have had emboli. Most of them are in auricular fibrillation.

Certain additional factors of importance in evaluating patients for operation are not included in this classification. As these jeopardize good results from surgery, they constitute relative contraindications.

1. Clinically, obvious active rheumatic carditis is a contraindication to surgical intervention. This position is taken because, in general, the degree of eventual residual clinical handicap is difficult to assess in the presence of active carditis. The patient may not require surgical intervention. Also, the effect of surgical intervention and valvuloplasty on the active disease as well as the effect of the active disease on the valvuloplasty have not been clarified. These questions will be answered in the course of time since active carditis is occasionally found unexpectedly at operation.

2. Severe aortic valvular disease sufficient to produce peripheral signs of aortic regurgitation or a definite enlargement of the left ventricle, due either to aortic regurgitation or to aortic stenosis, contraindicates the operation. It has been pointed out that severe aortic disease sufficient to cause left ventricular hypertension places an extra burden on the surgically created mitral flutter-valve. Recently, however, valvuloplasty has been done in the presence of mild aortic valvular involvement. In this connection, it should be noted that patients who are considered for operation frequently have a blowing diastolic murmur of slight to moderate intensity heard along the left border of the sternum. This may be due to functional pulmonic regurgitation (Graham-Steel murmur). The presence of such a murmur does not contraindicate operation providing the diastolic blood pressure at rest is greater than 50 mm. Hg.

3. Mitral regurgitation is a relative contraindication to operation. This is difficult to quantitate. The intensity of the systolic murmur at the apex is not a good guide. Very loud, high-pitched, musical murmurs are much more likely to accompany severe mitral regurgitation. Easy fatigability is a more prominent symptom than dyspnea. When there is enormous dilatation of the left auricle, mitral regurgitation of significant degree is likely to be present. This has been pointed out by others.1, 10 Auricular fibrillation is nearly always present. The presence of enlargement of the left ventricle in the absence of aortic disease or hypertension suggests the possibility of mitral regurgitation. Minor degrees of left ventricular enlargement are difficult to assess roentgenologically in the presence of a very large right ventricle. The electrocardiogram is more helpful than the roentgenogram in this connection. Marked pulsation of the left auricle also suggests such a diagnosis, but it is by no means an infallible sign. The presence of a so-called "insufficiency" curve10 in the pulmonary "capillary" pressure tracing obtained by cardiac catheterization is suggestive of significant mitral regurgitation. Such curves are not readily obtained, however, and their
interpretation is not simple. There may be a startling discrepancy between the clinician's impression of the degree of regurgitation and that of the surgeon as he palpates the fish-mouth funnel at the operating table. The surgeon too may gain an erroneous impression. For example, a low intraventricular pressure at the time of operation produces little or no palpable regurgitant jet; on the other hand, a feeble regurgitant jet could be due to a very large orifice. Either would give the surgeon the impression of minor insufficiency. It is important to develop accurate criteria for estimating mitral regurgitation for the present state of surgical correction of mitral regurgitation of major degree is not satisfactory. The patient who has a major degree of insufficiency but who is clinically stable should not have surgery now. All of the above clinical aids must be combined to make this quantitative estimate.

4. Auricular fibrillation or the history of definite peripheral emboli do not contraindicate operation. More than 60 per cent of our patients have been fibrillators. However, they probably make the immediate hazard of operation greater and the possibility of a peripheral embolus being dislodged at the time of operation somewhat more likely. We have encountered eight peripheral emboli occurring either at the time of operation or within 24 hours thereafter. Six of these patients were in auricular fibrillation at the time of operation and six had been in chronic congestive failure.

5. Extensive valvular calcification has never deterred us from carrying out valvuloplasty but it must be construed as an additional hazard. The first hazard is in relation to clot forming on such leaflets after they have been mobilized and the second factor is that such leaflets often are quite difficult to mobilize perfectly. The bases of such leaflets are always flexible but clumsy; bulbous calcific margins do not swing open and shut freely.

6. Advanced age obviously is associated with certain variations in the physical state that influence the surgical risk and result. Older patients have, in general, had their disease longer, the incidence of intra-auricular clot is higher and their recuperative power is less. While the most dramatic rehabilitation occurred in one 55 year old man bedridden for 18 months and in constant edema and congestive failure, age above 50 requires special attention. Those between 30 and 40 have done best. There have been only five patients under 30. They have all done well.

7. Organic tricuspid stenosis may make the operation more hazardous and the surgical result less favorable. Clinical evaluation of stenosis and insufficiency is difficult. Cardiac catheterization may be helpful in making this distinction.

8. Associated disease such as arteriosclerosis, hypertensive cardiovascular disease, asthma or other complicating and debilitating states are obviously important. These are of infinite variety and merely mean that after consideration of all factors the good clinician must add "his clinical impression" for or against the decision to intervene.

In a sense this classification constitutes a definition of some contraindications and indications for surgery. Group I patients do not need surgery and therefore this state constitutes a contraindication. Group II disability may justify waiting for improved surgery because the illness is static but if that static disability is unacceptable to the patient, it may constitute a reasonable justification for surgical intervention. To date, we have not felt that these patients should have operation now.

Those patients in group III are the ideal candidates for surgery now as the prognosis without intervention is bad and the risk of valvuloplasty is low (less than 10 per cent) considering the severity of the disease. The high mortality rate in group IV (less than 40 per cent) will be shown to be less hazardous to the patient than his disease at that phase of the life cycle; therefore, this terminal state does not contraindicate surgery. However, it argues strongly for prophylaxis in the form of surgery before this condition develops.

Detailed postoperative results as regards objective changes including blood flow, pulmonary pressures, pulmonary function and clinical condition will be reported separately.

It should be stated, however, that patients in group III have all been markedly improved and all have maintained or increased their degree of improvement over the period of observation (up to three years). It is common in this group of patients, who were seriously limited to stair climbing with difficulty or even to essential bed and chair existence before operation, to skate, ski, bicycle or dance after operation. (Rehabilitation in skating or dancing returns earlier than full housework for understandable reasons.)

The degree of improvement in most of the patients in group IV has been much less though it is often dramatic. Most have been able to resume sedentary occupations or light housework and have required infrequent or even no further mercurial diuretics. In no instance has a patient who survived operation had poorer cardiac function.
The Present Responsibility

Certain patients can be selected whose present course is benign (group I) but who of course may degenerate to one of the more serious categories at any time. If properly followed and observed, these patients should not have surgical intervention at this time. Some of these patients will never require surgical intervention. Furthermore, there is a real possibility that surgery can render this group a substantial disservice (a) incident to the risk and discomfort of an unnecessary operation, (b) incident to the valvular alteration itself, and (c) incident to the sacrifice of the auricular appendage in the event that subsequent operation should become necessary.

In group II, patients whose degree of disability is static, selection of cases for operation must be made on the basis of the discomfort and limitation in the individual case. The risk of operation in this group is not great; it can be carried below 5 per cent. The chance of relieving the handicap is good. On the other hand, if patients in this group are not materially discommoded by their illness, it is entirely possible that better valvuloplasty may be available within the next year or so. We have deferred surgery in this group, placing the patients on a waiting list and meanwhile insisting on careful clinical check lest they slip into group III.

Group III. The risk in this group is relatively low, below 10 per cent in this series, whereas the benefits are considerable. These patients are usually restored to comfortable, useful lives. This is because they get good, functional valvuloplasty before there is irreversible damage in the lung, myocardium and liver. In short, they are treated before they have slipped into group IV or die. These patients in group III constitute the ideal and urgent candidates for surgery at this time. They represent the most serious responsibility of the cardiologist in protecting the patient against fatal issue that is possible at any time while in this group or before he undergoes further progression into group IV. In our 48 patients of group III, there have been but four deaths.

Group IV. This, like group II, again represents a borderline group but in quite another way. These patients are suffering from a malignant disease. During the early phases of this study we preferred to take our surgical candidates from the group of dying patients. A control group demonstrates how group IV patients fared without surgical intervention. The control group constituted patients in group IV acceptable for surgery but who did not have it for various reasons, such as refusal of surgery on the part of the patient or the patient’s family. There were 19 of these patients and 17 died within one year, 15 within six months. Thus, it becomes apparent that it is fair to call this a terminal or malignant phase. There were 39 patients in group IV who were operated on and 14 died from this intervention. Of course, these results are far better than those in the control group and if we were discussing carcinoma of the stomach or liver we would be delighted with such salvage rates. However, in this malignant phase the results are being improved.

It becomes apparent that we must continue to explore the degree of usefulness of a technic established as beneficial in some instances until we clearly define the limitations of the method. At the same time, we are trying to improve the method and therefore the benefits of surgery in group IV. These efforts at improving the surgery of the terminal stage must continue until the physician accepts his responsibility consistently in earlier phases of the disease. The difference in mortality that patients face between group III and group IV is not inconsiderably a responsibility of the physician.

The classification of patients as to severity of disease and evaluation of patients as surgical risks still has not been reduced to a mathematical formula embracing all variables, in spite of our efforts. These equations remain only as good as the man applying the measure. The authors frequently find themselves in disagreement concerning classification of their patients.

* Three additional patients have died from other causes, subsequently.
SUMMARY

An attempt is made to define the physician's responsibility in the selection of patients for the surgical treatment of mitral stenosis.

The anatomy of the normal mitral valve is discussed. A classification of the morphologic types of mitral stenosis is presented. The evolution and present position of finger-fracture valvuloplasty are outlined in the light of other indirect and direct methods of surgical treatment. The life cycle or clinical course of patients suffering from mitral stenosis is discussed, and a clinical classification of such patients is presented. These patients are divided into four groups:

Group I. Benign (murmur without handicap).

Group II. Handicapped (nonprogressive).

Group III. Hazardous (progressive).

Group IV. Terminal.

It is felt that surgical intervention is justified in group IV because it presents a salvage rate of better than two-thirds of the patients, in contrast to a mortality rate of approximately 90 per cent without surgery. Unfortunately, irreversible changes in lung, liver and myocardium prevent maximum recovery in this group although they run the highest mortality risk.

Group II patients, the handicapped, may now be operated or not depending on the degree of disability. Presumably surgical techniques will improve and patients who have minimal handicap that is well borne and stable could reasonably be advised to wait for further technical improvements. On the other hand, good surgical treatment is now available and group II patients with marked handicap can be offered relief at the present time.

Group III patients are considered urgent indications for surgery now, inasmuch as their course under medical treatment is hazardous and their rehabilitation through surgery is excellent and in some cases even dramatic. The surgical mortality in this group is less than 8 per cent.

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The Responsibility of the Physician in the Selection of Patients with Mitral Stenosis for Surgical Treatment

DWIGHT E. HARKEN, LAURENCE B. ELLIS, LEWIS DEXTER, ROBERT E. FARRAND and JAMES F. DICKSON III

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